

The Evolution and Biology of Sex

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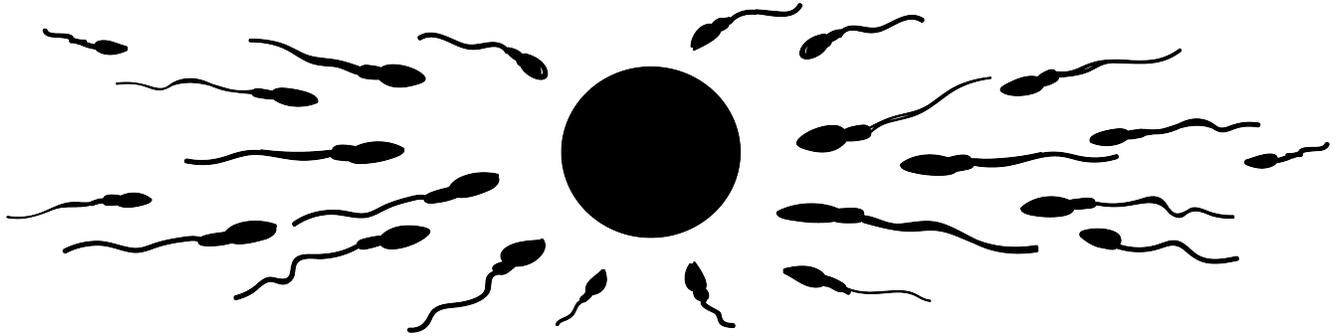


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When you approach any icon in the textbook,		
Icon	Description	What you should focus on
	Goals & Objectives	The goals and objectives for each chapter are meant to serve 2 functions: 1. To prepare you for the upcoming reading 2. To convey our expectations for what you should be able to do at the end of the reading
	Points to Ponder	Some questions in each chapter either require deeper thought or do not have obvious answers, but rather are simply points to ponder.
	Biology is Sexy	Sometimes the content may not seem overtly related to sex, however, everything we discuss is related to sex.
	Read More	We want to share a lot of interesting material with you, but some of the content is not directly related to the chapter's goals and objectives.
	Check Yourself	Throughout each chapter there will be opportunities for you to test your knowledge before proceeding to new content.
	Wrapping It Up	We begin each chapter with a biological problem and return to the problem at the end of each chapter in an attempt to wrap it up.

Chapter 1: The Nature of Science & The Nature of Sex



[1.0 Introduction](#)

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1.0 Introduction

Science is all about knowledge

Science is all about asking questions, generating new knowledge, and asking refined questions on the basis of that knowledge. We can study science through the lens of sex–sex appeal, sexual reproduction, sex and gender, and sexual orientation—as there are many questions regarding the topic that remain unanswered.

Consider the following questions

- **Do animals besides humans have sex for fun? If so, which ones?**

The authors of this text have often heard people claim that humans are exceptional in being the only, or one of the very few, animals that have sex for fun. But is this true? And how would anybody know whether other animals are having fun during sex? To look at the question from a different angle: why else would an animal have sex? Are they actually engaged in family planning?

- **Why do young men masturbate so much?**

Most people masturbate, but young men between the ages of 12 and 25 take all the prizes for masturbation frequency. While the masturbation question may seem silly (“because it’s fun, of course!”), it’s actually worth asking. Masturbating requires energy, wastes sperm and energy-rich ejaculate, and the associated erections and ejaculate can cause embarrassment in some contexts (e.g. dormitories, [buses](#)). We’ll discuss masturbation, in an evolutionary context, in other chapters.

- **Is homosexuality inherited?**

The factors that influence sexual attraction are still far from clear. While we’ll look at studies that argue for a genetic basis to at least some forms of same-sex sexual attraction, we’ll discuss studies that consider many non-genetic factors as well. In addition to addressing the question of homosexuality, we’ll encourage you to consider who is asking the questions (for example, why might some individuals feel compelled to seek a genetic basis for homosexuality? Why might others want to refute genetic arguments?), and why.

- **What’s the function, if any, of the female orgasm?**

Human female orgasm is a mystery in many respects. It can take many different forms, is not required for conception, and may occur outside of a “typical” sex act. Scientists have attempted to tackle the question of the function of the female orgasm, as well as the issue of whether female orgasm is evolutionarily *adaptive* (i.e., does it increase an individual’s reproductive success?).

- **Does the HPV vaccine lead to more teenage sex?**

The human papillomavirus (HPV) is associated with several deadly cancers, thus many health professionals advocate vaccinating adolescents prior to the onset of sexual activity. However, this recommendation involves parents making a decision that, for some, seems like giving their children “permission” to be sexually active. Thus, there has been some question about whether receiving the vaccine will make an individual more likely to engage in sexual activity, especially at an early age. We’ll discuss this question in the following pages. But first, what do you think?



Points to Ponder

- Does the HPV vaccine lead to more teenage sex? What do you think?
- How would you go about gathering evidence to answer this question?
- How would you address any of the above questions?

These questions, and limitless others, all lead to a central tenet of this text: **SEX, AND SCIENCE, ARE EVERYWHERE.** Sex affects human lives along many dimensions—from how we interact as societies, to individual health, to evolutionary success. We’ll use the evolution and biology of sex to address science as a discipline—both a body of knowledge and a way of creating knowledge. By the end of our discussion, you’ll be able to address—if not actually answer—all of the above questions, as well as many others.

Important Note: Sex-positive versus pro-sex

The authors of this text are working from a “sex-positive” viewpoint. By “sex-positive,” we mean that we will not be vilifying consensual sex in any form. We view sex as a natural product of biological forces, expressed in a diversity of presentations, and ideally associated with healthy individuals and societies. This is different from being “pro-sex.” We hope to never make recommendations or express opinions about individual choices related to sexual activity, including your ability to choose abstinence or express asexuality.

Please keep this “sex-positive” viewpoint in mind during your reading, our in-class discussions, and your interactions with peers. You should be able to express your own opinions and be heard, while respecting the opinions of others, but in the end, our class is a “sex-positive” space.

1.1 Chapter Objectives



Learning Objectives

Our primary goal for this chapter is for you to gain an understanding of the basic elements of the scientific process—from asking questions that can be addressed by observation and experimentation to interpreting data to making evidence-based recommendations. By the end of your reading and our in-class discussion, you will be able to:

1. Define the following terms:
 - **hypothesis**
 - **experiment**
 - **controlled variables**
 - **prediction**
 - **independent variable**
 - **dependent variable**
 - **scientific theory**
 - **control group**
2. Explain the distinctions between a question, a hypothesis, and a prediction.
3. Explain the distinction between hypothesis and theory.
4. Describe examples in which correlation does not necessarily imply causation.
5. Evaluate data in light of a stated hypothesis.
6. Given a specific example, draw conclusions based on evidence.

1.2 Nature of Science Overview

Science is a fact-based way of understanding natural phenomena. Science is really two things: (1) a collection of facts that have been established through observation or experimentation; and (2) a process for advancing knowledge about the natural world. We'll discuss both established facts and science process in this text, but our emphasis will be on *science as a process*.

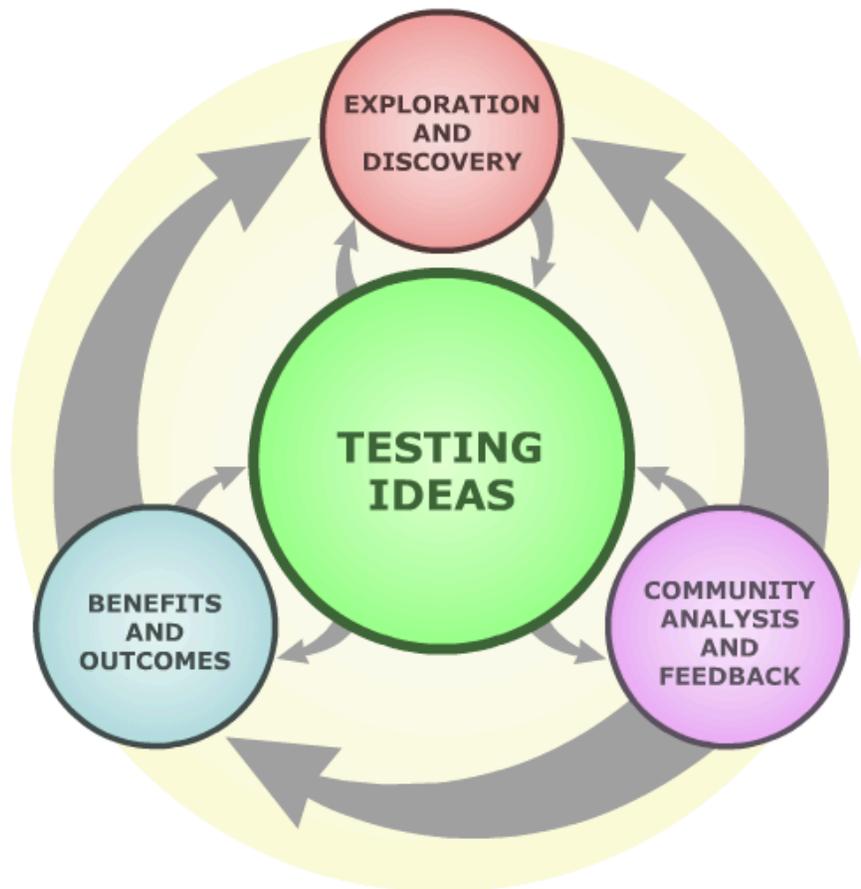


Figure 1.1 How science works: The flowchart.

1

The process of science

Simply, the process of science includes making observations, generating questions and hypotheses about these

1. Copyright 2017 by The University of California Museum of Paleontology, Berkeley, and the Regents of the University of California. Used with permission. "How science works: The flowchart.." Understanding Science. University of California Museum of Paleontology. 3 January 2017 <http://www.understandingscience.org/article/scienceflowchart>.

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observations, making and testing predictions, and revising the collective understanding through communication of our findings.

A **hypothesis** is a proposed explanation for some sort of natural phenomenon (such as male masturbation, female orgasm, or same-sex sexual preferences). By definition, a hypothesis sets the stage for further exploration, either through more observations or experimentation.



Check Yourself



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<https://open.lib.umn.edu/evolutionbiology/?p=145#h5p-7>

Which of the following are testable hypotheses for the above questions?

- Parents who disapprove of premarital sex are less likely to choose HPV vaccination for their offspring than do parents that are more accepting of premarital sex.
- Homosexual behavior is genetic, in that homosexual individuals are more likely to have family members with same-sex sexual preferences.
- The human female orgasm has the function of being associated with more successful copulations; specifically, women who orgasm are more likely to conceive during sex.
- Young men masturbate frequently because frequent masturbation is a form of “sex practice” that makes men better sex partners.

1.3 Experimental Design

An **experiment** is a procedure done in a controlled manner for the purpose of addressing a stated hypothesis. We'll discuss and conduct many experiments in class and lab during this course. Note that we can have experimental and control conditions (for example, you could compare frequent masturbators to a "control" group that is prohibited from masturbation) as well as **controlled variables**. You will want to keep as many factors as possible the same, to focus on those things that interest you.

For example, to focus on the factors associated with sexual preference, you might compare a group of heterosexual individuals to a group of individuals who are strictly homosexual. You will find your work easier if you compare individuals from similar cultural backgrounds, for example, or people of similar ages, raised in similar situations, etc. You might compare heterosexual Latino men, between the ages of 25 and 34, from Southern California, to homosexual Latino men, of the same ages and from the same area; in this example, you have controlled for four factors that otherwise vary: sex, ethnicity, age, and geographic location. Controlling these variables through careful study design will help you focus on your primary question.

Many hypotheses are open to a variety of observational and experimental tests. We can specify how we're testing our hypothesis through **predictions** that clearly state the type of evidence that will support our hypothesis. For example, one of the hypotheses to explain frequent male masturbation is "Young men masturbate frequently because frequent masturbation is a form of 'sex practice' that makes men better sex partners." A prediction arising from this hypothesis could be "females rank the sexual performance of frequent masturbators more highly than they rank the performance of men who do not masturbate (or who masturbate infrequently)." With this prediction, you can get a good idea of how to test the stated hypothesis.

Specifically, you can survey partners about the sexual performance of a sample of men—some who you know are frequent masturbators, and some who you know are not. Perhaps these partners could assign sexual performance scores, for example, on a scale of 1 (bad) to 5 (excellent). If frequent masturbators are associated with higher sexual performance scores, you have support for (but not proof of) your hypothesis. But really, you're just getting started! This one hypothesis is open to a variety of predictions, and each prediction can often be tackled in several ways.

We'll use the terms **independent** and **dependent** to describe variables in our studies. The **independent variable** is the one that is manipulated, or varies as part of the study design. How frequently a male masturbates could be an independent variable in the example above—you aren't necessarily manipulating this variable, but you are collecting these data as a way to test the stated hypothesis. The **dependent variable** is a factor that may change as a result of changes in the independent variable. In the above example, sexual performance scores are the dependent variable, and we have predicted that they will vary (increase) as masturbation frequency increases.

Scientists employ many tactics for interpreting the data they collect. One way is to report all the data in a table [note: these data were made up for this discussion!]:

Rating Scale: 1 - 5 (1 is bad and 5 is excellent)	
Frequent (> 5 times per month)	Rare (5 or fewer times per month)
4	3
3	4
5	3
4	2
2	3
4	3
4	4
3	2
5	3
3	2

Table 1.1 Sexual performance scores for frequent and rare masturbators.



Points to Ponder

Looking at these data, what do you think? Can these data be used to support our hypothesis, or not? In other words, do these data meet our prediction?

1.4 Interpreting Data

In this course, we'll look at a lot of graphical representations of data, often combined into averages as shown below.

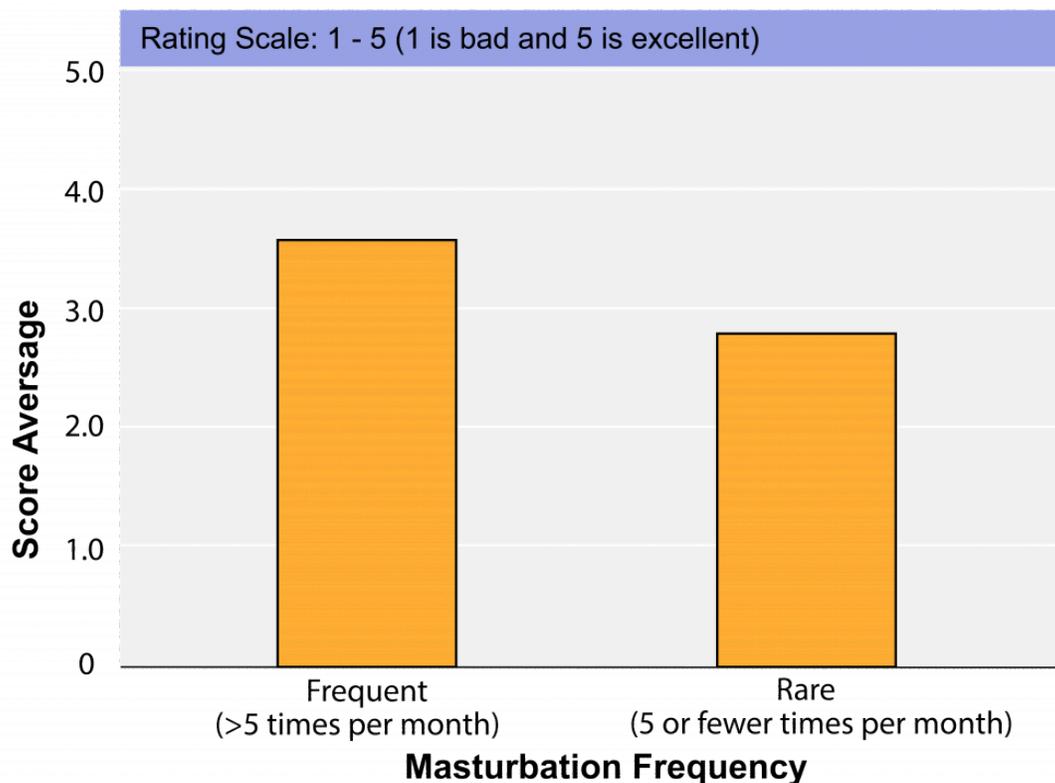


Figure 1.2 Sexual performance score average

Now, what do you think? Can these data be used to support our hypothesis, or not? In other words, do these data meet our prediction? When you look at these data, the difference is certainly in the direction that matches our prediction, but how do we know if this difference is not just a fluke?

Scientists often use simple statistical tests to evaluate trends in their data. They can use statistical tests to determine if any observed differences, like those above, are “significant”—that is, are these differences likely to hold true if the experiment is repeated?

From hypothesis to theory

If many rounds and methods of observation and experimentation serve to support a hypothesis, these lines of evidence can create a framework called a **scientific theory**. Yet, even theories are subject to revision via the process of science. For this reason, we discourage use of the terms “prove” and “proven” in science.

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Read More

You'll find an excellent overview of the nature of science at http://undsci.berkeley.edu/article/intro_01.

1.5 Stating a Hypothesis

Cervical cancer is cancer of the cervix, the lower part of the uterus. Globally, cervical cancer is the fourth most common cancer in women; however, in developing countries, cervical cancer is the third most common cause of cancer death in women.

Where is the cervix?

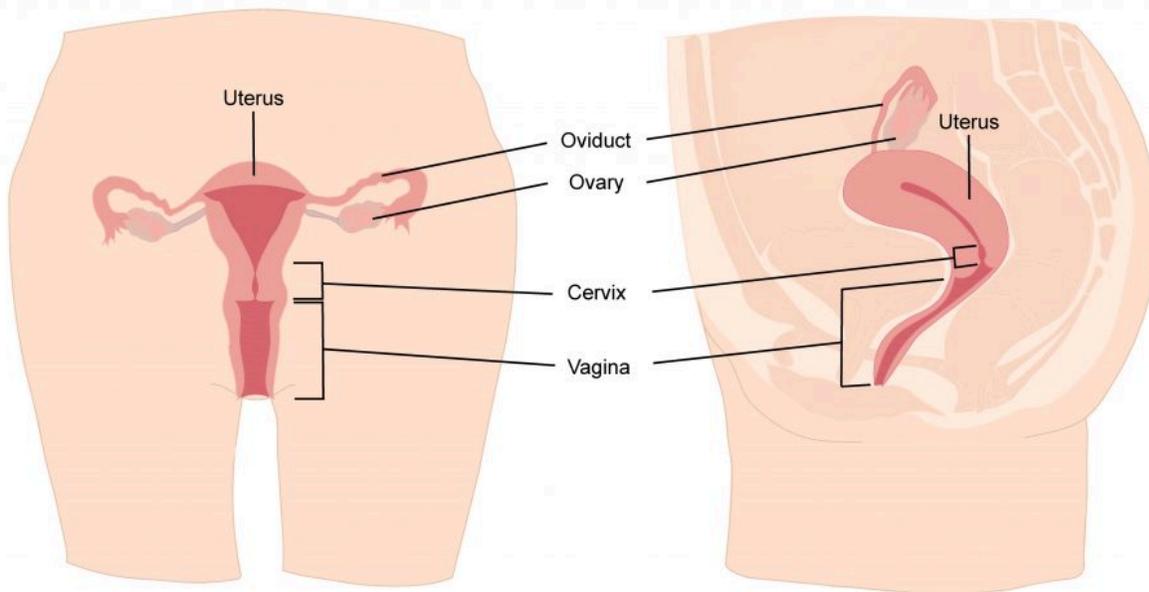


Figure 1.3 Schematic drawing of female reproductive organs, frontal and side view

What causes cervical cancer?

In the 1970s, some observations led researchers to suspect a connection between oral contraceptives (i.e., birth control pills) and cervical cancer. Thus, they developed a working hypothesis about the cause of cervical cancer as follows: oral contraceptives have a cancer-causing effect on the cells of the cervix.

Several population-level observational studies were conducted to detect whether cervical cancer was associated with the use of oral contraceptives. For example, Louise Brinton and her colleagues analyzed data on cervical cancer patients from 24 hospitals over a two-year period. These patients were compared to matched controls that were selected by randomly dialing telephone numbers to identify women that matched cancer patients in terms of age, ethnicity, and region of the country. Brinton and her colleagues interviewed 481 cancer patients and 801 controls, collecting information about health history, sexual behavior, and use of contraceptive.



Check Yourself



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<https://open.lib.umn.edu/evolutionbiology/?p=162#h5p-8>

1.6 Correlation Does Not Equal Causation

In the previous example, you may have selected “*Oral contraceptive usage is correlated with cervical cancer*”. This statement is accurate and does not imply that using the Pill necessarily *leads* to cervical cancer. Nor do we have any reason to think that Brinton’s study was flawed. She used a relatively large number of participants to look for trends, and did her best (using randomly generated phone numbers) to find a random sample for her control group. However, it is understandable that, when this study (and others like it) came out, many people were tempted to think that using oral contraceptives led to cancer. These individuals were falling victim to the idea that correlation implies causation; this misconception is powerful and has created a lot of trouble for scientists.

Note that some correlations are positive: increase/decrease in one variable is correlated with increase/decrease in the other variable. For example, there is a strong correlation between *decreasing* divorce rates in Maine and *decreasing* consumption of margarine (Figure 1.4):

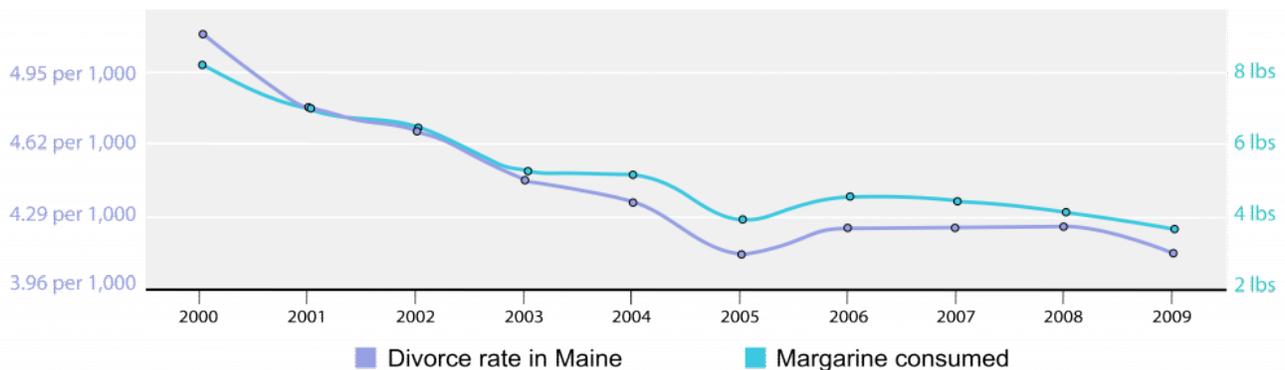


Figure 1.4 Divorce rates in Maine correlates with per capita consumption of margarine.

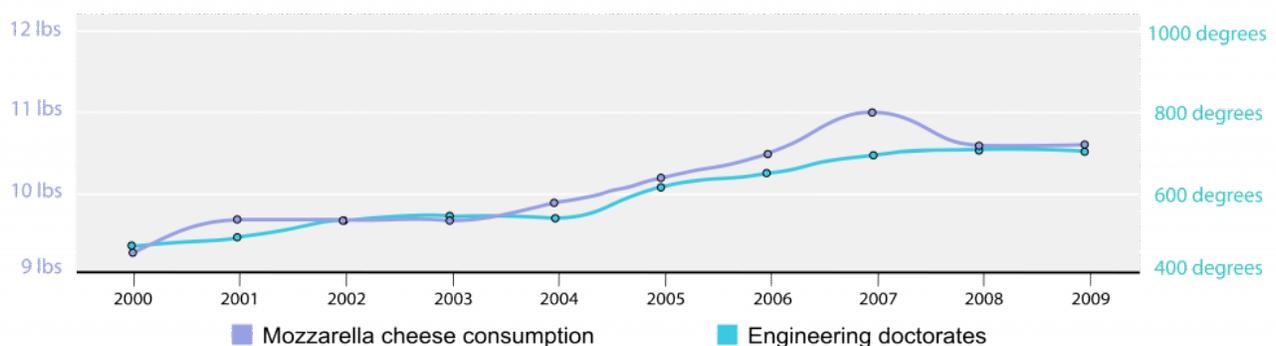


Figure 1.5 Per capita consumption of mozzarella cheese correlates with Civil engineering doctorates awarded.

Note the source! The above two examples are from a [fun!] website called “spurious correlations,” so they aren’t meant to be taken seriously, however the data are real: <http://www.tylervigen.com/spurious-correlations>

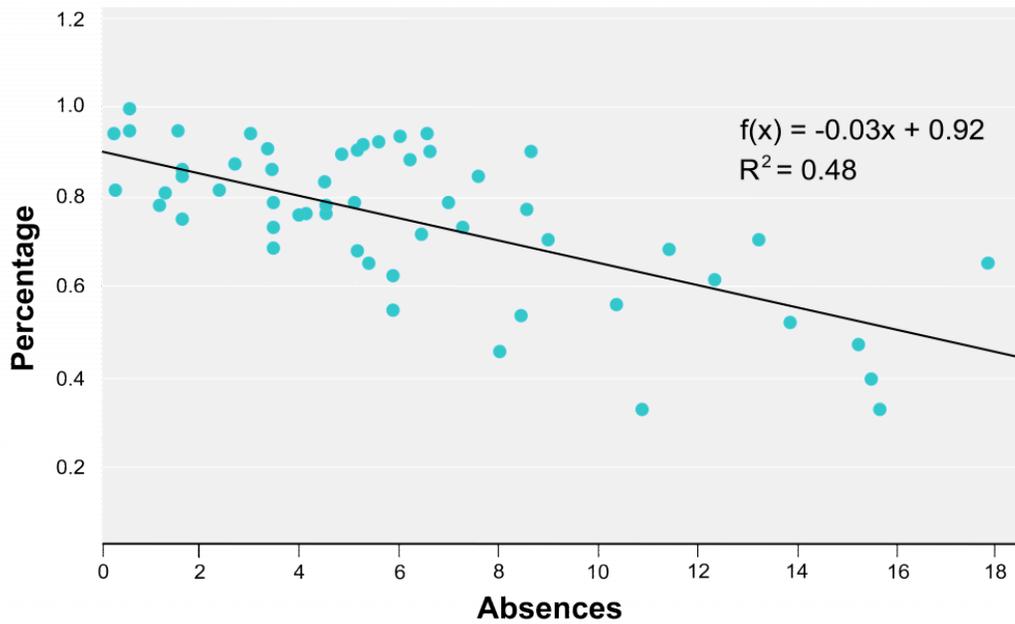


Figure 1.6 Absences versus percent score

Some correlations are negative, whereby an *increase* in one variable is correlated with a *decrease* in the other. The data above compare course absences with percentage performance in the course.



Check Yourself



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1.7 Exploring Correlations

When faced with a compelling correlation, such as the relationship between the Pill and cervical cancer, scientists must challenge themselves to consider whether the correlation suggests causality (e.g., the possible relationship between absences and course performance), is meaningless (e.g., presumably, the relationship between divorce and margarine), or whether the correlation may provide clues to other potential causes. For example, could using the Pill be associated with anything else that might be potentially carcinogenic? What do you think?

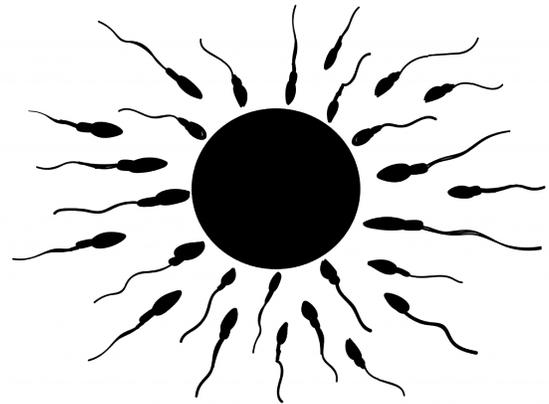


Figure 1.7 Egg and sperm cell

In her discussion of these findings, Brinton noted *“In addition, there was some evidence that pill associations were most pronounced among women who had never used barrier methods of contraception or who had histories of genital infections...”*

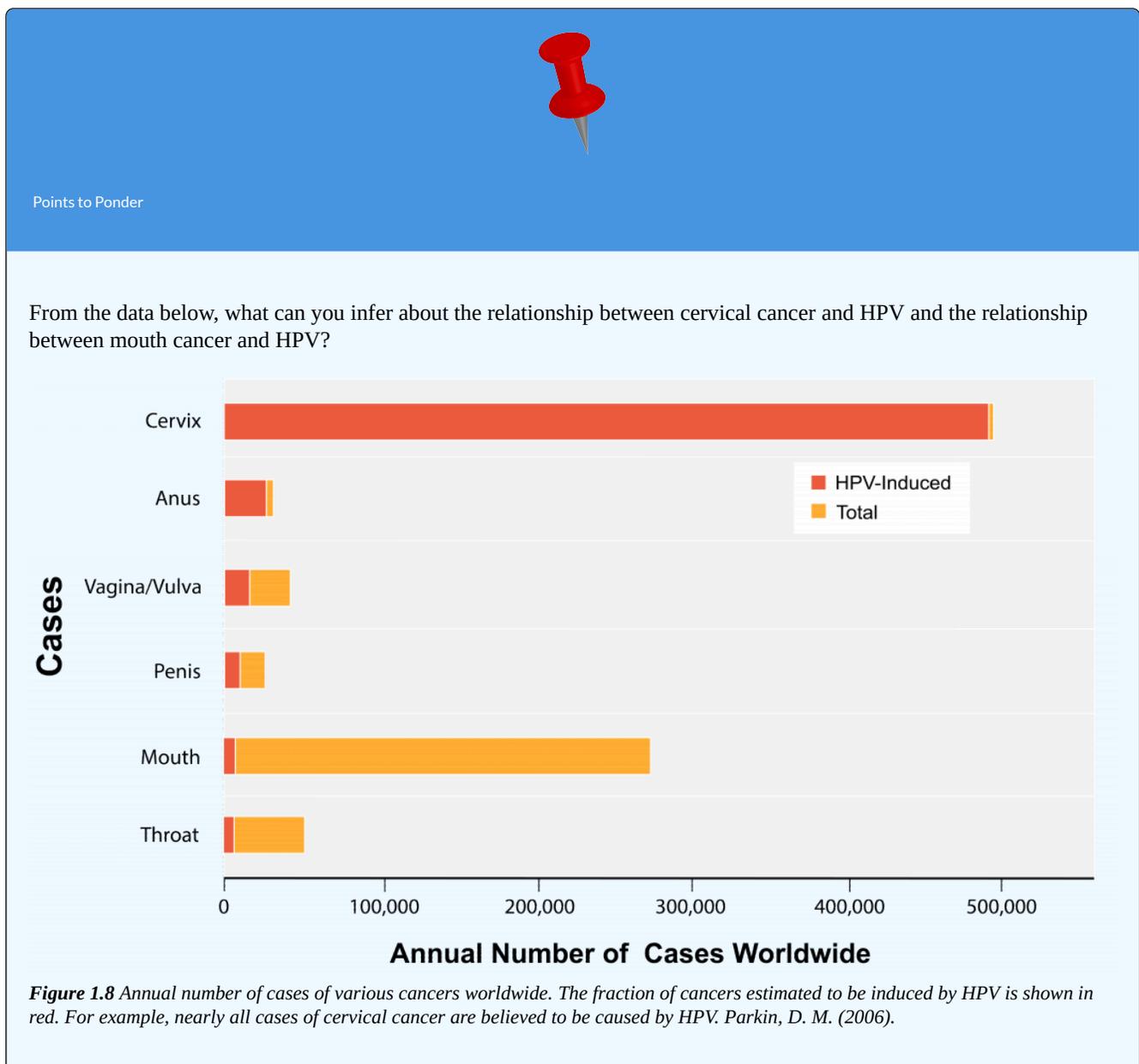


Points to Ponder

Given this statement, and what you may already know about cervical cancer, what were some obvious follow-up research possibilities?

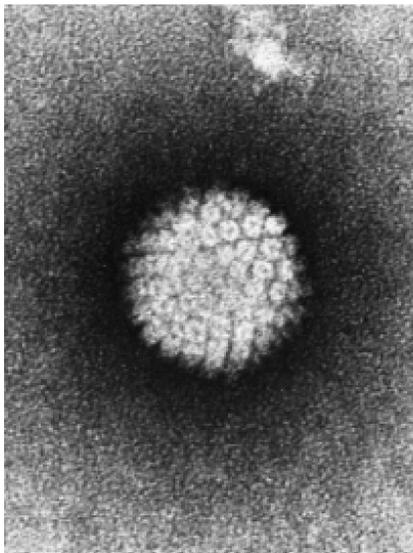
1.8 Human Papillomavirus (HPV) and Cervical Cancer

Following the observations of Brinton and others, scientists began to investigate a possible association between various infectious diseases and cervical cancer. Many years and studies later, we now know that HPV is associated with at least 90% of all documented cervical cancers. Smoking cigarettes also appears to place individuals at risk of developing cervical cancer. Some still suggest that using the Pill may be a contributing factor as well. However, there is compelling evidence implicating HPV as the cause of many types of cervical cancer. In fact, HPV is connected to other cancers as well—the following graph shows the annual number of cases of various cancers worldwide.

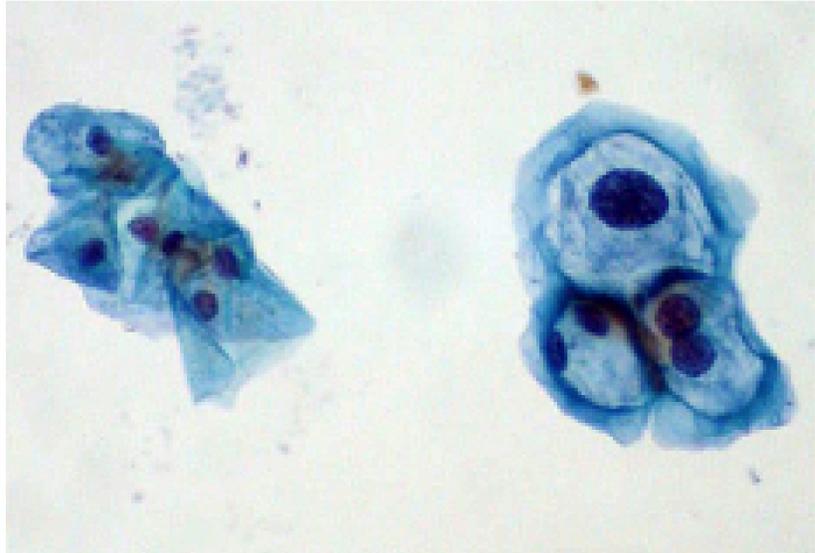


What is HPV?

Human papillomavirus (HPV) is a disease-causing virus and sexually transmitted disease, of the same name, that is typically diagnosed by the presence of genital warts. However, warts can occur in other places (e.g., the throat), and many individuals with HPV are undiagnosed. Therefore, it is important for women—whether they have been diagnosed with HPV or not—to have routine screenings (i.e., Pap smears) for cervical cancer. HPV prevention is challenging because there are many different strains of HPV. Consequently, the vaccines we currently use are designed to target several variations of the disease.



a)



b)



c)



d)

Figure 1.9 Human Papillomavirus (HPV) **a)** Electron micrograph of HPV **b)** Normal vs. HPV-infected cells. Pap smear – normal cells on left, HPV-infected cells on right **c)** HPV Warts in the throat, diagnosed at Milpark Hospital in 2015 **d)** Genital warts on penis

1.9 Does the HPV vaccine lead to more sex in teenagers?

In the chapter introduction, we mentioned that many health professionals advocate vaccinating adolescents against HPV prior to the onset of sexual activity. The two most common forms of the vaccine (Cervarix and Gardasil) have been shown, through a series of long-term studies, to be effective at preventing most types of HPV. However, some parents feel like selecting HPV vaccination for their kids is a way of giving their teenagers “permission” to be sexually active. Is there any evidence that this is the case? Specifically, does receiving the vaccine make an individual more likely to engage in sexual activity, especially at an early age?

Several studies have addressed this question. For example, in 2012, Nicole Liddon and colleagues published “Human Papillomavirus Vaccine and Sexual Behavior Among Adolescent and Young Women” in *The American Journal of Preventative Medicine*. Read their summary, below, and consider the following questions.

Background: Vaccines to prevent certain types of human papillomavirus (HPV) and associated cancers are recommended for routine use among young women. Nationally representative reports of vaccine uptake have not explored the relationship between HPV vaccine initiation and various sexual behaviors.

Purpose: Explore sexual behavior and demographic correlates of HPV vaccine initiation from a nationally representative survey of adolescent and young adult women.

Methods: In 2007-2008, a total of 1243 girls/women aged 15-24 years responded to questions about receiving HPV vaccine in the National Survey of Family Growth (NSFG). In 2010, demographic and sexual behavior correlates were evaluated in bivariate and multivariate analyses by age.

Results: HPV vaccine initiation was higher among those aged 15-19 years than those aged 20-24 years (30.3% vs 15.9%, $p < 0.001$). No differences existed by race/ethnicity for those aged 15-19 years, but among women aged 20-24 years, non-Hispanic blacks were less likely than non-Hispanic whites to have received the HPV vaccine (AOR=0.15). HPV vaccine initiation was greater for those with insurance regardless of age. HPV vaccination was not associated with being sexually active or number of sex partners at either age. Among sexually active adolescents aged 15-19 years, those who received HPV vaccine were more likely to always wear a condom (AOR=3.0).

Conclusions: This study highlights disparities in HPV vaccine initiation by insurance status among girls/women aged 15-24 years and by race/ethnicity among women aged >19 years. No association was found between HPV vaccination and risky sexual behavior.

-from Liddon NC, Leichter JS, Markowitz LE. Human papillomavirus vaccine and sexual behavior among adolescent and young women. *Am J Prev Med.* 2012;42(1):44-52. DOI: [10.1016/j.amepre.2011.09.024](https://doi.org/10.1016/j.amepre.2011.09.024)



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<https://open.lib.umn.edu/evolutionbiology/?p=197#h5p-10>

Liddon's studies were followed by similar work refuting the idea that women with HPV vaccine would be more likely to contract other sexually transmitted infections (such as chlamydia or herpes). In fact, a study by Tanya Mather and colleagues found that vaccinated women were more likely to have positive attitudes about safe-sex practices. Thus, the evidence collected thus far seems to discredit the idea that HPV vaccination leads to more sex, or to risky sexual behavior.

1.10 Science is a social endeavor

One of the key points raised by the cervical cancer example is that science is a social endeavor. For example, scientist may be motivated to study some topics and not others. Like all people, scientists are flawed and can make mistakes. Therefore, it is critical that science is open to replication and revision. It took many individuals many years of study, involving thousands of individuals, to establish HPV as a cause of many cervical cancers. And our knowledge is *still* imperfect.

Also, scientific findings matter to people. If the Pill really were the main cause of a deadly cancer, then many women would need to re-think their choice of contraceptives. Alternatively, many women might have decided to forego contraceptives altogether, possibly resulting in many unplanned pregnancies, higher abortion rates, etc. Further, if the HPV vaccine really does lead to more teenage sex, then many parents might opt not to have their children vaccinated, for a variety of reasons (religious, cultural, etc.).

As it is, there are still social and medical challenges associated with HPV vaccine. For example, in the United States in 2016, only 6 out of 10 teenage girls had started the vaccination series:

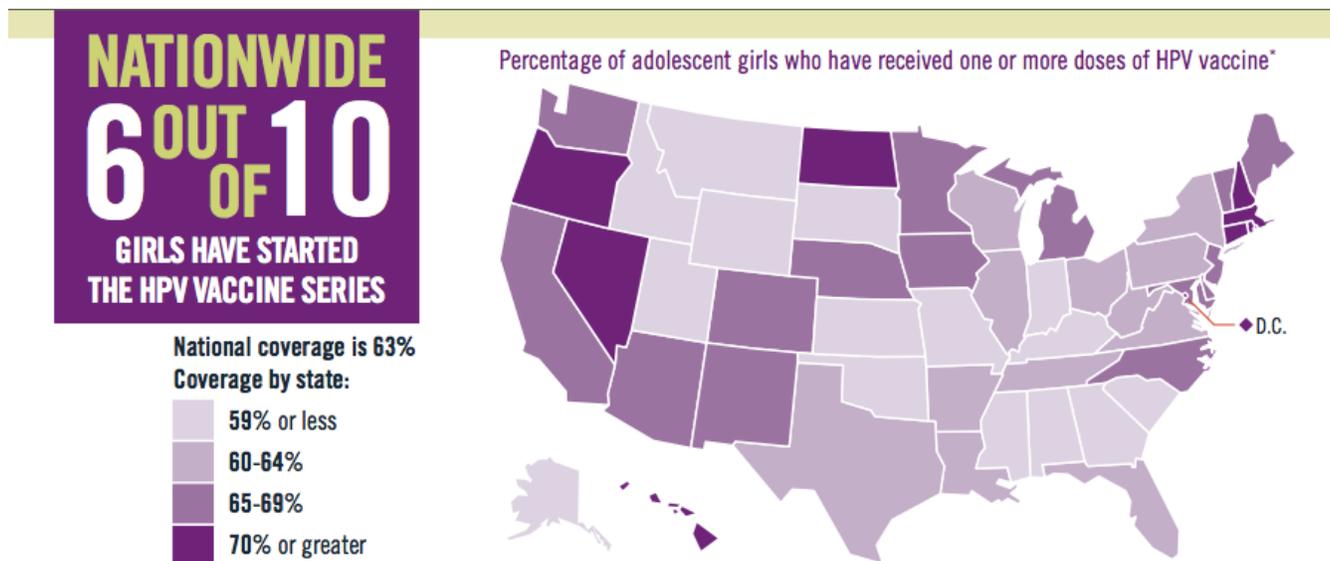


Figure 1.10 Nationwide 6 out of 10 girls have started the HPV vaccine series.

1

While there is obviously room for improvement with teenage girls, the data are grim for boys: Only 5 out of 10 teenage boys had started the series in 2016. This statistic may be due to parent concerns about teenage sex, or perhaps a lack of understanding at the population level of how vaccinations work. In order to make dramatic reductions in the incidence of cervical cancer, the majority of males *and* females must get vaccinated against HPV. Perhaps better education—about herd immunity and the connection between HPV and cancer—is warranted.

1. U.S. Department of Health and Human Services, Centers for Disease Control and Prevention. Public Domain. <https://www.cdc.gov/hpv/infographics/vacc-coverage.html>

NATIONWIDE
5 OUT OF 10
BOYS HAVE STARTED
THE HPV VACCINE SERIES

National coverage is 50%
Coverage by state:

- 39% or less
- 40-49%
- 50-59%
- 60% or greater

Percentage of adolescent boys who have received one or more doses of HPV vaccine*

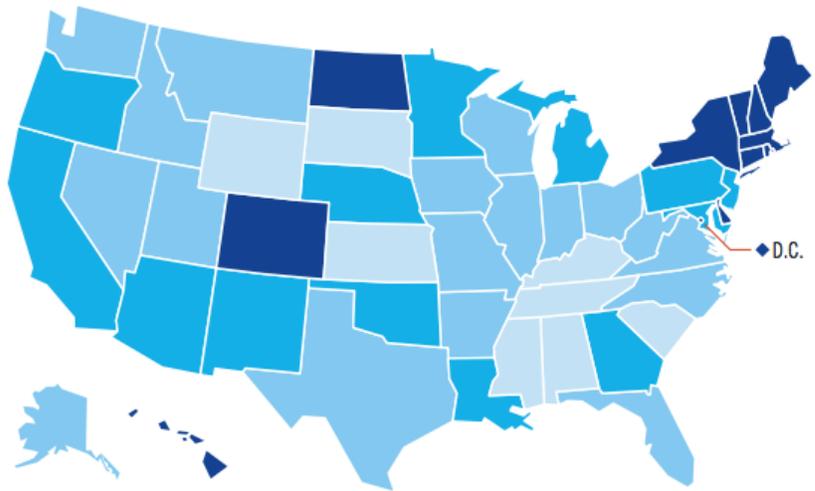


Figure 1.11 Nationwide 5 out of 10 boys have started the HPV vaccine series. ¹

1.11 Wrapping Up: Why use sex to study biology?



Why use sex to study biology?

The authors of this text maintain that by studying the evolution and biology of sex, we can understand much of the science of biology. Further, we think sex is fascinating and suspect many of our students feel the same way.

Critically, sex can help us illustrate many key features of science in general, specifically biology. For example:

a. Learn biology, save a life. Simply, biology is the study of life. By studying life, we can understand how to preserve our own lives, and how to live our lives to the fullest. And we've already discussed one example (e.g., HPV and cervical cancer) in which the connection between sex and death is evident. We'll discuss many others in the following chapters.

b. Science: it's all about asking questions. While we'll certainly present some established facts in these pages, we will emphasize questions and the processes by which scientists answer them, rather than focusing on answers themselves. And sex is a perfect vehicle for this discussion, because we still don't know many things about sex and sexual reproduction. We don't know the purpose of male masturbation, exactly how a single sperm is "selected" to fertilize an egg, how to effectively combat HIV (a virus we've been fighting for over 30 years), how and why homosexuality evolved, why sex and gender can vary so much, whether female orgasm serves a reproductive function, the occurrence of asexuality, and so on...For all of the above, we have ideas and testable hypotheses (that we'll discuss), but we don't have answers.

c. Science (or biology, specifically) is a social endeavor. However, we'll have many opportunities to illustrate that scientific questions are raised and addressed by people, often restricted by limited resources (time, money), often biased (hopefully unintentionally), and likely prone to error. We will encourage you to trust the process of science (one that is subject to review, replication, and revision) rather than the individual scientists themselves.

As we conclude this chapter and prepare for in-class discussion, be sure to return to the chapter's [goals and objectives](#).

Please note that we'll have many opportunities to exercise the above skills throughout the course. However, we hope you can begin the discussion with a basic understanding of the process of science. In the following chapters, we hope you'll develop an appreciation of how the study of sex can give you a good foundation in the basics of biology, specifically evolution, molecular genetics, and reproduction.

Chapter 2: Evolution



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2.0 Introduction

The Mystery of HIV Resistance

Acquired Immune Deficiency Syndrome (or AIDS) refers to a suite of symptoms associated with Human Immunodeficiency Virus (or HIV). Untreated individuals infected with HIV have a compromised immune system and are unable to combat common infectious agents. Basically, the virus enters the t-cells of the human immune system and commandeers these cells for its own purposes. Thus, the immune system is re-purposed to make more HIV, rather than fight infections. Without treatment, people with AIDS often suffer and die from the combined effects of several common pathogens—e.g., pneumonia, tuberculosis.

The impact of HIV infection is devastating: approximately 30 million people have died from AIDS-related causes, and at least that many are currently living with AIDS. In 2010 alone, 1.2 million adults and children died from AIDS in sub-Saharan Africa. And today, nearly 20 million children have lost their parents to AIDS.

Understandably, the discovery that some people are resistant to HIV infection was met with considerable enthusiasm. Follow-up work revealed that resistant individuals have a genetic variant, or allele, for a specific gene—CCR5. This gene codes for a cell-membrane protein, also called CCR5, which typically facilitates the incorporation of HIV particles into host immune cells (see Figure 1). Resistance to HIV is associated with one or two copies of a defunct version of the CCR5 gene. Being born with a copy of the non-functioning version of CCR5 is clearly desirable, given the death toll associated with HIV infection and AIDS.

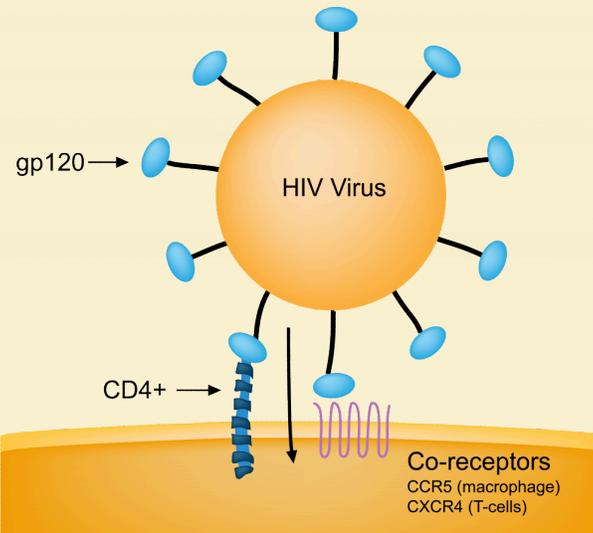


Figure 2.1 A simplified diagram of an HIV virus docking to a human t-cell (note the use of the CCR5 docking protein)

But there's a catch: the advantageous CCR5 gene is most common in Europe, especially in Northern Europe. For example, approximately 25% of Icelanders sampled had a non-functioning (and therefore desirable) version of the gene. In Britain, Spain, and Italy, the frequency of the resistant allele is 11%, 9%, and 5%, respectively. Yet in Nigeria, with over 3 million people living with AIDS, only 1 of 111 people sampled had a copy of the resistant allele. And in Kenya, where almost 7% of the population is infected with HIV, the resistant allele has not been detected.

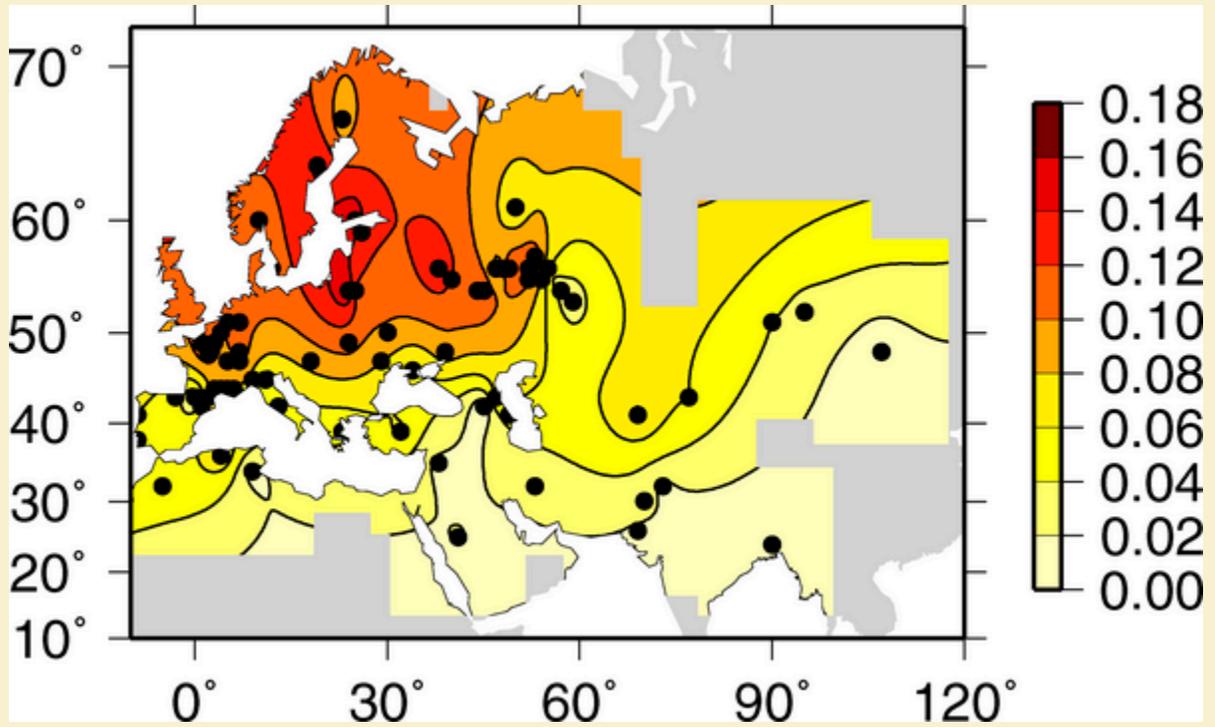
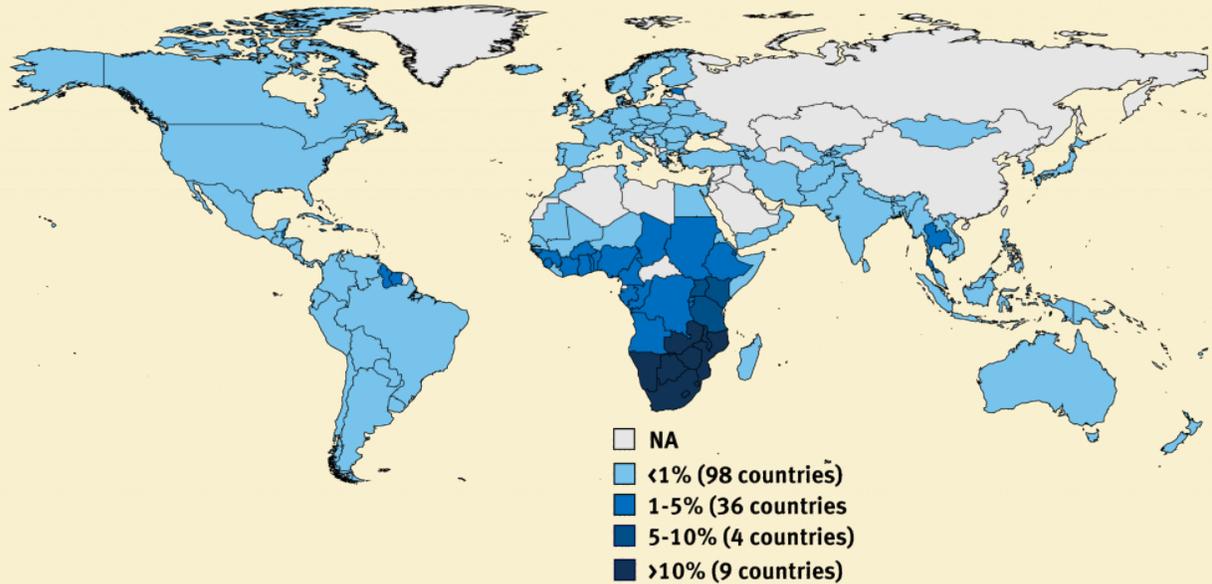


Figure 2.2 The global prevalence of HIV/AIDS

Adult HIV Prevalence Rate, 2012

Global HIV/AIDS Prevalence Rate = 0.8%



NOTES: Data are estimates. Prevalence rates include adults ages 15-49. The estimate for Sudan represents data for South Sudan. An estimate was not provided for Sudan.
SOURCE: Kaiser Family Foundation, www.GlobalHealthFacts.org, based on UNAIDS, Report on the Global AIDS Epidemic; 2013.



Figure 2.3 The global prevalence of the CCR5 mutation



Points to Ponder

- How is it possible that resistance is most common where it is least necessary?
- Why aren't there more resistant individuals in Sub-Saharan Africa, where the death toll is so great?
- Finally, can an understanding of evolution help us understand these harsh realities?

2.1 Chapter Objectives



Learning Objectives

Our goal for this chapter is for you to appreciate the fundamental tenets of evolution, and how evolutionary thinking frames much of our understanding of biology. By the end of this chapter and our in-class discussion, you will be able to:

1. Define the following terms:
 - **evolution**
 - **fossils**
 - **fossil record**
 - **natural selection**
 - **petrification**
 - **homologies**
 - **shared derived traits**
 - **analogies**
 - **convergent evolution**
 - **vestigial traits**
 - **biogeography**
 - **continental drift**
 - **mutation**
 - **gene flow**
 - **adaptation**
 - **fitness**
 - **sexual selection**
 - **genetic drift**
 - **founder effect**
 - **phylogeny**
2. Describe evolution and how it occurs.
3. Explain, using specific examples, several lines of evidence suggesting that evolution has occurred.
4. Articulate conditions that must be met for a population to evolve by natural selection.
5. Describe how evolutionary mechanisms can result in the generation of new species.
6. Cite evidence human evolution is ongoing.

2.2 Evolution Overview

What is evolution?

Simply, **evolution** is change over time. More specifically, evolution has occurred when there are genetic changes from one generation to the next in a population of organisms. Sometimes, it is possible to observe evolutionary change: for example, we can detect when a population of gonorrhea bacteria become resistant to a commonly prescribed antibiotic—the antibiotic is no longer effective against gonorrhea. However, some evolutionary changes are too subtle to be detected over the human lifespan.

Is evolution a fact or a theory?

Evolution is both a fact and a theory. When a change in a population can be observed, the observed change is evolution, and it is a fact. However, the mechanism by which the change occurred (such as gonorrhea's increased resistance to an antibiotic) is open to scientific scrutiny. We can hypothesize that gonorrhea became resistant through one of a few different evolutionary mechanisms, and we can then test appropriate predictions. In over 150 years of exploration, the evolutionary mechanisms discussed below have done an excellent job meeting predictions and explaining how life forms change. In fact, the evidence in support of these evolutionary mechanisms is so overwhelming, we can consider evolution among the most robust of scientific theories.



Consider the following questions

- What's an example of an observable evolutionary change?
- Define evolution.
- Why is a non-functioning version of the CCR5 protein potentially advantageous?

2.3 What is NOT evolution?

Evolution is a powerful idea that can be used to understand many natural phenomena, however it has limits. For example, individual organisms do not evolve; biological evolution is a population-level phenomenon, and cannot be used to explain changes in a single individual over the course of a life span. When individuals change over time, these changes may be due to development, conditioning, or learning, but they aren't evolution.

Evolution does not occur on demand, or to meet a pre-conceived goal. A dark insect on a light background may be visible to predators, and would likely benefit from a change that caused the insect to be lighter and thus concealed from danger. However, evolution can only change the frequency of characteristics that actually exist. Thus, if no light-colored insects exist in the population, then the frequency of light coloration cannot change. Similarly, resistance to HIV would be desirable in Sub-Saharan Africa, but resistance alleles do not occur there. If resistance ever does arise in HIV-plagued countries such as Botswana or Kenya, we might see a change in the frequency of HIV resistance in those populations. However, evolution cannot cause desirable features to arise on demand.

Evolution cannot be stopped. Populations are always changing, for better and for worse. Biologists often speak of evolutionary change that involves a population-level increase of desirable features. For example, inland plants that colonize a coastal area may become more salt-tolerant in their new habitat. But sometimes, useful features are lost. Human ancestors could synthesize their own vitamin C, but modern humans cannot. The loss of this ability has led to thousands of human deaths from scurvy, a debilitating disease that results from insufficient levels of vitamin C.

Evolution doesn't result in perfection. An insect that blends into its background may have extra protection against predators, but is still susceptible to disease-causing parasites, drowning in a flash flood, or getting squished by a human foot. Just like humans who must consume certain foods for their necessary vitamin C, or people who lack HIV-resistant genes, all organisms are biologically imperfect. Instead, we are the product of an evolutionary history that lacked foresight (and the inability to predict the consequences of vitamin-C deficiency), and evolutionary processes that could only work with the genes (such as non-functioning CCR5 alleles) that already existed in the population.



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<https://open.lib.umn.edu/evolutionbiology/?p=1207#h5p-11>

2.4 How do we know evolution has occurred? Fossil evidence.

Fossil evidence

Fossils are the preserved remains or traces of organisms that lived in the remote past (over 10,000 years ago). There are many different types of fossils that occur in various steps of completeness, collectively forming the **fossil record**, which provides a wealth of information about evolution:

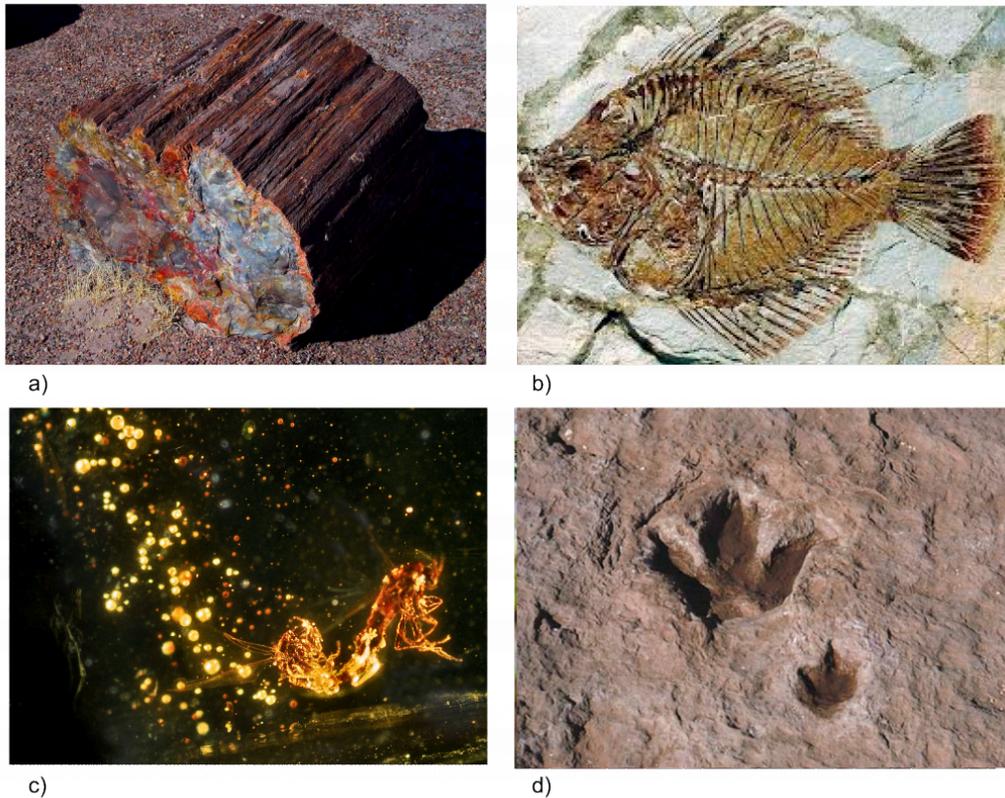


Figure 2.4 Examples of fossilized remains **a)** petrified wood from Petrified Forest National Park, Arizona; **b)** a fossilized flatfish; **c)** two 125-million-year-old dung midges, perfectly preserved in amber, are the oldest preserved mating pair in the animal kingdom; and **d)** fossilized dinosaur footprints.

- Some fossils are *mineralized remains* that result from a process in which living tissues are replaced by minerals such as quartz, calcite, and silica. In some cases, a near-perfect representative of an organism is captured in stone—this is called **petrification**. Petrified wood looks a lot like a tree trunk or branch, but if you touch it or kick it, you'll find out quickly that it's made of stone. We have learned a lot from petrified wood; for example, the discovery of fossilized tree trunks and leaves in Antarctica suggest the icy landscape was once covered by lush green forests.
- The remains of some organisms are only evident as casts or molds. Molds are impressions left by organisms, whereas casts represent molds that have been filled in by mineralization. In particular, sedimentary rock, which is formed by years of sand accumulating in layers on ancient sea floors, is an

excellent source of fossil remains of aquatic organisms. For example, recent discoveries of fish casts have clarified some details about flatfish evolution. Flatfish, such as flounders, have eyes on one side of their heads, posing the question: how did such a body plan evolve? In the past several years, many examples of ancient fish with “transitional” eye arrangements have been discovered; specifically, these fish had eyes that were arranged intermediate to those of flounders and typical bony fish.

- Some organisms persist long after death as **unaltered remains**. Unaltered remains have changed little since they died, and include hard, decay-resistant tissues such as teeth or shells. Soft tissues can also persist for thousands of years if they have been protected from heat or oxygen (both of which cause decay). Unaltered remains are typically found in ice, amber, or tar deposits. In 2012, an international team of scientists discovered two new species of small insects perfectly preserved in amber (fossilized tree resin) from over 100 million years ago. The discovery of these insects, called thrips, is exciting by itself, but the thrips also carried tiny pollen grains on their wings and abdomens. This is the oldest known evidence of **pollination**, whereby an animal carries a plant’s sperm (as pollen grains) to eggs. This is also the first example of pollination from the era of the dinosaurs.
- **Trace remains** are fossilized evidence of living organisms, but not the actual organisms themselves. Examples of trace remains include footprints preserved in rock, mineralized feces (or *coprolites*), and the molds of once-inhabited burrows. The recent discovery of reptile footprints, in a location that would have been hundreds of miles from the sea, suggests that reptiles have been able to move into continental interiors for at least 318 million years. This discovery pushed back earlier estimates for the movement of reptiles away from the sea by several million years.



Consider the following question

What are some possible limitations of fossilized evidence?

2.5 How do we know evolution has occurred? Comparative anatomy.

Comparative anatomy

Related organisms have similar bodily structures, or anatomy; for example, the skeletons of humans, rats, and bats are similar, despite numerous obvious differences. In fact, there is a bone-by-bone similarity in the bodies of these three animals. Anatomical similarities are particularly evident in a comparison of the forelimbs of the pterodactyl, bat, hawk, whale, and human (figure 4). These animals live in different types of environments, and use their limbs for different functions—flying, swimming, or grasping and throwing. Despite these different functions, all of these animals possess limbs with certain shared characteristics: a five-digit structure, a single large bone (*humerus*), two bones in the forearm (*ulna and radius*), and several wrist bones.

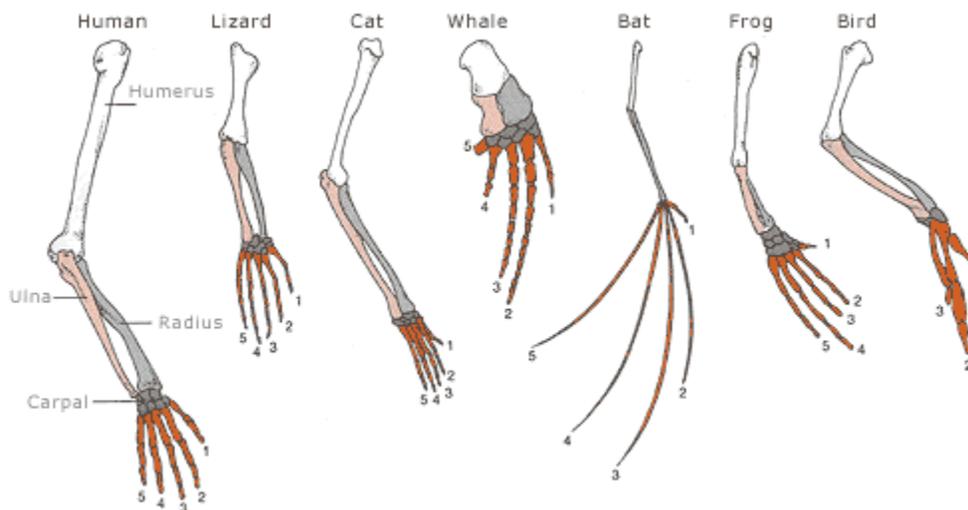


Figure 2.5 Homologies in vertebrate forelimbs.

In fact, all four-limbed animals (or *tetrapods*)—amphibians, reptiles, birds, and mammals—have similar limbs. Anatomical similarities, such as those that characterize tetrapod limbs, are used as evidence for common ancestry. In other words, these similarities suggest that pterodactyls, bats, hawks, whales, and humans all share a common ancestor somewhere in their distant past. Learning about shared ancestry provides an important clue to understanding an organism's evolution.





Figure 2.6 Leaves as homologous structures. Cactus spines, the leaves of a maple tree, and the cup-like “pitcher” of a pitcher plant are all modified from a common structure in an ancestor shared by all leaf-bearing land plants.

- Homologous structures** are anatomical features that different organisms share *as a result of a common ancestor*. The tetrapod limbs discussed above are homologous structures, or *homologies*. Because they evolved in an ancestor and are currently shared by different organisms, homologies are often called **shared derived traits**. Cactus spines, the leaves of a maple tree, and the cup-like “pitcher” of a pitcher plant are all modified from a common structure in an ancestor shared by all leaf-bearing land plants. While these leaves look different, and have evolved to serve different purposes, they are homologous structures and tell important stories about each plant’s history (figure 5).
- Analogous structures** are anatomical features that different types of organisms share, but not as a result of a shared ancestor. Instead, analogous structures, or analogies, are similar because the organisms have changed in response to similar environments. When distantly related organisms share features as a result of similar environmental pressures (and not because of common ancestry), we say that they have undergone **convergent evolution**. For example, arctic mammals such as foxes and snowshoe hares grow white fur during the winter months. White fur allows these organisms to blend into the ice and snow that characterizes their polar home, and presumably protects them from predation. However, foxes and snowshoe hares do not share a common ancestor *with white fur*. Of course they ultimately share a common ancestor, as do all mammals, but the fox lineage is full of non-white animals, as is the group to which hares belong. The winter white of arctic foxes and snowshoe hares is thus an analogous structure, due to convergent evolution in a white, wintry landscape.
- Vestigial structures** are anatomical features that are either no longer in use, or their use has been greatly reduced or altered. Vestigial structures, or *vestigia*, provide clues to an organism’s history by suggesting what features were useful in the past, and by linking an organism to other, related organisms. For example, many mammals exhibit *piloerection*, whereby muscles constrict around the hair follicles and the animal’s hair stands on end. If you’ve ever surprised or otherwise threatened a dog or cat, you’ve probably seen the results of piloerection. Humans are mammals too, but we’ve been reduced to a scant covering of body hair. When we are scared, we also constrict the muscles around our hair follicles, but this just gives us goosebumps (Figure 6). Goosebumps aren’t very scary, but as vestigia they link us to our mammalian relatives, and tell us something about our evolutionary history.



Figure 2.7 The Vestigial piloerection of humans leads to goosebumps...not very scary!

2.6 How do we know evolution has occurred? Genetic evidence.

Genetic evidence

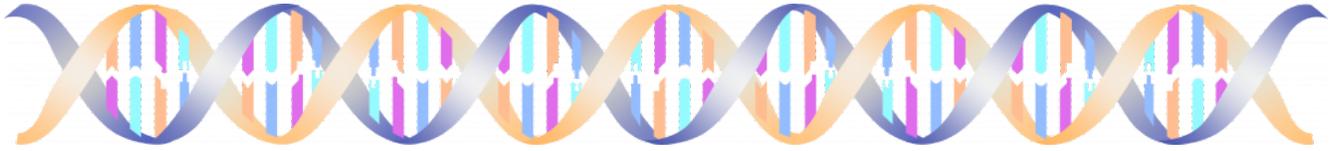


Figure 2.8 A double helix strand

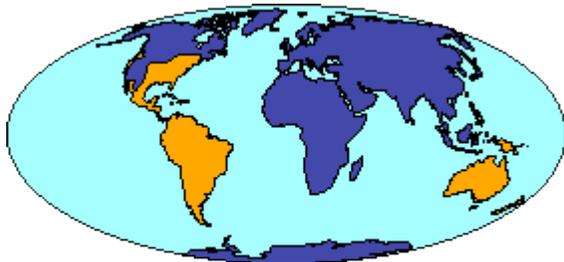
All living organisms share certain features, presumably due to our descent from a single common ancestor. These shared features are called *universal homologies*, and include our cellular organization (all living things are made of cells), our use of the molecule ATP to do work at the cellular level, and our common *genetic code*. The genetic code is a set of rules governing how the subunits of a gene (the nucleotides) correspond to the subunits of a protein (amino acids). Biologists use an understanding of the genetic code to study evolutionary relationships among different groups of organisms. They do this by comparing the proteins that are produced, or by looking for differences in the genes themselves. Genetic analyses may clarify differences between populations, such as the presence of the non-functioning *CCR5* gene (discussed above) that confers resistance to HIV. Some populations (for example, Icelanders) have a high frequency of the gene, and others (such as Kenyans) don't have this version of the gene at all—this difference is evidence for evolution in humans. Biochemical comparisons can also highlight what genes have been maintained, or *conserved*, over time. For example, a gene essential to sperm production is present in all sperm-producing animals—from sea anemones to leeches to humans. These animals share a common ancestor that lived 600 million years ago; thus, this highly conserved sperm-producing gene must have been pretty important during animal evolution.

2.7 How do we know evolution has occurred? Biogeography.

Biogeography

Biogeography, the study of the distribution of living organisms, addresses several evolutionary questions: How many types of organisms exist? Why are some types of organisms (e.g. insects) more abundant than others (e.g. mammals)? Why do certain organisms live in some places and not others? Why do islands have such distinct biodiversity compared to the larger continents? Why aren't there any polar bears in Antarctica? Why aren't there any giraffes in Hawaii? And so on....

Likewise, an understanding of evolution helps us appreciate the otherwise-perplexing global distribution of marsupial mammals. **Marsupials** are mammals in which females transport their young in distinctive pouches throughout their early infancy. In contrast, **placental mammals** have placental gestation and young that are born at a more developed stage. Marsupial mammals, such as kangaroos, opossums, wombats, and wallabies, occur in North and South America, Australia, and New Guinea, whereas the more-numerous placental mammals dominate the rest of the world. How can we explain these odd distribution patterns?



■ Distribution of marsupials today



Jurassic Period – 160 mya

Figure 2.9 Distribution of marsupials today versus Jurassic period

Knowledge of Earth's history is key to biogeography and evolutionary understanding. Indeed, a consideration of **continental drift**—the movement of the continents, over geologic time, as a result of the movement of plates in Earth's crust—is necessary to understand marsupial biogeography (figure 7). We also know, from fossil evidence, that marsupials originated over 150 million years ago in China, at a time when the Asian and North American landmasses were joined. Marsupials dispersed to South America, and from there to Antarctica and Australia, both of which were attached to South America. When Australia, Antarctica, and South America drifted apart,

each landmass carried a population of marsupial mammals. Over time, Australia's marsupials evolved into the bandicoots, kangaroos, koalas, and other marsupials that inhabit the continent today. When Australia moved closer to Asia, about 15 million years ago, placental mammals such as rats and bats colonized the landmass.

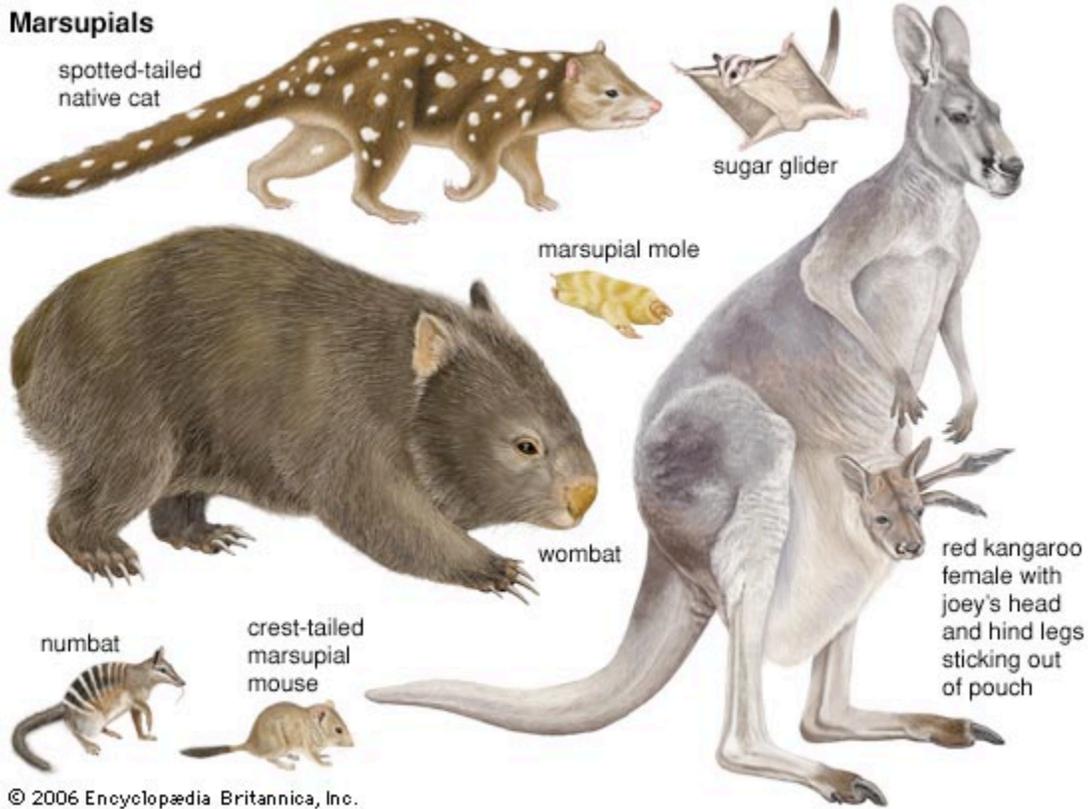
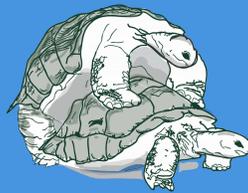


Figure 2.10 Marsupials



Biology is Sexy

The mystery of ape testes

In addition to homologies, analogies, and vestigia, comparative anatomy can inform our understanding of evolution by simply shedding light on which structures are most important in which organisms. For example, a comparison of great-ape genitalia can be an interesting exercise. The great apes include the gorillas, orangutans, chimpanzees, and humans. The great apes are unified by several shared derived traits, including bone structures that enable us to walk upright (although the non-human apes can only do so for a few steps at a time), opposable thumbs, three-dimensional vision, and large brains. We differ, however, in several features, including our mating habits.

Male gorillas fight amongst themselves—sometimes to the death—for dominance in a community; the dominant male then enjoys a harem of sexually available females. Chimpanzees are promiscuous, with both males and females

copulating with several other chimpanzees in a single day. And then there are the humans, exhibiting various mating strategies (from harem-building to serial monogamy to life-long monogamy) that appear to be somewhat dependent on culture. But what was the original human mating system? Did we come from an ancestral human that was monogamous, promiscuous, or somewhere in between?

The answer may lie partly in the testes, where sperm are produced in a sac-like scrotum hanging outside of the male's body. Animals display considerable variability in testis size. For example, within bats, testes volume varies greatly among species: the yellow-winged bat's testes make up only 0.11% of the bat's total body mass; in contrast, the Rafinesque's big-eared bat has testes that make up 8.38% of its body mass (why is it called the big-eared bat anyway?) And in the great apes, chimp males have the largest testes, relative to body size; gorillas have the smallest. For the promiscuous chimps, large testes may be mandated by sperm competition, whereby males must compete, through their sperm, for access to the female's limited supply of eggs. If a female mates with several males, each individual male is thrust into competition with the others, and no male can compete without an ample supply of sperm. This demand for more sperm is met by larger testes. Sperm from gorilla males, on the other hand, do not need to compete with sperm from other males, once a male gorilla has established dominance and secured a harem of available females no other males are mating with the females, therefore reducing advantage of producing huge quantities of sperm. A reduced demand for sperm appears to have resulted in smaller testes.

Human males have testes that sit somewhere in between those of the gorilla and those of the chimpanzee. What does this type of comparative anatomy tell us about our ancestral mating habits? We certainly don't appear to have been as promiscuous as the chimps, but we probably can't rule out sperm competition altogether. Human testes size, together with other evidence of our evolution, suggests that we have functioned somewhere in between the two extremes. Perhaps we have been largely monogamous, with low—but significant—levels of extra-pair copulation (or "cheating").

Interesting tales of ape genitalia don't end with the testes. The human penis is remarkable in being much longer, relative to body size, than the penis of the other great apes. An interesting observation and a story for another time...

2.8 The Importance of Variation

Variation is essential

Without differences between the individuals in a population, populations cannot change over time—that is, they cannot evolve. Some features must exhibit variability so that change is possible. This variation arises when novel genes are produced as **mutations**. In other words, mutations—random genetic changes—are the ultimate source of variation in changing populations.

When a novel mutation arises, the population has changed; thus, *mutation alone is a mechanism of evolution*. However, the occurrence of a single random mutation is unlikely to have a profound effect on a population. Other evolutionary mechanisms—such as gene flow, drift, and selection—play a larger role in shaping the diversity we see on earth today.

However, without this pre-existing and recurring variation, we would not have an evolutionary story to tell.

2.9 Mechanisms of Evolution: Gene Flow

Gene flow is a profound evolutionary mechanism

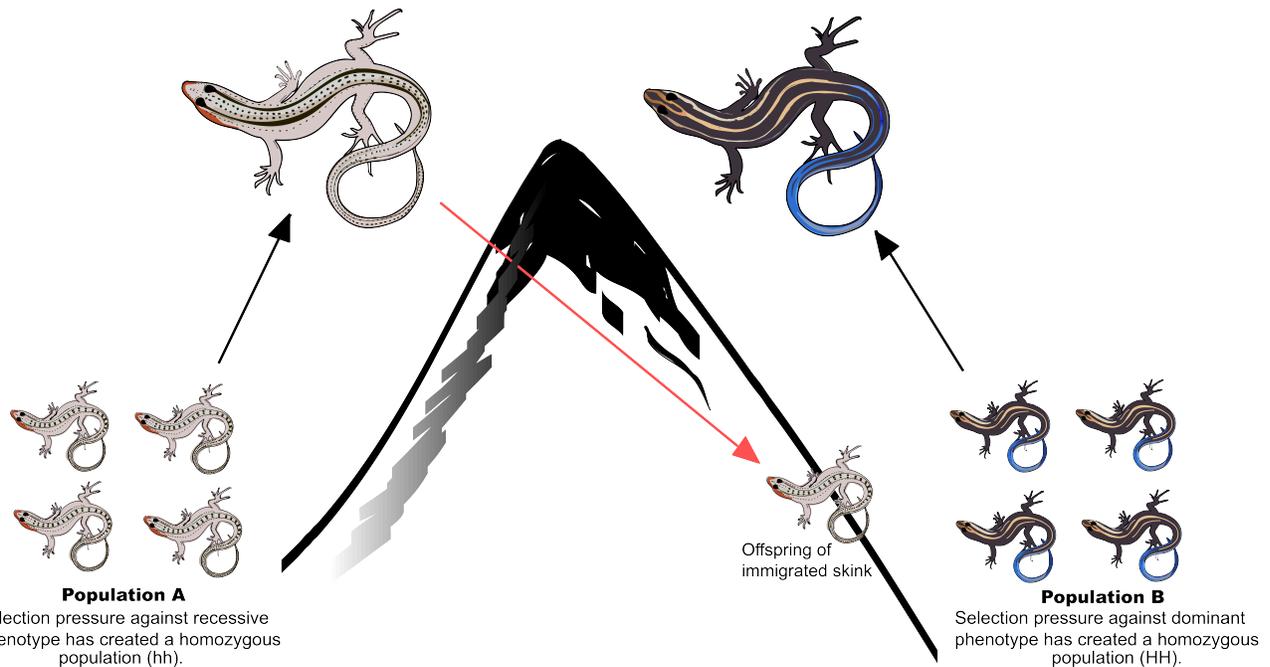


Figure 2.11 Gene flow helps create diversity

Gene flow, or *migration*, occurs when individuals move between populations. When this happens, some of the novel mutations in one population will migrate to the new population with the migrating individuals. The original population may become less diverse as a result of losing one or more of its individuals. Similarly, the migrants are likely to make their new population more diverse through the introduction of new gene variants. There are many examples of populations evolving through gene flow. An appreciation of gene flow can help us understand the global distribution of the CCR5 mutation in humans (discussed above, in the introduction to the chapter). The CCR5 mutation confers resistance to some forms of HIV, yet is not most common in areas with a high prevalence of HIV and AIDS. The mutation is relatively new: biochemical and biogeographic evidence suggest an origin in Northern Europe approximately 1200 years ago. However, the mutation was distributed long before HIV and AIDS were relevant to human health. In fact, the mutation's distribution pattern mirrors the Viking migration of the 9th through 11th centuries (Figure 8). Thus, we can hypothesize that Vikings carried the mutation with them as they conquered new territories, and passed the mutation to their descendants. But why was this genetic feature prevalent in Vikings? We'll develop that story further, below.

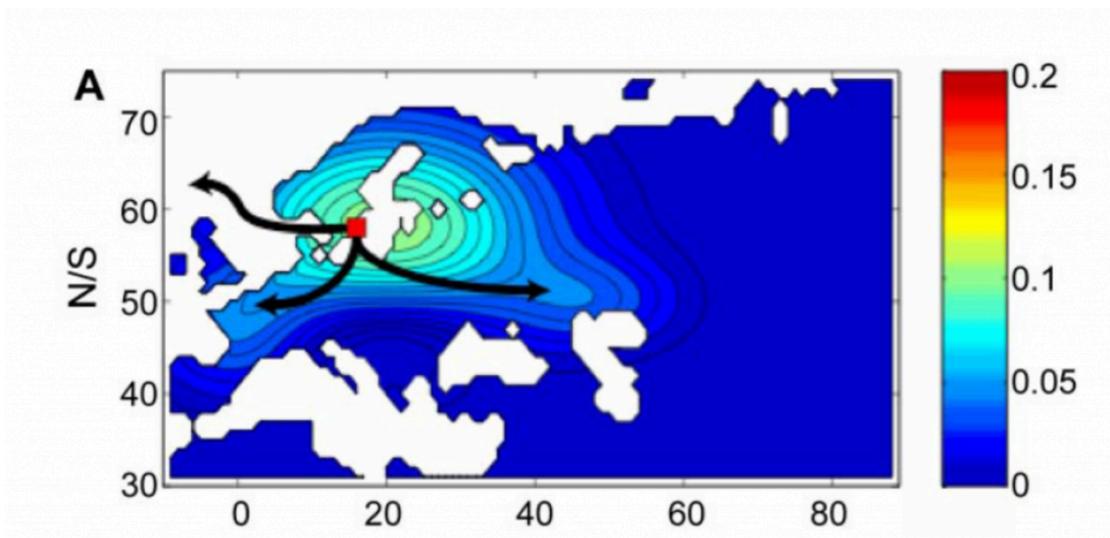


Figure 2.12 Did the Vikings distribute the CCR5 mutation?

2.10 Mechanisms of Evolution: Genetic Drift

With genetic drift, the key word is “random”

Genetic drift occurs when a population experiences random fluctuations in frequencies of genetic traits. The term “random” is key to an understanding of drift. If any heritable variation leads to genetic changes in a population, natural selection has occurred. Drift has occurred if these changes are unrelated to any heritable feature possessed by individuals in the population. Sometimes, as a result of chance events, certain individuals do not reproduce, and the population evolves as a result of drift. While natural selection results from aspects of an organism’s environment exerting “selective pressure” on the individual (e.g., the desert environment favors the spines of the cactus and the long ears of the fox), drift, by definition, is not a result of environmental pressures. Drift is common to small populations, such as those that colonize islands or other isolated habitats, or those that remain after large-scale disruptions (e.g. earthquakes, fire). Think about it: the random loss of 20 iguanas from a large population of 1 million iguanas is bound to result in fewer overall genetic changes than is the random loss of 20 iguanas from an island population of 100 iguanas.

One form of drift is the **founder effect**, which results when a small part of a population (the “founders”) moves to a new location, and the small variety of genes of the founders mean that the new population is different from the original population. Ellis-van Creveld Syndrome (a form of dwarfism that involves short stature, extra fingers or toes, and possible heart defects) and several other genetic conditions that are typically rare in large populations, are common in the Pennsylvania Amish population in the United States because of drift—specifically, because of the founder effect. This group was founded relatively recently by a few individuals who presumably had genes for these conditions. The population has grown, but has not been subject to the diversifying effects of gene flow (that is, individuals rarely enter the population from another population). Therefore the current population has these conditions in much higher frequency than the population from which they migrated.

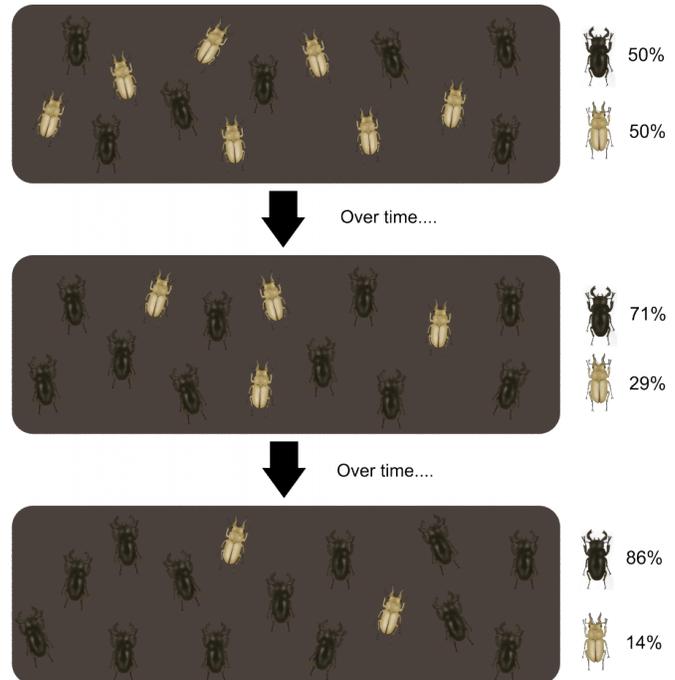
2.11 Mechanisms of Evolution: Natural Selection

Natural selection leads to adaptive evolutionary change

Any feature that benefits an individual in its present environment is considered *adaptive*, and the feature is referred to as an **adaptation**. Simply, individuals who possess an adaptation are more likely to survive and reproduce than are individuals without the adaptation. If some or all of the adaptation is inherited, we expect individuals with the adaptation to survive and pass the feature to their offspring in greater numbers than those in the population who do not have the adaptation.

Natural selection refers to adaptive evolutionary change or change that occurs when heritable adaptations confer a survival and reproductive benefit that, in turn, changes the genetic makeup of a population. Natural selection can change populations, species, and whole groups of organisms.

If you consider the speed at which some populations can grow, and the fact that resources are limited, it should be clear that there must be some culling mechanism working to limit population sizes. This mechanism is natural selection: individuals who inherit adaptations simply out-compete (by out-surviving and out-reproducing) individuals that do not possess the adaptations. Evolutionary “winners” are those individuals that survive to reproduce and pass on adaptations to their offspring.



Beige beetles have been selected against while black beetles have survived and reproduced.

Figure 2.13 Evolutionary development of beetles. Which beetles have been selected against?



Consider the following questions

- Give an example of evolution by gene flow.
- What is the relationship between adaptation and natural selection?
- What are some examples of adaptations?
- What is the relationship between natural selection and evolution?

- How might you determine whether a certain individual is an evolutionary “winner”?

2.12 Adaptation

Adaptations come in a variety of forms:

- Adaptations can be *anatomical* features, such as the strong caudal (or tail) fin of fish that cannot survive without strong swimming. The large ears of the desert fox is an adaptation that helps the fox dissipate heat in its hot environment. The modified leaves of the cactus, its sharp spines, are also adaptations for desert life; these spines limit the plant's loss of water in its arid climate and serve as defense against potential plant predators. The plant-like features of the stick bug allow it to blend in to its surroundings, an adaptation that confers protection against predation.
- *Biochemical* features, such as proteins, or the genes that code for proteins, can be adaptations. Genes for egg-yolk protein, vitellogenin, are present and functioning (producing egg-yolk protein) in fish, reptiles and birds. These genes for egg-yolk protein are adaptations that distinguish the egg-laying birds. Interestingly, the genes for producing vitellogenin exist in non-functioning, or *vestigial*, forms in all mammals with the exception of the egg-laying mammals, the *monotremes*: monotremes have one functional egg-yolk gene and one non-functional egg-yolk gene. These genes suggest a story about mammalian evolution. Namely, we began by nourishing our young with egg-yolk protein, like the modern-day reptiles and birds. As we developed the ability to lactate (i.e., feed our young with milk), we developed mutations that rendered us unable to make egg-yolk protein. With the ability to feed our offspring milk, the egg-yolk protein was no longer advantageous and therefore those with non-functional egg-yolk protein genes were not at a disadvantage. Thus a new group of organisms emerged without functional genes for egg-yolk production.
- *Behaviors* can be adaptations as well. A plant that orients its leaves to face the sun is responding, in an adaptive way, to its environment. A dog that barks at an intruder is exhibiting a protective adaptation, and the Cape ground squirrel that uses its own tail for shade is demonstrating a thermoregulatory adaptation. To see this tail in action, as a parasol and an anti-predator device, check out the following video. The arctic tern, which travels up to 60,000 kilometers from its arctic breeding grounds to the Antarctic, is implementing a behavioral adaptation that is energetically taxing but leads to reduced competition for resources, reduced predation pressure, and greater food availability.



Figure 2.14 Cape ground squirrel shading itself from the sun with its tail



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Fitness is the currency of evolutionary success

You can measure the effects of natural selection by determining an individual's reproductive success, or fitness. The evolutionary "winners" are those individuals who are best represented, genetically, in the next generation. A bird that lives longer than other birds, is more beautiful, and can fly further may seem like a winner. However, if he fails to reproduce, he won't have as much fitness as other birds that do have offspring. This concept of fitness is one that we'll return to throughout our discussions.

The inevitability of natural selection

Evolution by natural selection is as an inevitable consequence of life. If certain conditions exist, populations will evolve by undergoing natural selection. These specific conditions are:

1. *Organisms produce more offspring than the environment can sustain.*

Any population of organisms, reproducing at full capacity, will outstrip the available resources within a few generations. This leads to a competition for resources. This competition may be overt (e.g., a physical battle for resources or mates) or not (e.g., some individuals may be better at hunting or fishing or building nests). Among competing organisms, there will be winners and losers.

2. *Competing organisms exhibit individual variability.*

Variability between competing organisms may contribute to an individual's success or failure. For example, some finches may have adaptations that enable them to obtain food more efficiently than other finches.

3. *Some of the variability that organisms exhibit is heritable.*

If some individuals survive and reproduce because of inherited traits that the non-survivors lack, then the winners will pass these features on to the next generation, thereby altering the gene-frequencies of the population. This change in gene frequency, based on differential survival and differential reproductive success, is evolution by natural selection.

In summary, competition for resources and differential reproductive success inevitably lead to natural selection.



2.13 Sexual Selection

Sexual selection is a type of natural selection

Natural selection refers to evolutionary change that occurs when heritable adaptations confer a survival and reproductive benefit that, in turn, changes the genetics of a population. **Sexual selection** specifically refers to evolutionary change as a result of adaptations related to mate attraction, copulation itself, or mate retention. Sexual selection is at work when the peacock with the most elaborate feathers attracts the most peahens, and when male hercules beetles engage in combat (see figure below) for sexual access to females. Later in the course, we'll explore sexual selection further.



Figure 2.14 Male Hercules beetles using their large horns to fight each other

Is there a distinction between natural and sexual selection?

Sexual selection is a *type* of natural selection in which the adaptations are features related to mate choice, mate retention, and the reproductive act itself. With sexual selection, as with natural selection, adaptations can be anatomical (e.g., the long, corkscrew penis of the mallard duck), biochemical (e.g., the excessive production of testosterone by human males in the peak of courtship), or behavioral (e.g., the courtship song of the chickadee). As with natural selection, sexual selection involves adaptive change as a result of fitness benefits enjoyed by those with the adaptation(s). However, sexual selection does not involve adaptations that strictly concern survival, and

is therefore more limited in its scope. In other words: all forms of sexual selection are natural selection, but not all natural selection is sexual selection.

Read More

Read a cool story (featuring the University of Minnesota's own Marlene Zuk!) about natural and sexual selection in Hawaiian crickets at: http://evolution.berkeley.edu/evolibrary/news/061201_quietcrickets

2.14 Mechanisms of Evolution Overview

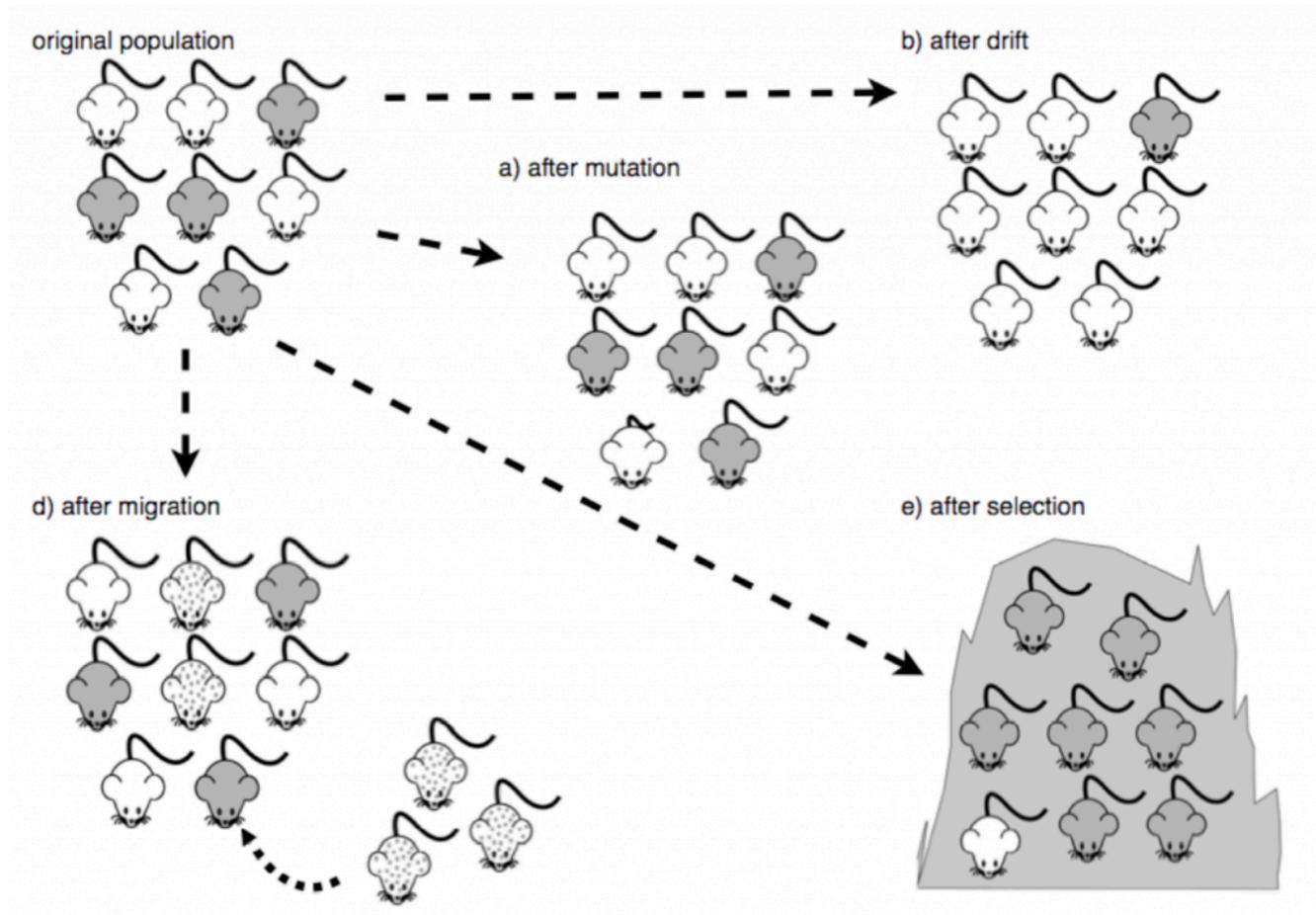


Figure 2.15 Populations evolve through mutation, migration (gene flow), drift, and selection. Image by Sehoia Cotner.



Consider the following questions

1. What are the differences between natural selection and genetic drift?
2. What is meant by the statement, “natural selection is inevitable”?
3. Is sexual selection the same thing as natural selection?

2.15 Biodiversity & Adaptive Radiation

Biodiversity is a product of evolutionary processes

Mutation and migration all play a role in generating diversity, and selection and drift eliminate some of this diversity. Over time, isolated populations of the same species can diverge

so much that they are considered separate species. For example, an ancestral population of lizards, possibly from mainland South America, colonized the Galápagos islands (in the equatorial Pacific Ocean) millions of years ago. After many generations of mutation, drift, and selection, the colonizing population was sufficiently different from the parent population to warrant classification as a different species. This pattern was then repeated throughout the islands, with migration, drift, mutation, and selection all acting differently on different island populations. Thus, from one colonizing species, the archipelago now has several different species of what we call *lava lizards*. All of these lizards are *endemic* to the archipelago—that is, they are found nowhere else in the world (Figure 2.15).



Figure 2.16 An endemic lava lizard on one of the Galápagos Islands

Adaptive radiation occurs when one ancestral species leads to several descendent species as new habitats are colonized. There are many examples of adaptive radiation and endemism in island chains such as Galápagos (e.g., finches, mockingbirds, lava lizards, and *Opuntia* cactus) and Hawaii (e.g. honeycreeper birds [Figure 2.16], fruit flies, and silversword plants). Adaptive radiations are also common where habitats are fragmented (e.g., by mountain ranges, rivers, etc.)

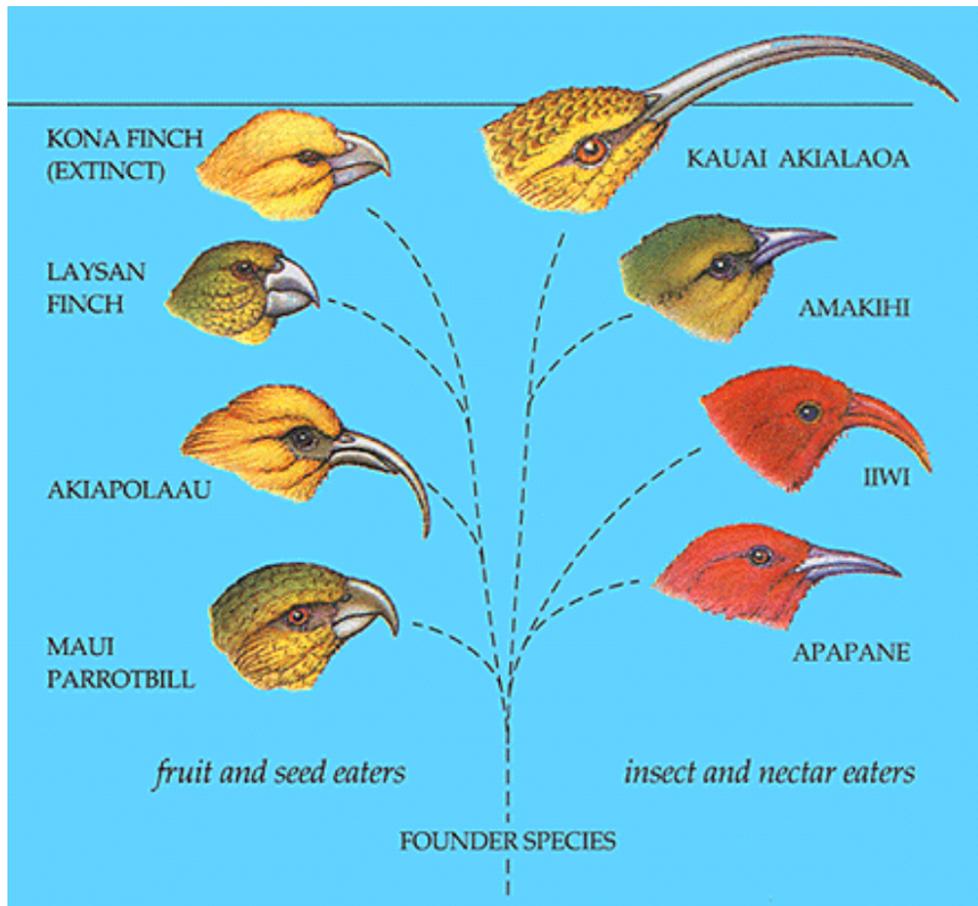


Figure 2.17 A few Hawaiian Honeycreepers, illustrating how diversity can result from the adaptive radiation of species from a single founding species.

2.16 Species & Phylogenetic Trees

What is a species?

It is surprising that, given the frequency and ease with which the term is used, there is no universally accepted definition of “species.” Historically, and in many contemporary uses, a **species** is defined as a group of actually or potentially interbreeding populations of individuals. This is the *biological species concept*; and its applications are vast. However, there are limitations to this species definition, as documented in the cases of asexual organisms and hybridizing populations. In addition, proponents of the *phylogenetic species concept* argue that the reproductive definition is evolutionarily irrelevant; instead, a species should be the smallest taxonomic unit, an intact group of organisms that share common ancestry. Yet the point at which one population is considered two distinct species is itself arbitrary, and based on perceived similarities—be they genetic, morphological, behavioral, etc. The situation is further confused by numerous other definitions of species.

In the typical model of speciation, a population is somehow subdivided into two or more **allopatric** (or physically isolated) populations. These populations experience different mutations, different selective forces and different random events of genetic drift, thus diverging in their evolutionary trajectories. Over time, the populations become sufficiently distinct to warrant identification as separate species. This distinction may be due to some sort of *reproductive isolating mechanism*, which can be either a *pre-mating isolating mechanism* or a *post-mating isolating mechanism*. Pre-mating isolating mechanisms are often behavioral differences (such as bird songs) or habitat preferences that prevent two individuals from courtship or copulation. Post-mating isolating mechanisms often result from genetic incompatibility, whereby offspring may result but they are not viable.

Phylogenies tell an evolutionary story

A **phylogeny** represents evolutionary relationships between different types of organisms. When we say “turtles, lizards, snakes, birds, and mammals are all amniotes (animals that reproduce with specialized, *amniotic* eggs), we are speaking *phylogenetically*.”

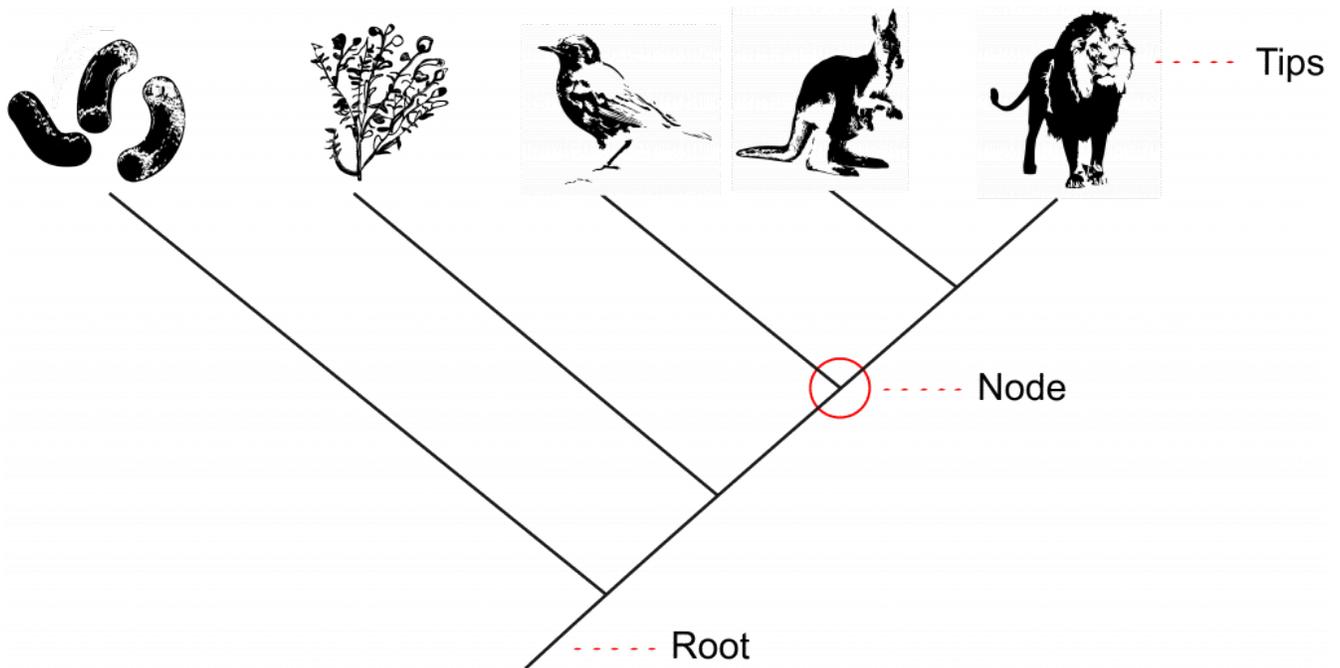


Figure 2.18 Phylogenetic trees demonstrate the evolutionary relationship between species

We use **phylogenetic trees** to visualize these relationships (Figure 12.8). In a phylogenetic tree, closely related organisms are joined by nodes. These nodes suggest common ancestry.

[Read More](#)

You'll read an article by David Baum and Susan Offner ("Phylogenies and Tree Thinking") that is an excellent introduction to phylogenies and the construction of phylogenetic trees.

2.17 Wrapping Up: Are Humans Evolving?



Are Humans Evolving?

Humans have been subjected to mutation, selection, drift, and migration

Humans, as living inhabitants of the planet, are subject to the same natural laws as all other living organisms. Evolution is no exception: the diversity of humanity today is the result of millions of years of biological evolution—mutation, selection, migration, and drift. This is not to say we aren't special in other ways! As the biologist George Gaylord Simpson said, "Evolution has no purpose; man must supply this for himself."

Return to the CCR5-mutation mystery

Thus far, we've established the following facts about the CCR5 mutation and HIV-AIDS:

- HIV-AIDS is a disease of the immune system, targeting the very cells that typically protect you from disease-causing agents or *pathogens*.
- Some individuals have a genetic variant that makes them resistant to some forms of HIV.
- Resistant forms of this *CCR5* gene are most common in Northern Europe, where HIV is not a strong selective force; in contrast, resistance is not found in sub-Saharan Africa and Southeast Asia, where HIV is a strong selective force.
- The resistant gene appears to have been spread by the Vikings, during their expansion through Europe in the 9th and 10th centuries.

But why was the genetic variant, or allele, present in the Vikings at all? The level at which we see this allele in Viking-invaded territories suggests selection *for* the CCR5 mutation; that is, it seems to have conferred some sort of benefit, but long before HIV was present in humans. Various hypotheses have emerged to explain the prevalence of the mutation, but many scientists agree that the nonfunctioning CCR5 protein must have protected the Vikings' ancestors against a pathogen similar in structure to HIV. One suggested pathogen is smallpox, as the steady rate of death from smallpox that has afflicted European children since the origin of the allele could have provided the necessary selective pressure to result in the frequencies of the gene that we see today (Figure 2.19)

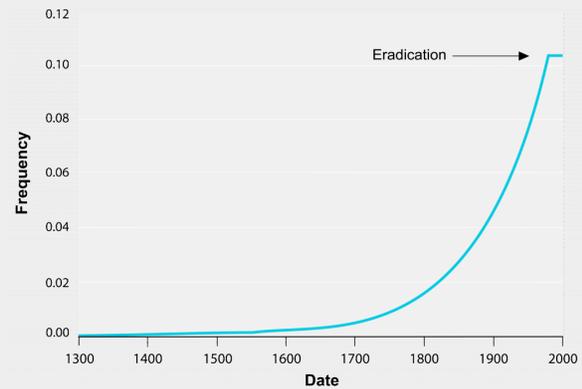
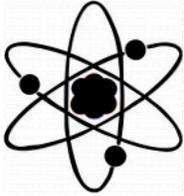


Figure 2.19 A change in P , the frequency of a dominant resistance allele, generated by smallpox mortality. A total of 680 years of smallpox are required for P to reach 10%. (From Galvani and Slatkin, 2003)

As we conclude this chapter and prepare for in-class discussion, be sure to return to the chapter's [goals and objectives](#).

Chapter 3: The Ingredients of Life



[3.0 Introduction](#)

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[3.2 What are living things made of?](#)

[3.3 Sugars](#)

[3.4 Nucleic acids](#)

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[3.7 Cells](#)

[3.8 Looking closer at organelles](#)

[3.9 Wrapping up: Revisiting the egg](#)

3.0 Introduction

Structure and function: a look at the egg and its parts

If you enjoy an egg for breakfast in the morning, when you are eating the yolk, you are eating a large single cell. What does that mean? What is a cell and what is it made of? What are the building blocks of life? The answer to these questions depends on the scale at which you are looking. In short, living things are made up of one or many cells, cells are made up of many different kinds of molecules, and molecules are made of atoms. In this chapter you will read about these basic ingredients of life. We will start with the smallest ingredients (atoms and elements), then examine the combination of atoms into molecules, and then explore cells, which are made up of molecules arranged in a highly organized fashion.

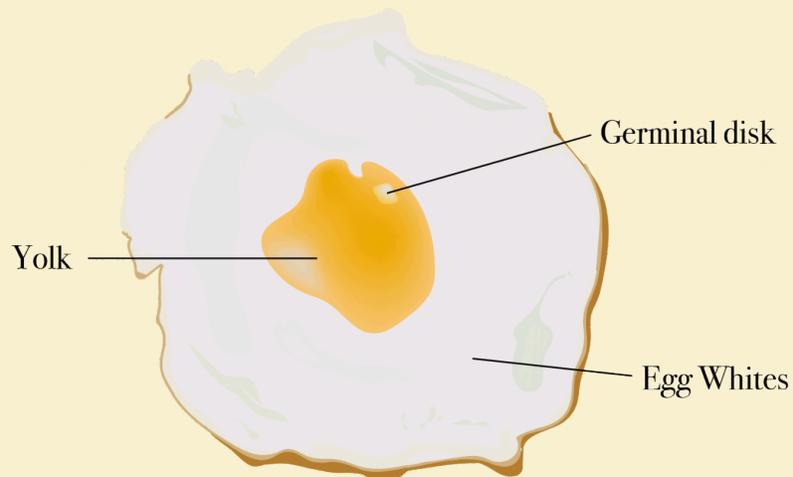


Figure 3.1 Visual breakdown of an egg and its parts. See any similarities to a cell?

3.1 Chapter Objectives



Learning Objectives

Our chapter goal is for you to have a basic understanding of the chemical foundations of life, as well as a basic understanding of how these chemicals interact in living organisms. By the end of this chapter, you will be able to:

1. Define the following terms:
 - **Cell**
 - **Element**
 - **Atom**
 - **Proton**
 - **Neutron**
 - **Electron**
 - **DNA**
 - **RNA**
 - **Prokaryote**
 - **Eukaryote**
 - **Organelle**
 - **Nucleus**
 - **Mitochondria**
 - **Chloroplast**
 - **Molecule**
 - **Chromosome**
2. Give examples of sugars, fatty acids, nucleic acids, and amino acids (note: you will NOT be expected to draw any of the molecules we discuss, however you should be able to recognize molecules by category).
3. Identify which elements are represented the most in living organisms.

4. Explain the varied functions of proteins.

3.2 What are living things made of?

If we took a sample of all living matter, put it in a blender, and further broke it down into its simplest parts we would have the **elements**. Currently 94 elements have been identified on earth. All matter that we know of is made up of one or more of these elements. You may have seen these elements listed in a periodic table like the one below (note the table has 118 elements – those past number 94 have been made by researchers and may or may not exist in nature).

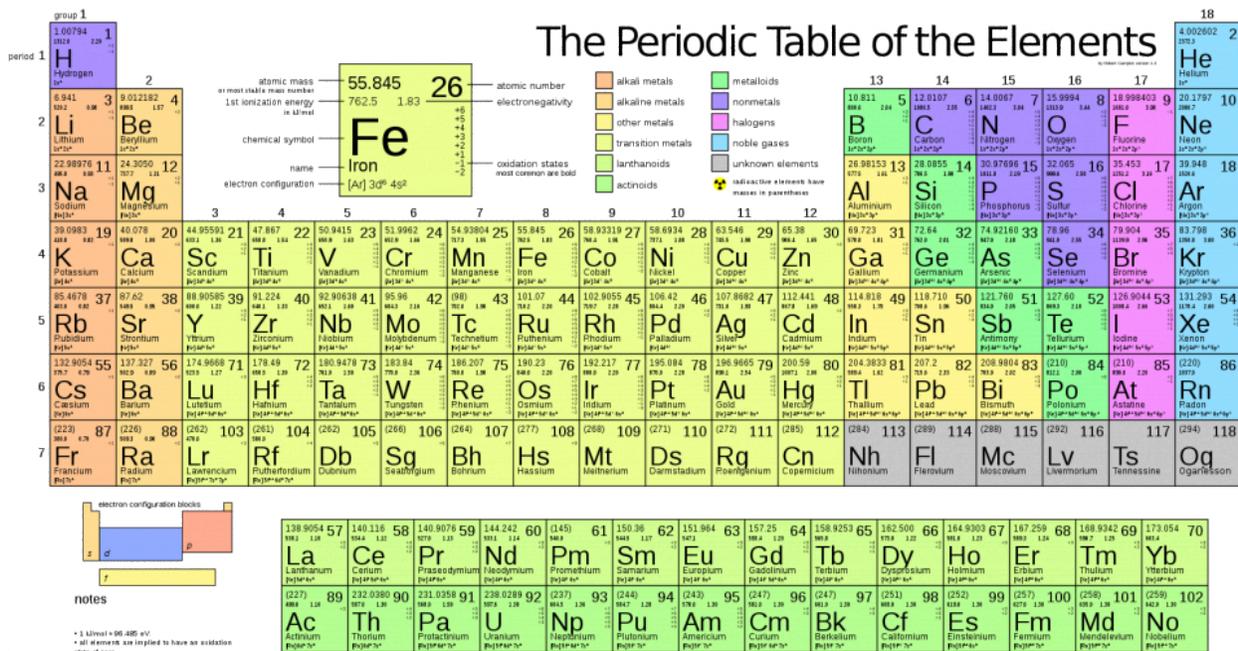


Figure 3.2 The Periodic Table of the Elements.

1

However, out of those 94 elements, the vast majority (over 98%!) of the human body (and the bodies of other plants and animals) is made up of only 6 of them. These most common biological elements are carbon, hydrogen, oxygen, nitrogen, calcium, and phosphorus.

1. By 2012rc. Own work. Notes and font fixed: The Photographer, CC BY 3.0. <https://commons.wikimedia.org/w/index.php?curid=8757312>

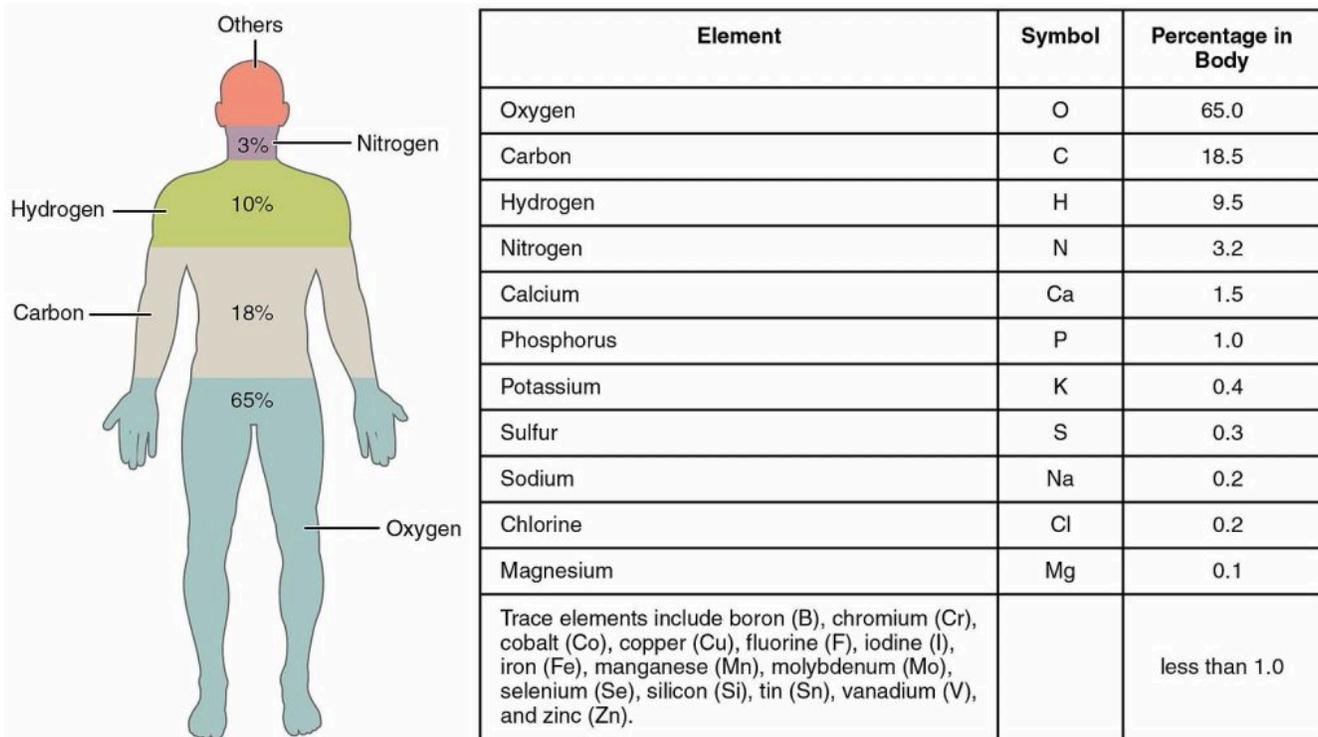


Figure 3.3 The main elements that compose the human body are shown from most abundant (by mass, not by fraction of atoms) to least abundant.

2

What we are made of

Astronomer Neil DeGrasse Tyson speaks about the beauty of what we are made of in this video:



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When you look at the ingredients of the universe the #1 ingredient is hydrogen, next is helium...oxygen, carbon nitrogen. ... What are we made of?... The fact that you rank the atoms in the human body, with the exception of helium, which is chemically inert...(look at the top ingredients of the human body)...[it] matches the universe....

So we've learned in the last 50 years that, of course, not only do we exist in this universe, it is the universe itself that exists within us."

One tiny piece of an element is called an **atom**. An atom is the smallest portion of an element that has the properties of that element. An atom contains a nucleus with positive and uncharged particles (**protons** and **neutrons**, respectively), and has negatively charged **electrons** that circle the nucleus.

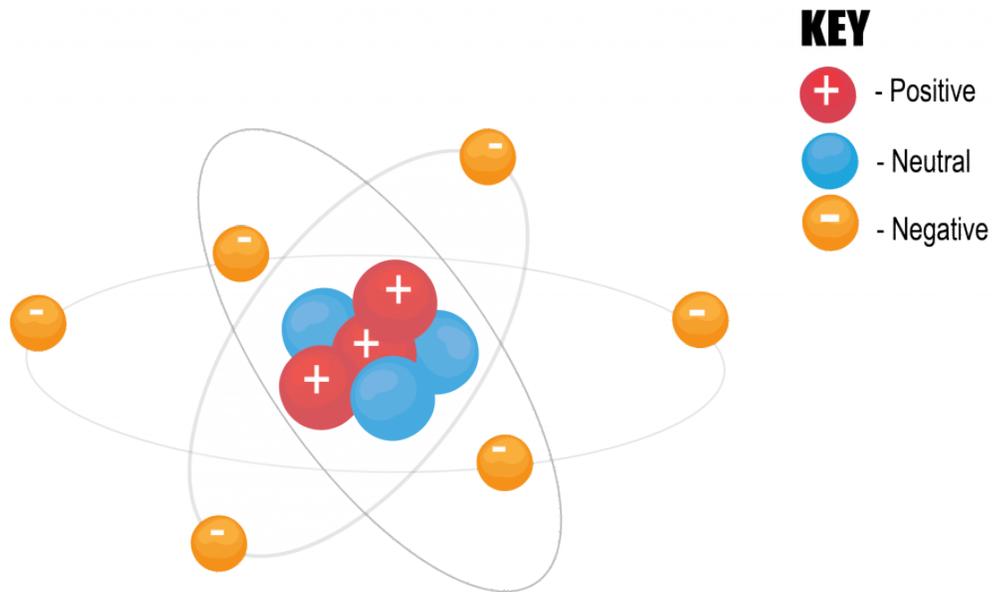


Figure 3.4 An atom's charges

The electrons of one atom can interact with electrons of other atoms and form chemical bonds. When two or more atoms of the same or different elements bond together they form molecules. The way these atoms are arranged into molecules impacts their properties and functions. One of the simplest molecules you may be familiar with is water, a molecule in which two hydrogen atoms are bound to an oxygen atom.

In biological organisms there are some important categories of molecules (called biomolecules) that are found in all forms of life. We will highlight a few of the biomolecule categories here, including:

- Sugars
- Nucleic acids
- Amino acids
- Fatty acids

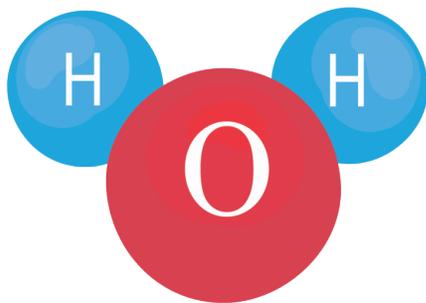


Figure 3.5 A water molecule

3.3 Sugars

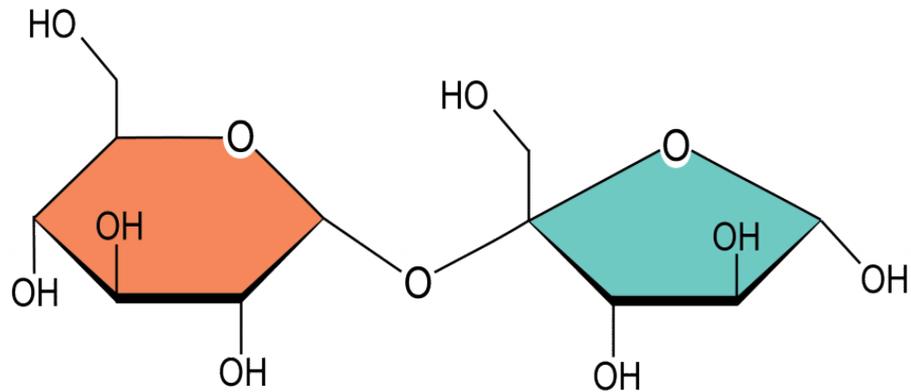


Figure 3.6 Image of a sucrose molecule

Sugars are composed of carbon, hydrogen, and oxygen atoms joined together to form one or more rings. There are many different types of sugar whose names may be familiar to you (glucose, fructose, lactose). The table sugar you might add to your coffee is called sucrose. Sucrose is actually a combination of two simple sugars: glucose and fructose. The sugars ribose and deoxyribose are essential parts of nucleic acids (discussed next).

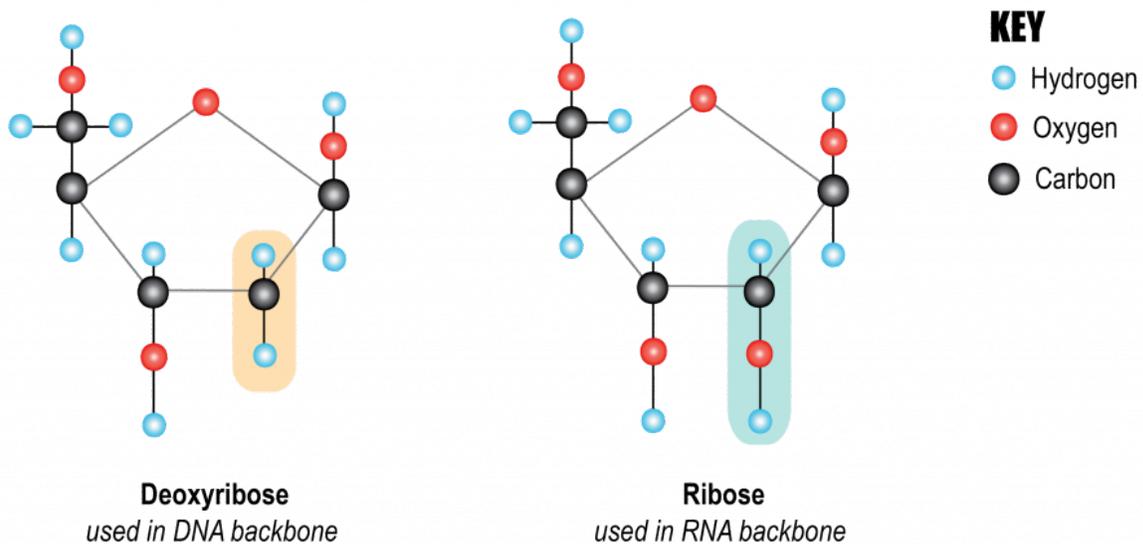


Figure 3.7 Image of a deoxyribose versus a ribose molecule

3.4 Nucleic Acids

Nucleic acids are the molecules that form deoxyribonucleic acid (**DNA**) and ribonucleic acid (**RNA**). DNA and RNA molecules carry information from location to location within an organism and from generation to generation. In short, DNA and RNA are information-containing molecules that are the blueprint for all life.

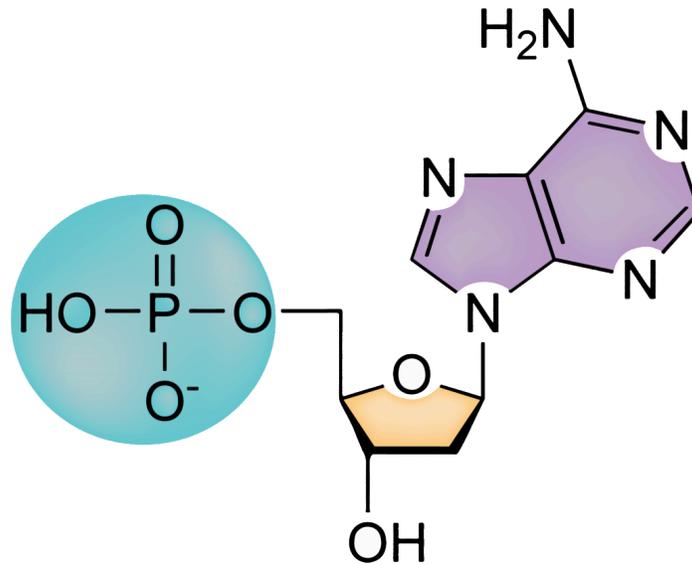


Figure 3.8 A nucleotide is made up of a phosphate, a sugar and a base.

The structures of both DNA and RNA consist of a strand of alternating sugars and phosphate molecules (a phosphorus atom surrounded by 4 oxygen atoms) along the “backbone” of the molecule. Attached to each sugar is a nitrogen-containing ring structure called a base. One phosphate, sugar, and base bonded together make up a **nucleotide**. These nucleotides join together to create the long stretch of nucleic acid that forms DNA or RNA.

DNA

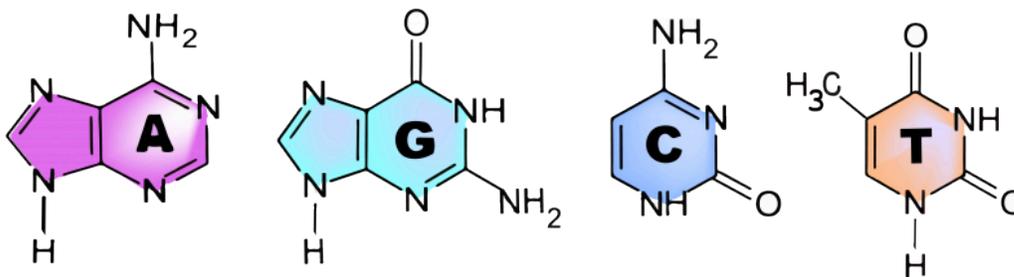


Figure 3.9 The four DNA bases – adenine, guanine, cytosine, and thymine.

In DNA there are four bases: guanine (G), adenine (A), thymine (T), and cytosine (C). DNA exists as a double-

stranded molecule, with two of these strands of nucleic acid paired together by interactions between the bases. In DNA, the A's pair with T's and C's pair with G's.

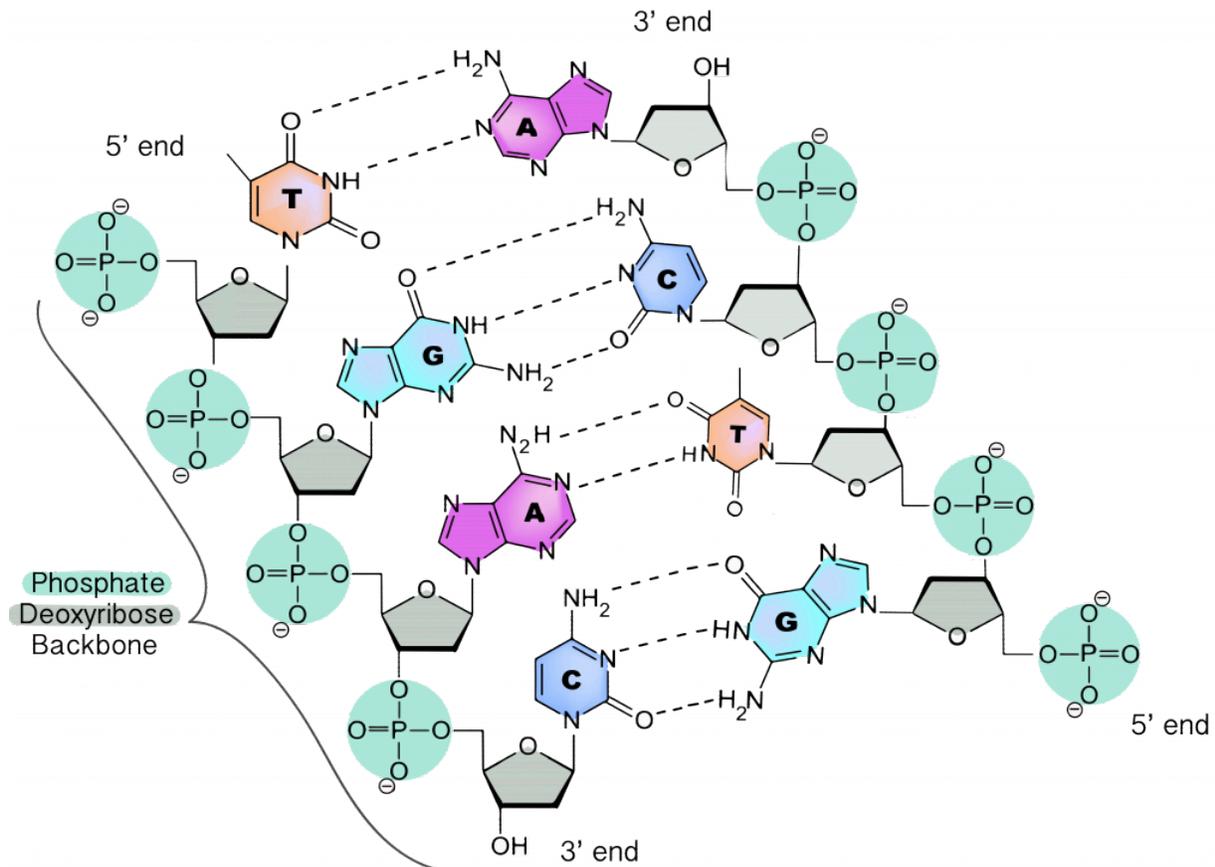


Figure 3.10 Image of DNA molecule showing its ladder-like structure

Thus, the DNA molecule can be thought of as a twisted ladder with the side rails of the ladder as the sugar-phosphate backbone, and the rungs of the ladder as the interacting bases.

If all of the DNA in a single human cell were stretched out, it would be about 6 feet long! However, DNA in a human cell is packaged into 46 separate strands, or chromosomes. These chromosomes spend most of their time in the cell in a messy (think plate of spaghetti) arrangement.

DNA is the molecule of inheritance for most biological organisms (there are some viruses that use RNA instead). Your shared DNA with family members has a lot to do with shared characteristics you may have with your biological relatives.

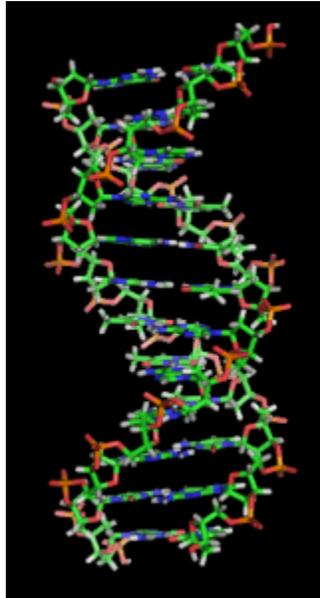


Figure 3.11 Another drawing of the DNA molecule showing its structure as a double helix (or twisted ladder). Again, the sides of the ladder are the sugar-phosphate bonds and the rungs of the ladder are the nitrogen-containing bases.

RNA

RNA molecules are synthesized from a DNA template. The RNA molecule is very similar to DNA in that it has a sugar-phosphate backbone (except in this case the sugar is ribose), and each sugar is linked to a base. However, in RNA there is no thymine (T); instead RNA uses the related base uracil (U) in its place. Also, RNA exists in a single strand.

See the video below for a comparison of DNA and RNA:



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RNA is important for several functions that have to do with the linking of another class of biomolecules, *amino acids*, into a structure called a *protein*.

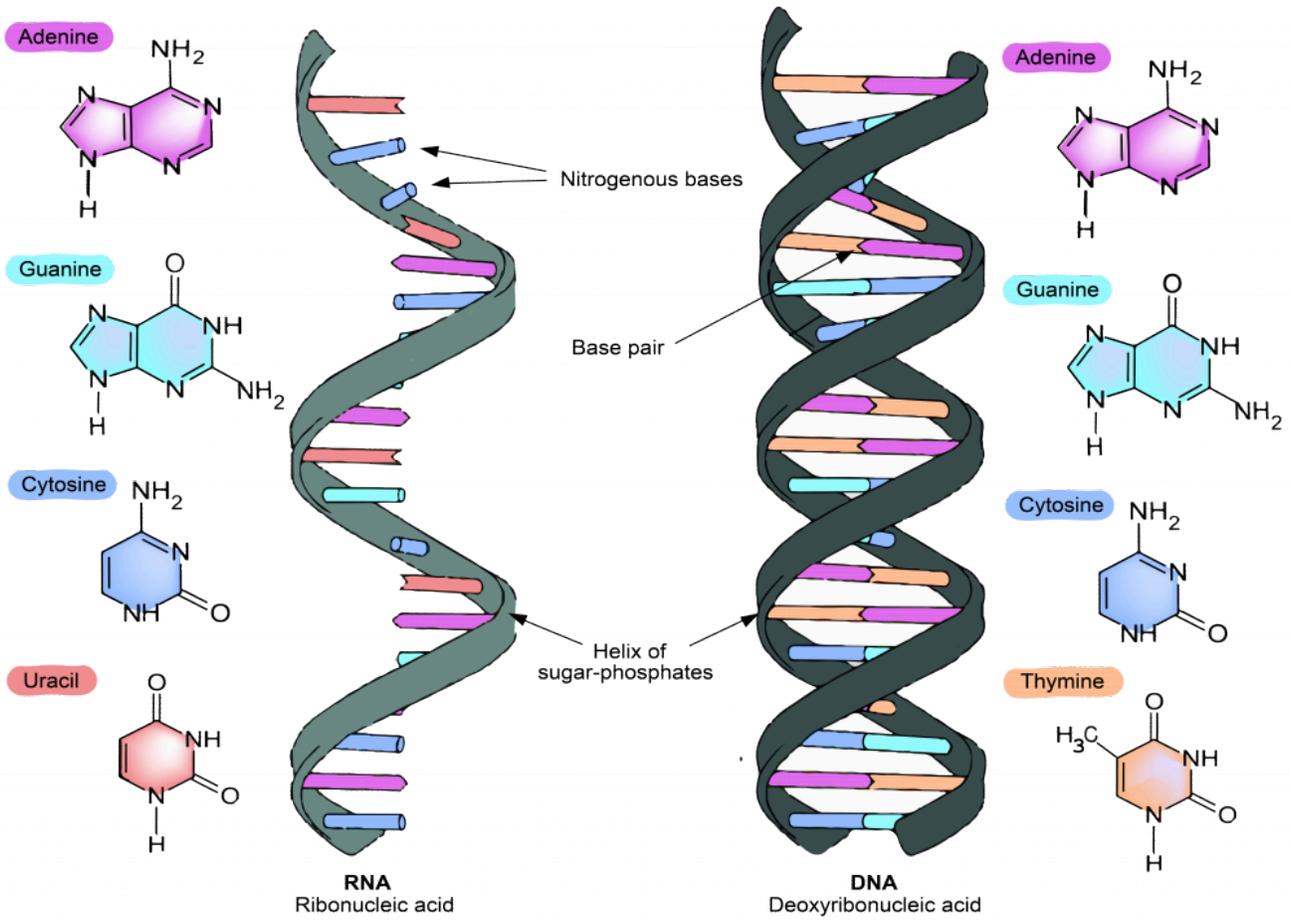


Figure 3.12 RNA versus DNA strands

3.5 Amino Acids

Amino acids are building blocks that join together to form proteins

Amino acid molecules have a central carbon-hydrogen molecule attached to three parts:

1. the carboxylic acid group, containing carbon and oxygen
2. the amino group, containing nitrogen and hydrogen
3. the side chain (the R group), which varies by amino acid and contains one or more of the following: hydrogen, carbon, oxygen, nitrogen, sulfur, phosphorus, sulfur, and selenium. The 21 common amino acids with their various side chains are depicted below.

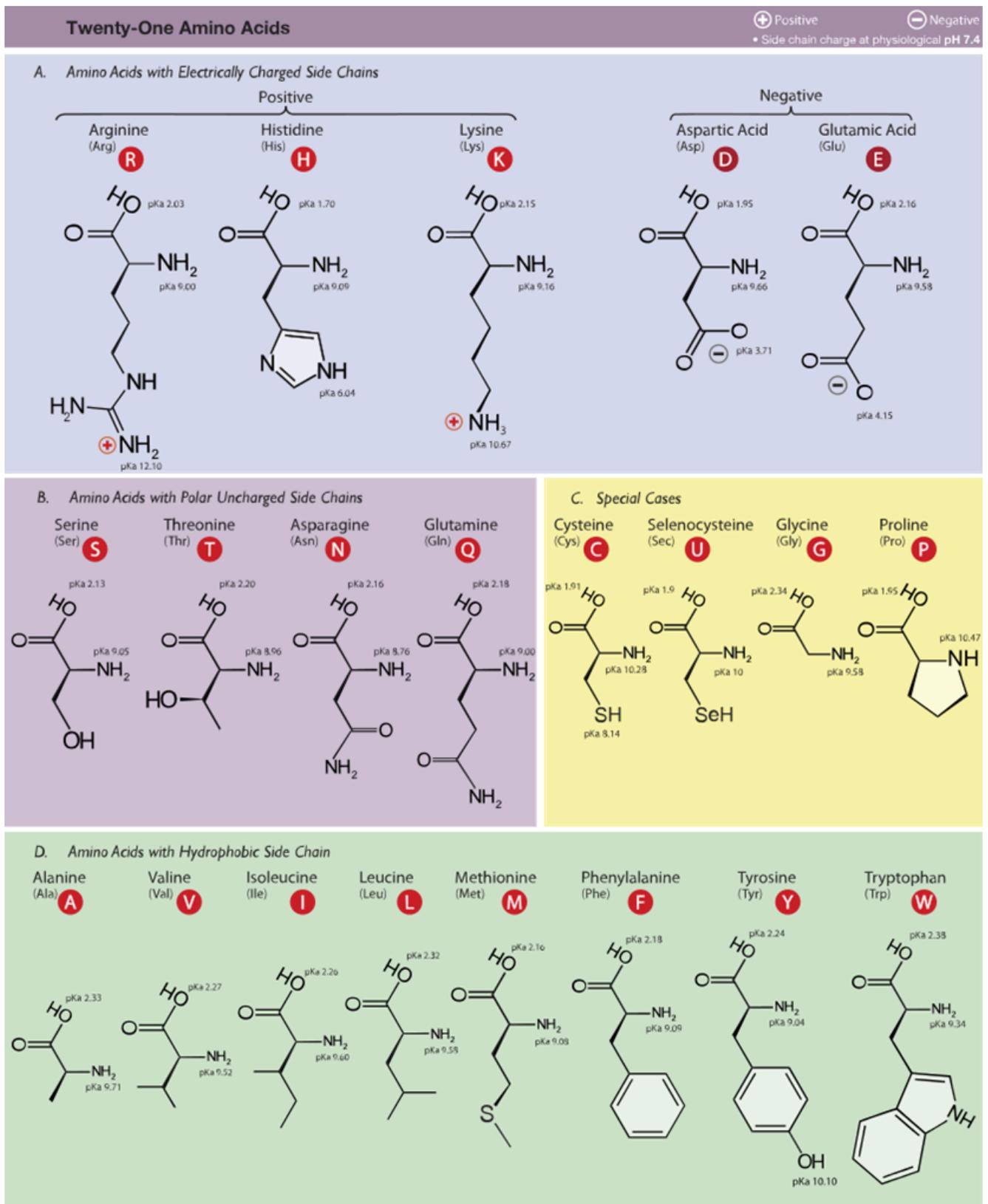


Figure 3.14 The 21 amino acids used by living organisms as building blocks for proteins.

The amino acids pictured above combine to form proteins when the carboxyl group from one amino acid and the amino group from another chemically react and form a bond. RNA helps guide the amino acid assembly to create a chain of amino acids called a **polypeptide** or a **protein**. This chain of amino acids folds into a functional protein based on the properties of the amino acid side chains. The resulting proteins can have a variety of functions in an organism based on each protein's shape. Watch this short video describing the function of various proteins:



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3.6 Fatty Acids

Like sugars, fatty acid molecules are composed of carbon, hydrogen and oxygen atoms. Fatty acid molecules have chains of carbon and hydrogen. These molecules are *lipophilic*, meaning that they will only mix with other fatty molecules and do not mix with water.

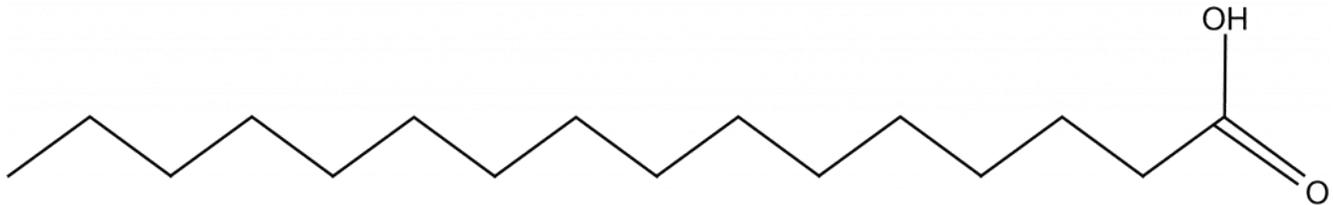


Figure 3.15 Palmitic acid, a fatty acid that has 16 carbon atoms, with associated hydrogen atoms, attached to oxygen atoms. Like the name suggests, this fatty acid is a major component of palm oil, but it also makes up large portions of human fat and breast milk.

Fatty acids can react with other small molecules to form triglycerides or phospholipids. Triglyceride and phospholipid molecules and other derivatives of fatty acids are used in organisms for energy storage. Another function of phospholipids is to form membranes that enclose cells and organelles within the cells.



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3.7 Cells

All living things are composed of one or more **cells** (viruses are an exception to this, but the jury is still out on whether or not they are actually alive...). Some organisms are one cell, while others are made up of dozens to quadrillions of cells (the estimated number of cells in a blue whale is greater than 100 quadrillion!)

So what actually defines a cell? A cell is a phospholipid-membrane-enclosed living structure that can replicate and uses biomolecules (often sugars) for energy.

There are two main categories of cells: prokaryotes and eukaryotes. **Prokaryotes** are visually simpler, in that they have cytoplasm (the inside stuff), are enclosed by a membrane, and do not have smaller enclosed structures (called **organelles**) inside.

Prokaryotes are generally single-celled organisms (there are a few exceptions that we will not discuss here). Two classifications of prokaryotes are Bacteria and Archaea. While these two types of prokaryotes look similar under a microscope, they are actually only very distantly related to one another (they branched off from a common ancestor over three billion years ago!).

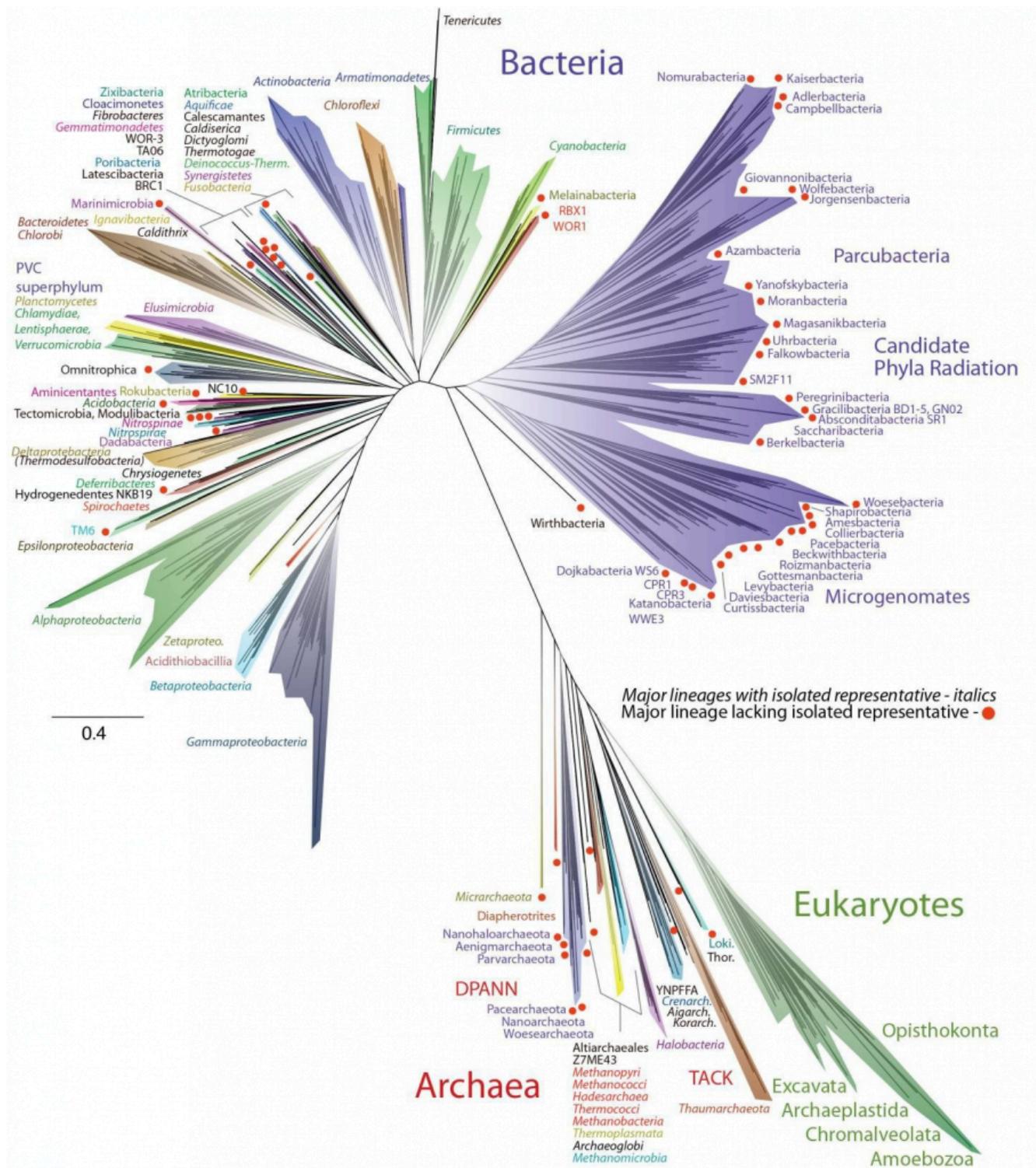


Figure 3.16 Scientists' most current understanding of the relatedness of organisms. Note that archaea and eukaryotes are closer together than archaea and bacteria. Figure from Hug et al. 2016.

Eukaryotes have a more complicated cell structure. Eukaryotes contain organelles within the cell including the nucleus, mitochondria, chloroplasts (in plants and algae) and others.

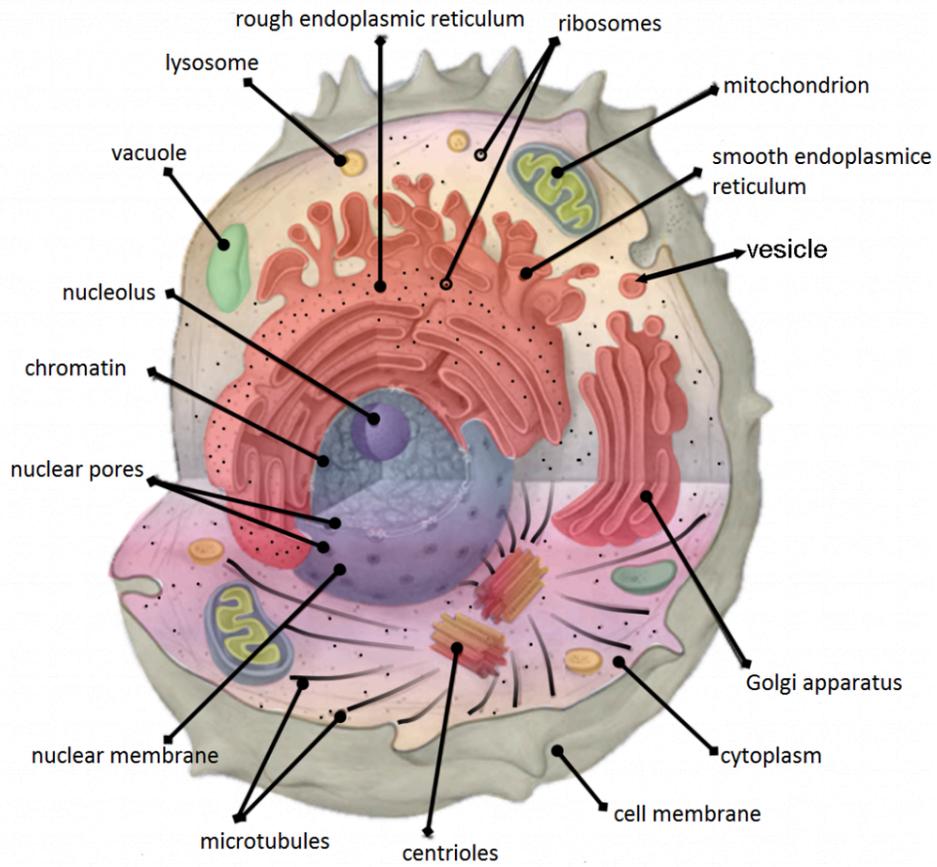


Figure 3.17 Schematic of an animal cell.

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Eukaryotes can be single-celled organisms or multicellular organisms (including all plants and animals).

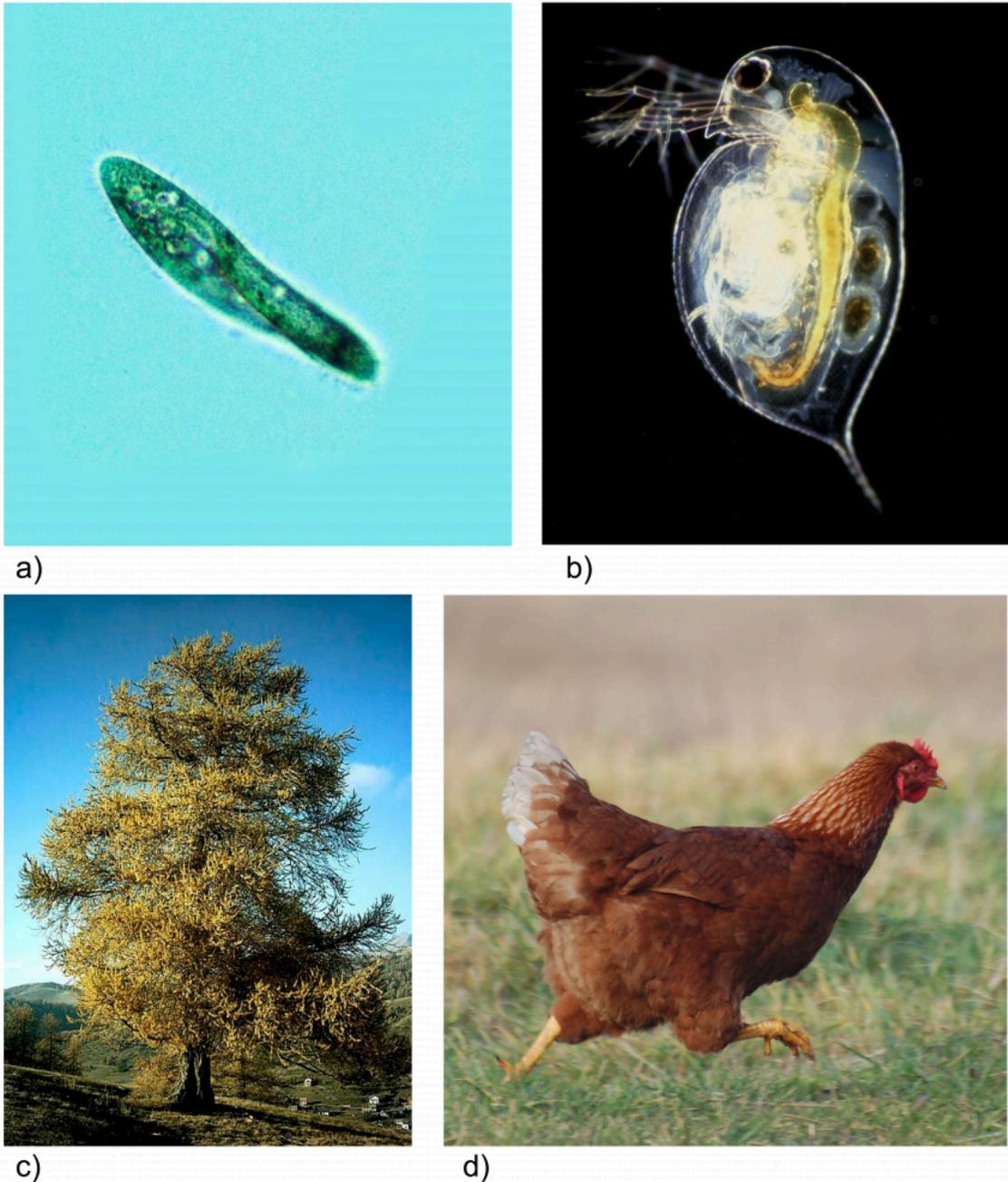


Figure 3.18 Examples of single-celled and multicellular organisms. **a)** a eukaryotic single-celled organism called a paramecium. **b)** a microscopic multicellular animal called a daphnid. **c)** a chicken (multicellular animal) and **d)** a larche (multicellular plant)

All sexually reproducing organisms are eukaryotes. For that reason, we will focus on eukaryotes. Inside eukaryotic cells is a membrane-bound organelle called a nucleus, which is surrounded by other membrane-bound organelles called mitochondria. See the video in section 3.8 for a description of all the organelles and their functions. This text will focus mainly on the **nucleus** and the **mitochondria**. The nucleus contains the vast majority of the cell's DNA. The mitochondria act as the cell's power-supply centers. Mitochondria take the energy from sugar and create high-energy molecules that the rest of the cell can use to do work and to replicate themselves.

3.8 Looking Closer at Organelles

Watch the following video about the functions of cellular organelles.



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3.9 Wrapping Up: Revisiting the Egg



Cracking the Egg

As discussed in the beginning of this chapter, the yolk of your breakfast egg is a single cell. Within the yolk is a structure called the germinal disk, which contains the nucleus and the cell's DNA. The yolk material (the yellow stuff) is part of the cytoplasm that contains lipids, proteins, and sugars that would be used for the developing chicken if the egg were fertilized (the eggs you get from the grocery store are likely not fertilized as the chickens were likely never near a rooster to fertilize them). The egg white is mostly protein and water, serving as a protective layer for the egg and for additional nutrients.

Your omelet is delicious and also has a lot of biology happening inside!

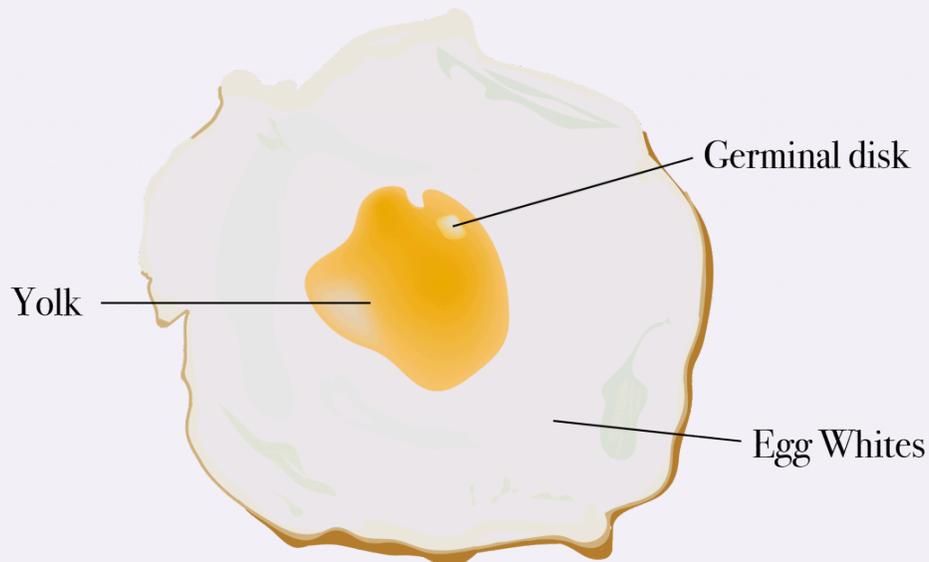


Figure 3.1 A breakdown of an egg

As we conclude this chapter and prepare for in-class discussion, be sure to return to the chapter's [goals and objectives](#).

Chapter 4: The Essentials of Genetics



[4.0 Introduction](#)

[4.1 Chapter objectives](#)

[4.2 An overview of basic genetics](#)

[4.3 Genotype to phenotype](#)

[4.4 Genes get around](#)

[4.5 Oral sex in cichlid fishes](#)

[4.6 See for yourself](#)

[4.7 Wrapping up: The science of paternity testing](#)

4.0 Introduction

Who's the daddy?



Figure 4.1 A local billboard sign on DNA paternity test.

How many men are actually raising kids that didn't come from the man's own sperm? Estimates vary wildly, from less than 1% to a scandalous 30%. Recent work with DNA testing puts the rate of "non-paternity" closer to 4% overall, with some studies citing less than 1% and others as much as 10%. Without their knowledge, these men may be investing time, money and affection in children they did not create. DNA paternity testing has become relatively common, and can be done with a simple and inexpensive kit purchased at many pharmacies.

DNA science has changed our lives dramatically. But what does "DNA science" mean? What is DNA? What does it do? And what role does DNA play in sex, evolution, and human behavior?

4.1 Chapter Objectives



Learning Objectives

This chapter provides a general overview of genetics, with the goal of introducing topics to be developed in future chapters. By the end of your reading and our in-class discussion, you should be able to:

1. Define the following terms:
 - **genetics**
 - **genes**
 - **nucleic acid**
 - **chromosome**
 - **protein**
 - **mutation**
 - **genotype**
 - **phenotype**
 - **codon**
 - **amino acid**
 - **allele**
2. Justify, with an example, this statement: genotype and the environment combine to make phenotype.
3. Explain the connection between gene, allele, chromosome, and DNA.
4. Describe, in general, what DNA does.
5. Explain the basic science of paternity testing.

4.2 An Overview of Basic Genetics

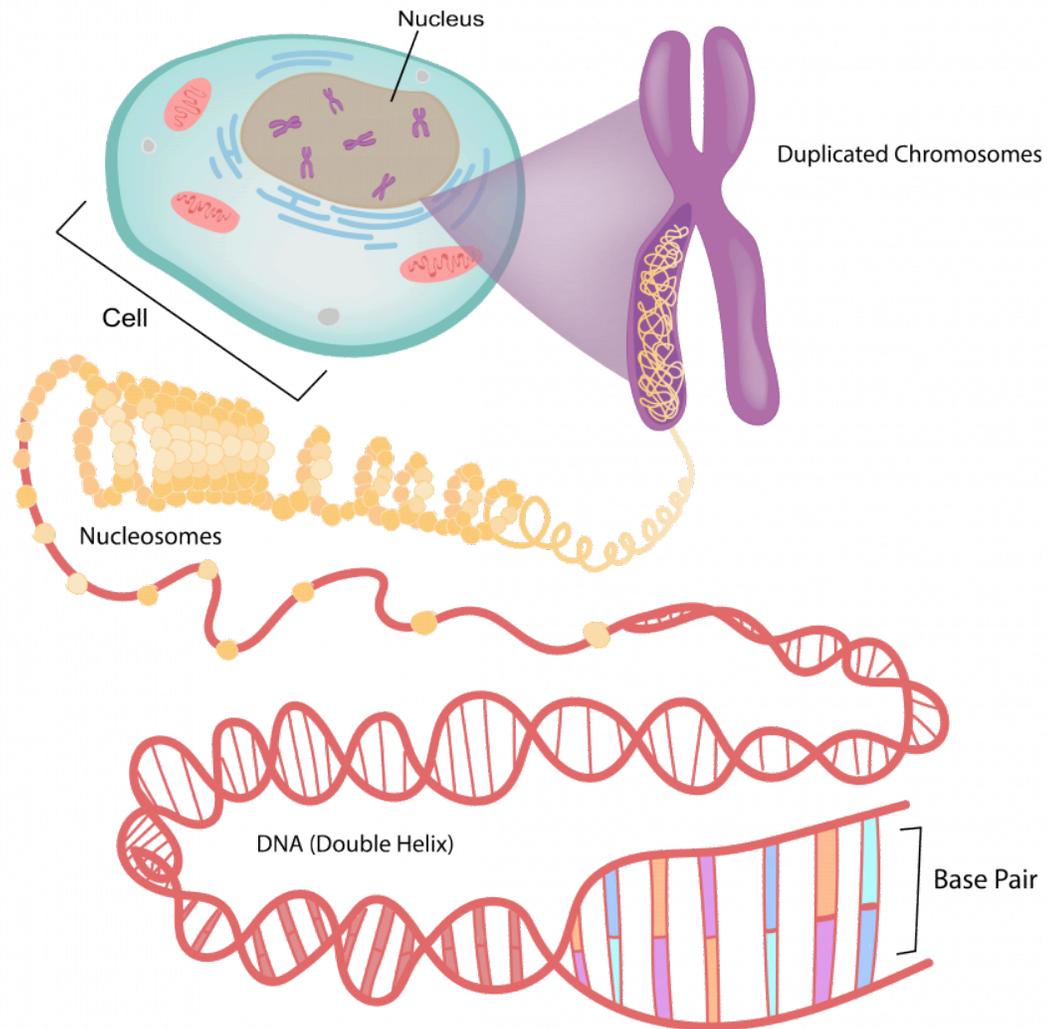


Figure 4.2 Deconstructed DNA

What unites us?

All living organisms share certain features. These include our cellular basis (all living things are made of cells), our use of ATP to do work in the cell, and the genetic code discussed in this unit. These features lend unequivocal support for a common ancestor for all living things. Moreover, the genetic code provides additional details about key events in evolution such as the origin of photosynthesis and the development of multicellularity.

What sets us apart?

Although we share many features, no two organisms are exactly alike. Even so-called identical twins are not the same in every way. And although humans use the same genetic code as do mushrooms and geraniums, we are actually quite different in many obvious respects. The science of **genetics** helps us understand not only what unites all living organisms, but also what sets individuals apart from each other. Specifically, genetics involves the study of **genes**—what they are, what they do, and how they are transmitted between generations. Simply, genes determine many characteristics of an organism. Genes are also units of heredity—they are passed to an individual through reproduction.

Chromosomes, genes, and DNA

Genes are the fundamental units of heredity, and they determine specific characteristics of an organism. Genes are made of **nucleic acids**, biological molecules that are found within structures called **chromosomes**. In many organisms, genes are located on chromosomes in the nucleus of the cell, in every cell of the individual's body (Figure 4.2).

The nucleic acids **deoxyribonucleic acid** (DNA) and **ribonucleic acid** (RNA) are large molecules found in the cells of all living organisms. DNA and RNA do their work through the synthesis of **proteins**, molecules that are specialized for various roles in the body—some proteins are involved in muscle contraction, others carry nutrients throughout the body, others fight infections, and so on. DNA and RNA also function in cell division and growth and conveying information across generations. Finally, DNA and RNA occasionally incur the very mistakes, or **mutations**, which make evolutionary change possible.

4.3 Genotype to Phenotype

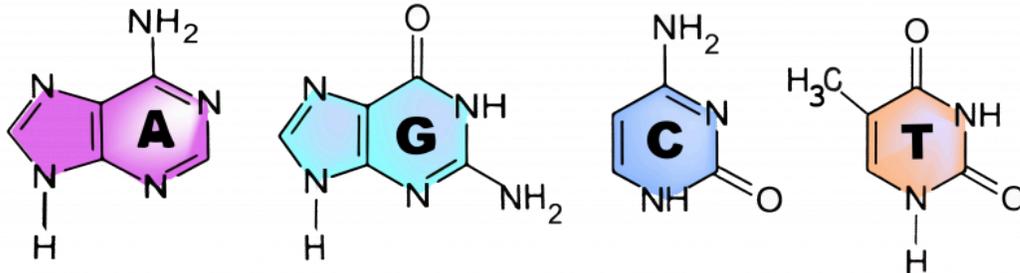


Figure 4.3 The four bases to a DNA strand

Simply, an individual's cells read genetic information in our DNA like words in a book, although DNA's "alphabet" is a mere four letters—Adenine (expressed as "A"), Guanine (G), Cytosine (C), and Thymine (T). These letters combine to form an individual's unique **genotype**, its total set of genes—but organisms are more than just a sequence of letters. In fact, identical twins, with almost the exact same genotype, may look and act very different from each other. An individual's **phenotype** is its observable characteristics, and the result of a lifetime of environmental influences acting on his or her genotype. For example, prolonged exposure to the sun makes some people tan, while others burn; how you react to the sun is a result of your genotype. Yet nobody is born tanned or sunburned. Rather, ultraviolet radiation from the sun activates your genes to produce pigments that will darken skin.



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4.4 Genes Get Around

Life cycles

Living organisms are characterized by growth and reproduction, and different types of organisms exhibit different **life cycles**. Most animals begin life as a single-celled zygote, which undergoes numerous cell divisions to become a fully formed individual. This individual develops into its reproductively mature adult form and produces **gametes**, sex cells such as sperm and eggs, which combine to form new zygotes. Plants and fungi exhibit versions of a two-part life cycle called **alternation of generations**. These life histories involve a multicellular stage that makes spores, and a multicellular stage that makes gametes.

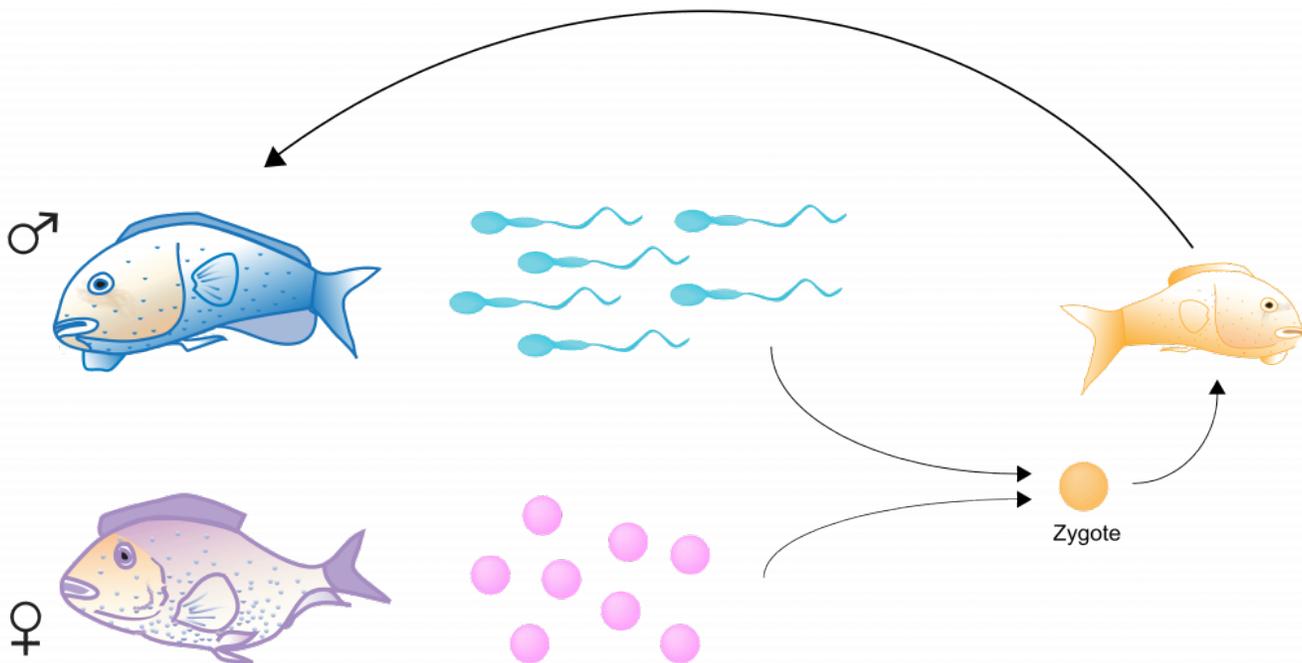


Figure 4.4 *The life cycle of a fish*

Heredity

A key part of genetics is **heredity**, the basic rules that govern how genes are transmitted from one generation to the next. Sexually reproducing individuals inherit a unique combination of genes from their parents when sperm and egg fuse, soon after sex. Some characteristics are inherited simply, following the rules of **Mendelian inheritance**. However, most characteristics of an individual's phenotype are due to a complicated, often unpredictable, blend of genetic and environmental influences. For example, an individual's libido (or sex drive) is presumably influenced by genetic factors, such as the number of testosterone receptors their cells produce, as well as life experiences, the availability of sexual partners, nutritional status, and (ask any parent) how much sleep he or she is getting.

Genes are expressed as proteins

What does DNA do?

The information encoded in DNA results from how our cells read DNA's four-letter alphabet of G, C, A and T. Each three-letter sequence of bases can be read as a **codon**, a molecular word that can be translated into an **amino acid** (or **peptide**); the primary structure of a protein is merely a string of amino acids (or a **polypeptide**) that have been encoded by a string of DNA bases. Think of a gene as a sequence of DNA bases that codes for a functional sequence of amino acids. In this way, DNA codes for proteins!

Evolution occurs when changes in DNA can be observed in populations

When DNA is passed from one generation to the next, what is being transmitted is really the genetic code for making proteins. In a group of individuals of the same species, most of their coding regions, or genes, will be the same. However, novel genetic changes, or mutations, occur as a small part of every individual's genome. These mutations may not seem like much when they arise, and most mutations make no difference to an individual's phenotype, but they are the ultimate source of genetic variation for all living organisms. Where there is variation there is the potential for some changes within a lineage. There is also the potential for some individuals to fare better or worse than others; in other words, this variation leads to the differential survival and reproduction we associate with evolution by natural selection.

An organism that inherits two nonfunctioning copies of a gene (one from each parent) may be at a disadvantage. She may not survive to reproductive age, or he may be less successful at reproduction. In each case, the individual's genes are not well represented in the next generation. For example, a mouse that inherits two nonfunctioning gene variants, or **alleles**, for a coat color gene will not produce fur pigment. This light mouse may experience increased risk of predation on a dark background, and, depending on its environment, could be less likely to survive to reproductive age (figure 4.3). In some environments, however, the light mouse may be at an advantage.



Figure 4.5 Light and dark mice on different backgrounds



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4.5 Oral sex in cichlid fishes



Biology is Sexy

Cichlid (pronounced SICK-lid) fish are a diverse group of freshwater fish that occur throughout the tropics. They are small and not particularly tasty, so some of their unusual features may be overlooked. For example, some cichlid fish are “mouthbrooders,” whereby the females spawn and then suck their eggs into their mouths. This parenting strategy protects the eggs while imposing obvious costs: how do you forage for food without eating your own children?

Some cichlid mothers handle the feeding problem by spitting out the eggs, foraging, and then sucking the eggs back into her mouth for easy transport. One adaptation associated with this behavior is the mother’s tendency to pursue, and attempt to put in her mouth, anything that looks like an egg.

Enter the male cichlids that are characterized by egg-like shapes on their anal fins. A mouth-brooding female may respond to these “eggs” as though they were her own, and attempt to suck them into her mouth. And swimming past a mouth-brooding female, the male with the egg-shaped mimics puts himself in an excellent position to ejaculate into the female’s mouth.

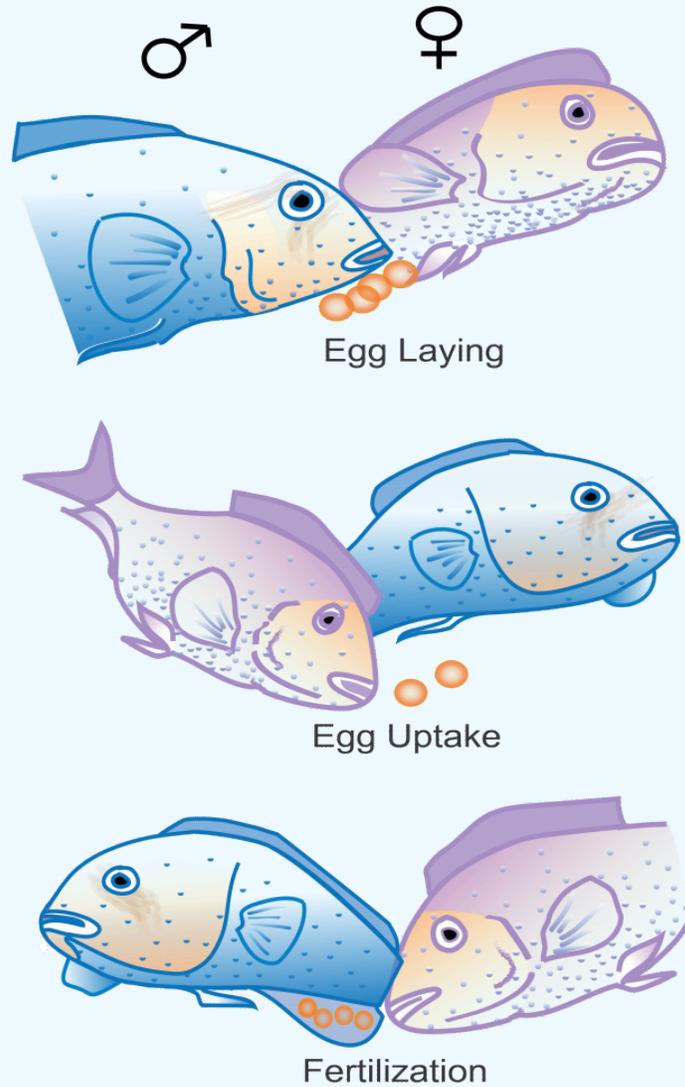


Figure 4.6 The fertilization cycle



Consider the following questions

Scientists have recently described the genetic basis of anal-fin egg mimicry in cichlid fishes. A single gene (*csf1ra*) that codes for a yellow pigment is active in fish with egg mimics, and appears to have evolved relatively recently.

- Did the female's egg-sucking behavior exert selection pressure on this gene?
- Can you think of an evolutionary benefit to the male's physiology (egg mimic) and behavior (ejaculation)?
- Can you think of any other organisms that have decorative devices that may have evolved to facilitate fertilization?



Check Yourself

Describe, using either of the examples discussed above (cichlid fish, mouse), the general function of DNA.



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4.6 See for Yourself

What can genes tell us about us about egg-laying mammals and the evolution of lactation?

Perhaps one of the most intriguing animals on earth is the platypus, a duck-billed mammal that lays eggs (figure 4). There are also a few *marsupial* mammals (e.g. kangaroos, opossums), organisms that complete gestation in specialized pouches. But most mammals gestate their young with the help of an organ called the **placenta**. Placental mammals include the primates (monkeys and apes), the carnivores (e.g., dogs and cats), the bats, and the cetaceans (e.g., dolphins and whales). The existence of three types of mammals raises several questions, including: which type of mammal evolved first—egg-laying (or *monotreme*), marsupial, or placental? Why don't we all lay eggs? And finally, how can genes help us understand mammalian evolution?



Figure 4.7 a) Platypus, a monotreme



b) Opossum, a marsupial



c) Tiger, a placental mammal

The answers may lie in **pseudogenes**. A pseudogene is a gene that is no longer functioning. Sometimes these genes are called *vestigial genes*, because—like the snake's limbs or the human

appendix—pseudogenes are vestiges of an organism's

evolutionary past. Like other vestigial traits, pseudogenes can help us understand evolutionary history. In general, pseudogenes arise when a gene mutates, resulting in reduced or eliminated expression, without destroying the whole organism. These residues of once-functional genes are presumably the result of relaxed selection for a given protein. For example, genes for the egg yolk protein, *vitellogenin*, exist as remnants in the mammals. Of the three vitellogenin (VIT) genes found in mammals, all three are nonfunctional in the marsupial and placental mammals, but one is still active in the egg-laying mammal, the platypus.

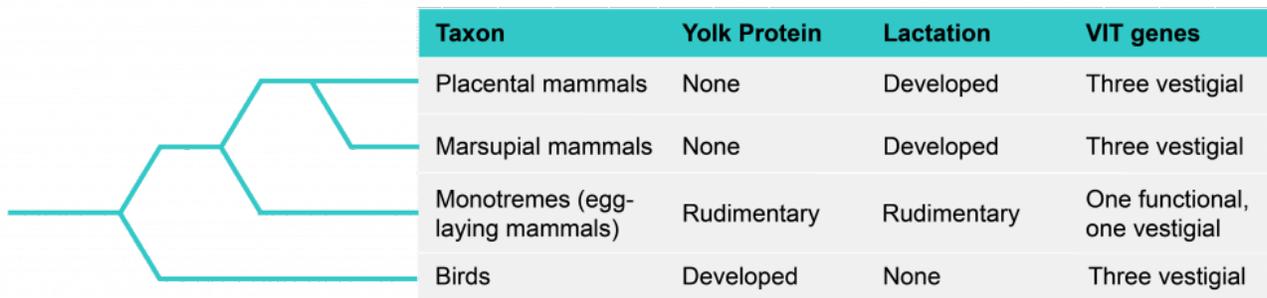


Figure 4.8 Phylogeny comparison

See for yourself: the figure above illustrates a phylogeny of the three types of mammals, using birds as an outgroup for comparison. For each taxon, or group of organisms, you can see the status of its yolk protein,

lactation, and vitellogenin genes. For example, birds lay eggs with extensive egg yolk protein; birds do not lactate, and they have three functioning egg yolk protein genes.

But how were placental and marsupial mammals able to give up production of yolk protein? Why didn't natural selection eliminate mammals with yolk-less eggs?

While mammals are known for lactation, their sister taxa—the reptiles and birds—do not produce milk. Could mammalian lactation have relaxed selection on the genes for yolk protein? Scientists studying the egg yolk genes also identified the genes coding for the milk protein *casein* in all mammalian groups, and hypothesized that the ability to produce milk may have reduced selection pressure on the egg yolk proteins. In other words, when young could get their protein from milk, egg protein was less essential; as the VIT genes mutated to inactive variants, selection did not eliminate individuals possessing these mutant alleles. According to this logic, the egg-laying platypus represents a developmental and evolutionary compromise: the platypus produces some milk and some yolk protein, but excels at neither. Perhaps it's no wonder we aren't overrun with egg-laying mammals.

As with all legitimate science, these findings raise a host of new questions. For example, why don't birds and reptiles, who also have genes for milk protein, have nipples and breastfeed their young?

4.7 Wrapping Up: The Science of Paternity Testing



The Science of Paternity Testing

Humans as a species are not especially diverse, genetically at least. Compared to our nearest relatives, the chimpanzees, we are quite homogeneous—that is, most of our alleles are the same within a population, and each human differs from other humans by, on average, 0.1% of their DNA. This may not seem like much, but if we have 3 billion bases in our genomes, then we expect that any two humans will differ by approximately 3 *million* bases. This population-level variation can make DNA testing fairly reliable. Paternity testing focuses on genes that are known to vary in the population. For each allele a child shares with the alleged father, the *probability of paternity* increases.

A DNA test is performed by collecting DNA, typically by using a cotton swab to gather cells from the inside of an individual's mouth. Samples are sent to a laboratory, where DNA is isolated from the rest of the cell tissue and amplified using a process called the **Polymerase Chain Reaction**. The polymerase chain reaction refers to a series of steps in which DNA is heated and cooled repeatedly. Heat-stable enzymes (isolated from a hot-springs-dwelling bacterium) catalyze the rapid production of billions of copies of DNA. A child's amplified DNA can then be compared to that of the mother and the alleged father. Several genes are analyzed in order to generate a Combined Paternity Index (figure 3). Many laboratories test fifteen or more genes, and boast probability of paternity confidence levels of 99.99%. If the child does not possess *any* of the male's alleles, the man will be excluded from paternity.

Confirming paternity can be beneficial for families. Men who are assured of paternity spend more time with their children, save more money for education, and are less likely to abandon the child's mother. Paternity testing can also be critical in legal cases that involve child support. But is paternity testing always a good thing? Could some test results have psychological costs for children and parents? In other words, do we pay a price to have such easy access to DNA technology?

CONCLUSIONS OF DNA PARENTAGE TEST: DNA PATERNITY INCLUSION

The alleged father is not excluded as the biological father of the child. Based on the genetic testing results obtained by PCR analysis of STR loci, the probability of paternity is >99.99% as compared to an untested, random man (prior probability = 0.5). Note: Results from privately collected (not witnessed) cases are for personal knowledge only and cannot be used as legal evidence of parentage or identity.

DNA Locus	Child		Alleged Father		Paternity Index
D8S1179	13	15	13	14	1.10
D21S11	30	31	31	-	6.84
D7S820	10	11	9	11	0.91
CSF1PO	10	12	11	12	0.80
D3S1358	16	18	16	18	3.65
TH01	9.3	10	6	10	14.70
D13S317	11	12	12	11	1.68
D16S539	11	-	11	14	1.76
D2S1338	17	23	16	17	1.52
D19S433	13	15	13	14	1.00
vWA	17	18	16	17	1.05
TPOX	8	9	9	-	3.33
D18S51	15	-	13	15	4.27
D5S818	12	-	11	12	1.52
FGA	23	-	23	24	2.94

COMBINED PATERNITY INDEX: 88127

PROBABILITY OF PATERNITY: >99.99%

Figure 4.9a) Interpreting paternity-test results where probability is greater than 99.99%

CONCLUSIONS OF DNA PARENTAGE TEST: DNA PATERNITY INCLUSION

Based on the genetic testing results obtained by PCR analysis of STR loci, the alleged father is excluded as the biological father of the child. The probability of paternity is 0%. Note: Results from privately collected (not witnessed) cases are for personal knowledge only and cannot be used as legal evidence of parentage or identity.

DNA Locus	Child		Alleged Father		Paternity Index
D8S1179	12	15	14	16	0
D21S11	30	31	32	33	0
D7S820	11	12	8	10	0
CSF1PO	10	-	11	12	0
D3S1358	15	16	15	17	0.79
TH01	7	9	7	8	0.62
D13S317	12	13	11	12	0.58
D16S539	9	12	9	-	2.24
D2S1338	17	21	16	18	0
D19S433	12	15	12	13	2.31
vWA	16	19	17	19	3.90
TPOX	9	11	12	13	0
D18S51	17	22	18	19	0
D5S818	11	12	7	11	1.09
FGA	21	22	19	26	0

COMBINED PATERNITY INDEX: 0

PROBABILITY OF PATERNITY: 0%

Figure 4.9b) Interpreting paternity-test results where probability is 0%



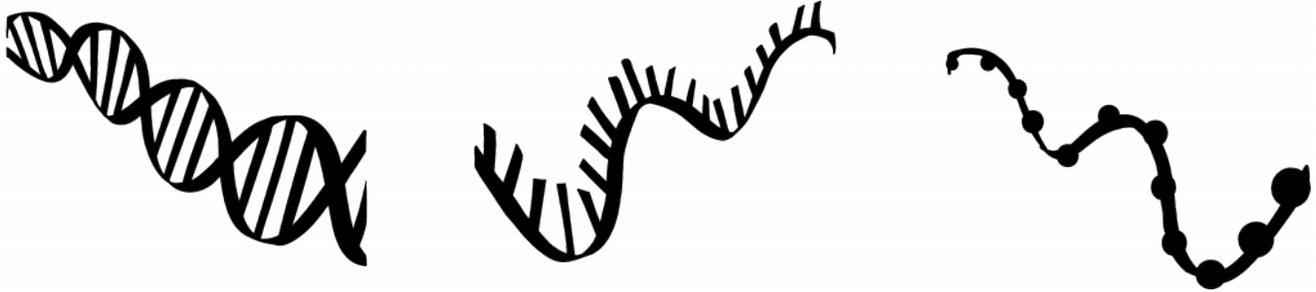
Consider the following questions

You have been told that individual X is the father of child Y, according to the results of a DNA test.

- What does that mean? How was this determined?
- If you are told that this relationship is certain, with 99.99% confidence in paternity, does this information add anything to your understanding? Explain.

As we conclude this chapter and prepare for in-class discussion, be sure to return to the chapter's [goals and objectives](#).

Chapter 5: Express Yourself! From DNA to Protein



[5.0 Introduction](#)

[5.1 Chapter objectives](#)

[5.2 The genetic basis of gene expression](#)

[5.3 Protein synthesis requires RNA](#)

[5.4 RNA is transcribed from a DNA template](#)

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5.0 Introduction

A Gene for Monogamy?

If you could test a potential mate to determine whether he or she would cheat on you, would you do it? Is such a test even possible? And are some individuals really more likely to cheat?

Monogamy is defined by sexual exclusivity— monogamous individuals have a single sexual partner for a given amount of time. Monogamy has several documented benefits, such as fewer sexually transmitted diseases, greater investment in childcare, and for humans, financial and emotional stability. But whatever the benefits of monogamy may be, it is a fact that many organisms that form pair bonds find ways to have sex outside of the bond—that is, many supposedly monogamous individuals cheat on their mates. Humans are no exception, and given the costs incurred by cheating, it makes sense to evaluate how faithful a potential partner will be.

Therefore, it is no surprise that reports of a “monogamy gene” are popular and intriguing. But does such a thing exist? How could a gene affect fidelity? And can we actually test a mate’s tendency to stray?

5.1 Chapter Objectives



Learning Objectives

Our goal for this chapter is for you to gain an understanding of the molecular basics of gene expression—the structure of DNA and RNA, along with how genes direct the synthesis of proteins, and how genetic mistakes (mutations) can affect these outcomes. By the end of your reading and our in-class discussion, you will be able to:

1. Define the following terms:

- **Nucleotide**
- **Purines**
- **Pyrimidines**
- **Nitrogenous bases**
- **RNA**
- **mRNA**
- **tRNA**
- **rRNA**
- **Transcription**
- **RNA polymerase**
- **Translation**
- **Polypeptide**
- **Mutation**
- **Substitution**
- **Deletion**
- **Frameshift**
- **Insertion**
- **Nucleic acid**
- **Chromosome**
- **Ribosome**
- **Heredity**
- **Amino acid**
- **Peptide**
- **Protein**
- **Genetics**

- **Zygote**
- **Codon**
- **Allele**

2. Describe the general structure of DNA.
3. Identify three types of RNA that are involved in gene expression.
4. Explain, using the genetic code, how DNA codes for proteins.
5. Describe a few of the many functions of proteins.
6. Derive the amino acid sequence of a polypeptide, based on DNA or RNA nucleotides.
7. Describe how different types of point mutations (substitution, deletion, insertion) can affect gene expression.
8. Evaluate whether there is such a thing as “gene for monogamy.”

5.2 The Genetic Basis of Gene Expression

A **gene** is the basic unit of inheritance. Genes consist of the nucleic acid DNA, large molecules packaged in dense cellular structures called chromosomes. Simply, **DNA (deoxyribonucleic acid)** consists of **nucleotides** that are themselves relatively simple: each nucleotide includes a sugar (**deoxyribose**), a phosphate ion, and a nitrogen-rich (or nitrogenous) “base”—the double-ringed **purines** guanine and adenine, and the single-ringed **pyrimidines** cytosine or thymine. Nucleotides are joined in a linear sequence that represents one of DNA’s strands. Genetic information is stored in the actual sequence of these nucleotides, and these sequences involve quite a lot of information. For example, the human genome, or full set of DNA, contains about 3 billion nucleotide bases, forming about 25,000 genes.



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DNA structure is fairly simple

One molecule of DNA involves two strands, joined at the bases, anchored by a “backbone” of repeating sugar-phosphate units, and wound in a helical conformation. Thus, DNA is often referred to as a “double helix.” Bases join together according to complementarity. Specifically, adenine (A) bonds with thymine (T), and cytosine (C) bonds with guanine (G). They combine through chemical interactions called **hydrogen bonds**, relatively weak associations that can be broken with heat or one of several enzymes.

In humans, two meters (~nine feet) of DNA are packed into each one of trillions of individual cells. Chromosomes are how cells have solved the problem of packing so much information (long strands of DNA) into a small, tightly compacted structure. DNA-binding proteins help to package these long strands of DNA into the chromosomes.

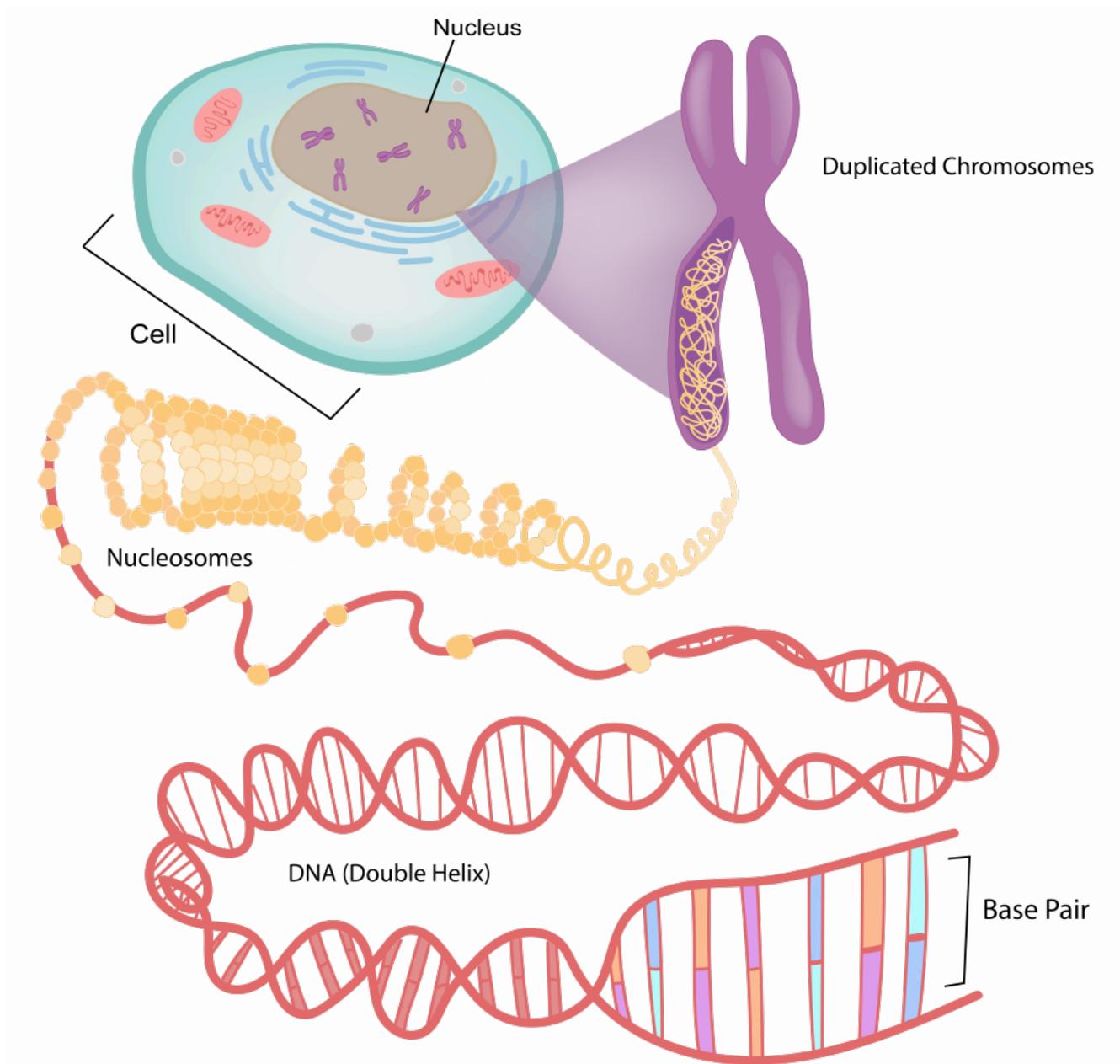


Figure 5.1 DNA in Eukaryote cell. Visualizing cells, chromosomes, and DNA. Chromosomes, found in the cell's nucleus, are a packaging solution for the massive amounts of DNA that make up your genes. Chromosomes are comprised of double-stranded DNA molecules, which themselves can be subdivided into genes, regions of DNA with a specific coding function.

DNA consists of paired bases of single-ringed (pyrimidine) and double-ringed (purine) **nitrogenous**, or nitrogen-rich, **bases**. The bases are visualized as letters at right. Their structures are represented below.

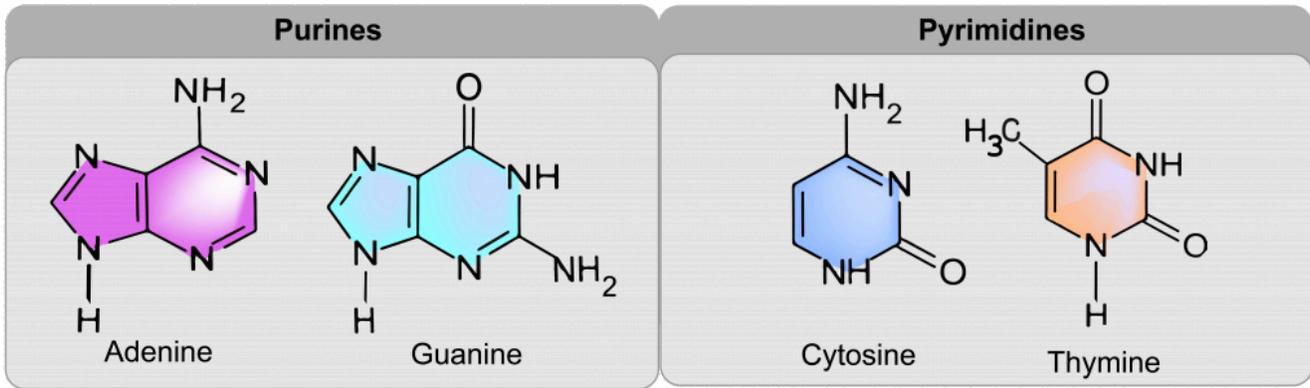


Figure 5.2 Purines versus Pyrimidines

5.3 Protein Synthesis Requires RNA

DNA is not the only nucleic acid involved in expressing the message in your genes. **Ribonucleic acid (RNA)** is another large molecule that is involved in protein synthesis. RNA is similar to DNA in that it also consists of a series of nucleotides anchored by a backbone of repeating sugars and phosphates.

RNA differs from DNA in that it is typically single-stranded, rather than double-stranded. Also, the **ribose** sugar in RNA's sugar-phosphate backbone is different from the deoxyribose in DNA. And although three of the four nitrogenous bases of RNA are the same as DNA (adenine (A), guanine (G), cytosine (C)), RNA has the base uracil (U) instead of thymine (T).

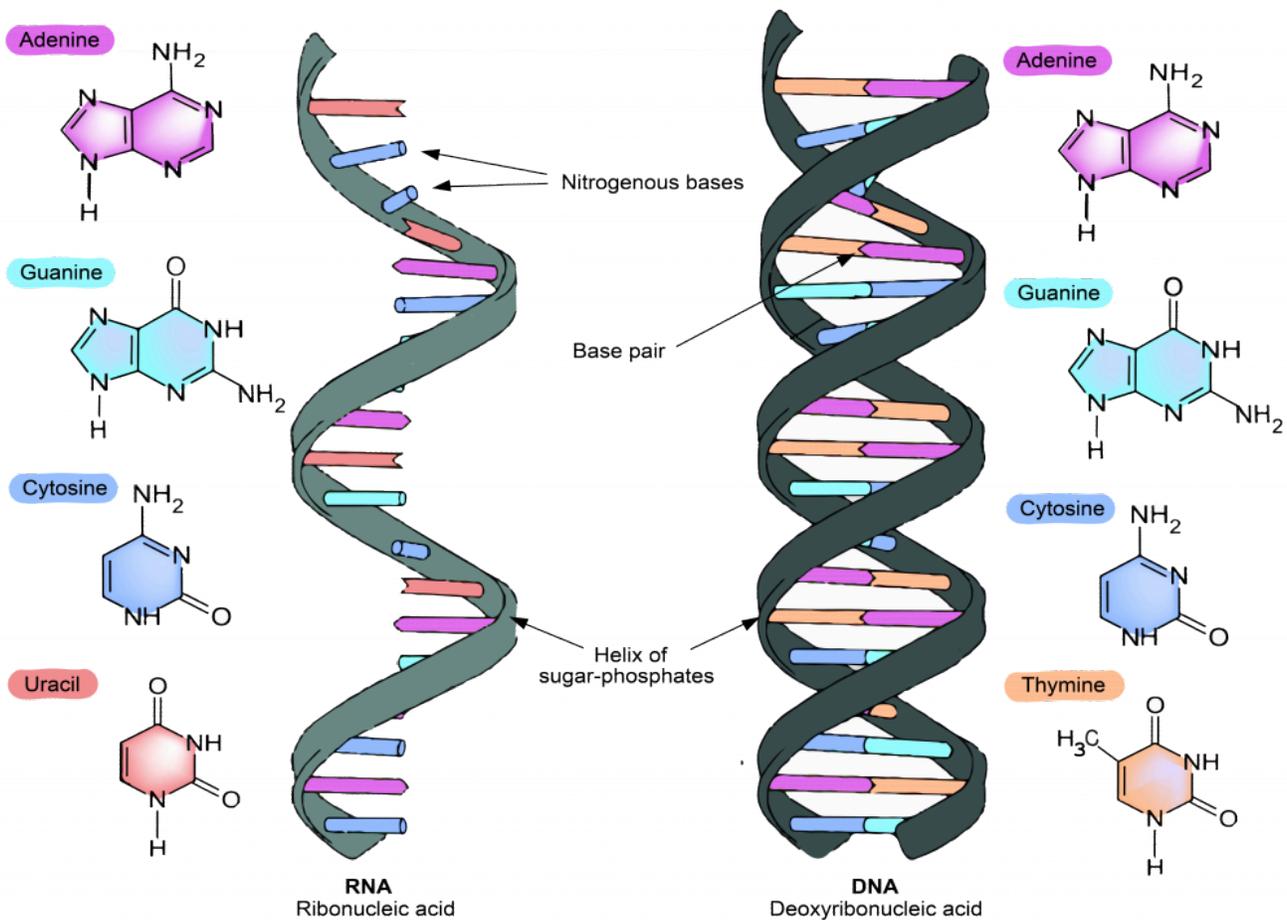


Figure 5.3 A comparison of DNA and RNA.



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There are three types of RNA involved in protein synthesis

RNA is more diverse than DNA, occurring in several different functional types, three of which are essential to making proteins:

1. **mRNA**, or messenger RNA, conveys DNA's genetic message in the form of an RNA transcript. An RNA transcript is a sequence of RNA bases complementary to the DNA bases in a gene that is being expressed through protein synthesis.
2. **tRNA**, or transfer RNA, aligns the mRNA transcript with amino acids, the fundamental building blocks of a protein. Each amino acid has a corresponding tRNA molecule, itself complementary to bases in mRNA.
3. **rRNA**, or ribosomal RNA, comprises much of the ribosome, a two-unit cellular structure that is the site of protein synthesis. Typically, human cells each contain several million ribosomes.



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List the three types of RNA that are involved in making proteins:

5.4 RNA is Transcribed from a DNA Template

RNA molecules originate from a DNA template, through the process of **transcription**. That is, a single strand of RNA is transcribed from one of the strands of a DNA helix. Namely, the DNA molecule unwinds at the site of the gene to be transcribed. **RNA polymerase** catalyzes the formation of an RNA molecule (or **transcript**) based on complementarity with DNA: where there is a guanine (G) in DNA, a complementary cytosine (C) is added to the RNA strand. However, where there is an adenine (A) in the DNA template, a uracil (U) is added to the RNA transcript. For example, the DNA bases TGCACA is transcribed to the RNA bases ACGUGU.

The transcribed RNA then either functions as mRNA, tRNA, or rRNA, with the roles described above. Either way, in eukaryotes such as plants, animals, and fungi, the RNA molecules are constructed in the nucleus of the cell. After transcription, RNA leaves the nucleus to continue protein synthesis in the cytoplasm of the cell.

In prokaryotic organisms such as bacteria, the mRNA transcript is immediately ready for its role in polypeptide formation. But in eukaryotes, extensive processing is required. RNA processing involves editing some nucleotides, removing non-coding regions of DNA, and preparing the transcript for recognition by the ribosome.

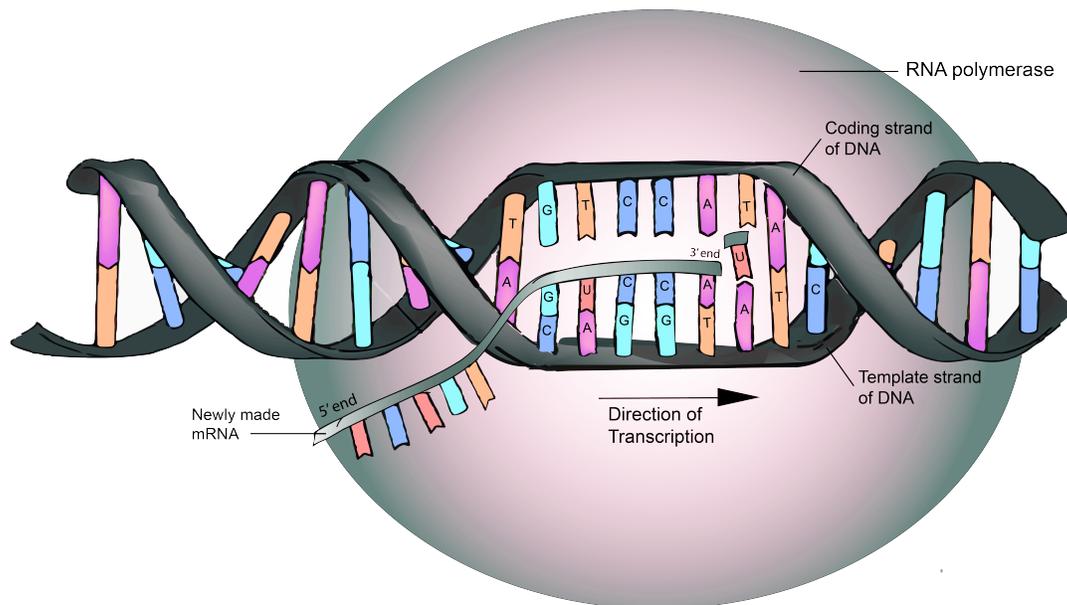


Figure 5.4 A single strand of RNA is transcribed from a DNA template.



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5.5 RNA is Translated into a Polypeptide

After mRNA is processed, the final transcript has a rendezvous with ribosomes and transfer RNA (tRNA). Once the mRNA attaches to the ribosome, **translation** begins. During translation, the transcribed mRNA is matched with specific tRNA molecules, which are each attached to a corresponding amino acid. These amino acids are bound to each other by peptide bonds forming a polypeptide. In this way, a linear sequence of RNA bases is translated into a linear sequence of amino acids.

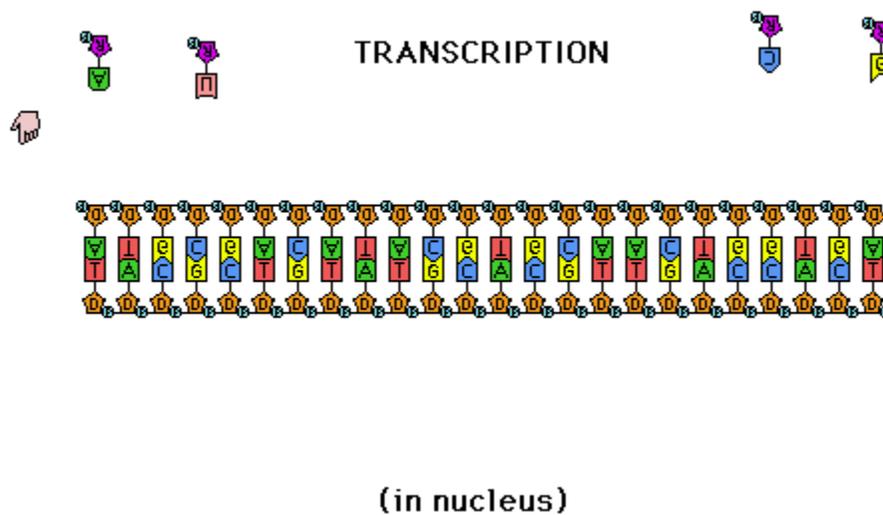


Figure 5.5 A simple animation of the process of transcription and translation in eukaryotes.



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What are amino acids?

Amino acids, the building blocks of proteins, are relatively small molecules that consist of an -NH_2 group, or amine, a carboxylic acid, or -COOH group, and a side chain (“R” in figure 6) that is specific to each amino acid. For example, the amino acid glycine is the simplest of the amino acids, with a single hydrogen (H) as its functional side chain. Although at least 500 amino acids have been identified, only 20 are found in the proteins produced by most organisms. When amino acids bind together through peptide bonds, the carboxylic acid group of one amino acid attaches to the amino group of another amino acid, creating a linear sequence of amino acids that is called a **polypeptide**.

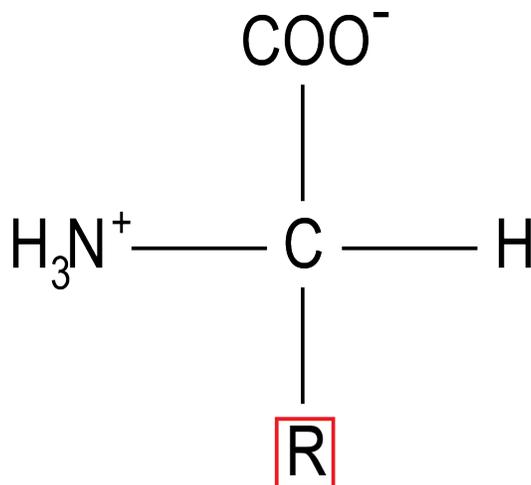


Figure 5.6 Generalized structure of an amino acid. Each amino acid has a different functional group, R.

5.6 What are proteins?

Proteins consist of one or more strings of amino acids called polypeptides. The actual sequence of amino acids in the polypeptide is the protein's primary structure. The hormone protein oxytocin is a small protein, consisting of only nine amino acids in a single polypeptide. Collagen is much larger, typically containing about 1,000 amino acids.

The way in which amino acids attract and repel each other gives proteins a characteristic shape. Some proteins consist of two or more polypeptides combined. And it is this final configuration that really determines how a protein functions. Simply, protein shape is the key to protein function.

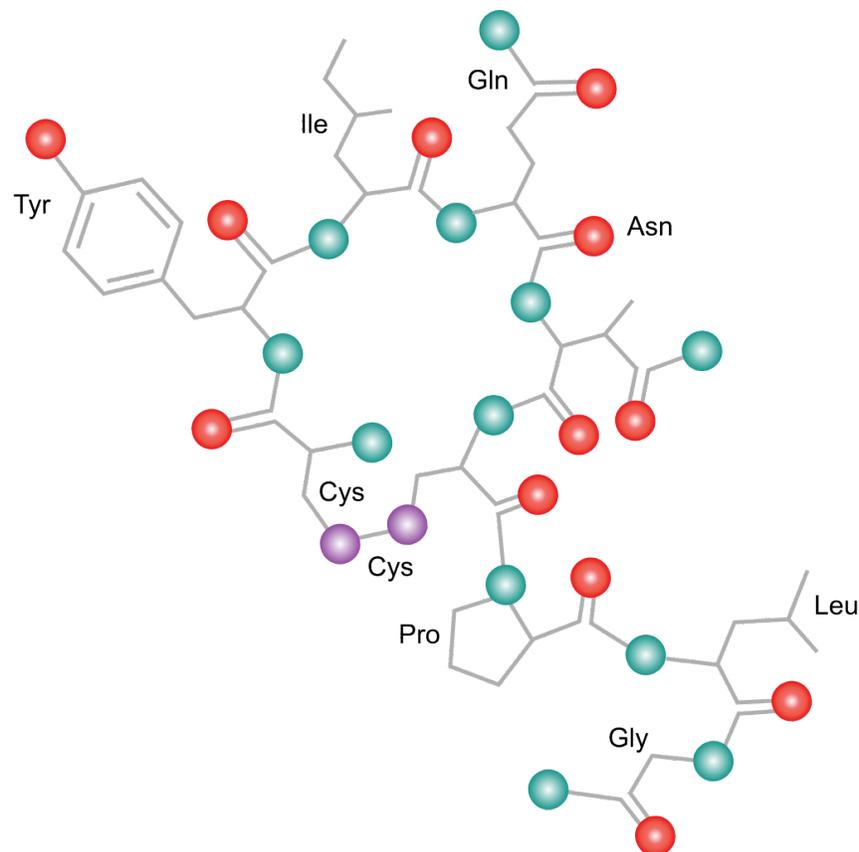


Figure 5.7 The primary structure of oxytocin is only nine amino acids. Each amino acid is represented above by a three-letter code; for example, Gly stands for glycine, Leu for leucine, and so forth. We'll discuss the many amazing functions of oxytocin later.

5.7 Proteins Take on Many Roles

Saying that DNA codes for proteins may seem a bit anti-climactic. After all, DNA is made out to be such a big deal in the news, television crime shows, paternity tests and= evolutionary stories. Surely it does more than just code for proteins!

DNA's importance is clear if you appreciate the diversity of protein types. Proteins can be structural components of a cell or act as cellular machines in the body. For a few examples:

Structural proteins include keratin, which strengthens hair, beaks, and feathers, and collagen, which is the main component of connective tissue and the most abundant protein in mammals.

Hormone proteins serve as messengers in the body. For example, oxytocin signals muscles of the uterus to contract during childbirth. Oxytocin is also involved in releasing milk for nursing infants, and has recently been studied for its roles in pair bonding, orgasm, and maternal behavior.

Transport proteins, such as hemoglobin, move molecules around in the body. Hemoglobin in your red blood cells transports oxygen throughout your bloodstream.

Enzyme proteins facilitate, or catalyze, chemical reactions in your body. For example, lactase is an enzyme that breaks down milk sugars. Another enzyme, aromatase, plays a key role in the synthesis of estrogens (sex hormones that are not proteins).

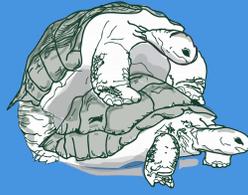
Receptor proteins bind to molecules that stimulate cellular activity. Receptor proteins occur in the cell's cytoplasm, the cell membrane, or the nuclear membrane. An example of a receptor protein is the sex-hormone binding globulin, which binds testosterone and estrogens in the body, rendering them biologically unavailable, or inactive. An increase in sex-hormone binding globulin is associated with lower levels of active sex hormones.

Pigment proteins absorb and reflect wavelengths of light selectively. The result is perceived as color. Melanin is a pigment protein responsible for human skin, eye, and hair color.

Contractile proteins such as actin and myosin orchestrate your muscle movement.

Antibody proteins are produced by the cells of your immune system to attack and destroy foreign invaders such as viruses and harmful bacteria.

Again, structure is key to protein function. Stringy, rope-like collagen is an excellent support protein, while the specific conformation of sex-hormone binding protein allows it to bind sex hormones in the body.



Biology is Sexy

Sex Proteins Keep Fruit Flies Energized

When it comes to proteins and sneaky sex schemes, male fruit flies deserve special recognition. In general, male fruit flies and virgin female flies like to sleep, enjoying long afternoon siestas that conserve energy and reduce exposure to predators and the sun. But after sex, females forego the siesta and increase their time foraging. This practice presumably allows the females to meet the energetic demands of producing eggs and supporting developing young. And it appears to be prompted by a “sex peptide,” a protein that males produce and release with sperm in the ejaculate. So, fruit fly males have a gene that is expressed as a protein that doesn’t affect the male directly—rather, the protein encourages his sex partner to stay awake, eat more, and provide for his young. Do other organisms produce proteins that target their sexual partners? Can you think of any examples? How might the genes for these proteins have evolved?



Figure 5.8 Fruit flies mating

5.8 Using the genetic code

DNA is transcribed into RNA, which is translated into a polypeptide. Simply, a series consisting of four bases (A,G,C,T) is transcribed and translated into a series of 20 amino acids. This shift from one chemical language to another is accomplished through the genetic code, a set of precise rules that govern how every possible sequence of three RNA nucleotides (a codon) corresponds to a specific amino acid.

The genetic code can be read as a table, whereby each three-letter codon is broken into its first, second and third RNA bases. These bases converge on a single amino acid, and this amino acid is the actual translated product of the original DNA nucleotides. For example, figure 6 tells us that the amino acid tyrosine (Tyr) corresponds to the mRNA codon UAC. The genetic code also contains an initiation (or “start”) codon, AUG, and three “stop” codons: UAA, UAG, UGA. The initiation codon also codes for the amino acid Methionine. Methionine (with Tryptophan) is one of only two amino acids that correspond to a single codon. Most amino acids correspond to several three- base `sequences. In other words, the genetic code can be very redundant.

		Second Letter					
		U	C	A	G		
First Letter	U	UUU } Phe	UCU } Ser	UAU } Tyr	UGU } Cys	U C A G	
		UUC } Phe	UCC } Ser	UAC } Tyr	UGC } Cys		
		UUA } Leu	UCA } Ser	UAA } Stop	UGA } Stop		
		UUG } Leu	UCG } Ser	UAG } Stop	UCG } Trp		
	C	CUU } Leu	CCU } Pro	CAU } His	CGU } Arg	U C A G	
		CUC } Leu	CCC } Pro	CAC } His	CGC } Arg		
		CUA } Leu	CCA } Pro	CAA } Gln	CGA } Arg		
		CUG } Leu	CCG } Pro	CAG } Gln	CGG } Arg		
	A	AUU } Ile	ACU } Thr	AAU } Asn	AGU } Ser	U C A G	
		AUC } Ile	ACC } Thr	AAC } Asn	AGC } Ser		
		AUA } Ile	ACA } Thr	AAA } Lys	AGA } Arg		
		AUG } Met L-Start	ACG } Thr	AAG } Lys	AGG } Arg		
	G	GUU } Val	GCU } Ala	GAU } Asp	GGU } Gly	U C A G	
		GUC } Val	GCC } Ala	GAC } Asp	GGC } Gly		
		GUA } Val	GCA } Ala	GAA } Glu	GGA } Gly		
		GUG } Val	GCG } Ala	GAG } Glu	GGG } Gly		

Figure 5.9 The genetic code consists of all the RNA codons and their associated amino acids.



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What else does DNA do?

Only 2% of the human genome codes for proteins, leading some scientists to refer to the remaining nucleotides as “junk DNA.” However, this moniker may be too harsh. Rather, it seems that much of your DNA is regulatory, and controls how much, or what kind, of a protein is synthesized. Some DNA is part of a gene that was active in our ancestors, but is no longer functional; for example, humans have several vestigial olfactory genes, suggesting that modern humans are not as dependent on our sense of smell as were our hominid ancestors. And some DNA was adopted from viruses that attacked human ancestors; these endogenous retroviruses say a lot about our evolutionary past with pathogens, but are not functioning genes.

It is also probable that many DNA sequences have functions yet to be identified. In 2012, a consortium of scientists reported on a decade-long project, The Encyclopedia of DNA Elements (ENCODE), suggesting that over 80% of DNA is somehow functional. ENCODE’s results have been applauded and criticized, a span of responses that highlights how DNA science is still in its infancy. Regardless of the exact percentages, it is clear that while much of our DNA does not directly code for protein, the human genome is far from being packed with junk.



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5.9 Many Genes are Highly Conserved

DNA expresses itself with astounding fidelity. For example, the protein cytochrome *c* (*cyt c*) and its corresponding gene are highly conserved, meaning that *cyt c* has changed little over evolutionary time. At ~100 amino acids long, *cyt c* is involved in aerobic respiration and is therefore part of every organism that depends on oxygen. Pigs, cows and sheep have identical *cyt c* molecules—amino acid for amino acid. Similarly, the cytochrome *c* of chickens and turkeys is identical, and ducks only differ from chickens and turkeys by one amino acid. In fact, researchers have inserted *cyt c* genes from fish, horses, humans and birds into the genomes of single-celled yeasts, and the yeasts have successfully produced *cyt c* protein.

Similarly, *hemoglobin*, the protein that conducts oxygen in our red blood cells, has changed little throughout evolution. Hemoglobin is also essential to most organisms that depend on oxygen, so presumably most changes (or mutations) to the hemoglobin gene were selected against. These conserved genes and protein products also provide insight into evolutionary relationships...a type of molecular evidence for evolution.

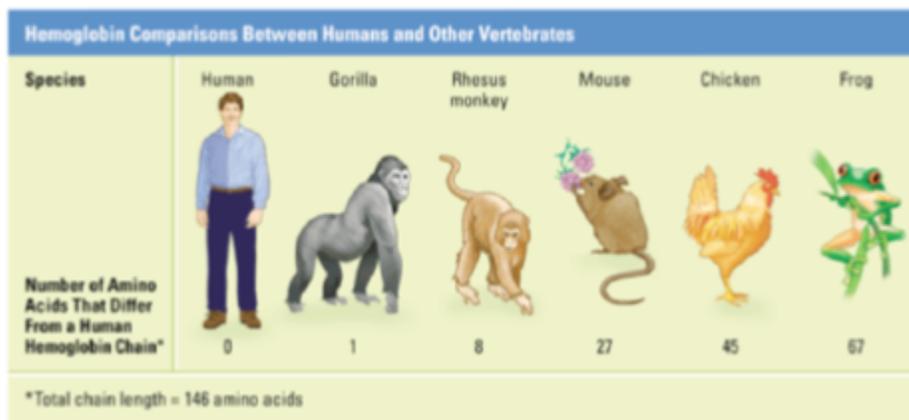


Figure 5.10 Hemoglobin evolution in several vertebrate animals. Note that humans differ from gorillas by only one amino acid in their total hemoglobin polypeptide. Thus, hemoglobin has been highly conserved throughout evolution.

5.10 Point Mutations Affect Gene Expression

Despite its fidelity, if DNA never incurred mistakes we would not have an evolutionary story to tell, and our world would not be characterized by such tremendous biodiversity. **Mutations** are mistakes that arise in DNA, either through random errors in DNA replication or through any one of a number of mutagenic agents—UV radiation, toxic compounds, etc. Mutations can affect whole chromosomes, large sections of chromosomes, or just a few nucleotides.

A type of mutation is the **point mutation**—a random change to one or a few DNA bases. These changes range from the “silent” mutation that has no effect, to mutations that alter amino acids without changing the fundamental nature of the protein, to mutations that render that gene nonfunctional, and may have detrimental effects at the organismal level.

Point mutations involve base substitutions, deletions, or insertions

A **substitution mutation** simply involves the replacement of one nucleotide for another. Substitutions may not change the amino acid sequence of a polypeptide. For example, a mutation in DNA that changes GAA to GAG will change the codon CUU to CUC; either way, the translated amino acid is leucine and the polypeptide is unaltered. Even some amino acid changes can have little effect on the expressed protein. However, simple substitutions can have profound consequences. For example, a simple substitution of a single base in the gene for hemoglobin results in the allele for sickle-cell anemia, a potentially fatal blood disorder.

A **deletion mutation** is caused by the removal of one or more nucleotides. A deletion of three bases will typically only affect one or two amino acids, but a deletion of one or two bases disrupts all the codons “downstream” of the mutation and affects the entire remaining reading frame of the gene (a **frameshift mutation**).

Many examples of the genetic disease cystic fibrosis are due to deletion mutations. Also, a deletion of 32 bases in a gene coding for a T-cell receptor protein (*CCR5*) alters the ability of HIV viruses to penetrate host cells. Individuals with this mutation (*CCR5-del32*) have decreased susceptibility to HIV infection, conferring some protection against AIDS.

Insertion mutations result from the addition of one or more nucleotides. Like deletions, insertions can affect the entire reading frame of the gene, or they can simply add one or more amino acids to the translated polypeptide. However, a number of three-nucleotide (or “trinucleotide”) repeat diseases exist and include Huntingtons disease and fragile X syndrome. In fragile X syndrome, several CAG triplets are inserted into the X chromosome. Individuals who inherit this disorder (especially males) may have profound mental retardation.

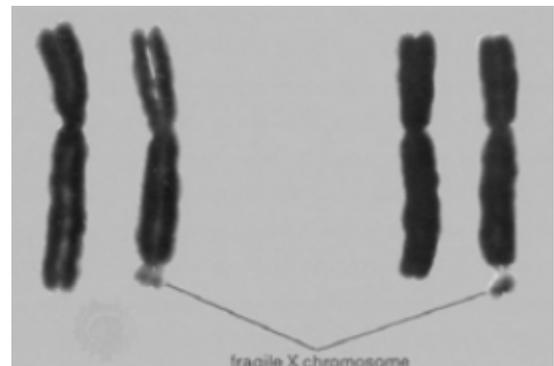


Figure 5.11 Two pairs of X chromosomes. In each pair, the chromosome on the left is unaffected, the one on the right has the mutation characteristic of fragile X syndrome. Fragile X is an example of a chromosomal mutation.

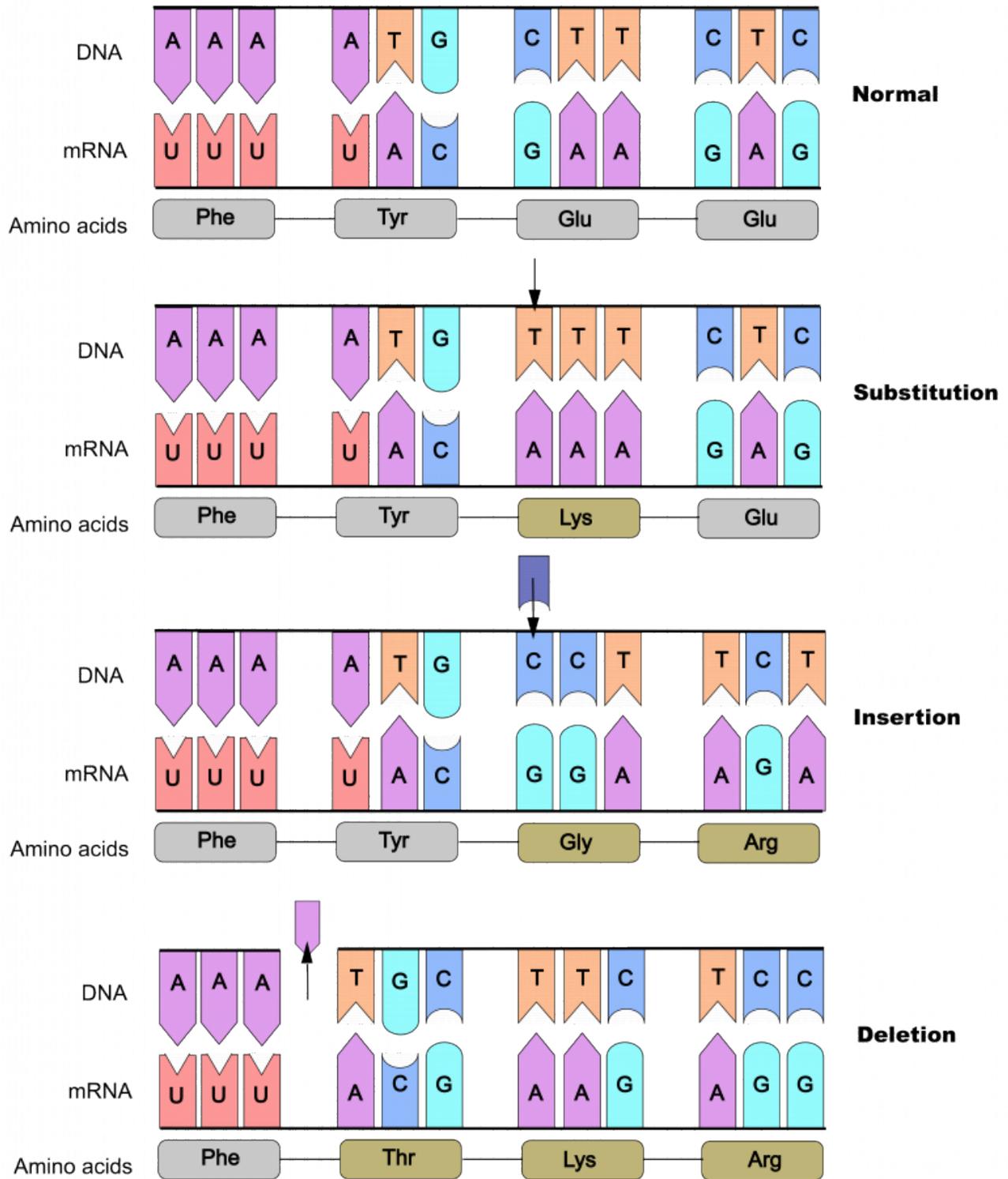


Figure 5.12 A summary of the types of point mutations discussed above.

5.11 Gene Regulation

How much and when a protein is translated (or how much the gene is expressed) is regulated by sequences of DNA called “gene regulatory regions”. These non-coding portions of the DNA interact with proteins and increase or decrease the amount a protein is expressed. Differences in gene expression are why an individual’s skin cells are different than nerve cells (even though both cell types contain the same DNA).

Additionally, as illustrated by monogamous and non-monogamous voles, small differences in sequences in the regulatory regions of genes in different organisms can create very different phenotypes, even when the DNA sequences for the coding portion of the gene are identical!



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Points to Ponder

Which of the mutations in the previous question cause the greatest change in the resulting polypeptide? Be sure you can explain your answer.

5.12 Wrapping Up: The Mystery of Monogamy



Vasopressin binding may affect partner fidelity in humans

In 2008, the media were abuzz with reports such as “Monogamy gene found in people.” Given the benefits of monogamy, and our social fascination with cheating mates, identifying a gene for monogamy would certainly be newsworthy. But how could a gene lead to monogamy?

Consider the prairie vole and the mountain vole, two species of small rodents found throughout North America. Unlike most mammals, prairie voles exhibit monogamous behavior, forming lifelong pair bonds, caring for each other and sharing pup-raising duties. Mountain voles do not form pair bonds. The males exhibit no parental care and the females abandon their pups after a short lactation period. The two vole species are over 99% genetically identical, so the key to their different mating systems must reside in the remaining 1%.

Early work suggested that a protein called vasopressin could be at the center of these behavioral differences. Vasopressin is a neuropeptide—a small polypeptide that acts as both a hormone and neurotransmitter, transmitting signals in the nervous system. Vasopressin is similar to oxytocin. Both vasopressin and oxytocin are known to play a role in regulating social behaviors, and brain images reveal that the two voles differ in the amount of vasopressin and oxytocin receptors.

Work on male voles indicated that the actual coding regions for vasopressin receptors are nearly identical in both species, but there is considerable difference in the regulatory regions flanking their genes. Scientists have speculated that these regulatory regions may lead to greater production of vasopressin receptors in prairie voles. An increase in vasopressin receptors leads to an increase in the activation of reward centers in the brain, leading to a sense of well-being that becomes associated with a mate and one’s offspring. Thus, an increase in vasopressin receptors increases the likelihood of forming and maintaining pair bonds.



Figure 10. The monogamous prairie vole (above) has spawned products such as the book at right.

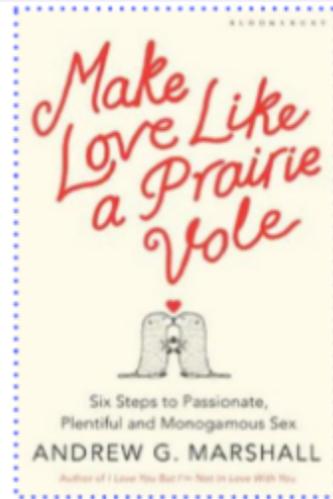


Figure 5.13 Monogamous prairie voles

But what about another mammal, human beings? Do humans have similar genetic mechanisms affecting pair-bond formation? This is where the 2008 study, mentioned above, comes into play. Hasse Walum and colleagues interviewed 552 people about the quality of their relationships and analyzed their vasopressin receptor genes. They found that, like the voles, men with a specific genetic variant in the regulatory region of the vasopressin receptor gene were less likely to be married and, if they were married, more likely to report marital problems than were their counterparts lacking this variant.

The authors of this study concluded their paper by noting that the presence of these associations “clearly does not mean that this polymorphism may serve as a predictor of human pair-bonding behavior on the individual level.” That is, these genetic differences aren’t everything: many men with the gene variant are happily married, and many without the variant report relationship problems. Also, numerous environmental influences affect fidelity. Some human cultures place a higher premium of monogamy, with subtle or overt penalties for those who cheat on a spouse. Couples who have sex regularly are more likely to stay together than those who have sex only occasionally (the amount of sex a pair has is itself influenced by age, economics, and general health). In sum, maintaining monogamy is complicated, and fidelity (like so many natural phenomena) is the result of a complicated mix of biological and/or environmental influences.

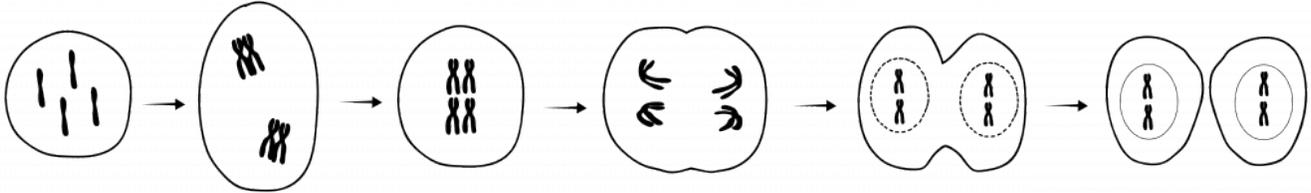


Points to Ponder

Looking at these data, what do you think? Can these data be used to support our hypothesis, or not? In other words, do these data meet our prediction?

As we conclude this chapter and prepare for in-class discussion, be sure to return to the chapter's [goals and objectives](#).

Chapter 6: Life Cycles, Cell Division, and Inheritance



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[6.4 Meiotic division results in sex cells](#)

[6.5 Meiosis I](#)

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6.0 Introduction

Dolly the Sheep

In 1996 Dolly the sheep became the first successfully cloned mammal. There was no addition of DNA from a father, no genetic “mixing,” just an identical copy of DNA from a mother. This accomplishment means many things, including the fact that we currently have the technology to mass-produce hundreds of copies of what seem to be the best and healthiest livestock.

But the scientific community seems to have little interest in setting up giant cloning labs. Instead, sex is viewed as desirable over asexual reproduction, or cloning. But why risk making a potentially inferior child when an ideal parent is available? With current genetic technology, humans can rewrite the genetic code of sheep. We have the capabilities to impart disease resistance, increase meat production and soon, we may be able to eliminate genetic disease. Why don't we just make a “super sheep” and copy it thousands of times? What does sex do for individuals, particularly to their genetic code, that's too important to replace with a really cool cloning lab?



Figure 6.1 Dolly the Sheep

6.1 Chapter Objectives



Learning Objectives

In this chapter, we'll discuss, broadly, two types of cell division—mitosis and meiosis. Specifically, we'll emphasize meiosis, and how meiosis is responsible for generating much of the diversity that we associate with sexual reproduction. Our primary goal is for you to understand *how* meiosis generates genetic diversity, and how the cells that result—sperm and eggs—combine to create unique individuals in the next generation.

By the end of your reading and our in-class discussions, you should be able to:

1. Define the following terms
 - **Mitosis**
 - **Meiosis**
 - **Gamete**
 - **Zygote**
 - **Fertilization**
 - **Haploid**
 - **Diploid**
 - **Homologous pair**
 - **Chromatid pair**
 - **Tetrad**
 - **Crossing over**
 - **Recombination**
 - **Independent assortment**
 - **Prophase, metaphase, anaphase, telophase, cytokinesis**
 - **Meiosis I and Meiosis II**
 - **Outcrossing**
 - **Genotype**
 - **Phenotype**
 - **Recessive**

- **Dominant**
 - **Mutation**
 - **Homozygous**
 - **Heterozygous**
 - **Punnett square**
 - **Sex-linked inheritance**
 - **Monoallelic inheritance**
2. Compare the processes of mitosis and meiosis
 3. Describe the progeny cells from both mitosis and meiosis, especially relative to the original cell
 4. Describe how meiosis generates genetic diversity via independent assortment and crossing over
 5. Use Punnett squares to predict inheritance probabilities for single-gene traits, including sex-linked traits
 6. Differentiate between the inheritance patterns of recessive and dominant alleles

6.2 Sex generates genetic diversity

As we discuss in [chapter 7](#), genetic diversity is the ultimate evolutionary defense. The more diversity in one's offspring, the more likely some of these offspring can survive challenges such as plague or famine. But how do we get that diversity? Do we just acquire mutations in our DNA? It's rare for a mutation to be helpful, and mutation is not the major way in which diversity is created in sexually reproducing organisms.

Instead, measurable genetic diversity is achieved in a safer way—a way of rearranging genes, which have already proven to be helpful, into innovative combinations. This is basically what sex is, and where sex comes into the evolutionary equation. Here we get a 'tried and tested' collection of genes from two parents who were, at a minimum, biologically successful enough to make it to adulthood, acquire a mate, and have a child. Sex creates novel combinations of DNA while still maintaining many of the genes that were helpful for an individual's parents. All the fascinating quirks of sexual biology that you read about in other chapters are really just strategies to help facilitate a single act: Fertilization.

Fertilization is the fusion of two sex cells (one sperm and one egg) into one cell, one that has the genetic potential to become a viable (or "functioning") individual. In this chapter we will explore how this process works and how the creation of these cells determines genetic identity. In other chapters we'll discuss the connection between copulation (or sex as an act) and fertilization, plus what happens after fertilization—gestation and birth.



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6.3 Mitosis is how most of our cells divide

Recall our earlier discussion of cell structure and the location of chromosomes ([Chapter 3.7](#)). Over 99% of the human cells in your body copy themselves using a process called **mitosis**. In mitosis the cell makes a near-identical copy of itself and splits in two. The two new cells have the same DNA as the original, or *parent*, cell and are nearly identical copies of each other. When your body creates new skin after a scrape or when a child grows taller, this is all due to *mitotic* cell division.

Prior to mitotic cell division, all the DNA in the nucleus makes a full copy of itself. During this process of **DNA replication**, the two strands of DNA in the double helix separate and each strand is used as a template to create a new strand. The two resulting DNA molecules each have one parent strand and one newly synthesized strand. This *semi-conservative* process of DNA replication is highly accurate, making less than one mistake (or **mutation**) every 200 million base pairs.

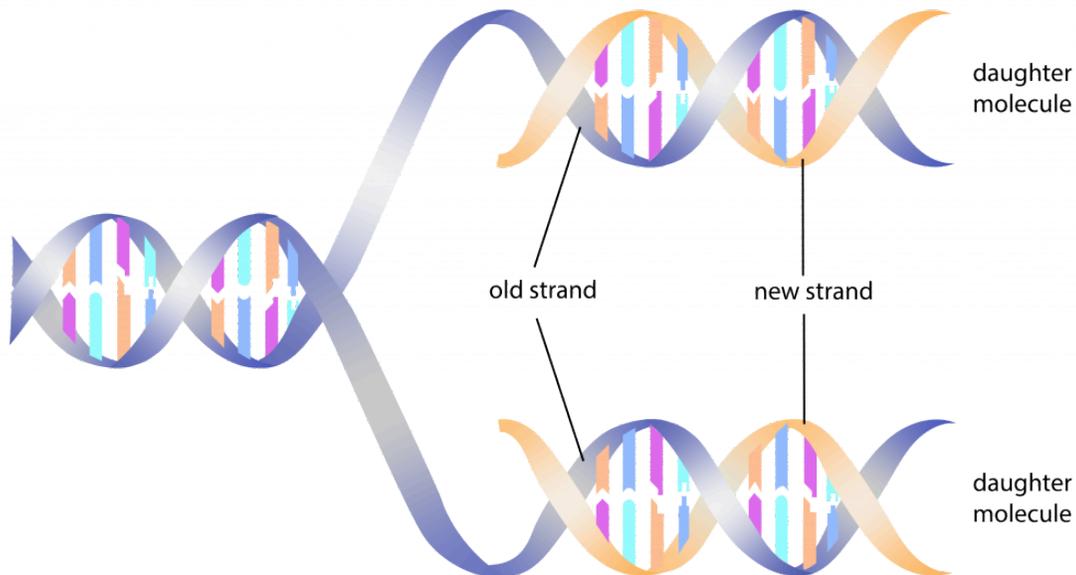


Figure 6.2 Parent versus daughter molecule

After DNA replication, the steps of mitosis can proceed. During the first step, **prophase**, the DNA of each chromosome condenses into tightly wound structures called **chromatids**. Sister chromatids, or *chromatid pairs*, contain the two new copies of the DNA—held together in the middle by a structure called a *centromere*.

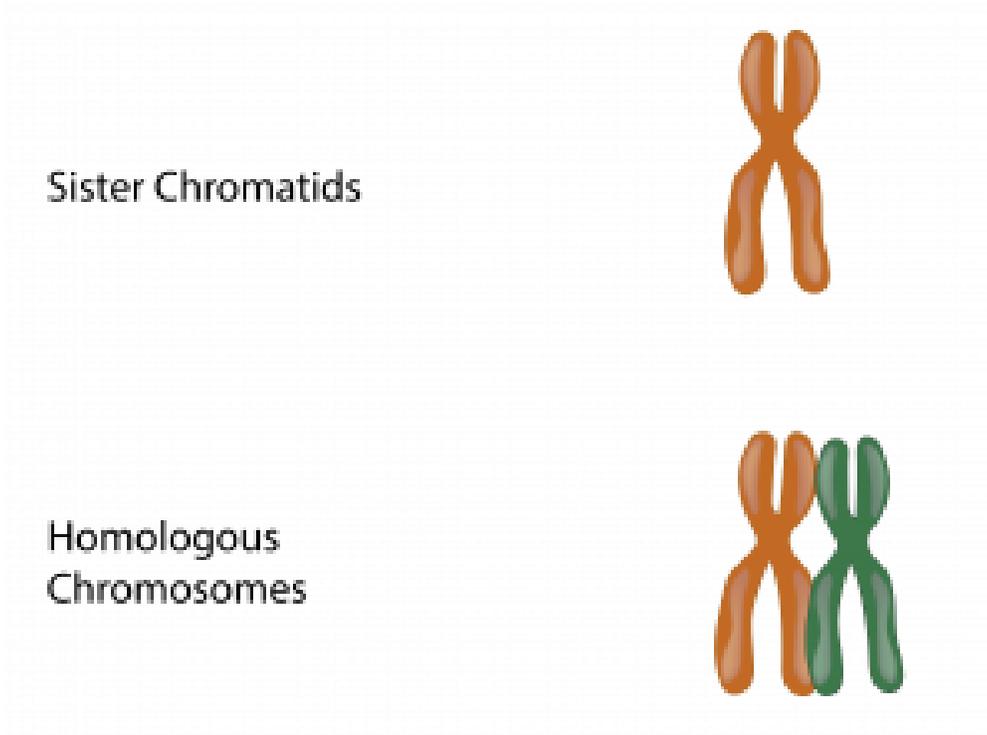
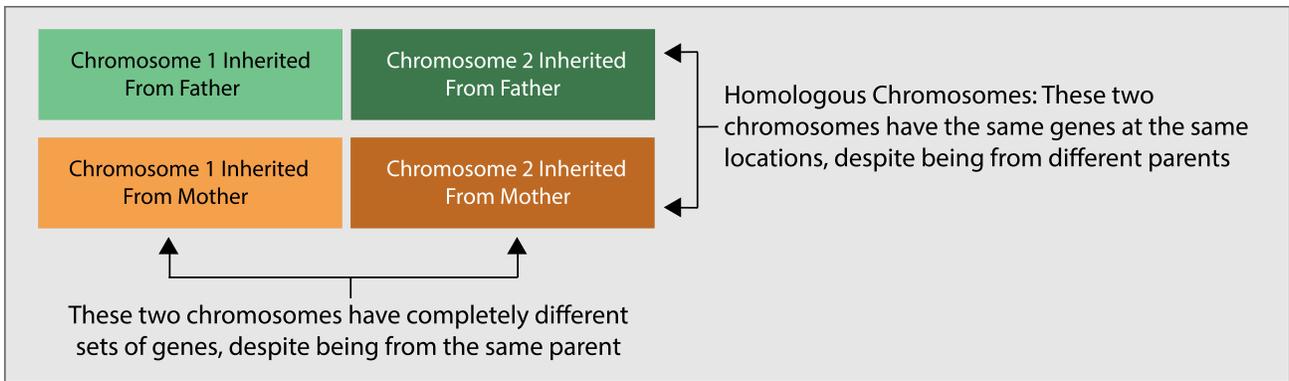


Figure 6.3 Homologous Chromosomes versus Sister Chromatids

During **metaphase** the chromatid pairs line up in the center of the cell. During **anaphase**, the sister chromatids are pulled apart to opposite sides of the cell by structures called microtubules. Finally, two new nuclei form around the DNA (**telophase**), and the cell divides down the middle (**cytokinesis**), forming two cells from the one. These two cells are identical to the original cell and identical to each other (aside from a small number of mutations).

Recall that most of the cells in your body have two copies of every chromosome, one inherited from your mother, and one inherited from your father. Together the two copies are termed homologues. When mitosis is completed the two new cells still have two sets of chromosomes or **homologous pairs**, just like the original. This concept of homologous pairs is important to our understanding of sex and the generation of genetic diversity. Pay attention to what happens with homologous pairs in the process of meiosis—described next.



MITOSIS OVERVIEW

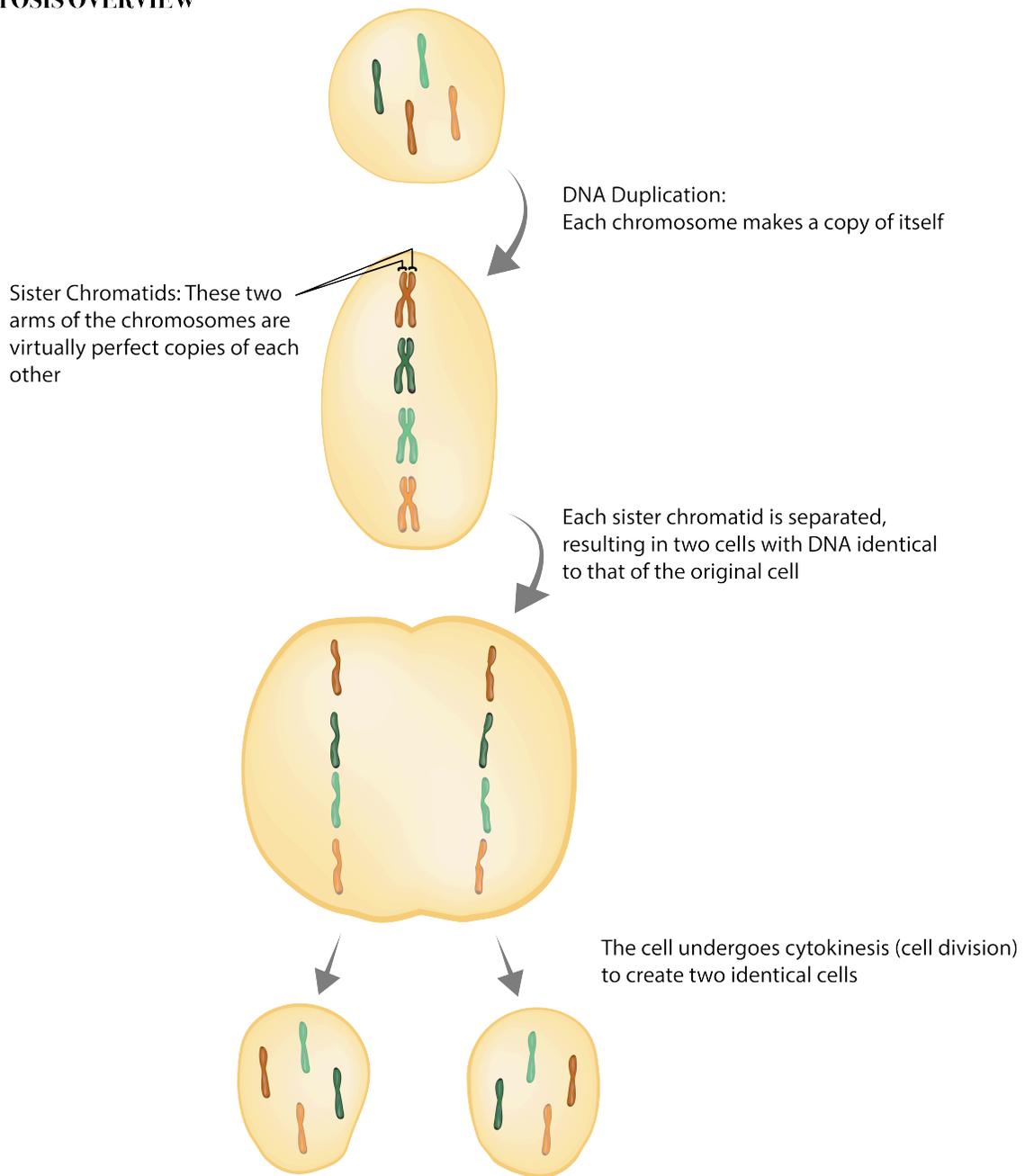


Figure 6.4 Mitotic Cell Division



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6.4 Meiotic division results in sex cells

Sex cells—eggs in females and sperm in males—can fuse to form a fertilized cell, or **zygote**. But how can two cells be fused without making a cell with too many chromosomes? Your skin, bone and brain cells all have two sets each of 23 pairs of chromosomes. Each set of chromosomes is called a homologous pair and in each homologous pair there is one chromosome that came from the mother and one from the father. That is, you have 23 homologous pairs of chromosomes in *almost* all of the cells in your body, resulting in 46 total chromosomes. In humans, if a cell with 23 pairs of chromosomes fused with another cell with 23 pairs of chromosomes, the new cell would have 46 *pairs* of chromosomes and 4 copies of each type of chromosome. For organisms that are diploid (meaning they have two sets of each chromosome) having more than two sets of chromosomes often results in problems with cell function. Instead, sex cells (sperm and eggs) have only *half* the number of chromosomes of a non-sex (or **autosomal**), cell. To achieve half the number of chromosomes, the germ cells (the cells that form sperm and eggs) undergo a process called meiosis. In human meiosis, each of the resulting cells has only one of each of your 23 chromosome pairs instead of 23 complete pairs of chromosomes. That is, each product of meiosis (egg or sperm) has only 23 chromosomes; egg and sperm cells do not contain homologous pairs.

To represent this with simple graphics, we represent meiosis here in an organism that has only two pairs of chromosomes. Keep in mind that for humans, there are actually 23 chromosome pairs undergoing these processes. Other organisms have different numbers of chromosomes: fruit flies have 4 pairs, or 8 total chromosomes; chickens have 39 pairs, or 78 total chromosomes; and chimpanzees have 22 pairs, or 44 total chromosomes.

A critical concept in our discussion of cell division and inheritance is that of **ploidy**, or the number of complete sets of chromosomes in a cell. Cells can be **monoploid** (1 set), **diploid** (2 sets), **triploid** (3 sets), and so on. In much of this chapter, we discuss humans; humans are diploid organisms, with two complete sets of chromosomes—one set from the father and one set from the mother—in almost all of their cells. The exceptions are the gametes, which are **haploid**. For diploid organisms such as humans, chickens, fruit flies, and chimpanzees, the haploid number is “1,” and refers to the fact that their sperm and egg cells have only one complete set of chromosomes. You’ll note that the term n is used to describe an organism’s haploid state; in diploid organisms, all the non-sex cells, or autosomal cells, are $2n$.

Understanding homology in humans

To complicate terminology further, only roughly half of humans have 23 homologous pairs. Women, who are genetically characterized by the presence of two X chromosomes, have 22 non-sex chromosomes and a pair of Xs. These X chromosomes have the same genes at the same locations and are thus homologues like the other 22 pairs of chromosomes. However, human males are typically characterized by having an X chromosome and a Y chromosome. These chromosomes are different in size, shape, and the genes they carry. Therefore, they are not homologues. Consequently, human males have 22 homologous pairs and one non-homologous pair of sex chromosomes—XY. It is typical for us to refer to humans—males and females—as having 23 pairs of homologous chromosomes, but now you know: it’s *actually* not quite that simple!]

6.5 Meiosis I

While mitosis involves one cell division, forming two cells from one original cell, meiosis involves two full cell divisions, forming 4 cells from one original cell. We call these two divisions meiosis I and meiosis II. Like in mitosis, meiosis begins after all of the DNA on all of the original cell's chromosomes has replicated. The chromosomes then condense into **chromatid pairs** or **sister chromatids**.

PROPHASE I. Meiosis is initiated in a stage called **prophase**. In prophase of the first meiotic division (**prophase I**), each of the chromosomes in the cell comes together with its homologue, forming structures called tetrads (the 'tetra' refers to the 4 chromatids of each duplicated homologous pair).

DNA Replication to Prophase I

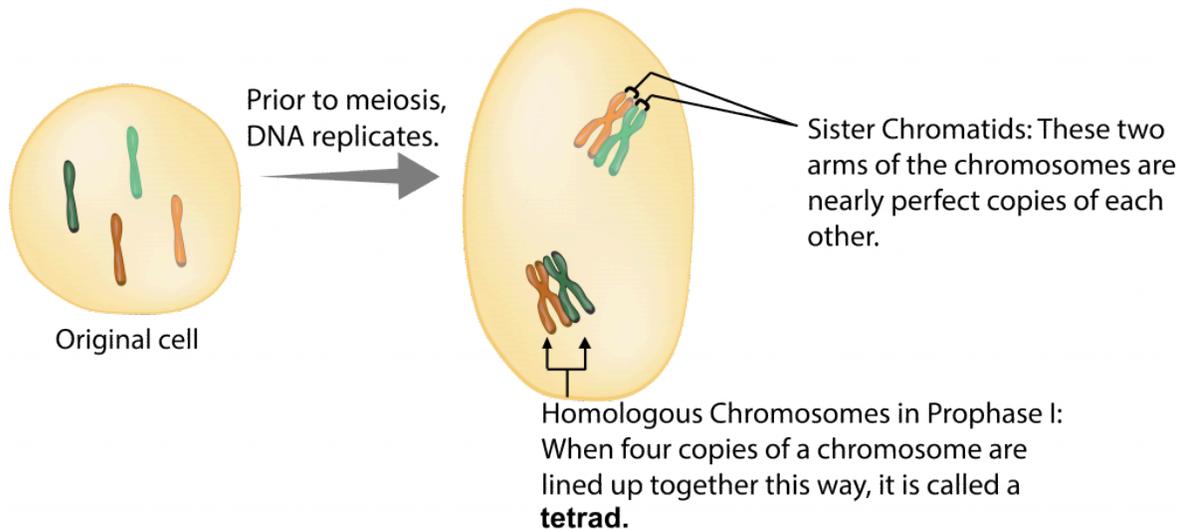


Figure 6.5 DNA Replication to Prophase I

METAPHASE I. In **metaphase I**, half the genetic identity of the potential offspring is determined. Specifically, all the homologous chromosome pairs, the tetrads, align in the middle of the cell. How they separate in the next stage is key.

Prophase I to Metaphase I

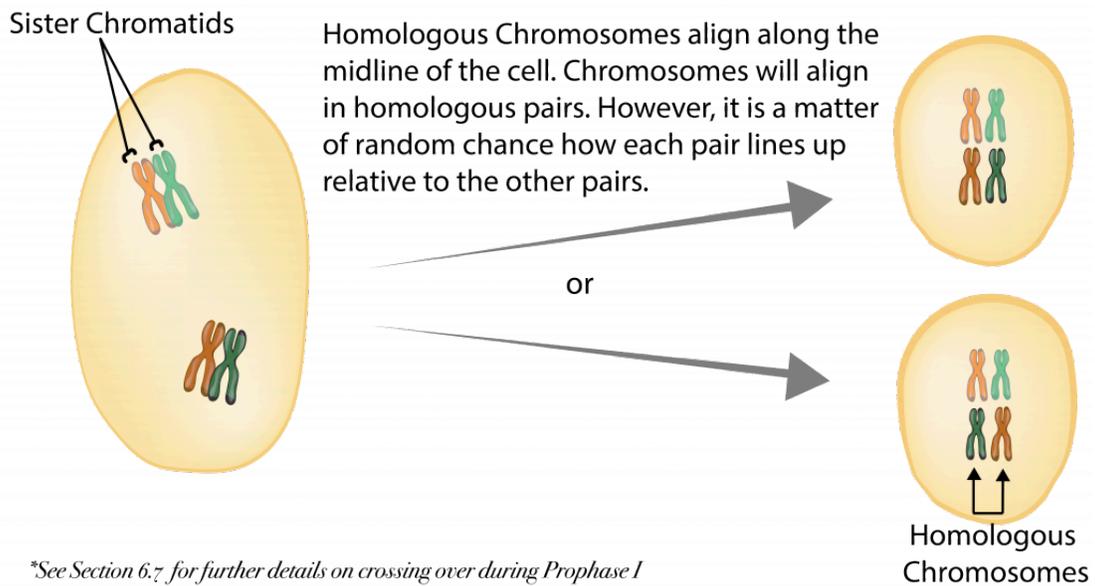
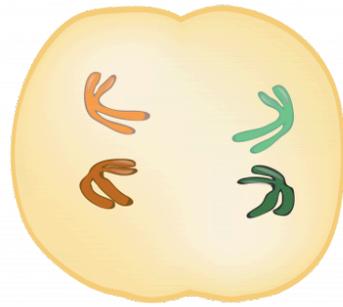


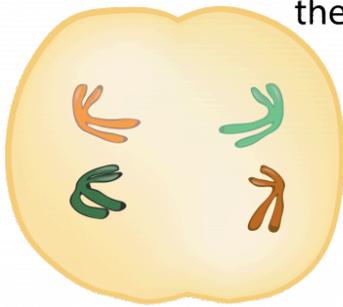
Figure 6.6 Prophase I to Metaphase I

ANAPHASE I. In **anaphase I**, each pair of homologues is pulled apart, with the chromatid pairs still intact, to assort to opposite sides of the original cell. The cell will cleave in the middle during telophase I & cytokinesis, forming two cells. Which of the chromatid pairs (paternal or maternal) go to each newly formed cell is a random function, depending simply on how the homologous pairs lined up during metaphase.

Anaphase I



or



In anaphase I, the homologous pairs separate. While the original cell is diploid, the resulting cells are haploid.

Figure 6.7 Anaphase I

Each new cell formed at the end of meiosis I will only inherit a chromatid pair from a single parent. **Independent assortment** is a term used to describe the process in which the homologous pairs divide into the daughter cells *randomly*. Given that humans have 23 pairs of chromosomes that can assort randomly, after this simple division there are 2^{23} , or over 8 million, possible combinations of chromosomes. The resulting sex cells are each a random mixture of both parents' chromosomes. Independent assortment is a major generator of genetic diversity, and a process that is unique to sexually reproducing organisms.

TELOPHASE I & CYTOKINESIS I. The first meiotic division concludes with **telophase I** and **cytokinesis I**. During telophase I, two nuclear membranes form and during cytokinesis, the cell will cleave in the middle to form two haploid cells.

Telophase I

Two nuclear membranes begin to form.

As a result of cytokinesis (cell division), we are left with two haploid cells that differ from the original cell.

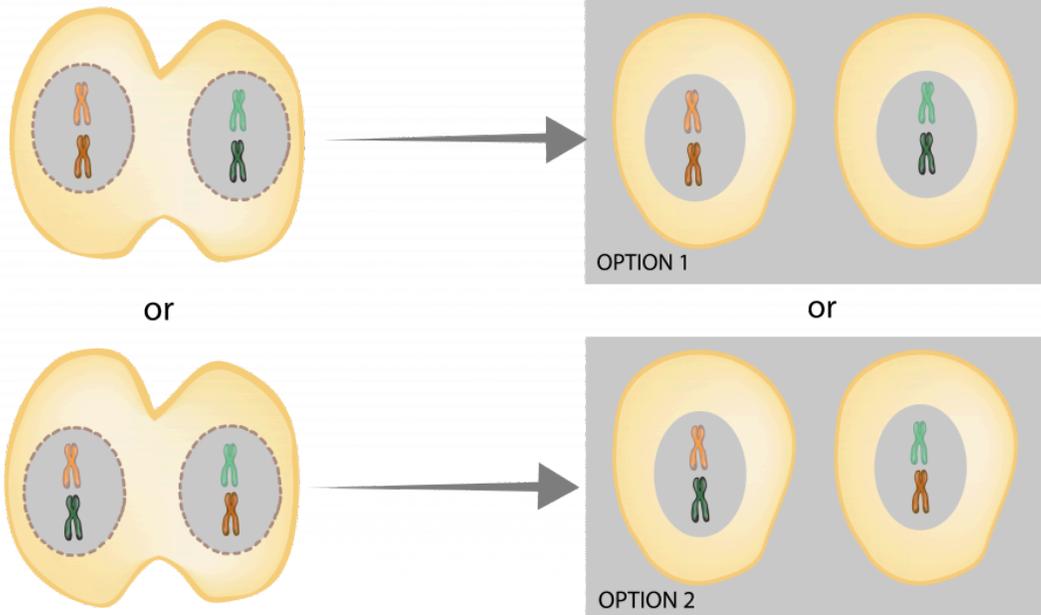


Figure 6.8 *Telophase I to Cytokinesis*

6.6 Meiosis II

After the first meiotic cell division, the resulting cells have been reduced from diploid to haploid because they now only have either a maternal or a paternal copy of each gene. However, each of these chromosomes still has two complete copies of DNA (the sister chromatids). The second meiotic division divides the sister chromatids, ensuring that only one copy is passed down.

MEIOSIS II

Following Option I from the previous page,

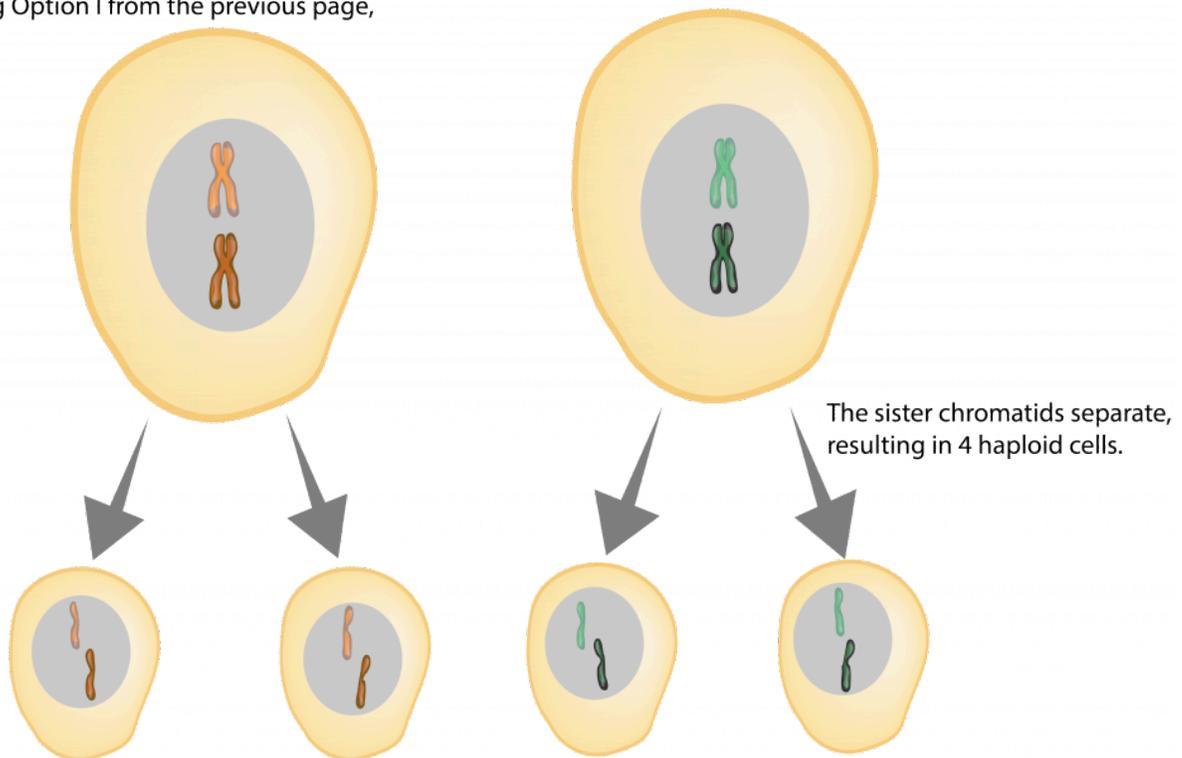


Figure 6.9 Meiosis II

In meiosis II the two cells from meiosis I are further divided into two cells each (for four cells total). Meiosis II looks a lot like mitosis: the sister chromatids separate and the chromosomes migrate into separate cells that are nearly identical to each other.

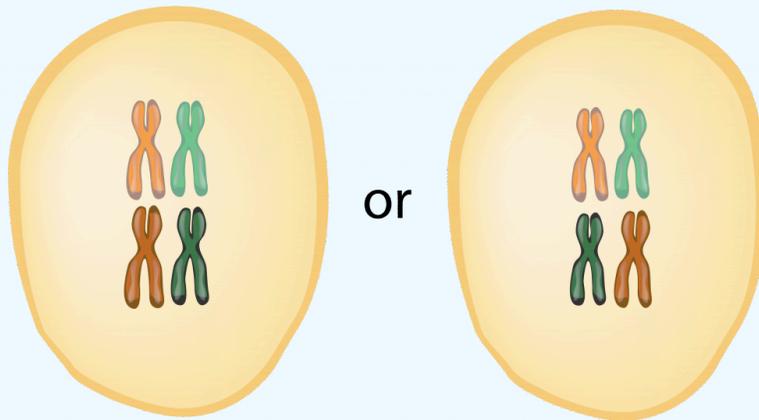
In a lifetime, a human female can produce hundreds of eggs and a human male hundreds of billions of sperm, essentially all of which will be genetically unique. However, most of these sex cells will never be used.



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6.7 Further genetic diversity is generated through crossing over

Independent assortment allows for the creation of genetic diversity without the dangers of mutation. That is not the only force that generates diversity. **Crossing over** or is another way that novel and diverse combinations of DNA are created.

Crossing over during Prophase I

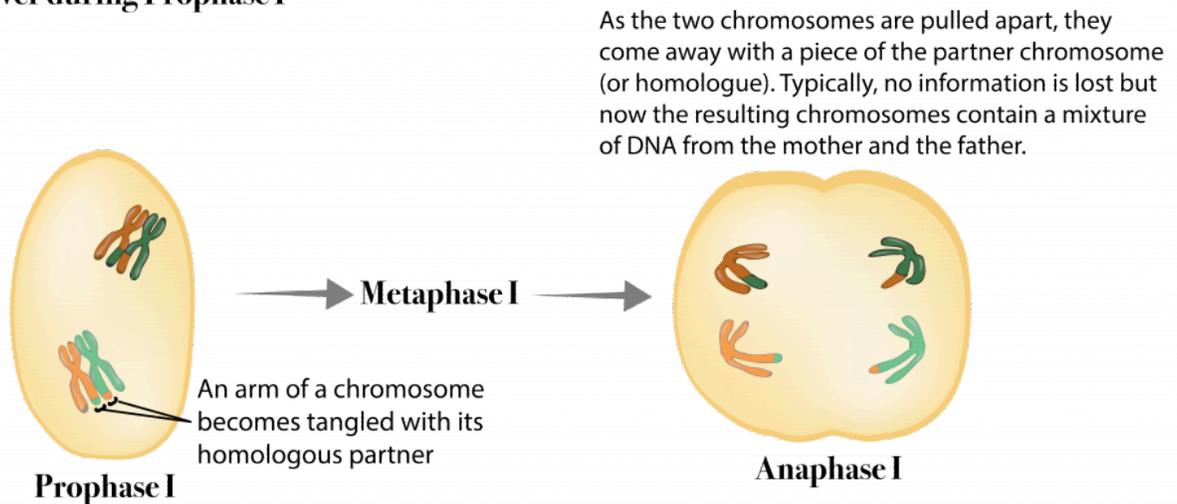


Figure 6.10 Crossing Over

Crossing over occurs in prophase I of meiosis, when the homologous pairs of chromosomes are together in **tetrads**. It is typical for an arm of one chromatid to get tangled with one of its homologues' chromatids. During disentangling in anaphase I, when the homologous pairs separate, the chromosomes will sometimes exchange portions of DNA. For example (and as depicted below), a chromosome that was originally entirely from your father could get tangled with your mother's corresponding chromosome, only to leave the tangle with most of the chromosome coming from your father but part of one of the chromatids coming from your mother's chromosome.

Although this process may seem chaotic, it is not random. Even when there is trading of DNA, each chromosome usually leaves with as much DNA as it started with, and the pieces of DNA that are swapped tend to contain complementary genes. Crossing over allows for the rearrangement of genetic code within the chromosome. In other words, in addition to the 2^{23} different cell types that can be created because of independent assortment, crossing over is an additional way your cells generate diversity in sperm and egg cells during meiosis.



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6.8 Pulling the pieces together

Meiosis + Outcrossing = Extreme diversity

Meiosis produces seemingly innumerable combinations of genes from a single individual. The randomness of meiosis is why biological siblings can be so different—they have unique combinations of genes, even though they came from the same parents. While the creation of sex cells is a key part of what makes sex such a great promoter of diversity, a significant amount of diversity comes from sex itself—specifically **outcrossing**, or mating involving two individuals.

Meiosis produces sex cells, each of which contains only one chromosome of each homologous chromosome pair. To make a genetically complete cell (with two copies of each chromosome pair), **fertilization** needs to occur. Fertilization is the merging of a sperm and an egg cell to make a cell with genetic material from both parents. This new cell, the zygote, contains a set of chromosomes, with a unique combination of genes, from both its mother and father.

Recap: Genotype, phenotype, and mutation

You have over 3 billion genetic “letters” (the nucleotides) that collectively represent your **genotype**. The genotype is the raw genetic data that you inherited and could potentially pass down to offspring. Your genotype, along with environmental cues and other factors that affect gene expression (see chapter 5) make up your **phenotype**. Simply, your phenotype is the expression of your genotype.

We can use an understanding of genetics to predict inheritance patterns of certain traits. You may be interested in the inheritance of traits that are advantageous (such as resistance to certain diseases), neutral (such as hair color), and harmful, or *deleterious* (such as disease traits). In the following section we discuss patterns of inheritance for different types of expressed traits, or phenotypes. Keep in mind that many traits are neutral, some traits are advantageous in some environments and deleterious in others, and some traits might be advantageous when an individual has one copy and deleterious when an individual has two.

6.9 Recessive traits are expressed when two copies are present

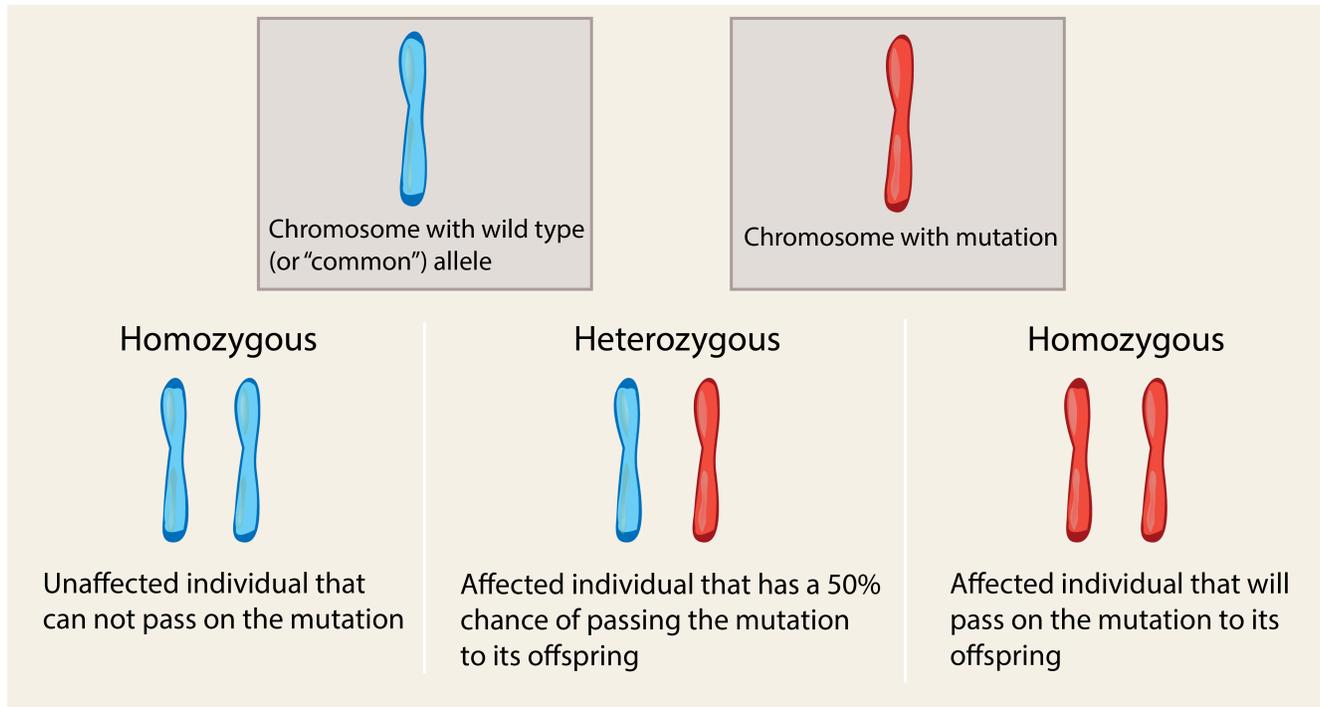


Figure 6.11 Recessive Mutation Chart

For many genes in your body, it doesn't really matter if one copy, on one of the homologues, has a mutation that reduces the function of the gene product or protein. Typically, the healthy copy can prompt the production of enough functioning protein to ensure normal development. In these cases, there won't be noticeable changes to an individual's phenotype unless the individual inherits two copies of the mutation. In other words, both alleles at a single locus code for proteins with reduced, altered, or no function. These conditions, which are only expressed when two alleles are inherited, are **recessive**.

A **carrier** is an individual who has a single copy of a recessive allele. In the case of a recessive trait, having only one mutated copy will not cause the individual to display the phenotype. These recessive inheritance patterns allow for an individual to carry potentially problematic mutations without suffering any of the consequences. Although carriers will not display the trait, they can pass the mutation on to children. If two healthy carriers both pass on a recessive trait, the phenotype will present in the individual offspring. One such example is Tay-Sachs disease, a disorder that is caused by recessive mutations in the HEXA gene, located on chromosome 15. Children with Tay-Sachs disease live for a very short period of time. However, this mutated HEXA gene still is able to persist in populations since carriers can be completely healthy, and grow up to reproduce.



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6.10 Dominant alleles can mask recessive alleles

In contrast to recessively inherited traits, some alleles are **dominant**. With a dominant trait, an individual will express the associated phenotype even with just one copy of the allele for the trait.

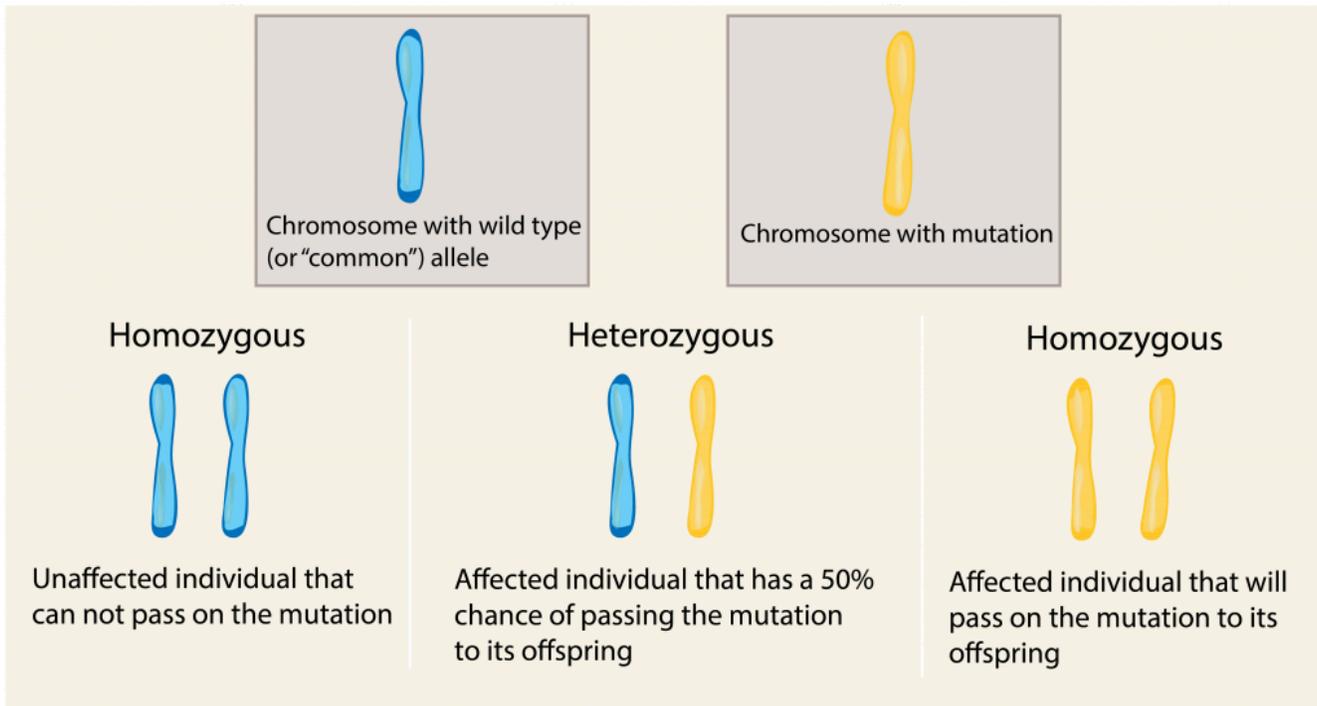


Figure 6.12 Dominant Mutation Chart



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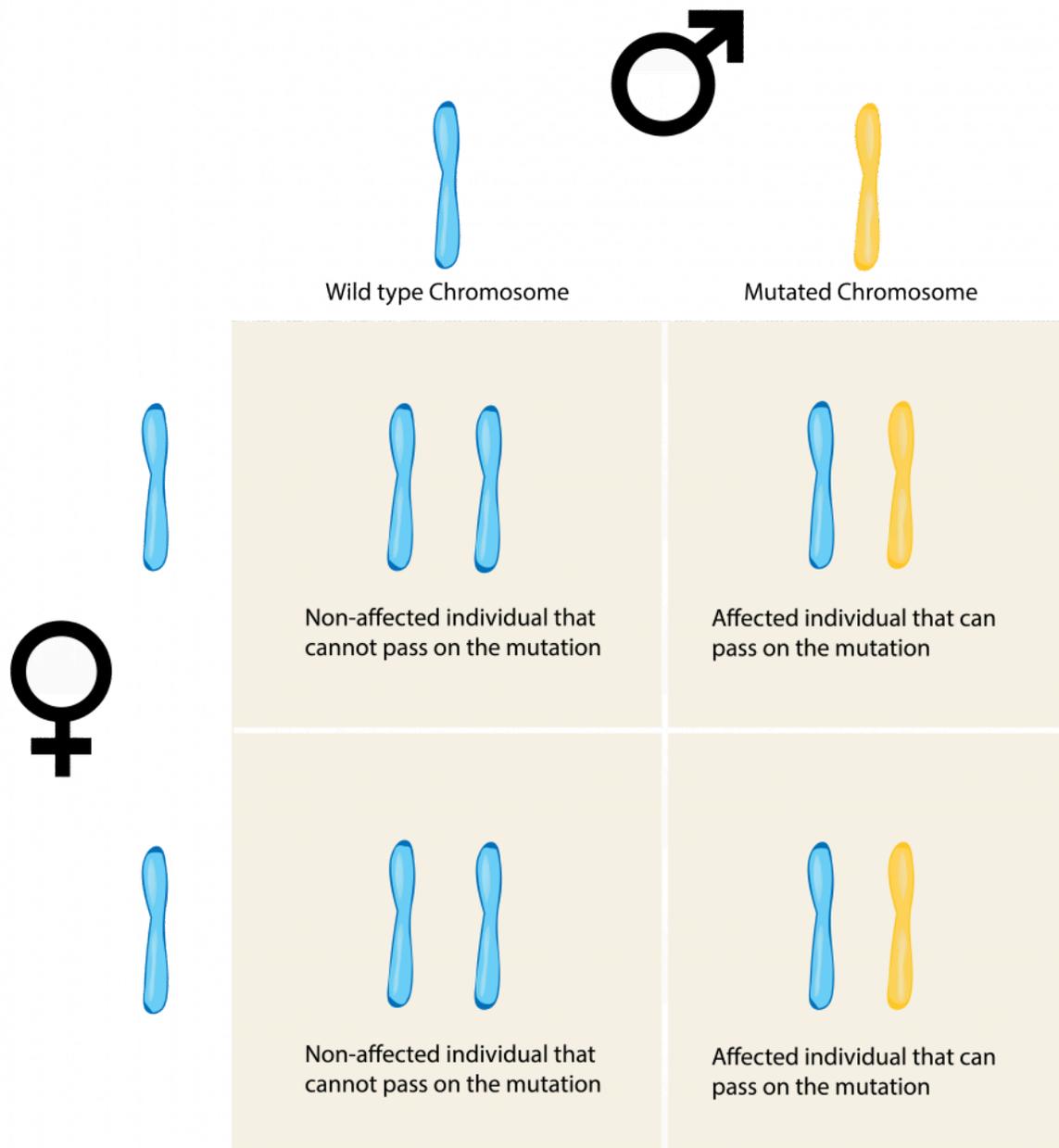


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6.11 Calculating the odds of inheritance

Punnett squares are visual tools to help us calculate the inheritance of a single trait, typically when given the parental genotypes. Each box in a simple matrix represents an array of potential genotypes for the offspring of two individuals.



There is a 50% chance that each new child from these parents will be affected by the mutation.

Figure 6.13 Dominant Mutation. Punnett Square

For simplicity, Punnett squares are actually annotated with letters representing only the gene in question, rather than entire chromosomes. The letters outside the box represent the possible *gametotypes* (gamete genotypes) for each parent—one in the left column and one in the top row. In a **monohybrid cross**, whereby we're focusing on inheritance probabilities for a single gene, a 2X2 matrix will be sufficient. Each parent will potentially pass down one of its two copies of the gene, creating up to four potential combinations. Upper- and lower-case letters are often used to represent different alleles. For example, an upper-case letter might denote a dominant allele, and a lower-case letter might denote a recessive allele.

The following Punnett squares track the inheritance of a gene for which the father has a mutation in only one of the copies of gene A, but the mother has no copies of the mutation. The mutated copy is denoted with a lower case *a* and the non-mutated, or **wild-type**, gene is denoted with an upper case *A*. There is a 50% chance that each parent will pass on each of the two copies. The mother will always pass on her dominant *A* allele, so we expect that all their potential offspring will have at least one *A*. The father is equally likely to pass on either the wild type *A* or the mutated *a*; therefore half the potential offspring have a second *A* and are **homozygous** (they have two of the same alleles) *AA*, and the other half have a mutated *a* and are **heterozygous** (they have two different alleles) *Aa*.

		Father's Genotype	
		A	a
Mother's Genotype	A	AA	Aa
	A	AA	Aa

Figure 6.14 Punnett Square



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		Father's Genotype	
		\bar{R}	\bar{r}
Mother's Genotype	R	RR	Rr
	r	Rr	rr



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6.12 Sex chromosomes

So far, the genes for the traits we have discussed occur on chromosome pairs 1 through 22 in humans. Almost every person has two copies of these chromosomes, one from their mother and one from their father. However, there is one exceptional set of chromosomes: the “sex chromosomes” (or pair 23; see [“Understanding homology in humans”](#)). These are known in humans as the **X and Y chromosomes**, because of their appearance.

Generally, female humans have two pairs of X chromosomes. Like chromosome pairs 1 through 22, each one of these X chromosomes came from one of each of her parents; they are *homologues*, and code for the same genes in the same locations, but are not genetically identical. For males it’s different. Males still inherit a single X chromosome from their mother. However, this X chromosome is paired with a mismatched Y chromosome that is inherited from the father. The Y chromosome is a fraction of the size of the X chromosome and it houses an entirely different set of genes than the X. The Y chromosome contains genes that provide the genetic instructions for the individual to become a male.

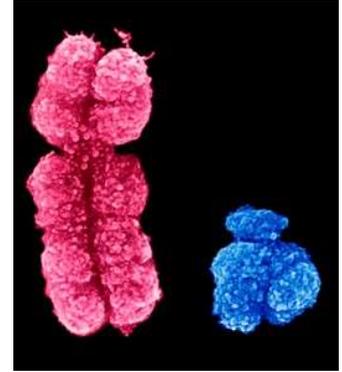
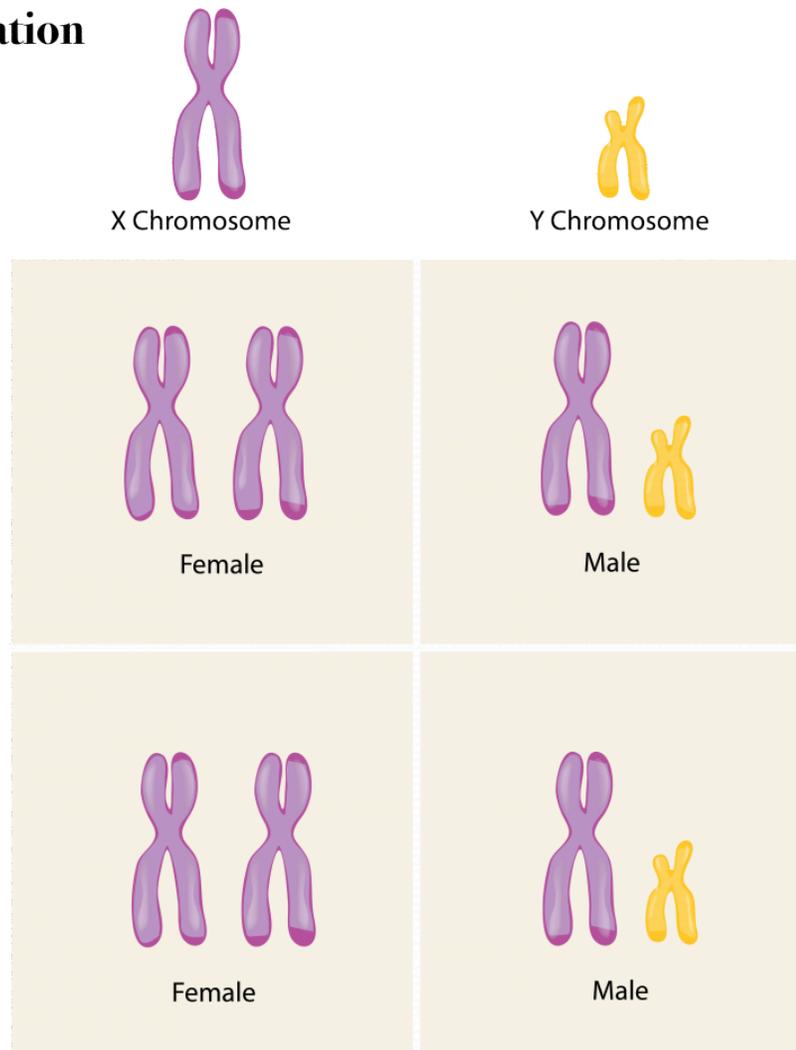


Figure 6.15 A colored image of the X (Pink) and Y (Blue) Chromosomes.

Sex Determination



There is a 50% chance of a male (XY) child and a 50% chance of a female (XX) child.

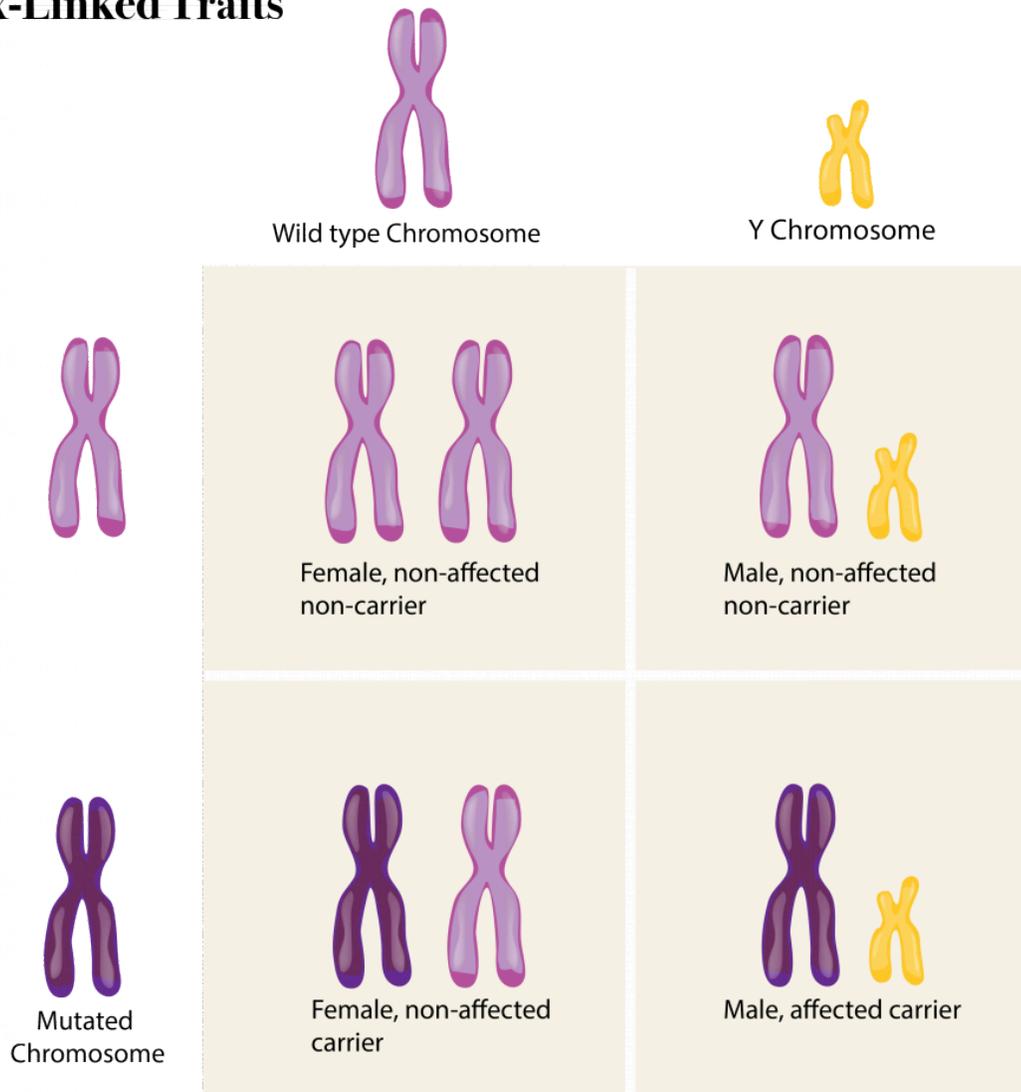
Figure 6.16 Sex Determination Punnett Square

Read More

Note that there are exceptions in which XY does not produce males and XX does not produce females. You can read more about various genetic conditions that can result in such conditions in [Chapter 8](#) and [here](#).

The non-matching chromosome configuration of the XY chromosomes makes for some unusual inheritance patterns. Recall that for most genes everyone has two copies—one on each chromosome. But for males, there is no second copy of genes on the singular X chromosome or the singular Y chromosome. This means that if a gene on a sex chromosome is defective or different, males have no second copy to compensate for its loss. Any normally recessive mutations will automatically present as phenotypes from the sex chromosomes of males. These traits that manifest themselves automatically in males are called **sex-linked traits**.

Sex-Linked Traits



There is a 50% chance of a male (XY) child and a 50% chance of a female (XX) child.

Figure 6.17 Sex-linked Traits Punnett Square



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6.13 Sex-linked inheritance

An example of a sex-linked trait is red-green colorblindness. The OPN1LW gene, which codes for a protein that allows humans to tell the difference between red and green, is located on the X chromosome. Without functional OPN1LW protein, a person will be red-green colorblind. In females, color blindness is a recessive trait because the possession of a single functional copy of OPN1LW is sufficient for normal

vision. Males, however, only have one copy of the X chromosome and thus only one OPN1LW gene. So, if there is a functional mutation on the OPN1LW gene of a male's only X chromosome, he will be colorblind. There is no corresponding gene on the Y chromosome to compensate for this loss of function. This difference in sex chromosomes explains why red-green colorblindness is rare in females, since females need to inherit two mutated copies if they are to be red-green colorblind. However, females must be **carriers** of colorblindness in order to pass the trait on to any of their sons.

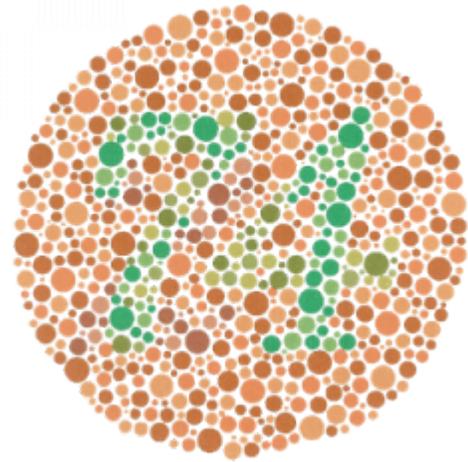


Figure 6.18 Color blindness example. Can you read the number on this colorblindness test?

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1. https://en.wikipedia.org/wiki/Ishihara_test#/media/File:Ishihara_9.png



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6.14 Moving beyond single-gene effects

In this chapter we've focused almost entirely on genetic patterns that result in **monoallelic traits**—traits in which a change in a single gene is enough to cause a change in phenotype. But monoallelic inheritance patterns are rare.

Most phenotypes are controlled by multiple genes. In general, when people talk about inheritance, they are likely discussing complex traits such as appearance, intelligence and personality. Commonly we hear phrases like “She’s got her father’s eyes” or “He’s got his mother’s temper.” Neither eye color nor temper are controlled by single genes. In fact, most of the meaningful traits that would influence an organism’s ability to survive and thrive are controlled by many separate genes. A complex trait such as intelligence, for example, requires the coordination of many biological functions and thus many genes working together *in concert with environmental influences*. Some have suggested that there are over 500 separate genes that may control human intelligence.¹ Traits controlled by many genes are called **polygenic traits**.

Because polygenic traits represent a mixture of genes, it is very difficult to find single genes that can significantly change a polygenic trait. Psychologists have developed detailed ways of measuring complex traits such as intelligence and mental illnesses. However, it’s much more difficult to study how these traits are passed down genetically. One such example is the disorder schizophrenia, an illness characterized by peculiar social behavior and confusion about reality. Schizophrenia is believed to be caused by a mixture of inheritable genetic mutations and triggers from the environment. People are more likely to have schizophrenia if they have a relative who has it, which suggests genetics play a role in susceptibility. But schizophrenia is also a polygenic trait and difficult to track genetically.

1. <https://www.nature.com/articles/s41380-017-0001-5>

6.15 Wrapping up: A return to cloning labs



A return to cloning labs

As you age, your DNA progressively accumulates mutations. Everything from the sun's rays to the food you eat can slowly modify your DNA. As we've discussed, most of these mutations don't affect protein production enough to alter phenotypes; in fact, most mutations are *silent* mutations. But if you live long enough, one of these random mutations will affect an important protein. If the wrong protein is altered, your heart could fail or you could develop cancer. This is why the older you get, the more likely you are to get cancer.

Sex offers a way around this problematic genetic decay. Only mutations that occur in the germ cells (the cells that undergo meiosis to create gametes) can be passed on to future generations. The mixing of parental DNA allows most recessive mutations to get masked by the other copy of DNA. Meiosis acts to create diversity and fertilization helps mask many potentially harmful side effects of that mixing.

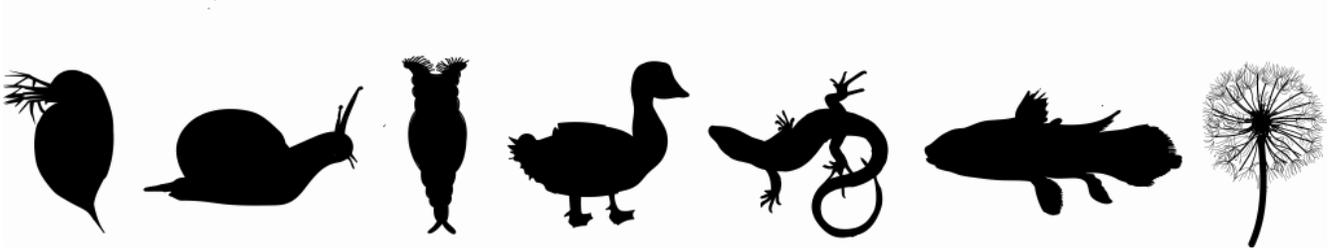
This is where Dolly the cloned sheep is at a disadvantage. She was a new sheep created with a copy of DNA from a cell from the mammary gland of an adult sheep. That cloning process is dangerous for two reasons. The first problem is her age. The cell from the original adult sheep was the product of many mitotic divisions and a (sheep's) lifetime worth of accumulated mutations and DNA damage. When Dolly was born, she was born with the accumulated DNA damage of a fully-grown adult. Dolly's cells showed signs of premature aging, which would not have been the case if she had been a product of sex; rather, she would have begun life with "fresh" DNA.

The second problem with skipping sex is what this does to the population. If all the sheep in a continent were clones of each other, they would all have the same DNA. This sameness would impart the population with identical genetic strengths and weaknesses. A single disease could wipe out the entire population. This disease susceptibility is one of the reasons farmers and horticulturalists are concerned about growing monocultures—single crops that are virtually genetically identical, even if they aren't necessarily clones. Genetic diversity helps a population be resilient in the face of change, and meiosis and outcrossing are the creators of this diversity.¹

As we conclude this chapter and prepare for in-class discussion, be sure to return to the chapter's [goals and objectives](#).

1. https://evolution.berkeley.edu/evolibrary/article/agriculture_02

Chapter 7: Why Sex?



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7.0 Introduction

Asexual, lesbian lizards

It is difficult to mistake whiptail lizard copulation for anything else (Figure 7.1). A lizard mounting another lizard from behind certainly suggests sex. However, whiptail lizards of the genus *Apidoscelis* are all females and reproduce asexually. Eggs are produced through meiotic division, but then undergo a doubling of chromosomes to return to a full set of chromosome pairs. This type of **asexual reproduction**, in which embryos develop from unfertilized eggs, is called **parthenogenesis** (from the Greek, meaning “virgin birth”), and is seen in many plants and animals.

These lizards are doubly intriguing because they may exhibit a stereotypical mating behavior; in fact, this behavior has been shown to increase the number of offspring the lizards produce (even though there are no sperm involved!).

Species like the whiptail lizards highlight the major problem with sex. There are many asexual organisms such as these lizards (figure 1), and they appear to be doing just fine without sexual reproduction. This ability to thrive without sex demands an answer to the question: why does sex exist? Why would any organism incur all the costs associated with sexual reproduction, if asexual reproduction has so many obvious advantages?



Figure 7.1 Female – female copulation in whiptail lizards (*Apidoscelis uniparens*). Note the scientific name: “uniparens” means “one parent.” Which female assumes the dominant “male” role and which female assumes a “female” role depends on which female is ovulating.

7.1 Chapter Objectives



Learning Objectives

Our goal for this chapter is for you to begin to appreciate that sexual reproduction is costly from an evolutionary perspective, and that there are hypotheses to explain why sexual reproduction exists in spite of these costs. Specifically, by the end of your reading and our in-class discussion, you will be able to:

1. Define the following terms:
 - **asexual reproduction**
 - **parthenogenesis**
 - **Muller's Ratchet**
 - **The Red Queen**
 - **major histocompatibility complex (MHC)**
 - **polygenic**
 - **polymorphic**
 - **codominant**
2. Identify three hypotheses for the persistence of sexual reproduction.
3. Where possible, list testable predictions associated with these hypotheses.
4. Identify evidence in support of specific hypotheses for the existence of sex.

7.2 Sex is a Problem

Between the cost of meiosis, the increased risk of disease transmission, and the cash required to buy dinner and a movie, sex is expensive! So why does it exist and why is it the major method of reproduction for multicellular organisms? The short answer to this question is, “nobody knows.” But like most short answers in science, that is not the interesting answer. Of the many hypotheses, a few consistently arise to explain the evolution and persistence of sex: 1) sex speeds up evolution; 2) sex leads to fewer mutations; and 3) sex is an evolutionary race between parasites and hosts.

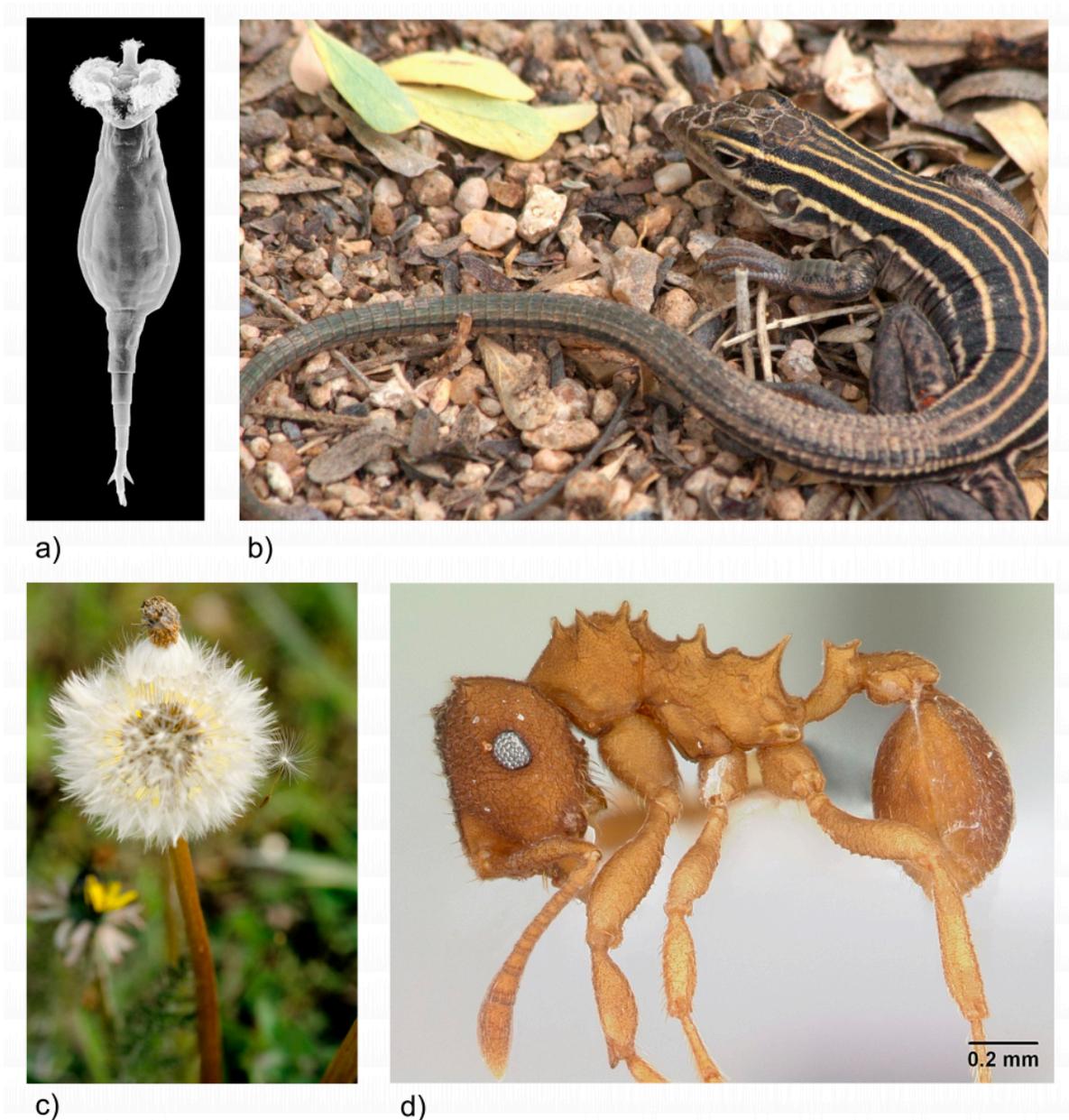


Figure 7.2 Various organisms thrive without sex. A few examples include **a)** the bdelloid rotifers; **b)** whiptail lizards; **c)** dandelions; and **d)** fungus-farming ants.

Does sex speed up evolution?

Several biologists have suggested that the purpose of sex is to speed up evolution. Between meiosis and exchanging gametes with another individual (or *outcrossing*), the process of sex produces a wide variety of offspring; this variety could potentially speed up the process of evolution.

One key prediction of this hypothesis is that species that evolve quickly have an advantage over species that evolve slowly. Unfortunately, this hypothesis is difficult to test, in that it makes a species-level claim about a mechanism (natural selection) that occurs at the individual level. An individual's fiercest competitors are those that are closely related and vie for the same resources. So imagine an asexually reproducing population that has a low-frequency variant that reproduces sexually. The sexually reproducing individuals would have to have some

sort of advantage over their asexual counterparts. Merely evolving at a more rapid pace does not necessarily give an immediate advantage to those individuals.

Additionally, while it may be true that sexually reproducing populations can change more quickly than asexuals, it does not necessarily follow that this is a good thing. Plenty of successful sexual organisms have changed little across long spans of time. For example, the coelacanth fish that exists today seems nearly identical to fossilized coelacanths that existed millions of years ago (Figure 7.3 and 7.4).



Figure 7.3 Image of a fossilized coelacanth from millions of years ago



Figure 7.4 Image of a coelacanth swimming today

Perhaps the largest problem with the idea that sex exists “to speed up evolution” is the premise that evolution itself is the goal. Change for the sake of change is not the point of evolution. In the case of natural selection, populations change in response to selective pressures. As part of this change, there are winners, to be sure, but there are also many losers.



Check Yourself



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<https://open.lib.umn.edu/evolutionbiology/?p=1329#h5p-32>

7.3 Does sex lead to fewer mutations?

Mutations are a fact of life. All organisms possess DNA that is different from the DNA they inherited, and this altered DNA, or mutation, is the ultimate source of all genetic variation. Some have suggested that one of the benefits of sex is its ability to rid the body of harmful (or *deleterious*) mutations.

Sex and DNA repair

Mutations arise from random errors in DNA replication: by the insertion or deletion of mobile genetic elements, or through the effects of various *mutagens* (e.g., chemical compounds or radiation) that damage DNA. However, most of the damage that occurs to a cell's DNA is repaired. The cell has numerous tools that it can use to fix the damage that can occur.

Mutagens such as radiation can cause single- and double-strand breaks in the double helix of DNA. If there is damage to only one DNA strand, the other strand can be used as a template for repair. More problematic is when both strands of DNA break; when this happens there is not a simple way to re-synthesize the lost DNA. In cells that are diploid (cells that have two copies of each chromosome), there is a complementary chromosome, or homologue, available—but in order to be useful, it must be close to the damaged DNA.

Chromosome pairs are close together in the first stage of meiosis, in which crossing over between the chromosomes occurs. In fact, many of the cellular tools used to repair double strand breaks in DNA are the same ones that facilitate crossing over (homologous recombination) during meiosis. So, if meiosis repairs damaged DNA, and meiosis is essential to sexual reproduction, perhaps sex exists because of the repair benefits of meiosis itself.

Sex and Ratchets

A ratchet works because it rotates in one direction, but not the other. Similarly, once a mutation occurs within a population, it is extremely unlikely to un-occur. Because mutations are relatively rare and occur randomly in an organism's genome, there is very low probability that there will be a "back mutation" or a second mutation in the exact same spot to undo the first mutation. Thus, over time, a lineage will likely accumulate increasing numbers of mutations, some of them harmful. This idea is often called "Muller's ratchet," after Hermann Joseph Muller, who hypothesized it, and the simple machine, the ratchet (figure 7.5).

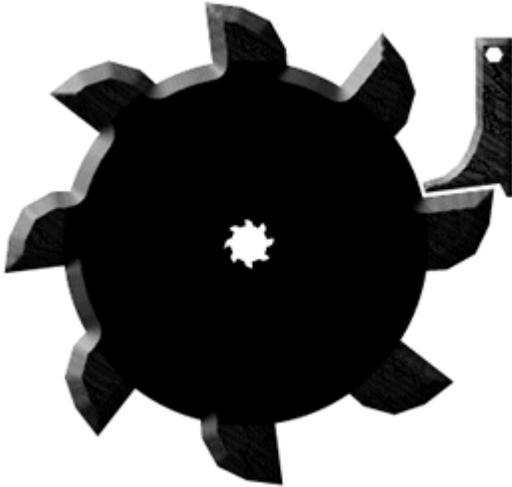


Figure 7.5 The ratchet works because it only rotates in one direction. This comes in handy when hoisting heavy objects, tightening screws, and loosening bolts. But what could this possibly have to do with sex?

According to **Muller's ratchet** hypothesis for the existence of sex, sexual reproduction is better able to eliminate harmful mutations from the genome. Creating a variety of gametes and combining those gametes with another individual (who would presumably have a different collection of mutations) results in some of the offspring carrying more harmful mutations than others. Presumably, the individuals or gametes with fewer harmful mutations survive and reproduce more successfully. In this way, through sex, a lineage has an opportunity to shuffle its genetic material, and produce some offspring with fewer harmful mutations (and the most beneficial ones). Thus, the shuffling of material that occurs during sexual reproduction essentially rotates the ratchet backwards.

1

Evidence supporting Muller's ratchet

A testable prediction associated with Muller's ratchet is: *Mutations accumulate more rapidly in asexually reproducing organisms than in sexual organisms.* To investigate this hypothesis, scientists study organisms that occur in both sexual and asexual forms. One such animal is the microscopic water flea (*Daphnia*; figure 7.6), which can be maintained in clonal or sexual lineages.

A comparison of strains of water fleas that reproduce asexually with those that occasionally have sex revealed that the asexual strains have more mutations. Specifically, the asexual water fleas have a higher proportion of substitution mutations that result in amino acid changes. This comparison supports the hypothesis that sexual reproduction reduces the accumulation of potentially harmful (or *deleterious*) mutations. Similar observations have been made in sexual and asexual lineages of freshwater snails.

2



Figure 7.6 This microscopic water flea (*Daphnia magnirostris*) can reproduce sexually or asexually



1. Image by ZabMilenko, CC BY 3.0. <https://commons.wikimedia.org/w/index.php?curid=49717967>

2. Image by: Paul Hebert. CC BY 2.5, Functional Genomics Thickens the Biological Plot. Gewin V, PLoS Biology Vol. 3/6/2005, e219. doi:10.1371/journal.pbio.0030219. <https://commons.wikimedia.org/w/index.php?curid=1428600>

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<https://open.lib.umn.edu/evolutionbiology/?p=1332#h5p-33>

7.4 The Red Queen

What does Lewis Carroll's Red Queen have to do with sex?¹

In this scene from *Through the Looking-Glass and What Alice Found There* by Lewis Carroll, Alice and the Queen run with all their effort – yet make no progress. Such is the claim that sex allows organisms to avoid extinction by keeping up in a very odd sort of race.

Alice never could quite make it out, in thinking it over afterwards, how it was that they began: all she remembers is that they were running hand in hand, and the Queen went so fast that it was all she could do to keep up with her: and still the Queen kept crying “Faster! Faster!”, but Alice felt she could not go faster, though she had not breath left to say so.

The most curious part of the thing was, that the trees and the other things round them never changed their places at all: however fast they went, they never changed their places at all: however fast they went they never seemed to pass anything. “I wonder if all the things move along with us?” thought poor puzzled Alice. And the Queen seemed to guess her thoughts, for she cried “Faster! Don't try to talk!”

Not that Alice had any idea of doing that. She felt as if she would never be able to talk again, she was getting so much out of breath: and still the Queen cried “Faster! Faster!”, and dragged her along. “Are we nearly there?” Alice managed to pant out at last.

“Nearly there!” the Queen repeated. “Why, we passed it ten minutes ago! Faster!” And they ran on for a time in silence, with the wind whistling in Alice's ears, and almost blowing her hair off her head, she fancied

“Now! Now!” cried the Queen. “Faster! Faster!” And they went so fast that at last they seemed to skim through the air, hardly touching the ground with their feet, till suddenly, just as Alice was getting quite exhausted, they stopped, and she found herself sitting on the ground, breathless and giddy.

The Queen propped her up against a tree, and said kindly, “You may rest a little, now.”

Alice looked round her in great surprise. “Why, I do believe we've been under this tree the whole time! Everything's just as it was!”

“Of course it is, “ said the Queen. “What would you have it?”

“Well, in our country, “ said Alice, still panting a little, “you'd generally get to somewhere else – if you ran very fast for a long time as we've been doing.”

1. Public Domain, <https://commons.wikimedia.org/w/index.php?curid=2040219>

“A slow sort of country!” said the Queen. “Now here, you see, it takes all the running you can do, to keep in the same place. If you want to get somewhere else, you must run at least twice as fast as that!”

“I’d rather not try, please!” said Alice...

Through the Looking-Glass and What Alice Found There by Lewis Carroll

Is sex part of an arms race?

In one human generation, HIV (the virus that causes AIDS) will reproduce over a million times. Given how natural selection works—via heritable variation and differential reproduction—human beings don’t stand a chance against this virus. How can we possibly adapt to such a fast-moving target? For that matter, how can any longer-lived organism compete with a quickly reproducing and quickly evolving enemy? Many of these enemies, or **pathogens**, such as viruses and bacteria, are also numerous and difficult to detect—invisible to the naked eye, they can enter a host’s body silently and reproduce with a fervor while their victims remain blissfully unaware. Given these challenges, how can any host organism defend itself against its would-be attackers? According to one hypothesis, outwitting pathogens is the whole point of sex.

The Red Queen

We are in the midst of an *evolutionary arms race*, in which host and parasitic pathogen must constantly adapt. Parasites must adapt to the host’s natural defenses, and host populations are under pressure to keep up with their ever-changing parasites. This reciprocal evolution between two types of organisms (in this case, host and parasite) is a type of **coevolution**. According to the **Red Queen Hypothesis**, sex exists as a mechanism for keeping up with rapidly coevolving pathogens. By generating genetic diversity, sex makes host organisms a moving target. Like Alice and the Red Queen in Lewis Carroll’s novel (Box 3), both host and parasite are running a race in which neither makes any observable progress. Yet, if the host organisms didn’t change dramatically with each new generation (if they didn’t have sex), they might go extinct.

Parasites adapt to exploit the most common type of host. Therefore, a host that can produce offspring that have novel defenses against parasites would have an advantage over an organism producing clones—simply by making offspring that are different.

7.5 Testing the Red Queen Hypothesis

The Red Queen hypothesis—that sex evolved to combat our coevolving pathogens—can be tested by analyzing a few key predictions of this hypothesis:

1. Sex is most beneficial where there is a high risk of infection
2. Pathogens are more likely to attack common phenotypes (for example, clones) in a population, as opposed to the less-common counterparts (such as those that resulted from sex)
3. In sexually reproducing populations, individuals choose mates that maximize diversity in their offspring

Note that all of these predictions implicitly rely on the heritability of being healthy (in this case, the ability to combat pathogens); specifically, parents must be able to pass along to their offspring genes for avoiding pathogens. Testing these predictions has resulted in several lines of evidence supporting the Red Queen hypothesis.

Prediction 1: Sex is most beneficial where there is a high risk of infection

An excellent system for testing this prediction involves a flatworm parasite in the genus *Microphallus*, a duck, and a small mud snail (*Potamopyrgus antipodarum*; Figure 7.7). This species of snail is able to reproduce sexually or asexually. The extent of sexual reproduction in a population of snails can be quantified by counting the number of males—asexual snails are all female.

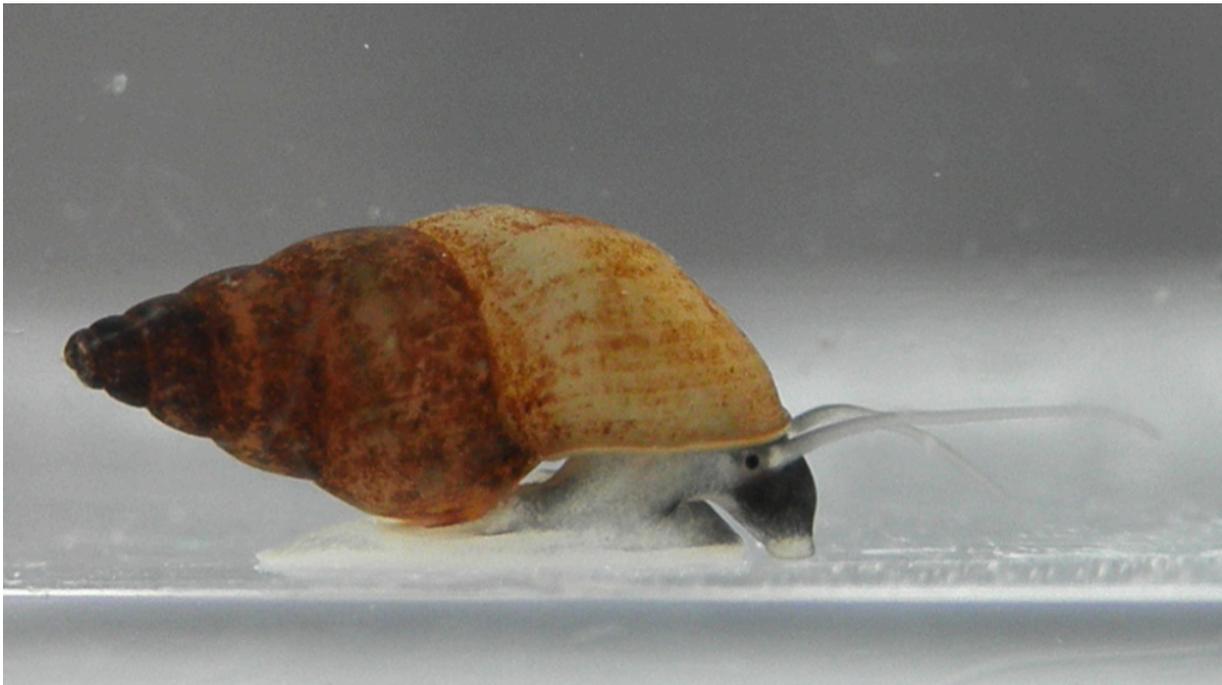


Figure 7.7 *Potamopyrgus antipodarum*, a small snail that lives in muddy shallows in New Zealand freshwaters)

The flatworm's life cycle begins inside of the snail, where the worm emerges from its egg. Infected snails are consumed by ducks. Once in the duck's intestine, adult worms have sex and produce eggs. Flatworm eggs are released, with duck feces, into the water, where they are ingested by snails and the cycle continues (Figure 7.8). Snails are harmed by this flatworm, largely because a symptom of infection is sterilization (the flatworm's scientific name, *Microphallus*, translates to "small penis").

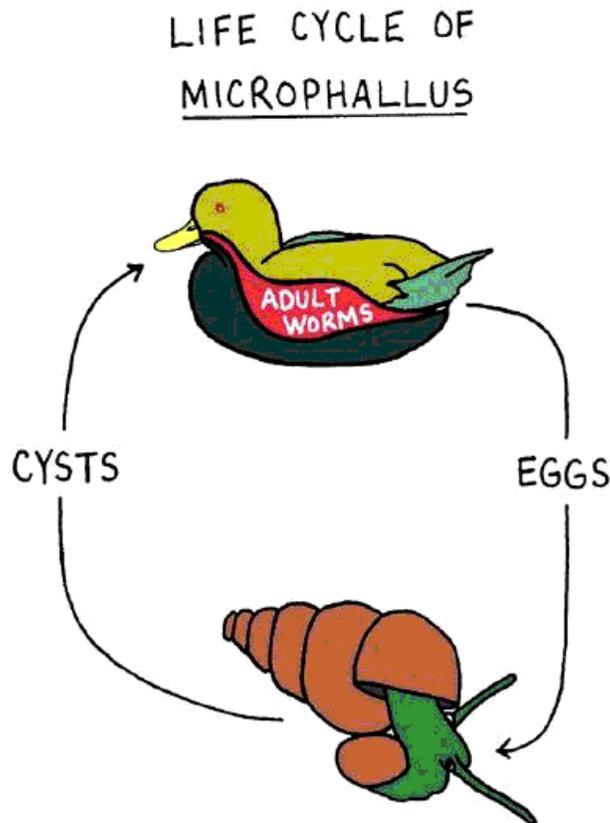


Figure 7.8 The life cycle of *Microphallus*

Observations of this system in two New Zealand lakes (Alexandrina and Kaniere) revealed that snails are more likely to be sexual (measured by frequency of males) in shallow waters, where ducks feed, than in deeper waters, where ducks do not feed (Figure 7.9).

1. By Michal Mañas - Mañas M. (2014). "Photo of the day (35): Potamopyrgus antipodarum". Blog about gastropods. <http://gastropods.wordpress.com>
<https://gastropods.files.wordpress.com/2014/10/potamopyrgus-antipodarum.png>, CC BY 4.0, <https://commons.wikimedia.org/w/index.php?curid=36715581>

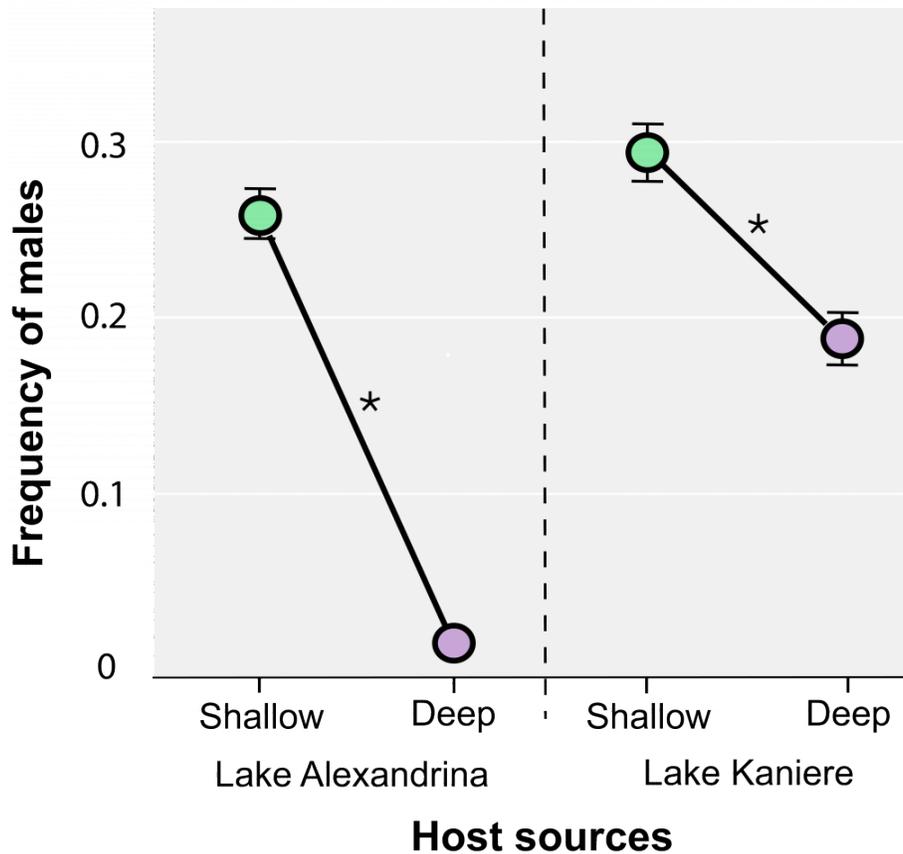


Figure 7.9 Infection rates in shallow-water and deep-water snails.

These results suggest that coevolutionary pressure is greater on the snails in the shallows, presumably because the feeding ducks effectively “close the circle” on the worm’s life cycle. Finally, higher infection rates in the shallows indicate that, in support of Prediction 1, above, sex is most beneficial where there is a high risk of infection.

Prediction 2: Pathogens are more likely to attack common phenotypes in a population, as opposed to the less-common counterparts

In the Mexican desert there are isolated pools inhabited by a species of minnow. Within these pools, populations of asexually reproducing individuals exist alongside sexually reproducing individuals. Fish in these ponds exhibit “black spot disease,” which is caused by a parasitic flatworm. Investigators have observed the frequency of sexual and asexual fish and the number of black spots in each type of fish in these ponds. Clonal fish are likely to have the most common phenotype in these ponds (as they are genetically identical to each other), while the sexually reproducing fish will have a wide variety of infrequent phenotypes. As the Red Queen predicts, the common type of fish (usually one of the clonal species) had the highest number of parasitic spots. In ponds where there was a genetically diverse, sexually reproducing population, the sexual fish had fewer spots.



Figure 7.10 Evening primrose blooms

Additional evidence comes from the evening primrose (figure 7.10), a flowering plant that—like the minnows, snails, and water fleas discussed above—exists in sexual and asexual forms. Evening primrose can be damaged by mildew from a pathogenic fungus. The plants produce an enzyme protein called *chitinase* to defend themselves against this fungus. A recent comparison indicated that the sexually reproducing primrose plants had greater variety in the gene that codes for chitinase than did the asexual plants. In addition, the overall amount of chitinase expressed was higher in the sexual plants than in the asexuals. Finally, the researchers found that the plants that were more resistant to mildew damage had higher fitness (they produced more fruit, and thus more offspring) in the presence of that pathogen. In evening primrose, greater diversity in a key gene renders an individual less susceptible to a pathogen, supporting the prediction that parasites are more likely to attack the most common phenotype in a population, and providing additional evidence for The Red Queen.

Know Your Pathogens

A pathogen is something that infects and causes a fitness cost in another organism. Pathogens come in a wide variety; some of them are not even considered living!

Prions – Prions are non-living infectious agents that are misfolded proteins.

Viruses – Whether you consider viruses alive or not depends on your definition of life. Viruses are protein-encased DNA or RNA entities that hijack a cell’s replication machinery to reproduce. Viral infections include influenza, HIV, HPV, and herpes.

Fungal pathogens – Fungi are responsible for a variety of infections including mildew, thrush, athlete’s foot and smut.

Bacteria – Bacteria are prokaryotic organisms that occur everywhere. There are more bacteria in and on you than there are cells in your body. Fortunately, the vast majority of bacteria are benign. However, some bacteria cause problems such as urinary-tract infections, some kinds of pneumonia, ear infections, pertussis (whooping cough), chlamydia, gonorrhea, and syphilis.

Protists – Protists are single-celled eukaryotes that cause diseases such as malaria and amoebic dysentery.

Animals – Common animal pathogens include lice, many types of worms, and parasitic wasps.

Prediction 3: In sexually reproducing populations, individuals choose mates that maximize diversity in their offspring

If there is a fitness advantage to diversity, parents can best maximize their offspring’s potential (and have more grand-offspring) with careful mate choice. There are numerous examples of organisms preferring mates that increase offspring diversity, and shunning mates that might do the opposite. Even many hermaphrodites, with both male and female sex organs, seek other hermaphrodites for copulation...even if they are capable of self-fertilization.

An excellent model for studying mate choice is Atlantic Salmon, an important commercial fish that lives its life in the ocean and returns to freshwaters to mate (or *spawn*). Sofia Consuegra and Carlos Garcia de Leaniz compared offspring diversity of salmon that were mated in a commercial fish hatchery (and unable to choose their mates) against that of salmon allowed to choose mates in the wild. The hatchery-spawned fish exhibited lower diversity than did the wild-spawned fish. Furthermore, hatchery-spawned fish displayed a greater number of roundworm parasites (*Anisakis*) than did their wild-spawned counterparts (figure 7.11). These results support the prediction that individuals will choose mates that maximize diversity in their offspring. Also, this work adds fuel to The Red Queen hypothesis by illustrating a potential benefit to Atlantic Salmon—namely, parasite avoidance.

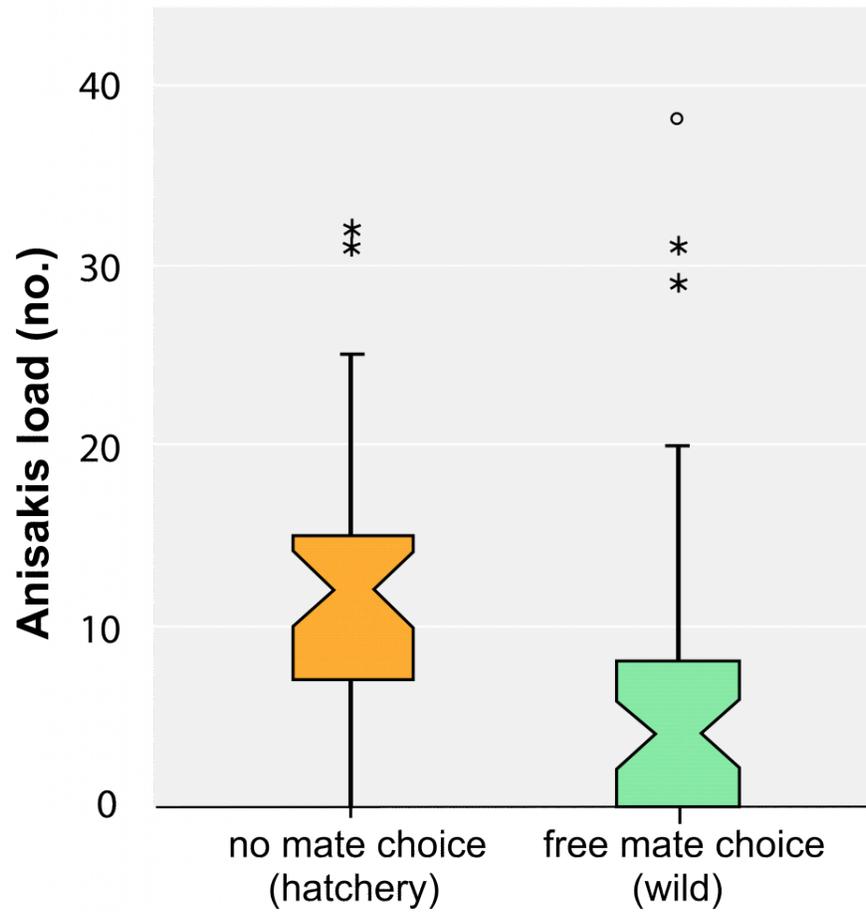


Figure 7.11 The offspring of fish mated in a hatchery have significantly more roundworm parasites than the offspring of fish mated in the wild.



Check Yourself



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<https://open.lib.umn.edu/evolutionbiology/?p=1340#h5p-34>

7.6 Major Histocompatibility Complex (MHC) Proteins

Scientists studying mate choice, offspring diversity, and pathogen avoidance often find themselves investigating a class of proteins called the **Major Histocompatibility Complex**, or MHC. MHC proteins are an important part of the vertebrate immune system. MHC proteins help a body recognize a potential pathogen and mount an immune response against the pathogen. The variety of MHC proteins an individual has determines the variety of antigens (foreign molecules) to which he or she can respond. Thus, greater diversity of MHC proteins should lead to a stronger immune system and greater avoidance of pathogens (See Box: [Know your pathogens](#)).

MHC proteins are the result of MHC genes, themselves an extremely diverse part of vertebrate genomes. For example, humans have over 100 MHC genes, spanning over three million bases on chromosome six. Over half of these genes are known to have an immune function. In addition, MHC genes are **polymorphic** (they have several possible alleles at each gene) and **codominant** (whereby each allele at each gene is expressed). Thus, parents can increase diversity in their offspring by choosing mates with different MHC alleles (figure 7.12). This preference for distinct MHC genotypes has been observed in mice, rats, sand lizards, and humans.

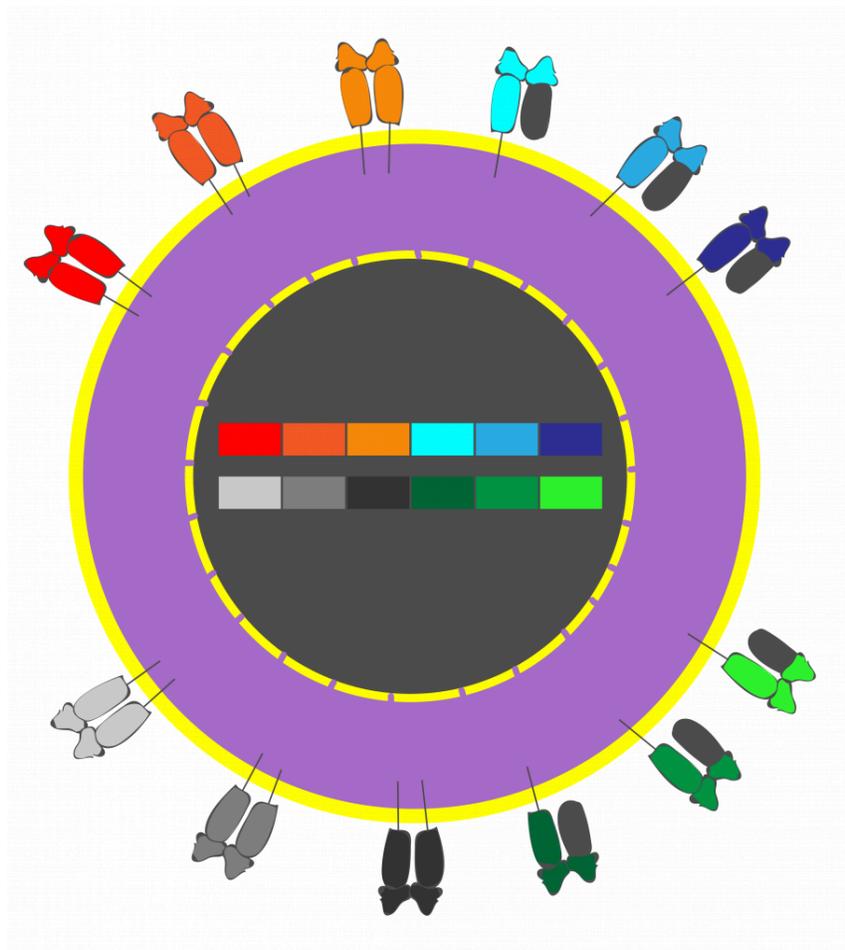


Figure 7.12 Choosing a mate that differs from you at his or her MHC genes will result in offspring with a greater diversity of MHC alleles. Each color represents a different MHC gene.

Paternity in sand lizards

Female sand lizards (figure 7.13) mate indiscriminately—that is, they seem to be willing to mate with any male who presents himself. One might expect with this type of mating strategy that the paternity of her offspring would be random among the males she has mated with. However, this is not the case. Rather, the male that differs the most from the female at MHC alleles has the highest chance of fathering baby sand lizards.



Figure 7.13 Male and female sand lizards

Some have speculated that however promiscuous the sand lizard is, her reproductive tract is more discriminating; in other words, selection for genetically dissimilar sperm occurs within the female sand lizard reproductive tract.

Even stranger, when a female sand lizard mates with two males, the second male adjusts the amount of sperm-bearing semen he ejaculates. Specifically, the second male appears to evaluate the degree of MHC-relatedness between the previous male and the female. If the first male is similar to the female, the second male has a larger ejaculate than if the first male is less similar to the female. This suggests that not only can the female sand lizard detect and discriminate based on MHC similarity, but a sand lizard male can detect the MHC type of the previous mate and adjust his ejaculate accordingly.

But how does he “know”? Key to many studies of MHC-related mate choices is a sense of smell. Apparently, many vertebrates (such as birds, fish, and mammals) can detect MHC complementarity using scent. Sand lizards are no exception; in fact, a characteristic of sand lizard mating is the male’s tendency to sniff the female’s genitalia prior to copulation. This behavior makes sense if the male can sniff out important information about rival males. After all, this information may ultimately increase his fitness.

7.7 Sniffing Out Complementarity in Humans

Evidence for human mating preferences is, by its very nature, complicated. Who we choose for mates is affected by factors such as appearance, grooming, education, class, dancing ability and preferred sports team. However there is evidence that human mate choice might be linked to MHC genes. A number of experiments have suggested that human females prefer the scent of males who are dissimilar to them in MHC (Box 4). Moreover, women rank a man's scent as the most important factor (more important than sight, sound and feel) in mate choice. In a fascinating twist, one study has shown that people who share MHC genotypes choose similar perfumes, suggesting that perfume preferences might serve to amplify your MHC display.

In another study, researchers analyzed the alleles of couples at several MHC genes and asked them questions regarding sexual attraction and sexual activities. They found that the higher the proportion of shared MHC alleles a couple have, the less sexually receptive the female is to her partner. Also, the more similar a female is to her partner, the more likely she is to be attracted to males other than her partner, especially while she is most fertile (that is, when she is ovulating). Moreover, the couples with a higher proportion of shared MHC alleles were more likely to have a greater number of sexual partners outside the relationship. These data suggest that humans, like non-human animals, use mate-choice strategies to select mates with different MHC alleles. In support of the above prediction, individuals choose mates that maximize diversity in their offspring.

To truly ascribe an evolutionary benefit to these MHC-related preferences in humans, there must be a fitness benefit to mate choices that rely on MHC complementarity. Alternatively, there may be a fitness *cost* to choosing a mate that is too similar at his or her MHC genes. Either way, this type of information is difficult to collect in human populations. However, a study of couples that suffered two or more miscarriages revealed that they were more likely to share MCH alleles than were couples without recurrent miscarriages.

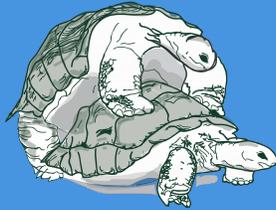
Obviously, we cannot ascribe all of human mate choice and reproductive success to MHC, nor would we attribute all of sand-lizard mating behavior to MHC alleles. However, there is evidence that humans show a preference for mates who have complementary MHC, they are more faithful to partners with different MHC, and there is a potential cost to poor mate choices. Collectively, these lines of evidence support The Red Queen hypothesis for the evolution and maintenance of sex.



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<https://open.lib.umn.edu/evolutionbiology/?p=1345#h5p-35>



Biology is Sexy

The Stinky T-shirt Experiments

Human mate choices are complicated! Recent work has attempted to isolate females' preferences for male scents. In a typical experiment, men are asked to refrain, from smoking, sex, spicy foods, and from using any scented products during the study period. In addition, the men are asked to sleep in the same t-shirt for two or three days. Female participants then smell the t-shirts that have been stored in sealed plastic bags and rate the attractiveness of the smell.

On average, women rate the smell of the t-shirts of men whose MHC genotype was distinct from their own as significantly more pleasant than those with a similar MHC genotype. Interestingly, this trend only held up for women not taking oral contraceptives. A recent phenomenon (Figure 7.14) is the interest in scent parties, in which people can rate the attractiveness of each other's t-shirts and then meet the person whose scent is interesting to them.

7.8 Wrapping Up: Sex and the Single Whiptail Lizard



The Single Whiptail Lizard

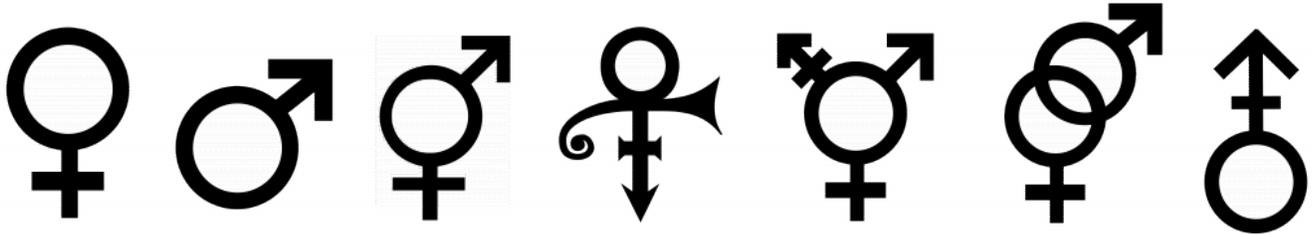
The asexual whiptail lizards discussed in the beginning of this chapter are a fascinating, recently evolved example of an organism that reproduces asexually. Comparisons of these lizards with their sexually reproducing relatives provide great experimental opportunities to explore the reasons for sex. In reality, there may not be only one reason sex exists; it may exist for several reasons, or sex may have evolved for one reason and been maintained for another reason. Regardless, the presence of bdelloid rotifers, whiptail lizards, evening primroses and other asexual organisms provides fascinating experimental fodder for those who are kept awake at night pondering the question, “Why sex?”



Figure 7.15 Whiptail lizard

As we conclude this chapter and prepare for in-class discussion, be sure to return to the chapter's [goals and objectives](#).

Chapter 8: Sex and Gender



[8.0 Introduction](#)

[8.1 Chapter objectives](#)

[8.2 Male versus female](#)

[8.3 What distinguishes the sexes?](#)

[8.4 Sex: It's about the gametes](#)

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[8.10 Understanding hermaphroditism](#)

[8.11 Wrapping up: What is gender?](#)

8.0 Introduction

Sex and Gender

The white-throated sparrow (*Zonotrichia albicollis*) is an easily overlooked little bird. It sings a simple song and is not too flashy; its main distinguishing features are the white and black or tan and black stripes on its head. However, scientists who have watched this bird's behavior and observed its chromosomes have learned fascinating information about this little bird that gives us insight into sex, gender, and the evolution of sex chromosomes.



Figure 8.1 The white throated sparrow: the white-striped morph.

1



Figure 8.2 *The white throated sparrow: the tan-striped morph.*

2

This bird has two color ‘morphs’ (or variations) of each sex. There are white-striped males and females and tan-striped males and females. There are consistent behavioral differences between the white- and tan-striped morphs. White-striped males and females sing a more melodious (i.e., prettier) song, are promiscuous (they mate with many partners), and are not too attentive to their nests. Tan-striped males and females are unremarkable singers, they are monogamous (they mate only with their partner), and fiercely defend their nests. To add a layer of complexity, the white-striped color morphs of both sexes mate almost exclusively with the tan-striped color morphs of the other sex. Do these birds have 4 sexes within one species? Or 2 sexes, but 4 genders?

3

2. Image: Melissa McMasters from Memphis, TN, United States https://upload.wikimedia.org/wikipedia/commons/2/27/White-throated_sparrow_%2825940489905%29.jpg

3. Image: Ltshears, https://upload.wikimedia.org/wikipedia/commons/7/73/BlueGill_002.jpg

Bluegill sunfish (*Lepomis macrochirus*) have two varieties of males and one variety of female. One male, the parental variety, builds and tends the nest and cares for the offspring, generally fathering most of the offspring in the nest. The other variety of male fertilizes some eggs in the nest through one of two alternative mating strategies: he can either sneak into the nest, fertilize and dart away (the “sneaker” tactic), or he may reside near the nest, but resemble the female (satellite males). These males will also fertilize some—typically a minority—of the eggs in the nest. Some scientists have referred to these two types of males—parental and alternative mating types—as two genders.

From these examples it is clear that some biological definitions of sex and gender are in order. These definitions are especially challenging because the terms *sex* and *gender* are often used interchangeably...by biologists and non-biologists alike.

Simply, sex is the distinction between male and female that has to do with biology and anatomy. Sex is often (but not always) binary (meaning there are two forms). The role individuals play in society (gender) is often influenced by sex, but not in a simple way, and this role is not binary. In this chapter we will explore the biology of sex and gender in humans and other organisms.

One artist’s rendition of the differences between sex and gender is here:



Figure 8.3 Bluegill in an aquarium.

The Genderbread Person v3.2 by its pronounced METROsexual.com

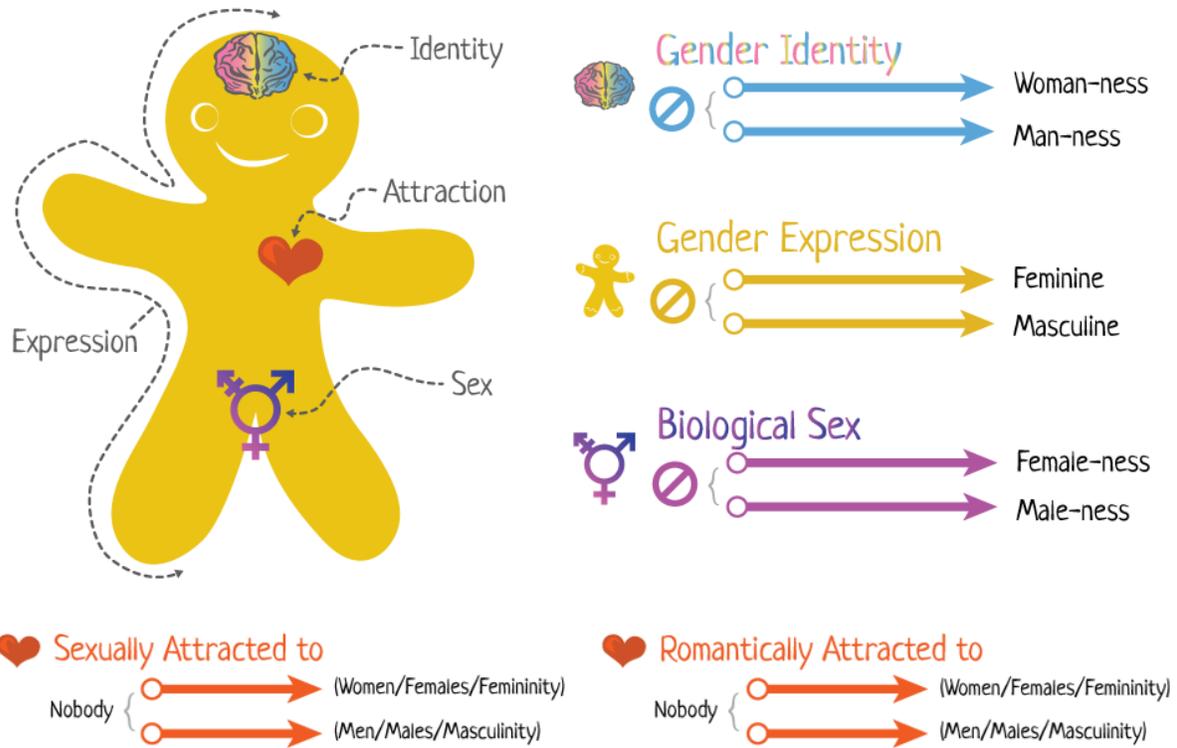


Figure 8.4 The Genderbread Person.

CAUTION: Please note that scientists' understanding of gender is in its infancy. Many of the things we'll say in this chapter will seem ridiculous in ten years. Our goal is to establish a shared vocabulary around sex and gender, and to give you a snapshot of this rapidly growing body of knowledge.

8.1 Chapter Objectives



Learning Objectives

In the following discussion of sex and gender, our goals are for you to realize that sex and gender are related, but distinct; and to understand that sex and gender are manifest in many ways, in an array of living organisms. By the end of your reading and our in-class discussion, you will be able to:

1. Define the following terms:
 - **sex**
 - **hermaphroditism**
 - **dioecy**
 - **gender**
2. Describe differences between sex and gender.
3. Cite examples of the non-binary nature of gender in humans and non-human animals.
4. List factors that determine sex in a variety of organisms.
5. Describe the development of sex organs in humans.
6. Explain some evolutionary pros and cons of hermaphroditism and why it exists in some plants and animals.

8.2 Male versus Female

Check out this lovely pair of superb fairywrens (*Malurus cyaneus*)



Figure 8.5 Pair of superb fairywrens (*Malurus cyaneus*)

1

and this mating pair of marsh fritillaries (*Euphydryas aurinia*),

1. https://commons.wikimedia.org/wiki/File:Male_and_female_superb_fairy_wren.jpg



Figure 8.6 Pair of mating marsh fritillaries (*Euphydryas aurinia*)

2

and these Parson's chameleons (*Calumma parsonii*)



Figure 8.7 Man holding up two Parson's chameleons (*Calumma parsonii*). Image: Charlesjsharp.

or these two grass snakes (*Natrix natrix*)



Figure 8.8 Two grass snakes (*Natrix natrix*) coiling around each other.

Each pair includes one male and one female. If you were asked to distinguish the male from the female in each of these examples, how would you do it? What sort of information would you need? Basically, how do we distinguish males from females?



Points to Ponder

Is there one unifying characteristic that sets males and females apart, in all organisms—plants, animals, protists, etc.—that have male and female individuals?

8.3 What Distinguishes the Sexes?

Is it the penis?

Your initial thought for what distinguishes male from female may be the presence or absence of a penis. And the presence of a penis would certainly help you distinguish between a male and female wolf, killer whale, or guinea pig. Yet, in the examples on the previous page, only the male snake, the male chameleon, and the male butterfly have a penis. The superb fairy wren males don't have penises, neither do male holly trees, goldfish, or seahorses. Plants, songbirds, and fish don't have penises, and seahorse and hyena *females* have structures that resemble a penis.

Is it the chromosomes?

You may have thought to look for chromosomes (long stretches of DNA) that can distinguish male from female. In a species in which male and female look similar, and in which males lack a penis, chromosomes may be helpful. However, not all organisms have sex determined entirely by genes on specific chromosomes. In the previous examples, the birds, butterflies, and snakes have chromosomes that can indicate sex, but the chameleons do not!

Is it body size?

Many organisms have a size difference between the male and female. The average male gorilla is about twice the size of the average female. Male northern elephant seals are three times the size of females.



Figure 8.9 Northern Elephant Seal

1

However, this pattern of size difference does not hold up for all species. The Oval St. Andrews Cross spider male is 1/10th the size of the female.

1. original image by Jan Roletto, uploaded 18:58, Feb 26, 2004 - de:Wikipedia by de>User:Baldhur, edited by Matthew Field - National Oceanic and Atmospheric Administration (<http://www.noaa.gov>), Public Domain, https://commons.wikimedia.org/wiki/Phocidae#/media/File:See_elefanten_edit.jpg



Figure 8.10 Oval St. Andrew's cross spider male (top) and female (bottom)

2

The tree frog female is also significantly larger than the male.



Figure 8.11 Mating tree frogs (*Polypedates leucomystax*) from Indonesia. The male is on top, the female is on the bottom.

3

For tarsiers and albatrosses there is no size difference between males and females.



Figure 8.12 Philippine Tarsier (*Carlito syrichta*)

4



Figure 8.13 Waved Albatros (*Phoebastria irrorata*) courting ritual.

5

8.4 Sex: It's About the Gametes

Attempting to find a distinguishing characteristic that can be universally applied across sexually reproducing organisms is challenging! But, biologically speaking, the distinction is quite simple—sexes can be distinguished, across the board, by **gamete** (or sex cell) size.

Some sexually reproducing organisms are **isogamous**, meaning they produce the same size gametes, and some are **anisogamous**, meaning they produce different size gametes. Isogamous organisms include species of fungi, algae and protozoa; some of these organisms have between 2 and thousands of “mating types” that can be thought of as sexes. But they aren't male and female.

Sexually reproducing anisogamous species generally have only two sexes. So how do we distinguish the males from the females? Scientists have created a definition of **female** that includes all the individuals that produce large gametes (eggs), those that produce small gametes (sperm) are **male**. Biologically, this large gamete/small gamete distinction between males and females is the only one that holds up well across many sexually reproducing species. Note: in humans, there are egg-producers that do not identify as female and sperm-producers that do not identify as male.

While sexually reproducing plants and animals generally have only two sexes, our ability to define individuals biologically as one sex or the other is complicated by our growing understanding of the sex varieties that exist. You can read here about some fascinating examples of our changing understanding of sex (<https://www.nature.com/news/sex-redefined-1.16943>).



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<https://open.lib.umn.edu/evolutionbiology/?p=1365#h5p-36>

8.5 Mechanisms of Sex Determination

What determines whether an embryo develops into a male or female? That depends on the organism. For many organisms the answer lies in the organism's DNA. For others it depends on environmental cues, and for some there is an interaction between environmental cues and genetic information.

Genetic Sex Determination

In humans and other mammals sex is determined genetically. Mammals have sex chromosomes—X and Y. On the mammalian Y chromosome is a gene called *Sry* that triggers differentiation into a male, with male reproductive physiology and male secondary (non-reproductive) characteristics. An embryo that has two X chromosomes and doesn't have an *Sry* gene will usually differentiate into a female, while an embryo that has an X and a Y chromosome will usually differentiate into a male. Other animals and most plants also have genetic sex determination.

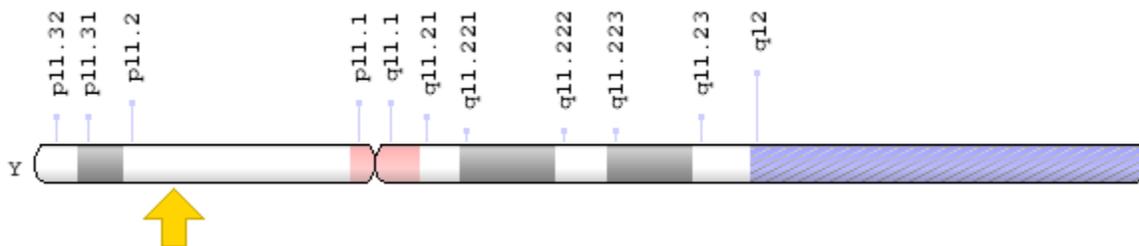


Figure 8.14 Y chromosome. Yellow arrow indicates the location of the *Sry* gene.

1

In birds and some reptiles those that have two different sex chromosomes (called WZ) are female, while those with two copies of the same sex chromosome (ZZ) are male. In other organisms such as wasps, the males arise from unfertilized eggs and therefore only have one copy of genetic information (they are haploid) while females arise from fertilized eggs and have two copies of genetic information (they are diploid).

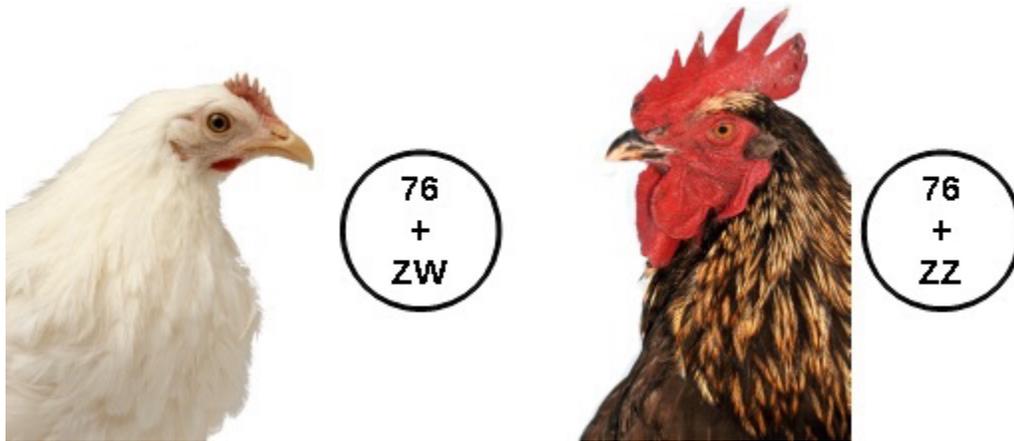


Figure 8.15 Illustration of avian chromosomal sex determination

2

Environmental Sex Determination

The sex of some other animals is determined by environmental factors. For example, the sex of many species of turtle, alligators and some fish is determined by the temperature at which the eggs are incubated.



Figure 8.16 Leatherback turtles hatching

For other species, sex is determined by social influences in the environment. For example, the green spoon worm (*Bonellia viridis*) is a marine worm that is emerald green in color.



Figure 8.17 *Bonellia viridis*, a marine worm

The worm is not sexually differentiated as a larva. If the larva does not encounter a chemical signal emitted by a female it will differentiate into a female, growing to about 8 cm in length. If the larva encounters a chemical the female secretes, it will differentiate into a male, remaining 1-3 mm in length. Then, the male will be sucked into the feeding tube of a female and spend its life with other males in the reproductive tract of the female.

Some species of fish are **sequential hermaphrodites**, meaning they are one sex during one portion of their life and then—depending on developmental or environmental cues—they become the other sex. For example, clownfish live in small groups with a breeding male and female and up to four non-breeding males. The female is the largest and the dominant fish in the group. If the female leaves the group (often by dying), the breeding male will differentiate into a female and one of the previously non-breeding males will become the new breeding male.

3. https://commons.wikimedia.org/wiki/File:Leatherback_Turtle_eggs_hatching_at_Eagle_Beach,_Aruba.jpg

4. Image: Sylvain Ledoyen (Own work) [GFDL (<http://www.gnu.org/copyleft/fdl.html>), CC-BY-SA-3.0]



Figure 8.18 Image of a Clownfish

5

Another environmental factor that can influence sex is the presence or absence of particular parasites. *Wolbachia* are a group of bacteria that live symbiotically in the cells of invertebrates. These bacteria have been shown to change genetically male insects into functional (egg-producing) females.



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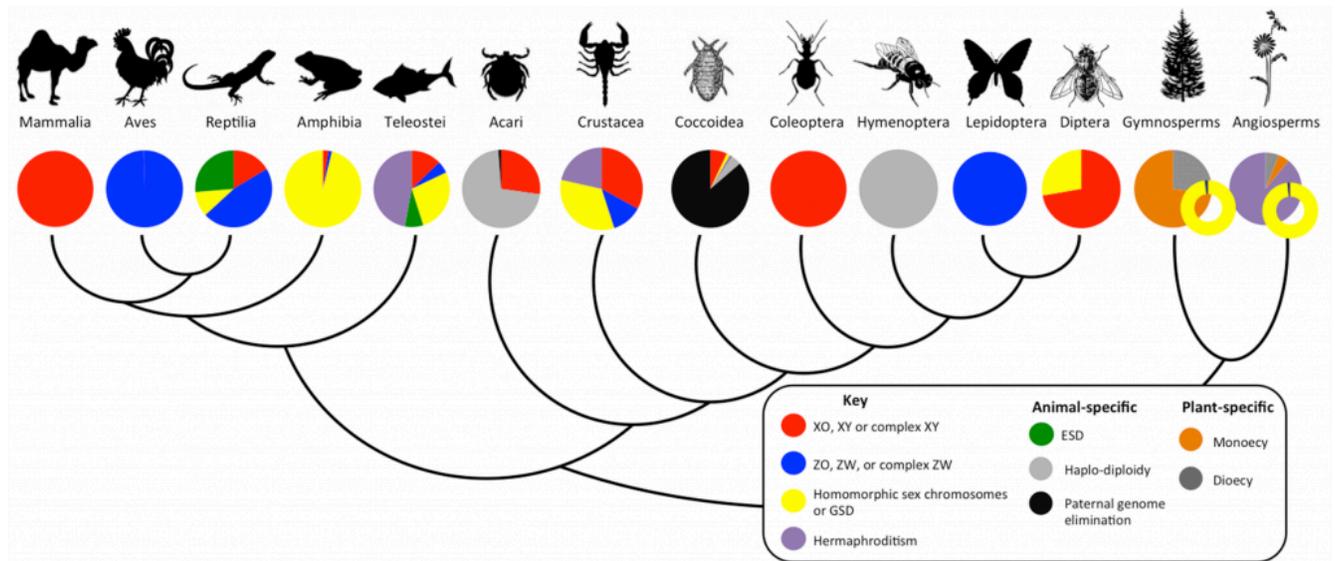


Figure 8.19 A phylogenetic tree

6

Sex determination mechanisms across various classes of organisms. XO, XY, ZO and ZW indicate chromosomal sex determination. GSD stands for genetic sex determination. ESD is environmental sex determination. Haplo-diploidy means that males are haploid (have only one copy of genetic information; resulting from an unfertilized egg) and females are diploid (have two copies of genetic information; resulting from a fertilized egg).



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8.6 Human Sex Development

When a sperm and egg join to form a zygote, whether that sperm carried an X chromosome or a Y chromosome determines the eventual sex of the developing fetus. However, for the first seven weeks of development a male and female fetus are indistinguishable.

Shortly after the seventh week, the presence of a gene called “*Sry*” on the Y chromosome directs the development of the testes. These new testes then begin to produce testosterone (a hormone associated with male development) and another hormone called Müllerian inhibiting hormone; together, these two hormones cause the disintegration of the female internal organs and further the development of male internal and external anatomy. In the absence of the *Sry* gene and the Müllerian inhibiting hormones, the female external and internal anatomy will form.

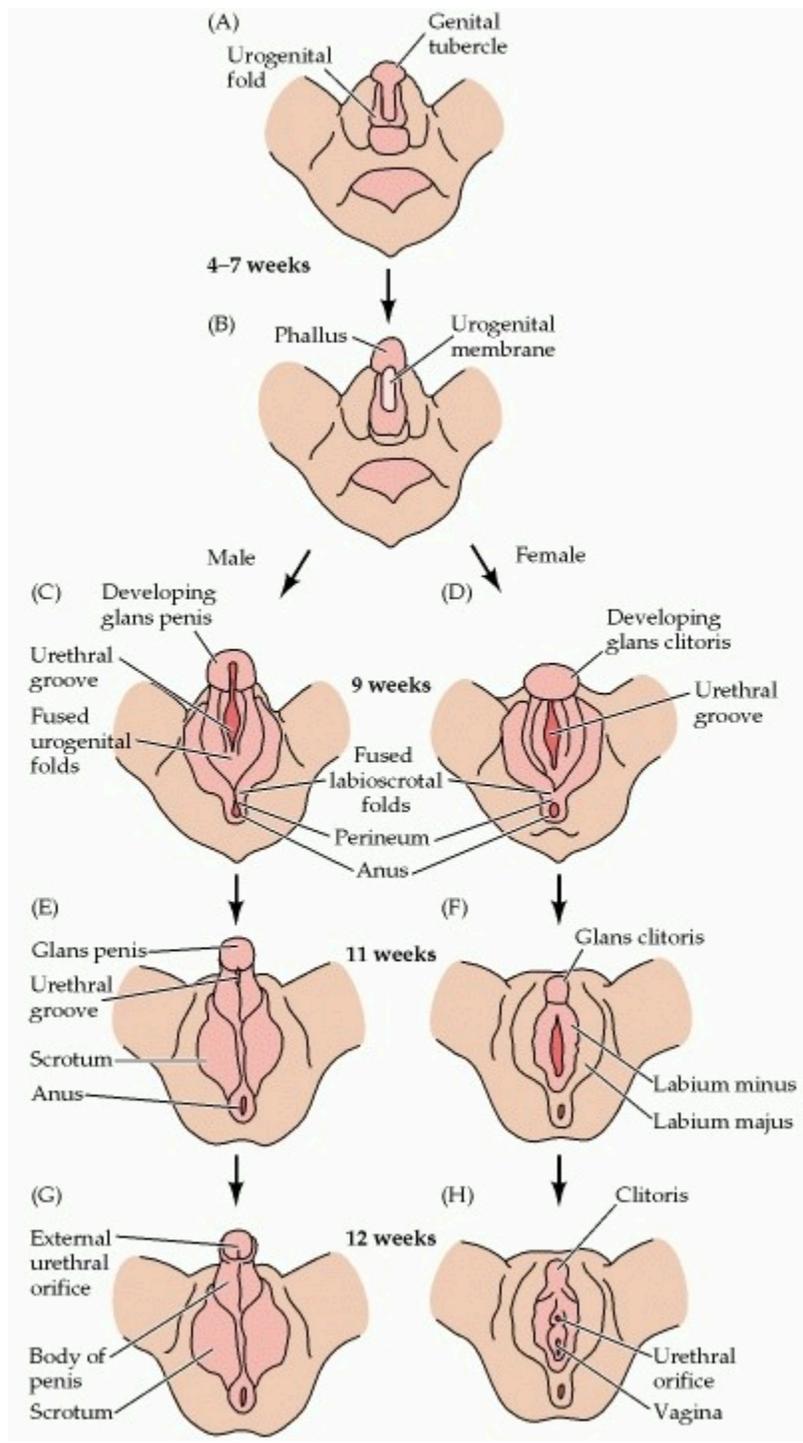


Figure 8.20 External genital development of a human fetus. Note the undifferentiated genitals at 7 weeks, and the increasing differentiation as development continues.

Read More

This [interactive website](#) will walk you through the steps of sex differentiation in males and females



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<https://open.lib.umn.edu/evolutionbiology/?p=1374#h5p-39>



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<https://open.lib.umn.edu/evolutionbiology/?p=1374#h5p-40>

8.7 Variations in Sex Development

There are genetic and environmental factors that can interfere with the usual development of reproductive organs and secondary sex characteristics in humans and other animals. Collectively, conditions of sex development that do not conform to typical male/female development are called **intersex** conditions.

Genetic factors

In humans there are medical conditions that lead to intersex development. Some of these conditions have known genetic causes, others are not known. You can read about various intersex conditions here – <http://www.pbs.org/wgbh/nova/body/intersex-spectrum.html>

Endocrine disruption

Exposure to hormones, hormonal mimics, or other compounds that can interfere with usual hormone signaling can alter reproductive anatomy and function. For example, tributyl tin is a substance that was added to marine paints. This compound prevented barnacles and other organisms from attaching to the bottoms of boats and other marine structures. Unfortunately, the leaching of this compound into marine ecosystems induces the growth of penises on female snails, interfering with female snail fertility. The use of tributyl tin in marine paints has been banned by international agreement due to its toxic effects on marine life and the potential for human exposure through seafood.

Endocrine disruptors are compounds that interfere with hormonal signalling. There are suspected endocrine disruptors in pesticides, fragrances, plastics, flame retardants, and many other products. Endocrine disruptors have been implicated in the rise in breast cancer, testicular cancer, genital abnormalities, and in the possible decline in the sperm count (the amount of sperm per ejaculate) in men today.

Endocrine disruptors are also suspects in the mystery of some unusual wildlife effects. For example, in the United States, large percentages of male fish in some species (especially large- and small-mouth bass) have been found to have eggs developing in their testicular tissues. These fish species are not sequential hermaphrodites (i.e., they are not known to change sexes). The cause of this phenomenon in the wild is unknown, however environmental exposure to estrogen mimics in the environment is a hypothesized cause.

Laboratory exposure to endocrine disruptors can lead to reproductive abnormalities. Laboratory studies have focused on suspected endocrine disruptors such as ethinyl estradiol (a substance in birth control pills that ends up in waste water), bisphenylA (a compound in some plastics), triclosan (an antimicrobial agent added to soaps, toothpaste, clothing, and other products that advertise themselves as antimicrobial), and atrazine (a weed-killer that is widely used on corn fields in the Midwestern United States; see box below). These studies have demonstrated that these compounds can cause feminization of reproductive organs, changes in behavior, and in some cases, full sex reversal in laboratory exposures.

Our understanding of what compounds could be affecting hormonal signaling in wildlife and humans is growing,

but with the tens of thousands of new synthetic chemicals being used today, it is challenging to know which chemicals to prioritize for study.

Read More

Here is a table compiled by the European Commission on the Environment of 553 chemicals that are suspected endocrine disruptors. http://ec.europa.eu/environment/archives/docum/pdf/bkh_annex_01.pdf

You can read about endocrine disruption from the perspective of the:

- U.S. Environmental Protection Agency, <https://www.epa.gov/endocrine-disruption/what-endocrine-disruption>
- World Health Organization, <http://www.who.int/ceh/risks/cehemerging2/en/>
- National Institute of Environmental health safety. <https://www.niehs.nih.gov/health/topics/agents/endocrine/>

8.8 Atrazine and Tyrone Hayes

Atrazine and Tyrone Hayes

Dr. Tyrone Hayes is a professor at the University of California at Berkeley who specializes in frog development. He has been studying the effects of the weed-killer Atrazine on frogs for 20 years. He started out this work as a paid consultant for the company that manufactures atrazine (now Syngenta). In his consulting work, he found some unexpected results, the atrazine-exposed male frogs had ovarian tissues in their testes and reduced larynx (voice box) size. The concentrations at which Dr. Hayes observed those effects are well below the concentrations that atrazine can be found in surface and well water in places where atrazine is used. Dr. Hayes was not allowed to publish the results of his studies that were funded by the atrazine manufacturer, so he resigned his position at the company, and redid the studies. The studies were published in 2002 and a feud between Dr. Hayes and Syngenta has been ongoing. In 2010 Dr. Hayes published another study showing complete sex reversal (genetically male frogs becoming fertile females) due to atrazine exposure.

Other studies by independent researchers on other organisms have shown endocrine disrupting effects of atrazine as well. However Dr. Hayes's particular results remain controversial. Despite attempts to ban it, atrazine remains one of the most commonly used pesticides. This map from 2014 shows the U.S. usage of atrazine.

1



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1. USGS. https://water.usgs.gov/nawqa/pnsp/usage/maps/show_map.php?year=2014&map=ATRAZINE&hilo=L

8.9 What about hermaphrodites – and why aren't they more common?

Some organisms contain male and female **gonads** (sperm or egg producing organs), and produce both sperm and eggs (see figure X for examples). These individuals are called **hermaphrodites** (in plants they are often called **monoecious**). In previous pages, you learned that some fish are sequential hermaphrodites (they change from one sex to the other). Other organisms are **simultaneous hermaphrodites**, meaning they can produce both sperm and eggs at the same time. Examples of such organisms include most plants, some types of snails, worms, slugs, and some fish. Can you imagine any advantages to one individual having the ability to produce both eggs and sperm?

Note: Hermaphroditism does not exist in humans. While some humans have intersex conditions, they are not hermaphrodites.



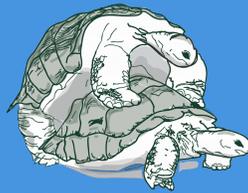
Points to Ponder

How might it be beneficial to an individual to produce both eggs and sperm? Similarly, can you imagine any costs associated with hermaphroditism?

8.10 Understanding Hermaphroditism

You can probably imagine a few benefits of **hermaphroditism** (having the ability to produce both eggs and sperm). For one, any individual could mate with any other individual in the population (this is **outcrossing**), expanding the pool of potential mates. Also, if a hermaphrodite could not find *any* mate, it could fertilize its own eggs (this is **selfing**). Furthermore, selfing hermaphrodites save a lot of energy that would otherwise be spent in seeking and acquiring a mate.

So why aren't more species hermaphrodites? There are several proposed explanations about why hermaphroditism is not more common. One is that male and female mating strategies are distinct (see chapter on sexual selection). It could be that there is an evolutionary advantage to fully pursuing female sex strategies or male sex strategies, and that compromising between the two is disadvantageous. Basically, this is a "specialist versus generalist" argument, in which the specialists are **dioecious** (meaning they only have one sex per individual) and the generalists are hermaphrodites. Indeed, some of the odd behavioral adaptations of hermaphrodites (see penis fencing flatworms below) are evidence of the dueling selective pressures in hermaphrodites.



Biology is Sexy

Penis Fencing Flatworms



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<https://open.lib.umn.edu/evolutionbiology/?p=1382#oembed-1>

The above video shows two ocean-dwelling hermaphroditic flatworms fighting to inseminate the other by stabbing the other with their penises (called penis fencing). In the end the one who gets stabbed has to wander off to find food – while the other gets to go in search of another mate.



Figure 8.23

inseminated.

¹Why do these worms have this behavior? Researchers who have explored this behavior suggest that the costs and benefits of being the sperm recipient, versus the sperm donor, affect the way penis fencing flatworms engage in these battles. In short, the sperm recipient (whose eggs are fertilized) has higher energetic costs associated with the reproductive event because they are providing the nutrient-rich egg. So when all else is equal, there is an advantage to being the one to inseminate, while avoiding being inseminated. However, one group of researchers from Florida researching a hermaphroditic sea slug found that when one slug was food deprived, the benefit to the food-deprived slug of being the inseminator was even greater, and in that case, the well-fed slug was best off being

Another suggestion for the evolution of separate sexes has to do with organelles (or small compartments within a cell) that contain DNA (mitochondria in plants and animals and chloroplasts in plants). Organelles are inherited only through the egg. Thus, all of your mitochondria are inherited from your mother (sperm do not contribute mitochondria to the embryo). For a gene in the mitochondrion or chloroplast, sperm production is an evolutionary dead end! Therefore genes have evolved in the mitochondria and chloroplasts that cause an organism to focus its reproductive efforts on egg production.

Imagine a landscape of organisms in which there has been this type of pressure to produce only eggs (no sperm). In this environment, if there were a lone variety that focused its reproductive efforts on sperm production, that organism would do very well! Negative frequency-dependent selection is when there is an advantage to being a rare type in a population. Thus, if there is an excess of egg-producing organisms, organisms that disproportionately produce sperm will be at an advantage. One of the hypotheses for the evolution of dioecy from hermaphroditism involves negative frequency-dependent selection and the evolutionary advantage of specializing in a rare gamete type in this situation.



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<https://open.lib.umn.edu/evolutionbiology/?p=1382#h5p-43>

Below are examples of simultaneous hermaphrodites.



a)



b)



c)



d)

Figure 8.24 a) Tiger lily; b) Earthworms mating; c) Hamlet Fish; and d) Banana slug.

2345

2. <https://commons.wikimedia.org/wiki/Lilium#/media/File:LiliumBulbiferumCroceumBologna.jpg>
3. https://upload.wikimedia.org/wikipedia/commons/f/f6/Mating_earthworms.jpg
4. https://commons.wikimedia.org/wiki/File:Butter_hamlet_fish.jpg
5. https://upload.wikimedia.org/wikipedia/commons/thumb/5/51/Banana_slug_in_the_Hoh_Rainforest.jpg/800px-Banana_slug_in_the_Hoh_Rainforest.jpg

8.11 Wrapping Up: What is Gender?



The concept of gender

Above we defined the sexes by the size of gamete produced. The terms sex and gender are often used interchangeably. However, for our discussions we will distinguish between the terms. The American Psychological Association states, “**Gender** refers to the socially constructed roles, behaviors, activities, and attributes that a given society considers appropriate for boys and men or girls and women. These influence the ways that people act, interact, and feel about themselves.” In short, gender has to do with the roles one plays in society and is often influenced, but not necessarily determined, by sex. Gender roles vary from culture to culture in humans. For a brief description of some of the varied gender roles across cultures, see Table 1. Societal roles in non-human animals are harder to interpret, however there is also evidence of more than two gender roles in a number of non-human animals (see discussion of white-throated sparrows and bluegill in the beginning of this chapter).

In recent years there has been growing popular awareness that people do not all identify as the gender commonly associated with the sex they were assigned at birth or conform to the concept of gender as binary (they do not strictly identify as man or woman). However, historic and linguistic evidence indicates that non-binary gender is *not* a new phenomenon and can be observed across human cultures in both modern and historic times (see Table 1). It is worth noting that while we learned earlier that there are genetic factors that can cause a person to have intersex characteristics, most people who do not fit in a gender binary or identify as the opposite gender from the sex they were assigned at birth, do not have any identifiable genetic condition associated with sex.

Read More

The link has some excellent tips on how to be supportive to someone who is gender non-binary or transgender.
<https://www.glaad.org/transgender/allies>

The white-throated sparrow and the bluegill and the great diversity of sex and gender presentations in humans and other sexually reproducing organisms illustrate that sex and gender are complicated. Our idea of sex as binary is imperfect at best. The idea that gender is beyond binary is supported by biology, sociology, and history.

Studies of and discussions of sex and gender can be controversial. Any discussion of sex and gender is almost certain to make at least some participants uncomfortable. However, there are fascinating aspects of the biology surrounding sex and the sociology surrounding gender that make exploration into these topics worthwhile. Our understanding of sex determination is growing rapidly, and our understanding of the relationship between sex and gender is in its infancy.

Location	Name	Description	Notes
Brazil	viado/travestis	male who dresses and presents in a feminine manner	
Europe	sworn virgins of the Balkans	female who participates masculine roles	sworn virgins stayed with their family and inherited family land (often sworn virgins were in families with no male heirs). Also worthy of note: there are a number of saints (St. Eugenia, St. Pelagius, St. Wilgefortis, St. Joan) who had varying degrees of assuming a masculine role in society
Hawaii and Tahiti	māhū	male who dresses and presents in a feminine manner	
India	hijra	born as male, ritually established as third gender. Often dress in feminine clothing. Intersex individuals were often included in this category.	Hijras play an important role in Hindu religious ceremonies
India	sadhin	female who wears men's clothes and cuts hair short	typically celibate
North America – Navajo	nádleeh	male or female who participates in society in roles typical of the other sex	
North America – Mohave	alyha	male who participates in society in feminine roles	At puberty there is an initiation ceremony during which the child chooses to dance as a female or male, if the child chose the female dance, gender was changed. After this time the alyha's genitals were referred to using terms for female genitals
North America – Mohaves	hwame	female who participates in society in masculine roles	
North America – Cheyenne	hetaneman	female who participates in society in masculine roles	
North America – Cocopá	warrhameh	female who participates in society in masculine roles	
North America – Maidu	suku	female who participates in society in masculine roles	
Philippines	bayot/bantut/bakla	male who adopts feminine roles and presentation	
Samoa	fa'afafine	male who adopts feminine roles and presentation	considered very important for family cohesion
Thailand	kathoey	male or female who participates in society in roles typical of the other sex. Intersex individuals are also included in this category	In recent years females who present as males are now typically referred to as "tom" from "tomboy"
Tonga	fakaletī	male who participates in society in feminine roles and presentation	
Tuva	pinapinaaine	male who adopts feminine roles and presentation	

Figure 8.25

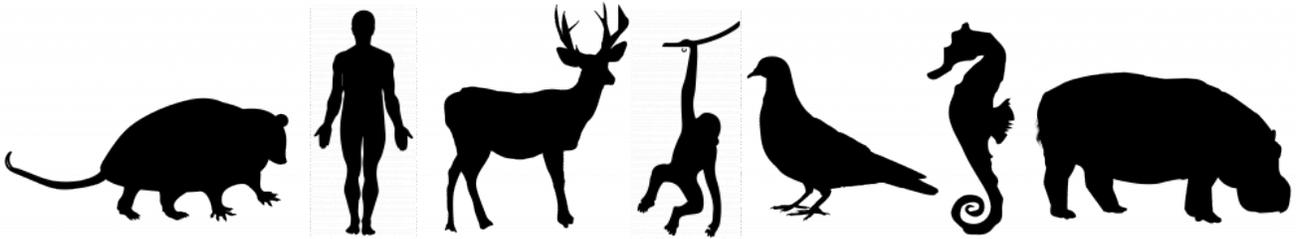
Read More

Most of the information in this table was from the Book Gender Diversity by Serena Nanda.

Note: some (but not all) of the gender roles here are also associated with sexual orientation. For brevity, sexual orientation was not included here. See Nanda's text for more detail.

As we conclude this chapter and prepare for in-class discussion, be sure to return to the chapter's [goals and objectives](#).

Chapter 9: Sex Ratios



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[9.1 Chapter objectives](#)

[9.2 Characterizing populations](#)

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[9.7 Testing Trivers-Willard in opossums](#)

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[9.10 Testing Trivers-Willard in humans](#)

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[9.12 Male bias and extreme sex ratios](#)

[9.13 Wrapping Up: Understanding human sex ratios](#)

9.0 Introduction

Understanding sex ratios

In Australia, there are approximately 100 males for every 100 females. In Bahrain country, there are 153 males for every 100 females (1.53 M:F). And in El Salvador, there are 92 males for every 100 females (0.92). A quick look at the map clarifies that, in human populations, sex ratios vary globally. Do you note any patterns in the image below?



Check Yourself



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Interesting sex ratios are not unique to humans, and in some non-human organisms differential sex allocation leads to dramatically skewed sex ratios. For example, in some population of the bluestreak cleaner wrasse, there is only one male for every 6-8 females.



Figure 9.1 Bluestreak cleaner wrasse

In jacanas, males may outnumber females by more than 2:1.



Figure 9.2 *Irediparra gallinacea*: Kununurra Lake, Kununurra, Western Australia.

How can we understand some of these wildly different sex ratios, in both human and non-human populations?

9.1 Chapter Objectives



Learning Objectives

Our objective for this chapter is to present some patterns related to observed male:female sex ratios, in both human and non-human populations. We'll also discuss several factors affecting sex ratios, and hypotheses for understanding skewed sex-allocation phenomena. By the end of your reading and our in-class discussion, you will be able to:

1. Define the following terms:
 - **operational sex ratio**
 - **sex allocation**
2. List the three key factors that influence population sex ratios
3. Describe the Trivers-Willard hypothesis for sex allocation, and cite evidence in support of this hypothesis
4. Explain alternative hypotheses for explaining skewed sex ratios in organisms with chromosomal (genetic) sex determination

9.2 Characterizing populations

Populations—groups of individuals of the same species—are defined by a suite of emergent characteristics, such as average age of first reproduction, average life span, and average number of offspring. These characteristics may vary from population to population; for example, the average age of first reproduction in humans varies from 19 in Mozambique to 31 in South Korea. However, these characteristics tend to vary more between species than they do within species. As an example, human birth rates are nothing like that of the Pacific salmon, which can produce over 10,000 offspring per individual.

In this chapter, we'll focus on another, often-puzzling aspect of populations—sex ratios. We'll talk about organisms in which the sex-ratios are typically 1:1, with relatively equal numbers of males and females (e.g., humans), and populations that are either consistently skewed (e.g., a 1:5 male:female sex ratio is typical of the American alligator; meaning for every male there are 5 females, notice that this means that the proportion of males is 1 male out of 6 total alligators or $1/6$ or 16.7%), or characterized by variability (e.g., the sex-ratio of peafowl populations varies quite a bit). Before we move on, let's make sure you are interpreting ratios correctly.



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9.3 An Introduction to Operational Sex Ratios

Operational sex ratio (OSR) is the ratio of sexually receptive males to sexually receptive females, (typically expressed as male:female ratio). The term “operational” refers to the idea that it is the sex ratio of sexually active, or sexually competing, individuals, that matters to much of our discussion. For example, a population’s OSR can affect the intensity of competition between same-sex individuals for access to mates.

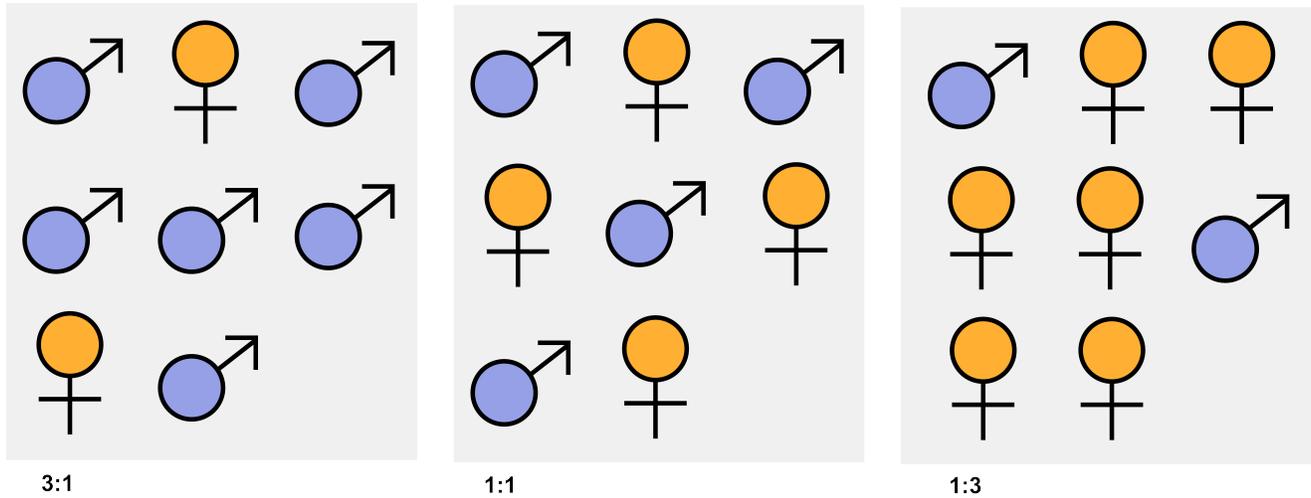


Figure 9.3 Various sex ratios



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9.4 Variety in Operational Sex Ratios

Ultimately, sex ratios are the result of:

- **sex allocation** (or, sex ratios at birth), which in humans favor males,
- differential mortality (in humans, females generally have less mortality), and are therefore favored) and
- differential migration (e.g., are males or females more likely to leave their home communities at maturity? This varies by species and populations).

Sex allocation: Sex allocation, or sex ratios at birth, can depend on many factors. In organisms that have chromosomally determined sex (i.e., biological sex is predicated on chromosomes), sex ratios are a function of meiosis, conception, and development. There is some conventional wisdom that in organisms with chromosomally determined sex, sex-ratios should be around 1:1 Male:Female. Why? Because, due to the genetic “lottery” of meiosis, half of the offspring conceived should be males, and half of the offspring conceived should be females. Let’s revisit meiosis, using the example of hippos, for just a bit:



Figure 9.3 Pygmy Hippopotamus at Zoo Lagos, Portugal

1



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<https://open.lib.umn.edu/evolutionbiology/?p=3317#h5p-90>

1. [https://en.wikipedia.org/wiki/Pygmy_hippopotamus#/media/File:Hexaprotodon_liberiensis_Lagos_Zoo_Portugal_\(3\).jpg](https://en.wikipedia.org/wiki/Pygmy_hippopotamus#/media/File:Hexaprotodon_liberiensis_Lagos_Zoo_Portugal_(3).jpg)



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<https://open.lib.umn.edu/evolutionbiology/?p=3317#h5p-91>

So, if half of the offspring conceived are male, and if gestation proceeds the same for male and female fetuses, then we expect the sex ratio at birth for these hippos to be 1:1. However, this is not always the case. We'll discuss interesting findings on hippo sex allocation soon. Keep reading!

Recall from other discussions (and summarized [here](#)) that many organisms do not experience chromosomal sex-determination. For example, many reptiles have temperature-dependent sex-determination—their sex is determined by the temperature at which they are incubated. In the American alligator, eggs that are incubated at higher temperatures (above 31°C) produce mostly males, while incubation below 31°C produces mostly females. Thus, a warming climate is altering sex ratios in the American alligator (and many other reptiles). In the green spoon worm, the presence of a female leads a neighboring worm to develop as a male, and many other organisms (e.g., clown fish, wrasses) can change sex depending on the surrounding sex ratios.

Differential mortality: When the sexes experience differential mortality, this will inevitably lead to differences in sex ratios. For example, in many ducks, the breeding season is especially hard on females. Copulation is often forced on females by males, and females can be vulnerable to deadly harm as a result. Also, simply laying eggs is energetically taxing, and females are more vulnerable to predation and disease during the egg-laying period. As a consequence, adult sex-ratios in many duck species are male-biased. And in organisms with intense male-male competition, adult sex-ratios can favor females, even if the sex ratios were 1:1 at birth.

Differential migration: Some adult sex ratios are affected by differential migration, which refers to any situation in which males or females are more likely to migrate—out of the population they were born into, into a new population. Both the home population and the new population can experience a change in sex ratios as a result. In the last few decades of the 20th century, a large number of Mexican men left Mexico in search of work elsewhere. As a result, the adult sex ratio in many areas of Mexico skewed female, leading to an increase in unmarried women and a lower birth rate. Similarly, many towns in North Dakota have become male-dominated, as jobs in the male-dominated fracking (a way to extract oil and natural gas from the earth) industry have boomed.



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9.5 The puzzle of skewed sex ratios at birth

A puzzle for many biologists is the occurrence of abnormal sex ratios (those that deviate from ~1:1) at *birth* in populations with chromosomally-determined sex. Consider the hippopotamuses from the previous page. Given what we know about meiosis, we'd expect half of the hippos conceived to have XY chromosomes (of the male sex) at birth, and half of the hippos to be XX (female) at birth. However, in captive populations of the endangered pygmy hippopotamus, only 41% of the offspring are male. This is a significant deviation from 50%, or from a 1:1 ratio. Studies of the sperm of captive pygmy hippopotamuses have indicated that the lowered sex ratio is due to males biasing X-bearing sperm in their ejaculates, although the mechanism through which that occurs is unknown.

Many other examples of skewed sex ratios, in organisms in which biological sex is determined chromosomally, have been documented. Evolutionary biologists have suggested that selection operates in a way to bias the production of offspring of the more advantageous sex. In some cases, the advantageous sex may be male, in others, it may be female. In the case of the captive pygmy hippos, scientists have suggested that—in the captive environment—the perception of a high population density leads to the production of fewer males. Fewer males means fewer rivals for territory. The following is from an article by Joseph Saragusty and colleagues:

“Why should males opt to produce more females? If a male is long-lived, the ownership of a territory being a pre-condition to mating success, and the habitat is saturated with occupied territories, then sons are unlikely to find an empty territory and are likely to compete with their fathers for territory ownership. Under such conditions, fathers have an interest in directing the sex of the progeny in a direction that will enable it to increase its own reproductive success and avoid father–son competition.”

1



Check Yourself



1. [Saragusty, J., Hermes, R., Hofer, H. et al. Male pygmy hippopotamus influence offspring sex ratio. *Nat Commun* 3, 697 (2012). <https://doi.org/10.1038/ncomms1700>]



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9.6 The Trivers-Willard hypothesis of sex allocation

Many studies of sex allocation have focused on the condition (or fitness potential) of the parents, or, more commonly, of the egg-producing parent (a term we'll use interchangeably with mother; "egg-parent" is more accurate and inclusive—especially with respect to humans). Specifically, according to an idea proposed by two scientists (Robert Trivers and Dan Willard; this idea is now called the Trivers-Willard hypothesis), mothers in good condition should bias their offspring in favor of males, and mothers in bad condition should bias their offspring in favor of females. This hypothesis has been succinctly (and maybe a little crudely) summarized as "when the going gets bad, have daughters." This sentiment rests on the presumption that poor-condition mothers will produce offspring that are in poor condition; further a poor-condition female can still achieve some reproductive success (thus giving you some grand-offspring), while a poor-condition male is more likely to be completely shut out from reproduction.

But why? Males are more likely to have highly variable reproductive success (some are wildly successful, but many have no reproductive success), while female reproductive success is often less variable (a highly-successful female will have fewer offspring than a highly-successful male; but fewer females will have no reproductive success). By this logic, a good-condition male can far outperform the productivity of a good-condition female. In short, the Trivers-Willard hypothesis is really a form of fitness "bet-hedging" on the part of the parents. A parent who is in good condition can maximize their fitness by having more [good-condition] sons than [good-condition] daughters; a poor-condition parent is better off having [poor-condition] daughters.



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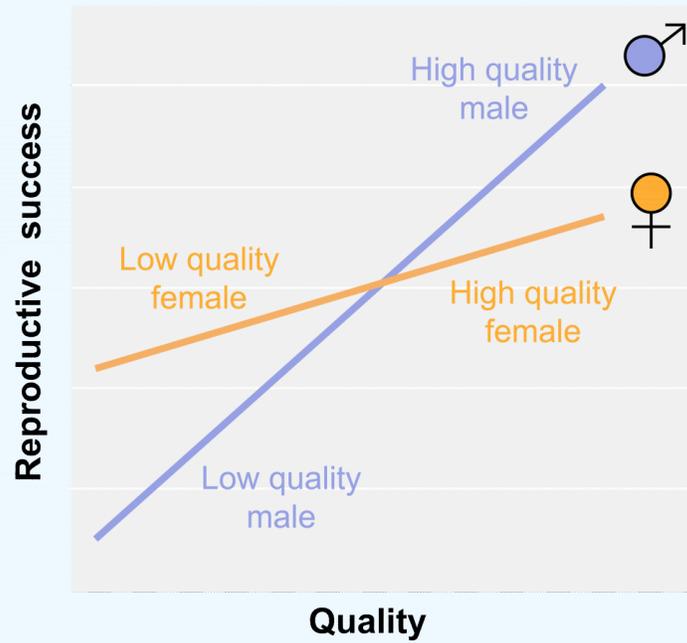


Figure 9.4 Reproductive success as a function of quality in the Trivers-Willard hypothesis. In this example, low-quality females are more successful than low-quality males, but high-quality males are more successful than high-quality females



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9.7 Testing Trivers-Willard in opossums

In the mid-1980s, two biologists devised a simple test of the Trivers-Willard hypothesis of sex-ratio allocation. Specifically, they trapped and marked (for future identification) 40 female Venezuelan opossums. They then supplemented the diets of 20 of these opossums, specifically by leaving a tin of sardines by their burrows every two days for several weeks. Then, they re-trapped the females, looked in their pouches (opossums are marsupials, meaning their young develop in pouches), and identified the sex of their offspring.

Figure 9.5 Opossum with her babies

Their data are summarized below.

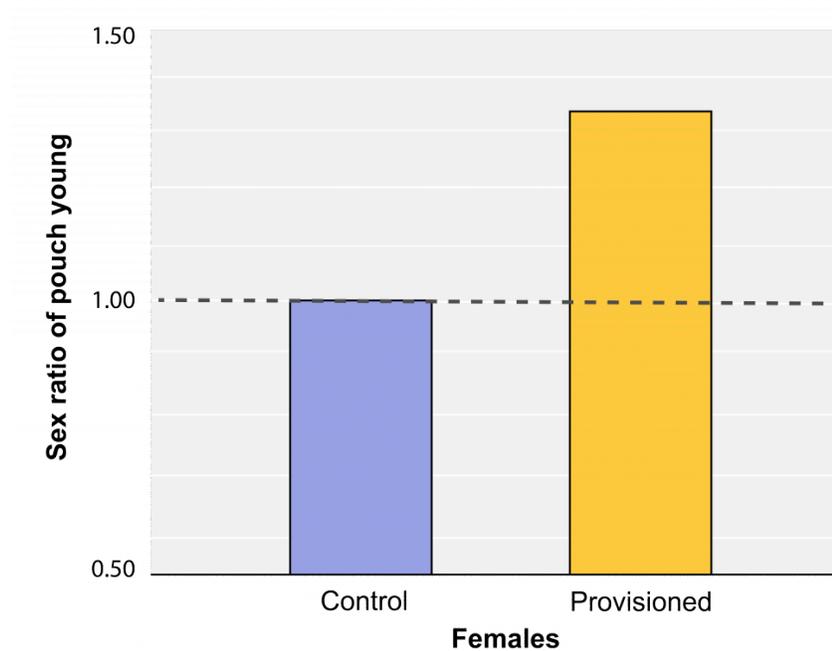


Figure 9.6 Sex ratios of pouch young from control and provisioned females. Sex-ratio difference is significant ($P(\text{one-tail}) = 0.007$, binomial test). Control females, $n=256$; provisioned females, $n=270$.



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Similar support of the Trivers-Willard hypothesis has been observed in similar studies with other organisms, but in some cases, these findings have not been observed. In some situations, explanations other than the Trivers-Willard hypothesis may better explain observed sex ratios. For example, in some organisms, males are bigger and demand more resources; it makes sense that a mother in poor condition would selectively miscarry males in favor of females.

9.8 Testing Trivers-Willard in red deer

There are other aspects of “condition” than nutritional status. For example, in the case of the Scottish red deer, maternal condition doesn’t predict the sex ratio of her offspring, but social status (in this case, dominance over other females) does. Consider the following data:



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So, what does a female achieve through these biased sex ratios? Is her fitness (or lifetime reproductive success, LRS) better off when she has sons (if high-ranking) or daughters (if low-ranking)? The figure below depicts the lifetime reproductive success (LRS) of the sons and daughters of red deer mothers, based on the mother’s social status.

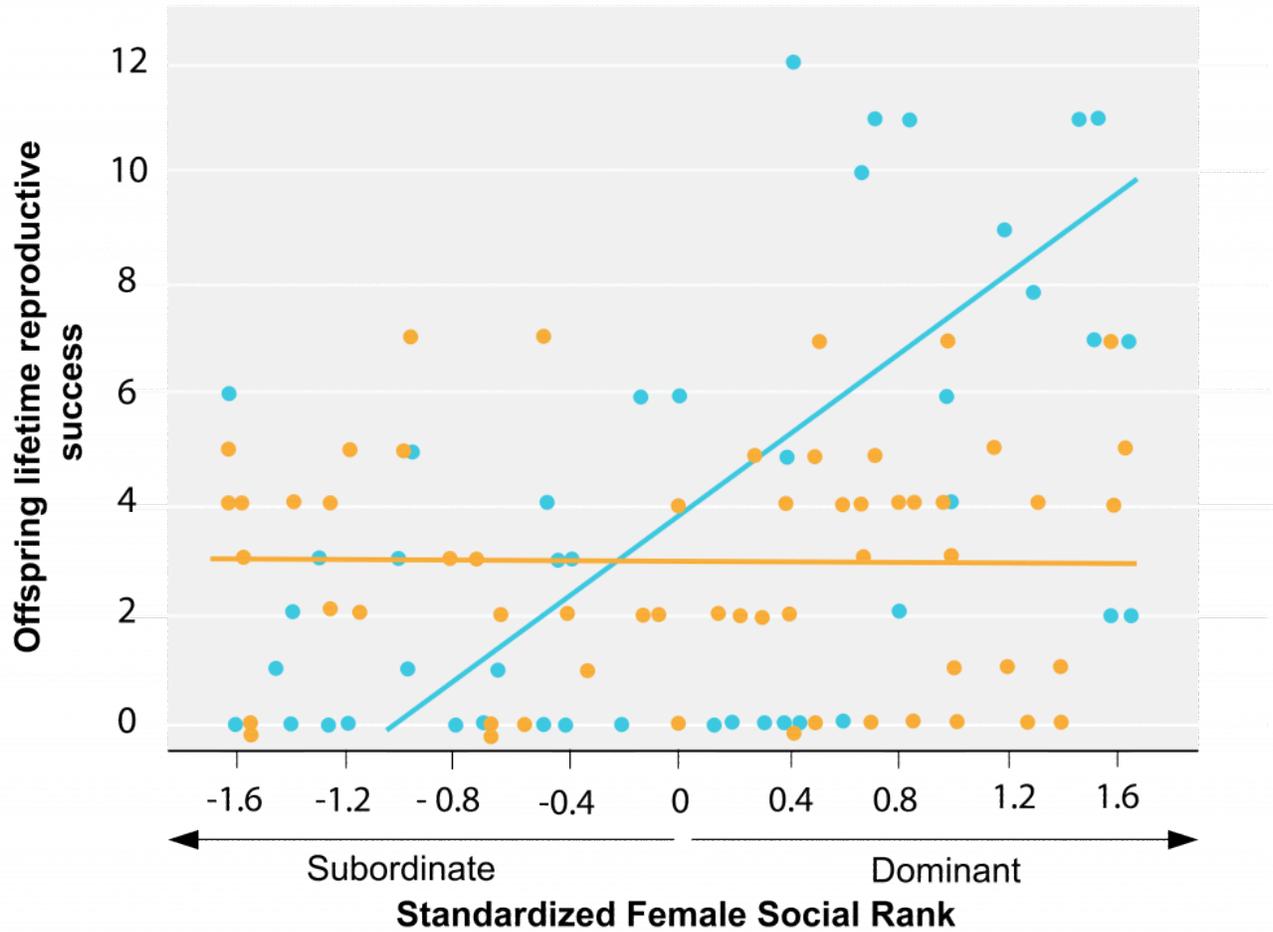


Figure 9.7 Lifetime reproductive success (LRS) of sons and daughters of red deer hinds in relation to their mother's social rank. See text for details (blue are males, orange are females). The slope shown is the reduced major axis.

9.9 Testing Trivers-Willard in spider monkeys

In her observations of Peruvian spider monkeys, primatologist Meg Symington recorded how, of twenty-one offspring born to the lowest-ranked female monkeys, twenty-one were female; of eight born to the highest-ranked monkeys, six were male; the monkeys who ranked in the middle had a roughly even sex ratio in their offspring. This should not be surprising, given the red deer example on the previous page. However, in monkeys, an additional factor to consider is which sex typically leaves home at puberty. In the case of the spider monkeys, it's the females.

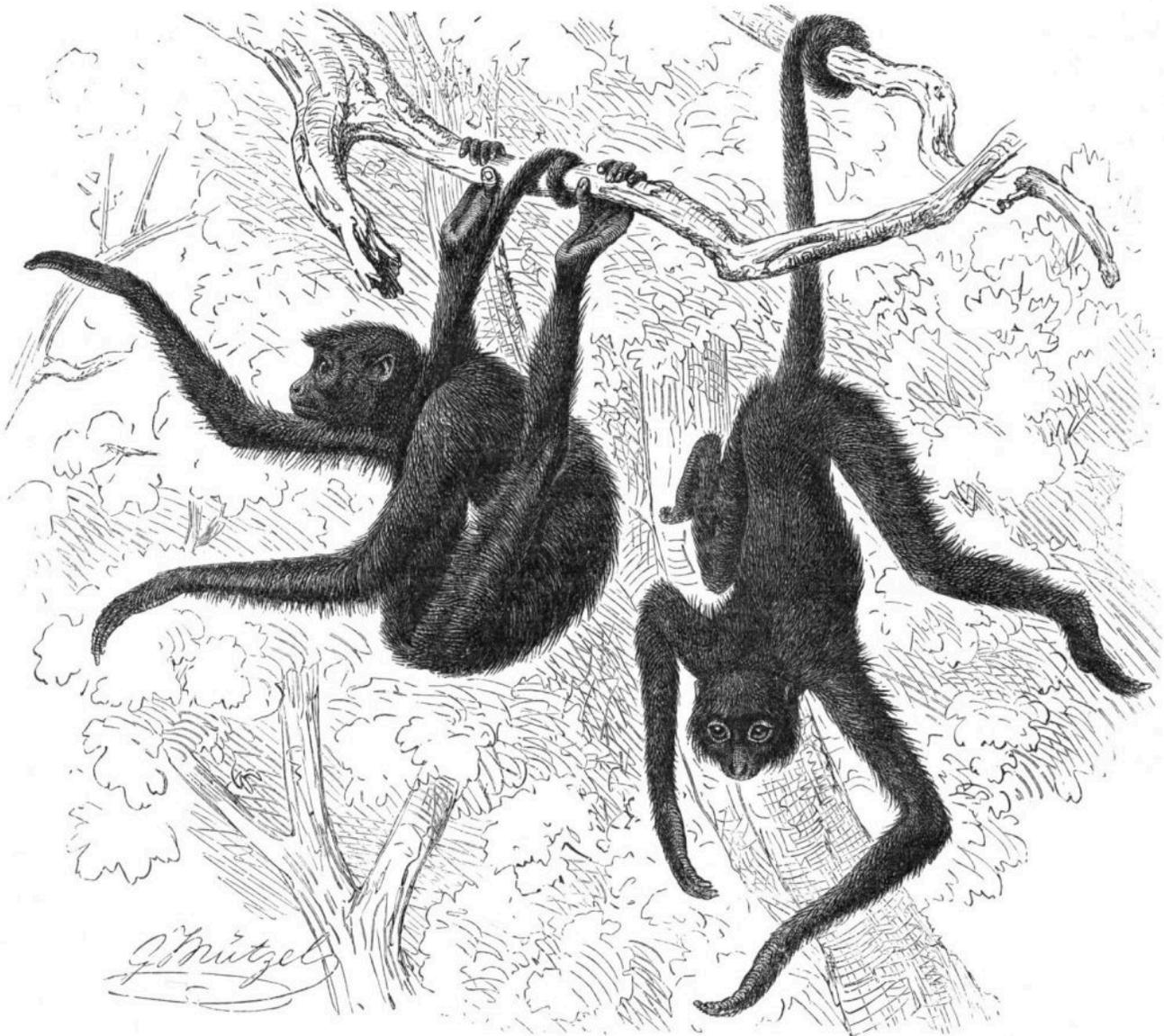


Figure 9.8 Illustration of a Klammeraffe

As Matt Ridley summarizes in the book, “The Red Queen” (p. 118):

“In most monkeys (including howlers, baboons, and macaques) males leave the troop of their birth and join another at puberty—so-called male-exogamy: In spider monkeys the reverse applies: Females leave home. If a monkey leaves the troop it is born into, it has no chance to inherit its mother’s rank: Therefore, high-ranking females will have young of whatever gender stays at home in order to pass on the high rank to them. Low-ranking females will have young of whatever gender leaves the troop in order not to saddle the young with low rank. Thus high-ranking howlers, baboons, and macaques have daughters; high-ranking spider monkeys have sons.”



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9.10 Testing Trivers-Willard in humans

So, does the Trivers-Willard hypothesis apply to human populations? In short, it's complicated. And it's always more difficult (or really, impossible) to conduct controlled studies in humans. However, there are certainly tantalizing lines of evidence that suggest that even in humans, mothers in good condition may tend to produce more sons than daughters. As merely a few examples:

- A multi-year study of children born to thousands of Danish mothers investigated the impact of stress on offspring sex ratio. The investigators noted that mothers who experienced an extremely stressful “life event” (such as the death of a spouse or child, a cancer diagnosis, etc.) during pregnancy exhibited a lower offspring sex ratio than did their less-stressed counterparts.¹
- Stress is high during times of war, and this stress has been hypothesized to be associated with a decline in the M:F sex ratio. For example, in summer 1991, Slovenia engaged in a brief war for independence. The data on human sex ratios in the country and in the capital of Ljubljana, from several years before, during and after, the war, are depicted below.²

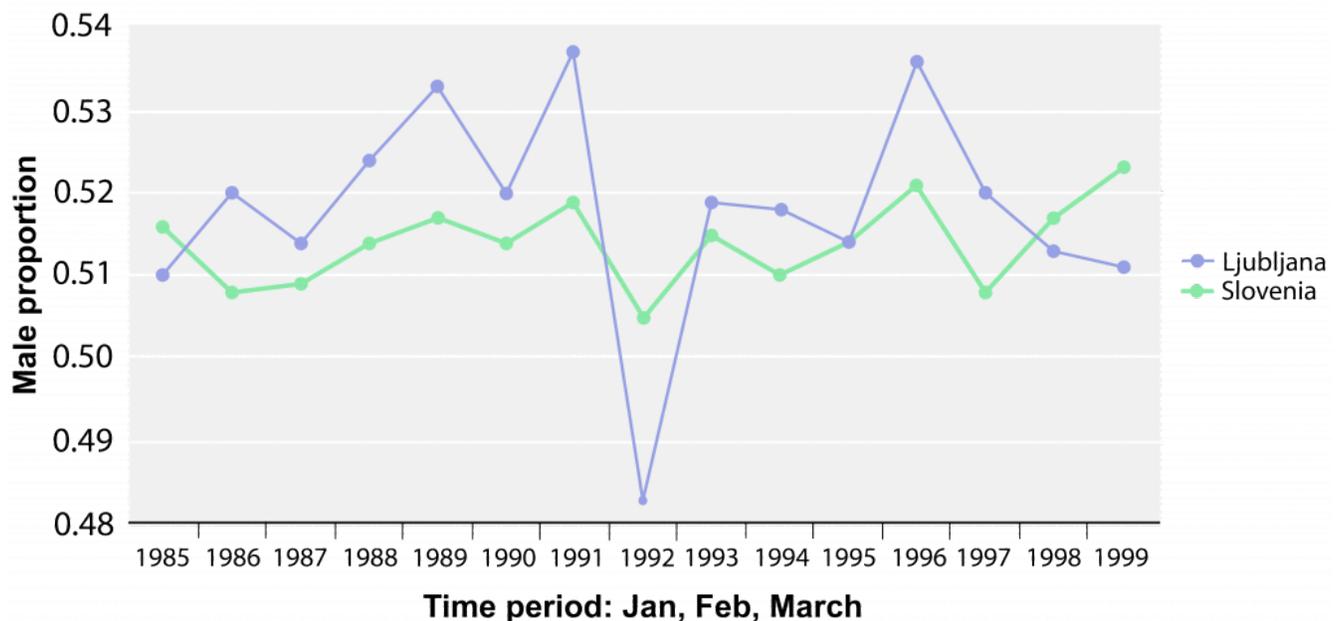


Figure 9.9 Male ratios between Ljubljana and Slovenia over the years.

1. Hansen, D., Moller, H., & Olsen, J. (1999). Severe periconceptional life events and the sex ratio in offspring: follow up study based on five national registers. *BMJ (Clinical Research Ed.)*, 319 (November 2008), 548–549. <https://doi.org/10.1136/bmj.319.7209.548>

2. Hansen, D., Moller, H., & Olsen, J. (1999). Severe periconceptional life events and the sex ratio in offspring: follow up study based on five national registers. *BMJ (Clinical Research Ed.)*, 319(November 2008), 548–549. <https://doi.org/10.1136/bmj.319.7209.548>



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- Stress is also high after natural disasters, and several lines of evidence suggest that enduring an extreme natural disaster can lead to a lowered sex ratio in a population. For example, [a catastrophic earthquake](#) shook Kobe, Japan, in January 1995. Consider these data, from the period afterwards, and note the pattern at approximately nine months later.³

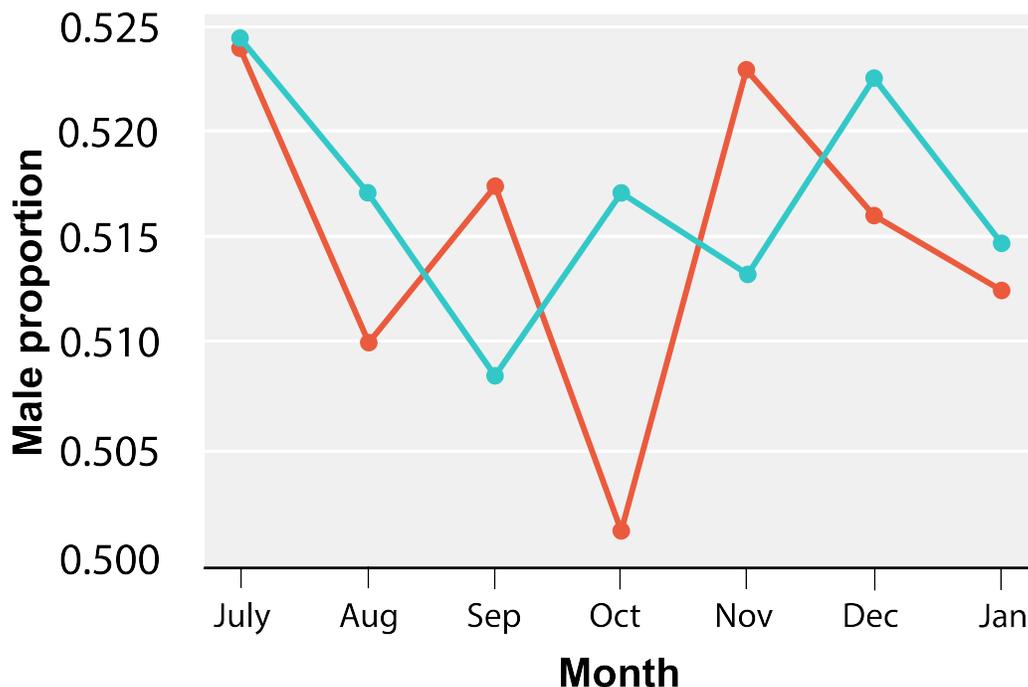


Figure 9.10 Male proportion among newborn infants in Hyogo Prefecture, Japan in the period from July 1995 to January 1996 (red line). For comparison the corresponding values, averaged, for the preceding 2 years, are indicated as the blue line.

- Another way to measure “condition” is through the mother’s general health. In many human

3. Hansen, D., Moller, H., & Olsen, J. (1999). Severe periconceptional life events and the sex ratio in offspring: follow up study based on five national registers. *BMJ (Clinical Research Ed.)*, 319(November 2008), 548–549. <https://doi.org/10.1136/bmj.319.7209.548>

populations, this can be a metric as simple as body weight—that is, does the mother typically get enough to eat? Several studies have examined the relationship between maternal weight and offspring sex ratio. For example, in Mhairi Gibson and Ruth Mace’s study of an agrarian community in southern Ethiopia, they documented a relationship between upper-arm circumference (which is itself a measure of health—i.e., strength, having enough to eat) and offspring sex ratio.⁴ Their data is represented here:

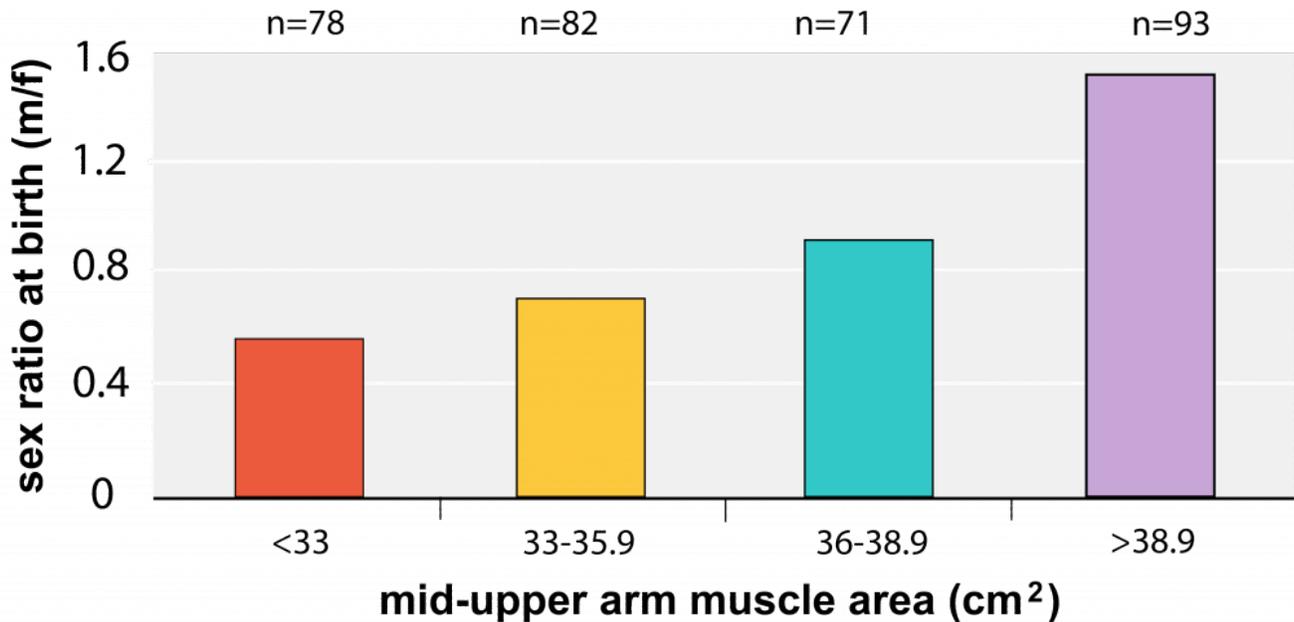


Figure 9.11 Sex ratio of the most recent birth for women by maternal mid-upper arm muscle area, or AMA ($n=324$; overall sex ratio, 0.88).



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<https://open.lib.umn.edu/evolutionbiology/?p=3459#h5p-102>

4. Gibson, M. a, & Mace, R. (2003). Strong mothers bear more sons in rural Ethiopia. *Proceedings. Biological Sciences / The Royal Society*, 270 Suppl (August), S108-9. <https://doi.org/10.1098/rsbl.2003.0031>

9.11 Beyond Trivers-Willard

Although the Trivers-Willard hypothesis provides a useful framework for understanding many sex-ratio phenomena, it is not the only explanation for altered sex ratios. For example, many studies have noted a correlation between temperature and sex ratio, with more sons being born in warmer years. These findings lead to follow-up questions, such as: how could temperature influence offspring sex ratio? (Note: this has been noted in organisms that do not have temperature-dependent sex determination.) Is this yet another example in support of Trivers-Willard, in which warmer years yield more food and mothers in better condition?

Further complicating matters is the observation, from a study of over 200 countries, that latitude is a significant predictor of sex ratio. Specifically, tropical latitudes, closer to the equator, have a lower sex ratio than those in temperate environments, generally speaking.

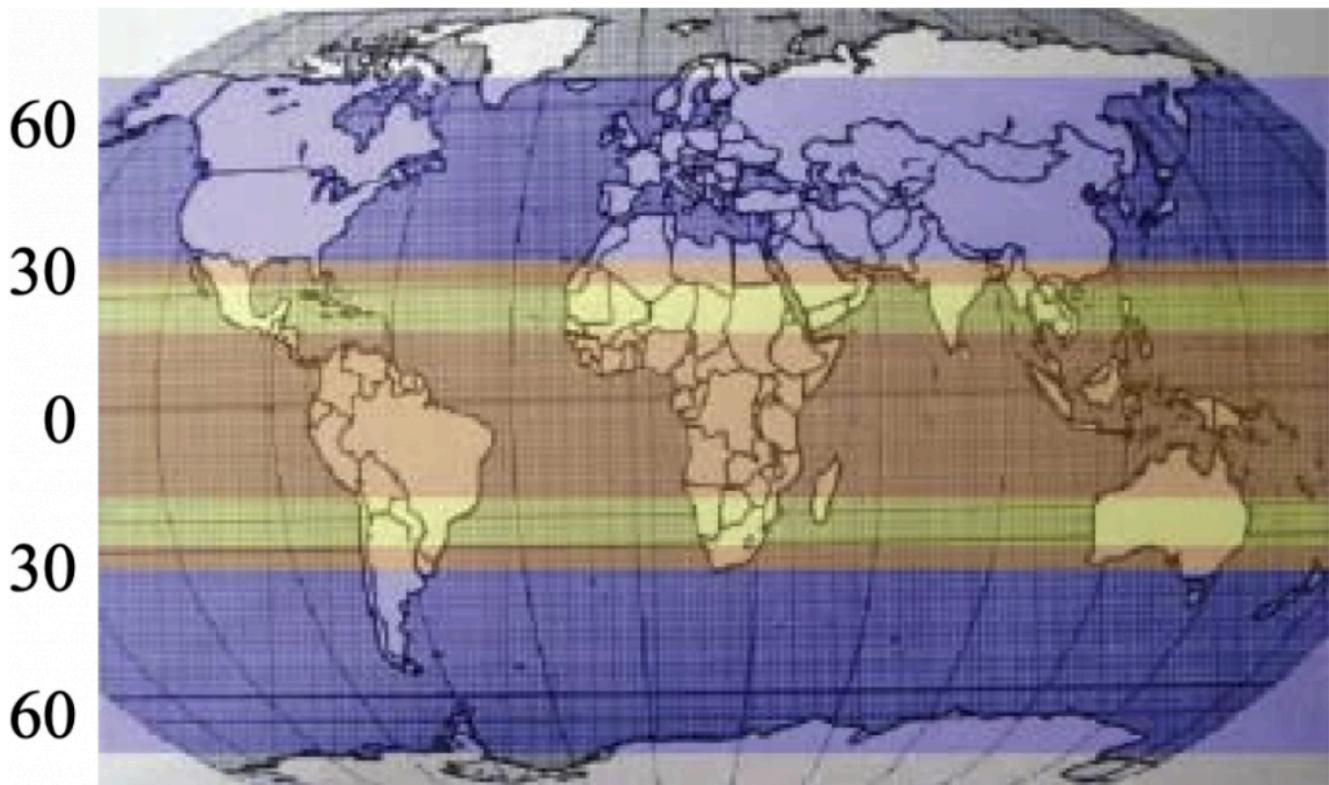


Figure 9.12 Color-coded average proportion of male offspring for each five degrees of global latitude, for 202 countries. All figure segments are color-coded (blue, more than 51.7% males; yellow, 51.2–51.7% males; orange, 50.7–51.2% males). All sex proportions were calculated using the average for 1997–2006.

1

It's difficult to separate the effects of temperature anomalies (episodes of temperature extremes), whereby warmer temperatures lead to more males, from this latitudinal effect, whereby fewer males are born in the tropics. However, studies of solar-cycle peaks and seasonality in sun exposure suggest that fewer males are conceived

1. [From: Navara, K. J. (2009). Humans at tropical latitudes produce more females. *Biology letters*, 5(4), 524-527.].

(or they are conceived and subsequently miscarried) during times of higher UV exposure. The suggested logic behind these findings is that UV, which is known to damage our DNA, may be especially hard on male genomes. Specifically, any detrimental mutation in either the male's X or Y chromosome will be expressed, because there is no homologue to mask the effects of the mutant.

And that's not all! Other investigators have suggested a correlation between sex ratios and parental age, socioeconomic status, and the presence in the mother of various illnesses such as hepatitis, schizophrenia, or multiple sclerosis. Sex-ratio allocation is complicated and fascinating, and our understanding of these phenomena is ever-changing.

9.12 Male bias and extreme sex ratios

Consider the data compiled by Abrevaya:¹

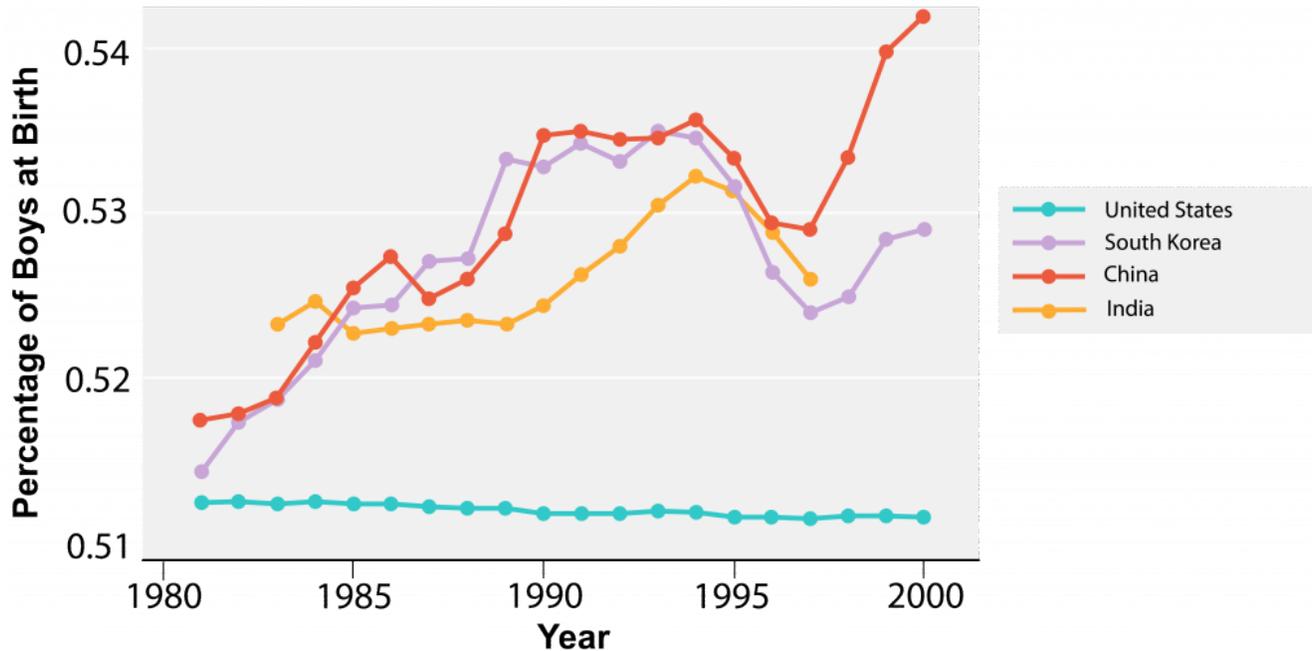


Figure 9.13 Likelihood of a Male Birth, by Country

Note a clear pattern of increasing male births in South Korea, China, and India, largely associated with increased access to ultrasound and selective-abortion technology. In one study of abortion clinics in India, of 8000 abortions that followed a sex-determination procedure, 7,999 were of female fetuses. In another study, not a single one of 250 identified male fetuses were aborted. In the city of Taegu, South Korea, a sex ratio of approximately 1.0 in 1980 became a sex ratio between 1.2 and 1.3 in 1990. That's roughly 125 males for every 100 females carried to term.

These modern techniques of sex-selection follow a history of neglect or infanticide of female offspring, practices that are well documented in the literature and have led to some dramatically skewed sex ratios. Some villages in India have documented ratios as high as 100:31. When these ratios are extrapolated to populations of tens of millions of people, the inevitable consequence is millions of “missing girls.” The following data summarize this phenomenon of millions of missing girls in Asia.

1. Abrevaya, J. (2009). Are there missing girls in the United States? Evidence from birth data. *American Economic Journal: Applied Economics*, 1(2), 1–34. <https://doi.org/10.1257/app.1.2.1>

Country	Calculated no. of missing females, in millions*
Afghanistan	0.5-1
Bangladesh	1.8-3.7
China	34-41
India	27-39
South Korea	0.2-0.3
Pakistan	2.6-4.9
Taiwan	0.4-0.6
Iran	0.8-1.2

Figure 9.14 Number of missing females for selected Asian countries, 2001

2

These data give rise to myriad additional questions: Why is there such a strong bias for sons? Does this bias exist outside of Asia? Do these preferences increase with decreasing fertility, such as during China’s “One Child Policy” of the recent past?

One thing that does seem fairly well documented is that these preferences do not seem to be driven by the mother. Several studies have illustrated that mothers have little expressed preference for sons over daughters, while fathers around the world tend to prefer sons. A study of sex ratios gives us never-ending food for thought!

Read More

Read more [here](#) and check out some interactive graphs!

2. [from Hesketh, T., & Xing, Z. W. (2006). Abnormal sex ratios in human populations: causes and consequences. *Proceedings of the National Academy of Sciences of the United States of America*, 103(36), 13271–13275. <https://doi.org/10.1073/pnas.0602203103>, using data from Hudson V.&Den Boer, A.M. (2004) *Bare Branches: The Security Implications of Asia’s Surplus Male Population* (MIT Press, Cambridge, MA) and Klasen, S. & Wink, C. (2002) *Popul. Dev. Rev.* 28, 285–312.]

9.13 Wrapping Up: Understanding human sex ratios



Understanding human sex ratios

Recall the examples of sex ratios in human populations by country, discussed in the introduction to this Chapter. In Australia, which has a sex ratio at birth of 1.06, has a total sex ratio close to 1.00, largely due to differential mortality throughout the life span (girls have higher survival). But what about Bahrain (1.54 M:F) and El Salvador (0.92 M:F)? A close look at Bahrain's age-structured sex ratios suggests that the high overall sex ratio is the result of immigration, especially that of younger men capable of manual labor in Bahrain's petroleum industry.

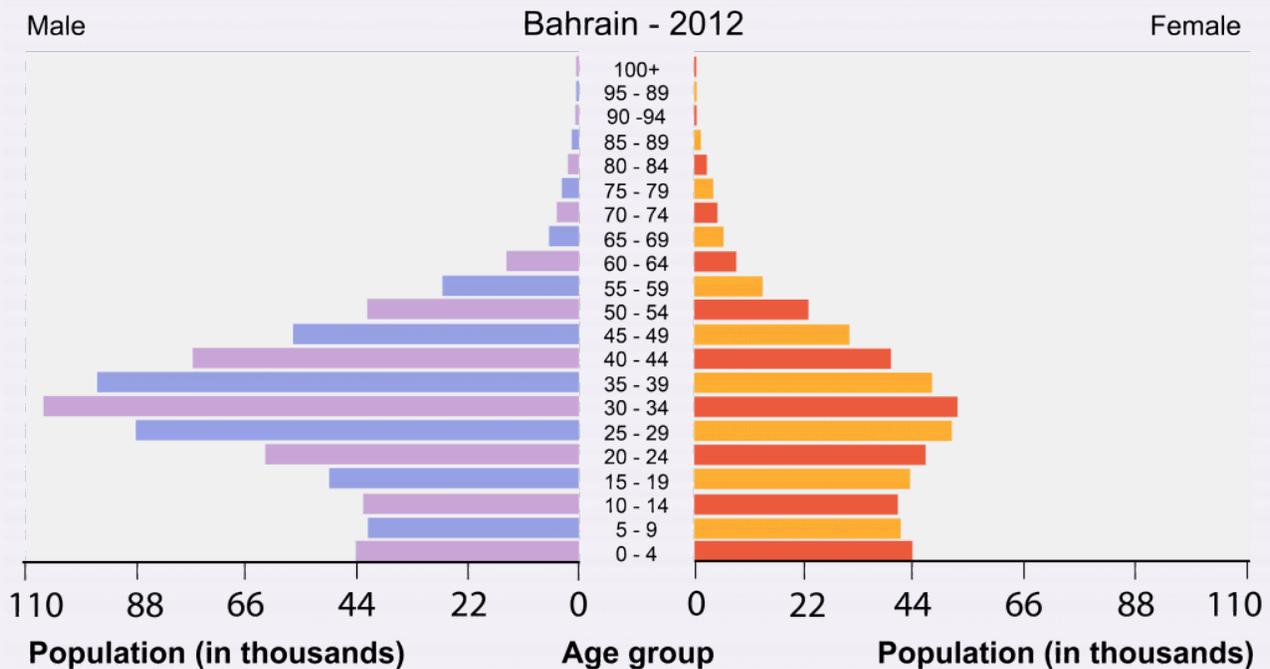


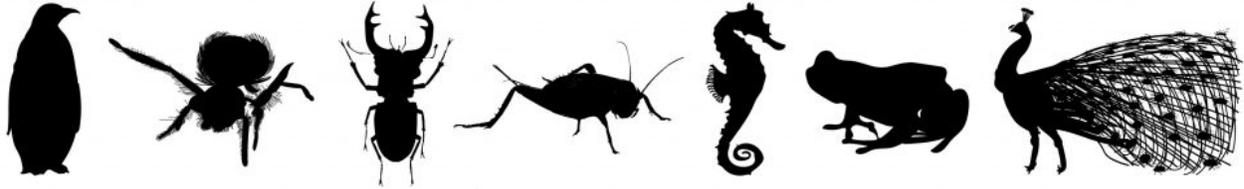
Figure 9.15 Male versus female population in Bahrain in 2012.

In El Salvador, a 1.05 sex ratio at birth is part of a 0.92 sex ratio overall. This low sex ratio is likely due to the sort of differential mortality (due to accidental death and disease) observed in males across the globe, as well as the high incidence of violent, gang-related crime (disproportionately impacting males) that has plagued this country for several decades.

Note that these proposed explanations are largely hypothetical. It's not possible to directly pinpoint the impact of a single factor on a population's sex ratio. What is clear, however, is that population sex ratios are context-dependent, due to multiple variables, and fascinating.

As we conclude this chapter and prepare for in-class discussion, be sure to return to the chapter's [goals and objectives](#).

Chapter 10: Sexual Selection (contributed by Rachel Olzer)



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10.0 Introduction

The case of the silent crickets

If you have ever tried to escape the sounds of the city at night, you probably realized that nature can be just as loud! All the insects buzzing, frogs croaking, owls hooting- these are the sounds of animals looking for love. Most of these sounds are emitted by individuals, often times males, seeking mating partners. The Pacific field cricket, *Teleogryllus oceanicus*, is no exception. Like most crickets, males of this species sing to attract females.



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<https://open.lib.umn.edu/evolutionbiology/?p=1761#audio-1761-1>

So then, how can we understand the presence of non-calling males? In a recently studied population of Hawaiian crickets, non-calling males made up 50% of the population on the island of Oahu and over 90% on the island of Kauai. Knowing how natural selection operates, how can we understand the presence of these non-calling males? How do these non-calling males pass on their genetic material to the next generation, if they cannot sing to attract a mate?

Selection has operated on sexually reproducing organisms in many ways, including ways specific to attracting and retaining mates. This type of selection is called sexual selection, and is the focus of this chapter. Read about sexual selection, and then we'll return to the problem of the non-calling male crickets at the end of the chapter.

10.1 Chapter Objectives



Learning Objectives

By the end of your reading and our in-class discussion, you will be able to:

1. Define the following terms:
 - **intrasexual selection**
 - **intersexual selection**
 - **anisomagy**
 - **parental care**
 - **secondary sexual traits**
 - **sexual dimorphism**
 - **cryptic female choice**
 - **sperm competition**
 - **satellite behavior**
 - **sexual mimicry**
 - **traumatic insemination**
2. Understand the differences between natural selection and sexual selection.
3. Identify the two forms of sexual selection and the types of traits that result from each process.
4. Explain why males and females have different reproductive strategies and how this can lead to sexual conflict
5. Describe the different behaviors that males and females engage in before, during, and after sex that are subject to sexual selection

10.2 What is sexual selection?

Natural selection describes a process in which individuals who are better at obtaining resources and escaping predation are more likely to survive and reproduce, leaving their heritable traits to future generations. Through this process individuals become more adapted to their environment and if this continues for several generations, there will eventually be a measurable change in the genetic composition of a population. This is what scientists define as evolutionary change.

But what about traits that do not directly aid in survival? Or better yet, what about traits that actually hinder survival? Picture the elaborate tail feathers of a peacock. These feathers, while visually stunning, make it nearly impossible for an individual to fly. Not only that, but these feathers also make it easier for a predator to spot a peacock from a distance and they can also be used by a predator to apprehend a peacock so it cannot escape. Rather than helping a peacock survive, their tail feathers actually make it more difficult to escape predation. So does such a trait evolve?



Figure 10.1 The peacock's tail is elaborate and beautiful, but can be used by predators to locate and trap them. So how can such a trait even evolve, if it is clearly detrimental to the peacock's survival?

1

This question can be answered by viewing this trait not as aiding in survival, but as a trait that aids in reproduction.

1. [Wikimedia CC-BY-SA-2.0](#)

Exaggerated traits, like the tail feathers of a peacock, are used to attract females for mating, ensuring that a male passes his genes on to the next generation.

employing a second adaptive mechanism through which evolutionary change occurs; this is called sexual selection. Sexual selection is a “special case” of natural selection in which individuals compete for mates in order to pass on their genes to future generations. Exaggerated traits like a peacock’s tail are used to attract females for mating, ensuring that a male passes his genes on to the next generation. In essence, sexual selection acts on an individual’s ability to successfully reproduce- even if that ability comes at a cost to survival.



Figure 10.2 Rather than aiding in survival, the peacock’s tail aids in his reproduction. Males adorn these elaborate traits to attract the attention of females.

2



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<https://open.lib.umn.edu/evolutionbiology/?p=1763#h5p-53>

10.3 How does sexual selection work?

Sexual selection, the process through which individuals compete for mates, primarily takes two forms: **intersexual selection** and **intrasexual selection**. Intersexual selection, often referred to as **mate choice**, involves individuals of one sex choosing among members of the opposite sex based on the attractiveness of certain traits that those individuals possess. Intrasexual selection, also called **mate competition**, involves one sex competing with members of the same sex for access to mates.

Typically, the sex that is choosing mates is the one that invests more in gamete production prior to mating. Conversely, the sex that is chosen is also the sex that fights with members of the same sex for access to mates, and is traditionally the one that invests less in gamete production. In many species females produce just a few large and costly eggs, while males produce many, small and less expensive sperm. Because of this difference in gamete production and investment, known as **anisogamy**, females are typically the choosy sex and males typically compete with other males for access to females.



Figure 10.3 Notice the difference between the size of the human egg versus the human sperm. The difference in gamete size and number can explain why females are choosy about which males to mate with. This is referred to as **anisogamy**.

1

Generally, it is unusual for males to be choosy about their mates. There are many reasons for this. Gamete production and investment is one reason. Another reason why females are typically the choosy sex has to do with the level of investment in offspring care, known as **parental care**. For example, following sexual reproduction and fertilization, most mammals develop within the body of their mothers. The developing offspring of most mammals then get their food and oxygen from the blood of their mothers through a spongy organ called the placenta. Even marsupial offspring, though not fully developed when born, are usually carried by their mothers in a pouch until

1. [Wikimedia](#) Public Domain.

they are able to walk on their own. I suspect that you have noticed a pattern here- mothers typically invest more than fathers when it comes to caring for developing offspring. This is another major reason why females are choosy about their mates.

However, in some animals, males provide a great deal of parental care to their offspring. For example, in emperor penguins each female produces a single egg. She then transfers the egg to her male mate and leaves to spend the winter in the open ocean in search of food and other resources. During the Antarctic winter, which lasts about four months, male emperor penguins huddle in groups, guarding their eggs and keeping them warm. An extreme example lies in seahorses, among whom males get pregnant and carry their offspring during development, after which they give birth to baby seahorses.

As a result of males investing a great amount of time and energy into caring for their offspring during development, some species of animals show a reversal in who is the choosy sex. For example, in many poison-dart frogs, males are the sole providers of parental care to developing offspring. As such, female poison-dart frogs will fight amongst each other in the presence of calling males, and some have been observed to court a single singing male in the field.

Historically, much of the research on sexual selection has focused on what happens between males and females prior to mating. For example, a lot of work has been dedicated to understanding how males signal to attract females, and what females look for in potential mates. However, it is important to note that sexual selection can occur before and after a female and male mate. Before mating, individuals will signal their quality to potential mates. After mating, individuals can bias paternity in their favor through various processes including cryptic female choice and sperm competition, which we will discuss later in this chapter.



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<https://open.lib.umn.edu/evolutionbiology/?p=1765#h5p-54>

10.4 Can we see markers of sexual selection in animals?

Just as with natural selection, sexual selection can lead to changes in the genetic composition of a population that can be seen through physical changes to the way an organism looks. Both mate choice and mate competition can lead to the evolution of elaborate traits, termed **secondary sexual traits**, because they are not the primary traits involved in sexual reproduction or sperm transfer. Instead these traits aid in the process of sexual reproduction by allowing individuals to gain access to mates. Typically, one sex possesses an elaborate secondary sexual trait or traits, but the other sex does not, a condition called **sexual dimorphism**. Both mate choice and mate competition can involve the evolution of secondary traits that are sexually dimorphic.

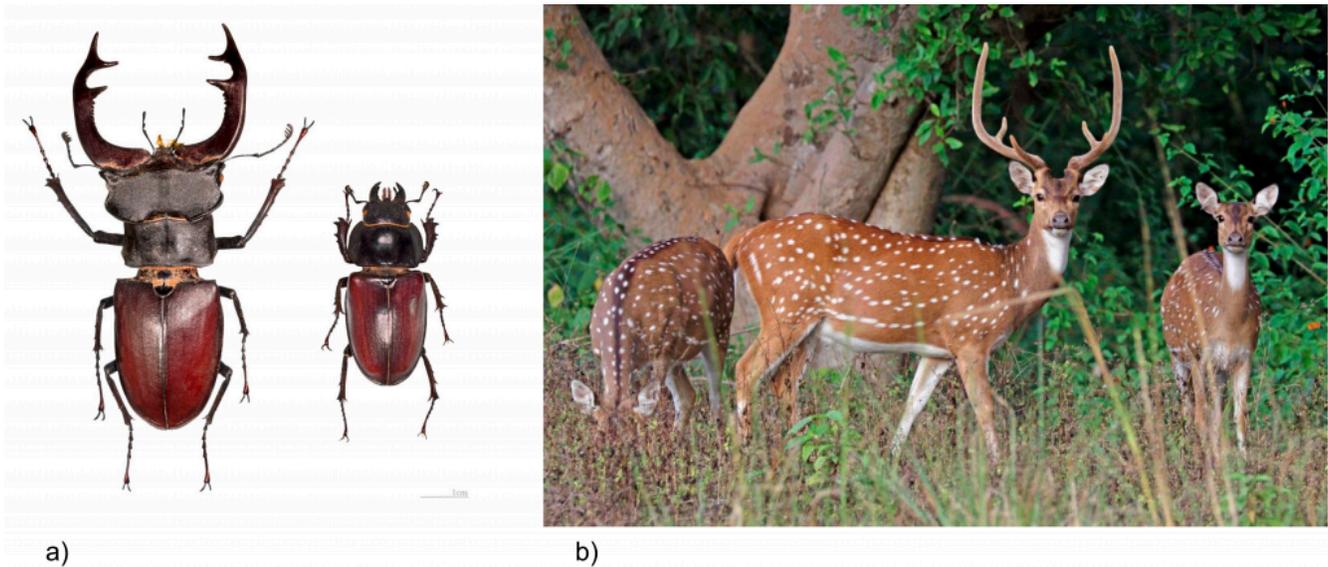


Figure 10.4 a) The stag beetle has horns that they use to stab rival males, helping them gain greater access to females and **b)** Many male deer possess antlers that are used during fighting contests.



Figure 10.5 Sexual dimorphisms can also include differences in color pattern between males and females. As the signaling sex, males typically possess elaborate color patterns that they use to attract the attention of females.

12

Traits that are subject to selection via mate choice are referred to as **ornaments or sexual signals**. Ornaments can involve different signal modalities, including **visual** signals like the bright colors of many birds and butterflies; **olfactory** (i.e. chemical) signals like the scent patches that many mammal species use to attract mates; **auditory** signals used by chorusing frogs and some insects like crickets; or even tactile signals like the **vibratory** signals used by some spiders when they tap their legs on the surface of a leaf to attract mates. Sexual signals can also involve multiple signal modalities. For example, male jumping spiders will often use both visual and vibratory signals when trying to attract females for mating.

Video of jumping spider male signaling to female:



1. [Wikimedia CC BY-SA 4.0](#)
2. [Wikimedia CC BY-SA 4.0](#)



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<https://open.lib.umn.edu/evolutionbiology/?p=1767#oembed-1>

10.5 Why be choosy about your mate?

As we discussed above, the difference in gamete size and production predicts that females should be choosy about whom they mate with because eggs are costlier to produce than sperm. Therefore, females have a vested interest in protecting their eggs from males who are less fit. A female simply has more to lose by making a bad choice about her mate.

How do females decide whether a male is a suitable mate? There are several, non-exclusive models of how and why mating preferences evolve. Broadly, there are two types of fitness benefits that drive mate choice: direct and indirect benefits. **Direct benefits** increase the fitness of choosy individuals through material resources. Males will sometimes provide females with a food gift before mating. These resources, called **nuptial gifts**, provide nourishment to females that they may not otherwise get. For example, male great grey shrikes- a predatory bird- will present prey items like rodents, other birds, lizards, and large insects to females immediately before mating. A female great grey shrike will choose a mate according to the size of the prey item presented to her. Such is the case in many insects and spiders where males present these nuptial food gifts to females in the hopes that she will choose to mate with him. In this way the size or quality of the nuptial gift can serve as another trait through which females can judge the quality of a potential mate.

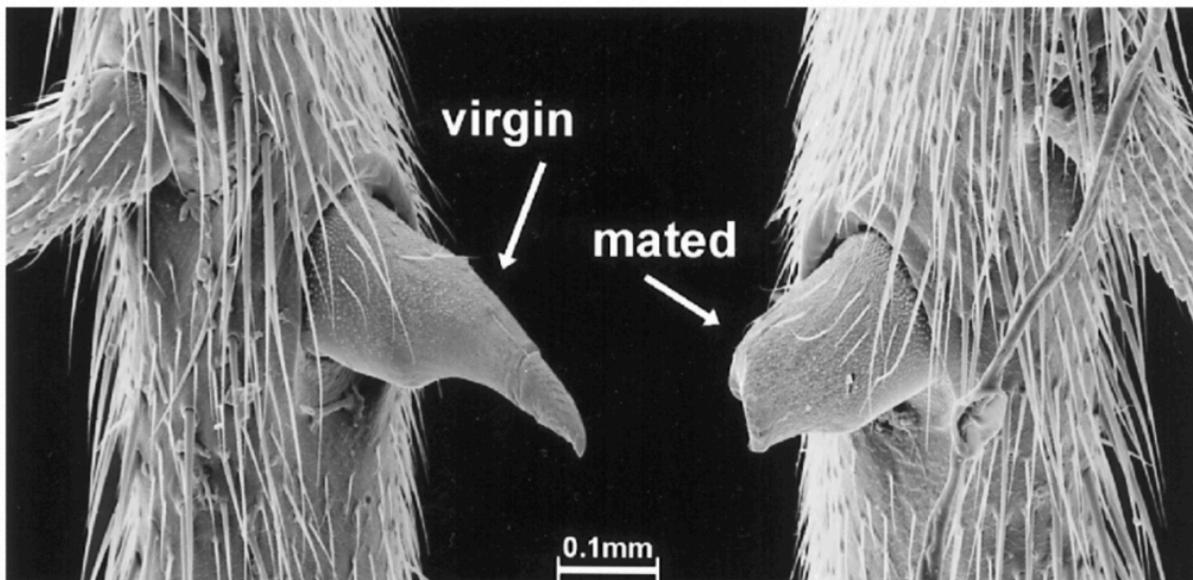


Figure 10.6 Here we see a magnified image of two ground cricket legs. This species of crickets engages in tibial spur feeding in which females will chew on a specialized structure on the leg and get nutrients that aid in her survival and reproduction. The leg on the left shows an intact tibial spur, the marker of a virgin male. The leg on the right is of a mated male, with a tibial spur that has been chewed on by a previous female mating partner.

1

1. [Kenneth M. Fedorka](#) and [Timothy A. Mousseau](#) "TIBIAL SPUR FEEDING IN GROUND CRICKETS: LARGER MALES CONTRIBUTE LARGER GIFTS (ORTHOPTERA: GRYLLOIDAE)," *Florida Entomologist* 85(2), 317-323, (1 June 2002). [https://doi.org/10.1653/0015-4040\(2002\)085\[0317:TSFIGC\]2.0.CO;2](https://doi.org/10.1653/0015-4040(2002)085[0317:TSFIGC]2.0.CO;2)

In extreme cases, males will even sacrifice parts or all of themselves to females. For example, in some species of ground crickets, females receive a nuptial gift by chewing on a specialized spur structure on the male hind tibia (i.e. leg) while mating. Most predatory species of preying mantids practice a type of extreme nuptial feeding known as **sexual cannibalism**, in which a female will eat her mate prior to, during, or after copulation. Most often, a female mantid will begin feeding by biting off the head of a male, as they would with regular prey. Because copulatory movement in males is controlled by nerves in the abdomen, not the head, removal of a male's head does not affect mating, sperm transfer, or proper fertilization. The reason for sexual cannibalism has been heavily debated. Experiments show that females on low quality diets are more likely to cannibalize her mates, compared with females given high quality diets. Some suggest that males that submit to females and are cannibalized gain a selective advantage by producing higher quality offspring. In any event, this type of sexual behavior is quite rare because the costs are often assumed to out-weigh the benefits, particularly for males. However, the high prevalence of sexual cannibalism in several species of spiders gave rise to the common name "black widow spider".



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The most popular example of sexual cannibalism is in mantids. Females will bite off the head of males, sometimes before even mating with them. However, if a female bites the head off a male after copulation has been initiated, mating typically continues until sperm is transferred.

Some types of nuptial gifts are considered a direct benefit, because they enhance a female's survival and reproduction. In contrast, **indirect benefits** increase the fitness of the offspring of a choosy individual. Typically, females who choose to mate with attractive males will benefit by producing offspring, specifically males, who are particularly attractive as well. As such, the attractive offspring will be more likely to attract females, and thus the choosy female's genes will continue to spread- the ultimate goal of all living organisms. For example, in some species of insects, like katydids, the nuptial gift provided by males is packaged with sperm. The package is an edible gift called a **spermatophore**. These extra nutrients in the sperm are assimilated by the female and are thought to enhance the fitness of the offspring produced, thus increasing the probability that both the female and male continue to pass on their genes. Because indirect benefits enhance offspring fitness, they are often thought to be an extension of parental investment that males can make for their offspring. Therefore, these types of benefits can also be used by females to judge the quality of a potential mate.

2



Figure 10.7 Nuptial gifts, especially spermatophores, are common in insects. Often the male katydid transfers a spermatophore to a female during mating. Wikimedia CC BY-2.0

10.6 Unconventional ways of finding a mate

Signaling to attract mates does not come without costs. For one, signaling can be energetically demanding. Think about how tired you might be after singing to a potentially unreceptive audience for an entire day. Now think about doing that every day for your entire life! Perhaps more importantly, signaling doesn't just attract attention from potential mates, it can also attract unwanted attention from predators and parasites. Remember how the peacock's tail attracted attention from predators like a tiger? This is a common problem in nature. For example, male Túngara frogs from the tropical rain forest sing to attract females. However, this singing also attracts an acoustically savvy bat.



Figure 10.8 Sexual signals do not just attract attention from potential mates, but they can also attract unwanted attention from predators and parasites. A famous example of this is in the Túngara frog found throughout the central and south American tropical rainforests. In this system, males sing in choruses to attract females. Wikimedia. CC BY 2.0

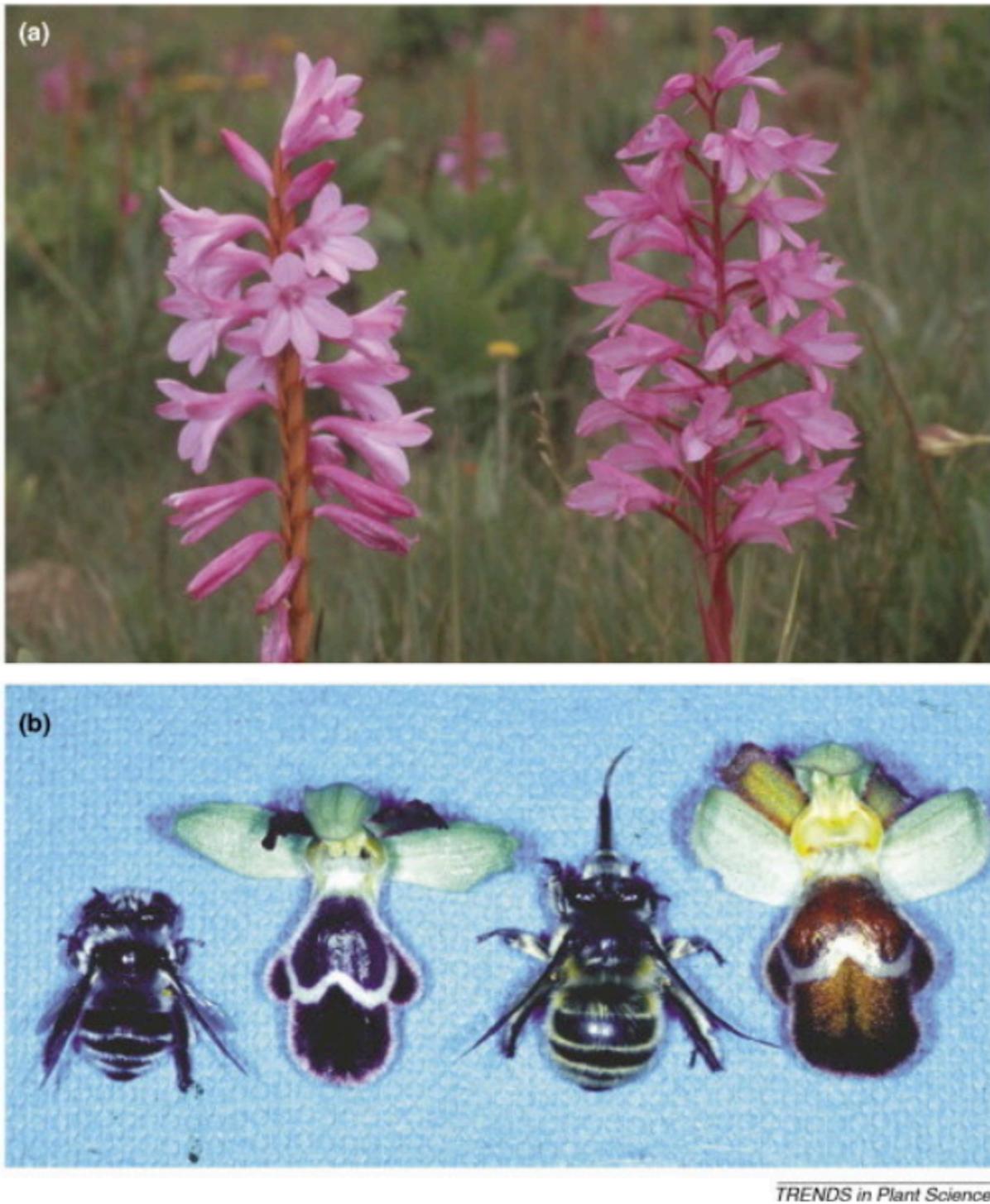
To balance the relative costs and benefits associated with signaling, individuals within a population will sometimes adopt alternative strategies for finding and attracting mates. These **alternative mating strategies** allow individuals to gain access to mates, sometimes in very sneaky ways, without expending a lot of energy and typically without increasing the risks of predation associated with signaling. Alternative mating strategies can take a number of forms. One such alternative strategy involves “sneaker males” that will steal mating opportunities from other males. For example, in some fishes, males will sneak into the territories of other males and attempt to mate with the females that a rival male is monopolizing. Variations on this behavior, referred to as **satellite behavior**, can be found in several species of fishes, frogs, and crickets. Alternative mating strategies can be employed by the choosy sex as well. For example, in some damselflies, males will harass and try to force

copulations with females. As such, some populations have evolved **sexual mimicry** in which one sex mimics the other sex in its behavior, morphology, or chemical signaling. In damselflies, females have evolved to mimic males in their appearance, allowing females to avoid harassment and continue to mate only with males that they actively choose.



Figure 10.9 Alternative mating strategies can be employed by the choosy sex as well. A common example of this is sexual mimicry in damselflies. In some species of damselflies females will mimic males in appearance in order to avoid harassment by males who want to mate. The female above is mimicking a male in color so that she will not be harassed by many males that she is not interested in mating with. Wikimedia CC BY-SA 4.0

Examples of sexual mimicry include the spotted hyena, certain types of fishes, birds, some species of insects, and even plants. In plants, especially orchids, flowers can mimic the mating signals of their pollinator insects to attract them. These insects are attracted and pollinate the flowers through pseudo-copulations or other sexual behaviors performed on the flower.



TRENDS in Plant Science

Figure 10.10 Sexual mimicry occurs in plants as well. Flowers will sometimes mimic the mating signals of their pollinator insects. These insects are attracted to and pollinate the flowers through pseudo-copulations or other sexual behaviors that they perform on the flower. The bottom picture shows an example of how two different species of orchids have an uncanny resemblance to their respective pollinators.

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Sexual mimicry can influence the social system of a species as well. The most common example is the spotted hyena in which female hyenas resemble male hyenas in their sexual anatomy. Females have an elongated clitoris that resembles a penis, and a false scrotal sac. These pseudo-penises, paired with high levels of certain hormones, result in highly aggressive and dominant females. Not only is there a dominance hierarchy, or ranking system based on aggression, among female spotted hyenas, but this dominance hierarchy extends to males as well. Females with the lowest social ranks are still more dominant than the highest-ranking males- an unusual social system among animals.



Figure 10.11 The most extreme example of sexual mimicry is perhaps the presence of a pseudo-penis in female spotted hyenas. Researchers have suggested that this sexual mimicry has had major effects on the social structure of the spotted hyena. For example, females have a much higher social rank in groups than males.

10.7 When males and females do not agree

Frequently, males and females have conflicting strategies for maximizing their reproductive success. These conflicting optimal strategies typically arise over the mode and frequency of mating and can give rise to sexual conflict. Remember that males and females often make very different investments in reproduction with females producing just a few, nutrient-rich gametes over which they are often very protective. In contrast, males typically produce many, motile, and less nutrient-rich gametes. This difference in gamete production usually means that females optimize their reproductive success by mating with just a few, high quality males, while males optimize their reproductive success by mating with as many females as they can in their lifetime.

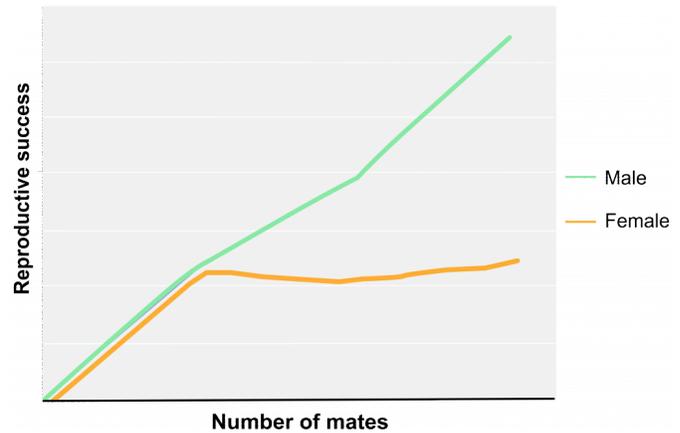


Figure 10.10 Males maximize their fitness by having as many mates as possible. In contrast, females maximize their fitness by having fewer, high quality mates. This difference in how males and females maximize their reproductive success can lead to conflict between the sexes.

Sexual conflict can lead to the evolution of sexually antagonistic behaviors. For example, males benefit by mating with multiple females, but females typically do not benefit from frequent mating. Thus, females will often exercise extreme mate choice, and males will sometimes try to circumvent this mate choice by using forceful mating tactics and coercive behaviors to achieve reproductive success.

One such forceful-mating tactic, called **traumatic insemination**, occurs exclusively in invertebrates, and involves a male inserting his genitalia into the non-genital tissue of a female. Essentially a male will stab a female with his genitals and release sperm products directly into her blood stream- yikes! This behavior can lead to fertilization because in most invertebrates, like insects, blood and other fluids circulate together (in contrast to that of many other animals, including humans, in which blood circulates in a different system from other fluids). Following traumatic insemination, sperm can migrate through the blood stream to a female's ovaries, resulting in fertilization. Most of the research on traumatic insemination has been carried out on bed bugs in which males have evolved knife-like genitalia that can stab through the hard exoskeleton of a female and transfer sperm directly into her body cavity and blood stream.

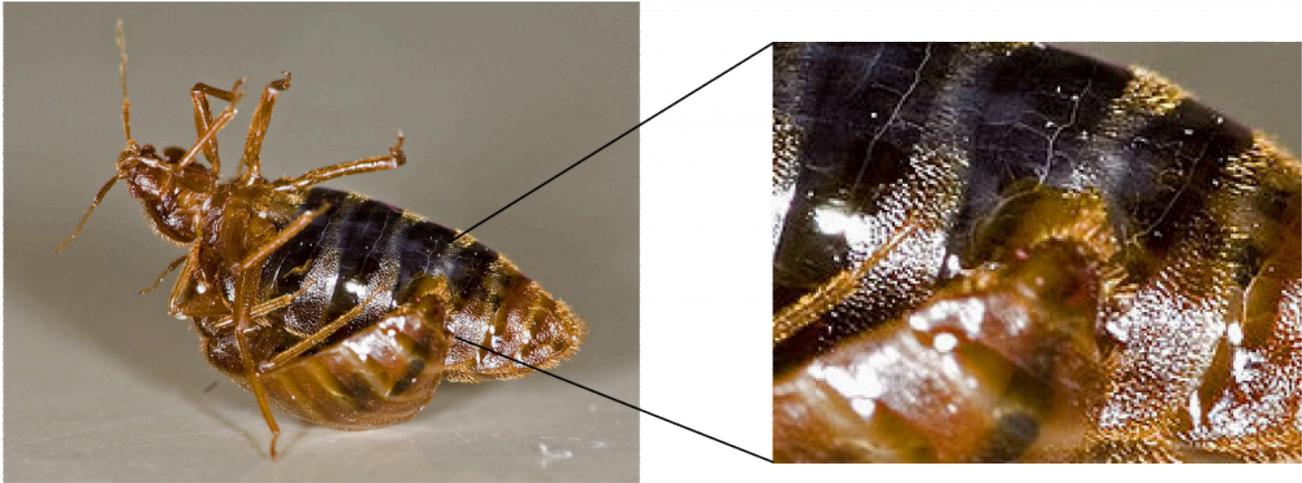


Figure 10.11 Traumatic insemination is perhaps best-studied in bed bugs. Males bed bugs use a knife-like penis to stab through the hard exoskeleton of a female's abdomen, injecting sperm into the non-genital tissue and releasing sperm directly into her body. This allows males to circumvent mate choice, thereby increasing their reproductive success by mating with as many females as possible. Updated version of: Wikimedia CC BY-SA 1.0

While it is easy to see how this behavior benefits the male, traumatic insemination has been shown to reduce female fitness because the number of offspring produced via this process is typically less than the number produced via non-traumatic means. Because of the reduction in fitness, females have evolved behavioral and physiological adaptations in response to these forced copulation attempts.

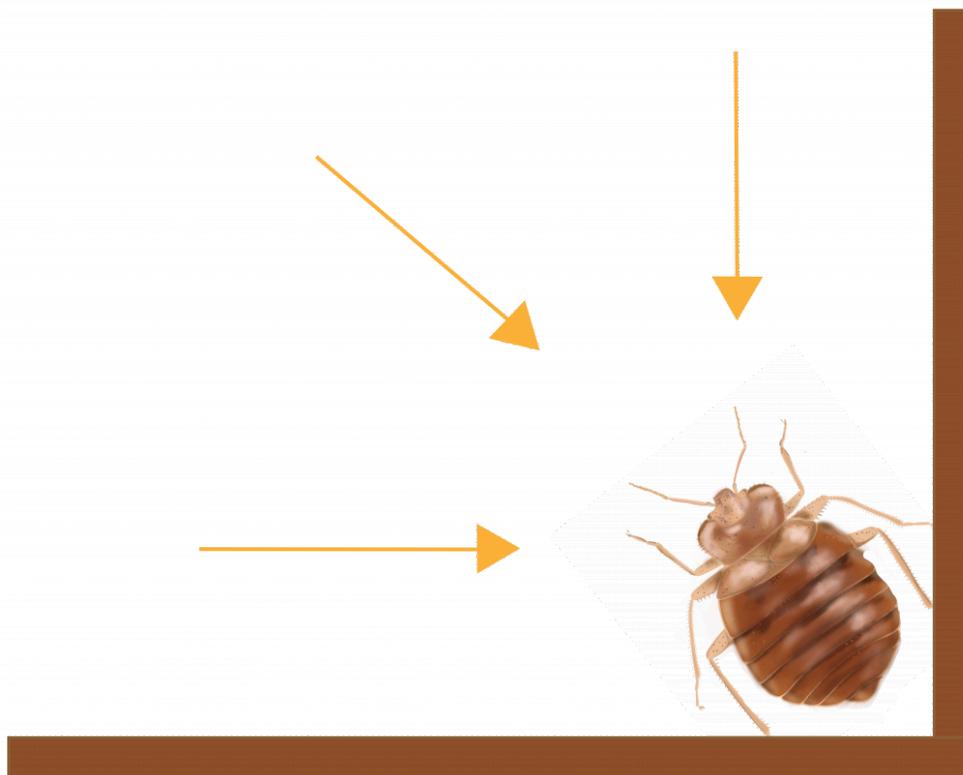


Figure 10.12 Because of the detrimental effects that traumatic insemination has on their health, female bed bugs have evolved adaptations to counter attempts by males to force mate with them. One of these counter-adaptations is called a “refusal position” in which females will back themselves into a corner, and essentially protect their abdomen from advancing males.

Sexual conflict can also present in the form of coercive sexual behaviors. As the term coercive implies, these behaviors are not mutually beneficial to males and females, but they evolve because the behaviors confer some advantage to one sex. For example, in lions, mature males will sometimes kill the young offspring of a female they wish to mate with. This behavior, called **infanticide**, typically results in the mature male becoming the new sexual partner of the offspring's mother, who would otherwise be unreceptive to a new mate. Once again, adaptations to counter this behavior have been observed. In lions, females will have multiple mates, confusing paternity for males in the population, making it difficult for a male to be certain that the offspring he kills are not his own.

The important thing to understand about sexual conflict is that this is often a cyclical process. This means that one sex may evolve a means for biasing reproduction in their favor (e.g. males attempting to mate more often by forcing females to mate with them), and in response, the other sex can evolve adaptations to shift reproduction back in their favor (e.g. females evolving refusal positions to counter attempts at forced copulation by males). Thus, this process is often referred to as an evolutionary arms race because any evolutionary change will only ever be relative progress. In other words, one sex might gain an advantage in the short-term, but no sex will have the upper-hand for the long-term.



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<https://open.lib.umn.edu/evolutionbiology/?p=1774#h5p-55>

10.8 Post-copulatory sexual selection

Remember that sexual selection does not come to a halt after animals have mated! If a female mates with multiple males, such that sperm from several individuals remains in her body for an extended period of time, sexual selection can continue long after a male and female have mated. Just like with sexual selection before mating, post-copulatory sexual selection occurs in two forms: **cryptic female choice**- an extension of mate choice; and **sperm competition**- an extension of mate competition.

Cryptic female choice

Using physical or chemical mechanisms, females can bias paternity and affect male reproductive success by choosing whether certain sperm are successful in fertilizing their eggs. The term “cryptic” is used to describe an internal, and thereby hidden, process that females employ to choose the sperm from males that they prefer. The research suggests that cryptic female choice is likely a consequence of sexual conflict regarding the frequency and mode of mating. While males increase their fitness by successfully mating with as many females as possible, and thereby fertilizing as many eggs as possible from different females, females can incur fitness costs associated with mating with many males. Cryptic female choice reduces these costs by allowing females to mate multiply (as males wish to do), but then only select sperm from the favorable males afterwards. Here, females benefit by influencing paternity in favor of the males they prefer- possibly because they provide some direct or indirect benefit to her.

Sperm competition

Sperm competition, an extension of mate competition, is the process by which sperm from two or more males compete for fertilization of a female’s egg or eggs. Sperm competition is often compared to having tickets in a raffle: a male has a better chance of having their ticket drawn (i.e. fathering offspring) if he has more tickets in the raffle (i.e. he releases more sperm per ejaculate into a female’s reproductive tract). Alternatively, males may not release more sperm, but instead they evolve faster, more motile sperm that allow an individual’s sperm to reach a female’s eggs first. Among the best evidence we have for sperm competition is the evolution of longer sperm tails in animals that have multiple partners.

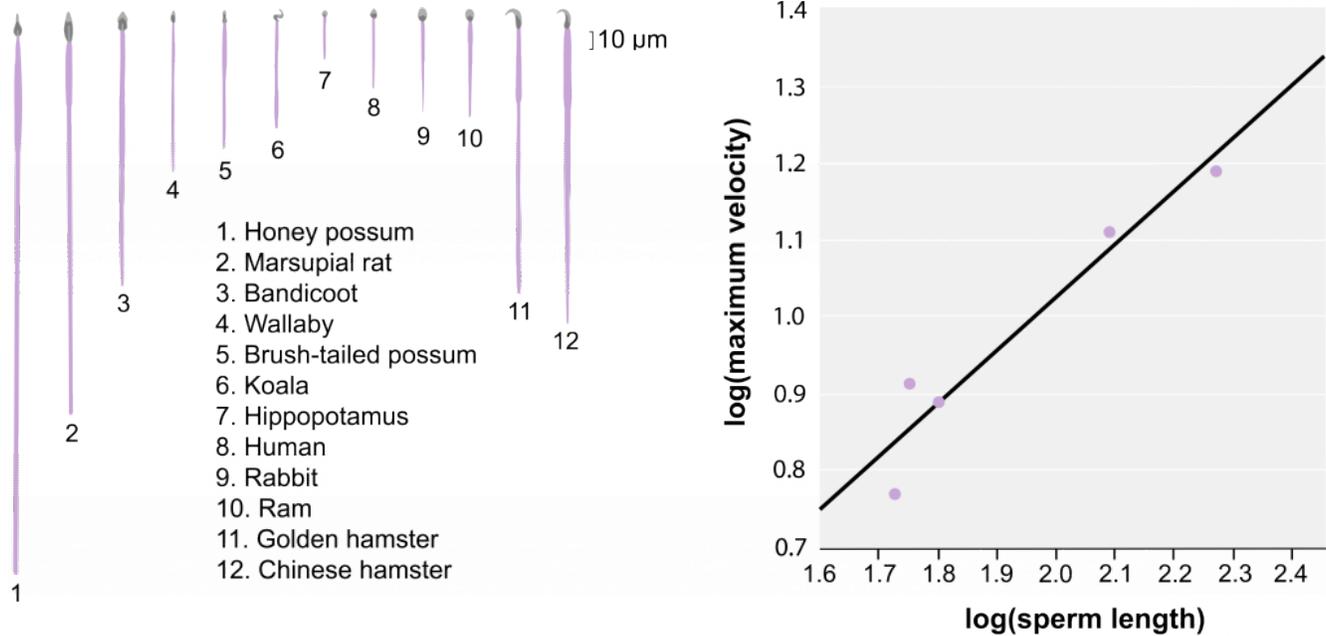


Figure 10.13 Among the best evidence for the presence of sperm competition in animals is the evolution of longer sperm tails in animals that have multiple mating partners. Longer tails are correlated with faster moving sperm. Males with longer, faster sperm can fertilize a female's eggs before other males are able to.

Remember that sexual conflict can give rise to antagonistic behaviors to bias paternity. Here, cryptic female choice is a way for females to bias paternity in favor of males that are better or that she prefers. However, we should expect that males, in turn, evolve in response to this cryptic choice so as to bias paternity in their favor.



Check Yourself

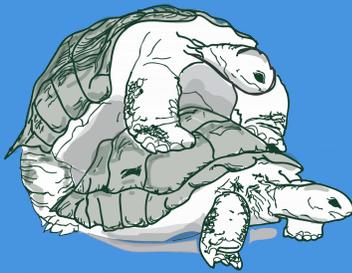


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<https://open.lib.umn.edu/evolutionbiology/?p=1776#h5p-56>

10.9 What is the evidence for sexual selection in humans?

Evolution by sexual selection has been invoked to explain a number of human anatomical features, which appear useless or detrimental to human survival. These include hairlessness, male facial hair, rounded breasts, pubic hair, and penis size. In the next few pages we will talk about some of the more interesting examples of traits that some scientists have argued are products of sexual selection: primary sexual traits (e.g. male penis size), female orgasms, large brains, our extensive vocabularies, and many aspects of our cultural behaviors.



Biology is Sexy!

Why do human men have nipples?

Early evolutionary biologists were keen to observe that humans, especially compared to nearly all other mammals, were much less hairy. Many hypothesized that because human females were much less hairy than their male counterparts, the loss of hair was due to selection by pre-historic human males for less hairy female partners. But if this were the case, and men were selecting for less hairy women, then why would hairlessness be present in human males, and not just human females? The answer will show up often as we discuss evolution- by-products of similar biological pathways in male and female animals can give rise to traits that are only selected for in one sex, but will often show up in the other sex. An interesting example of this is the presence of nipples in mammals. In most mammals, including human females, the nipple is important for transporting milk from inside to outside of the body. Given that human women are solely responsible for breast feeding, why do male animals, like human men, have nipples? Maybe this is not a question you have ever thought about; after all, it would be quite startling to suddenly see a handful of animals, including humans, without nipples. But evolutionarily, this question is quite interesting and the answer will come up a lot in our discussions of human evolution, so it's worth spending some time on it.

Have you ever noticed most animals have a similar body plan? For example, most animals have a set of eyes somewhere on their head, the head rests atop a thorax or torso, and legs protrude downward from the thorax. While aberrations from this body plan can happen, often as the result of mutation, most animals have this body plan. The reason is that all animals, in fact, all living things, evolved from a common ancestor. As such, the developmental pathways that give rise to the traits we see in animals are highly conserved. This means that across a wide variety of animal species, pathways sharing a common function, also share a common origin in the biological past. Evolution typically takes the path of least resistance and which organisms may not be completely optimized, we are often just “good enough” to survive and reproduce.

This same logic applies to the case of nipples in humans. The developmental pathway from embryo to fetus is highly conserved, with little variation in the early stages of embryonic development. Human fetuses do not develop sex-specific characteristics, like a penis or vagina, until certain genes are turned-on around week 7 of development. Because

nipples are not a trait that is determined by male or female-specific genes, all human fetuses will develop them. While they do not serve the function of nourishing offspring in males, they also do not affect survival and reproduction. Because they are not costly, nipples persist in men as it is not an evolutionary priority to get rid of them, and a re-working of the entire embryonic developmental plan would be extremely difficult.

Fun Fact: Why do humans have two nipples?

A good rule of thumb for mammals is to have twice as many nipples as offspring that you produce at one time. A female cow typically has two offspring at a time, and has four nipples; a small dog has eight nipples and a large dog has ten nipples because typical litter size is four to five pups at once. Thus, human females, who typically gestate one fetus at a time, have two nipples.

Large penises and breasts

Human penises are quite unique! Despite common slang terms that imply otherwise (e.g., “boner”), the human penis contains no bones. Unlike most of our closest evolutionary relatives, like chimpanzees and bonobos, human males do not have a penis bone, or baculum. Instead, human males must maintain an erection by pumping blood into the penis. Evolutionary biologists have speculated that the loss of the baculum in humans may be due to sexual selection by human females. Because the human erection relies on a type of hydraulic pumping system, erection failure can be an early warning of certain health conditions. Thus, human females are able to use the male erection as a clear sign of good health in potential partners.

Interestingly, the human penis is also much thicker than the penises of other great apes. Some have suggested that the evolution of the human penis towards a large size, both in length and diameter, has been the result of sperm competition. However, sperm competition typically favors larger testicles, not larger penises. Others have suggested that it is the result of mate competition, because a larger penis will be more efficient in displacing sperm from rival males during sexual intercourse. Support for this idea is limited. In fact, one study found that the amount of semen displacement during sexual intercourse was related not to penis size, but to the depth of pelvic thrusting. However, these researchers also stated that a longer penis would be more capable of leaving semen in less-accessible parts of the vagina, making it more difficult for subsequent males to remove or displace the semen.

Similarly, human females have much larger breasts than other primates. Because the additional fatty tissue in human breasts does not contribute to milk production, many think breast size evolved as a courtship signal. Many scientists think that large, round breasts and larger penises may once have served as signals of health and fertility, but many of these traits are now the product of “runaway selection”- a positive-feedback loop in which strong mate choice leads to the further exaggeration of a sexual trait.

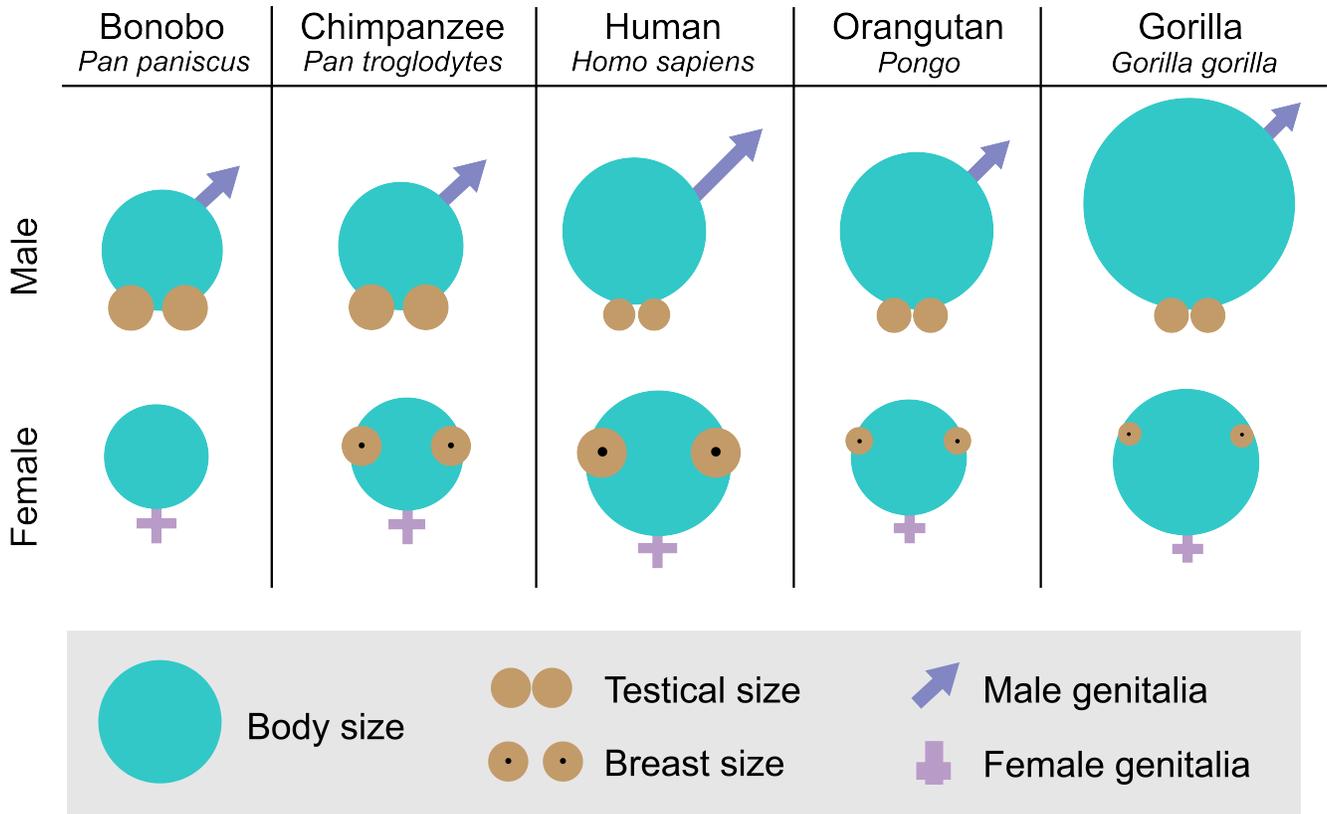
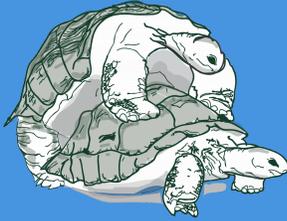


Figure 10.14 Humans have large penises and breasts relative to body size compared with other great apes.

10.10 What's up with the human female orgasm?



Biology is Sexy!

Considering that it happens all the time across the world, scientists are fairly clueless about the female orgasm. Among the most tantalizing of questions is, “what is the evolutionary significance of the female orgasm?”

It is widely thought that the male orgasm exists to encourage men to spread their sperm (i.e. genetic material) as much as possible. Some argue that women orgasm for the same reason: to encourage procreation. But in practice, compared with the male orgasm, female orgasm is extremely difficult to achieve and nearly 10% of women never have an orgasm in their lifetime. Furthermore, unlike the male orgasm, which is coupled with the release of sperm and other ejaculates necessary for reproduction, the female orgasm appears to serve no similar role. So then what is the purpose of the female orgasm? Why do women orgasm? Few questions regarding human evolution have been more difficult to answer than those regarding the female orgasm and for the most part there are two firmly opposed arguments for this phenomenon.

One hypothesis harkens back to a concept we previously discussed- women orgasm simply because men do; in other words, female orgasm occurs because of selection on males. This answer is similar to the reason why men have nipples because women need them to feed their children. Because men and women share a similar developmental pathway, whatever structure or process appears in one sex will necessarily appear in the other. Women develop similar erectile and nervous tissue that is necessary for orgasm in virtue of the strong and ongoing selective pressure on males for releasing sperm via coupled orgasm and ejaculation.

While most scientists agree that women probably started having orgasms as a by-product of men having them, why women still have orgasms is hotly debated. Some people think that this holdover from development continues to be the reason that women experience orgasm. The *holdover camp* claims that female orgasms are an incidental by-product of male orgasm, and that's it.

Another evolutionary holdover hypothesis involves ovulation. In some species, like humans, ovulation is spontaneous. In other species, females actually do not ovulate until stimulated to do so through hormonal surges during and after copulation. Scientists have recently proposed that human women orgasm because of an evolutionary holdover from closely related species that used orgasm as an ovulation-triggering event. In other words, human women orgasm not because of a holdover from male development, but because of a holdover from our early female ancestors.

Many scientists are unwilling to accept that the female orgasm does not serve an adaptive purpose. This group of scientists has proposed three broad categories for why women orgasm: pair bonding, mate choice, and enhanced fertility. The pair-bonding hypothesis suggests that female orgasm facilitates the bond formed between sexual partners, aiding in mate retention and bi-parental care. Others propose that women can judge the quality of potential long-term mates by whether or not orgasm is achieved during their sexual interaction. This last hypothesis states that the female orgasm actually does aid in reproductive success. Referred to as the “upsuck theory”, the idea is that during orgasm, muscles contracting and relaxing in the uterus and vagina create suction that moves sperm up the female reproductive tract, resulting in enhanced fertility.

We may never know the exact reason why women orgasm from an evolutionary perspective, but simply pondering the

question is interesting. Much of the fun for researchers is returning to scientific questions with new methods and with fresh eyes!

10.11 Is the brain another object of sexual desire?

It turns out that the sexiest organ of all might in fact be just between your ears—your brain! Since Darwin first contemplated about the evolution of humans and sexual selection, mate competition, primarily among males, has been considered an important force for evolutionary change in primates and other mammals. Even further, brain size and complexity has been regarded as a hallmark of primate and human evolution. While research on primates has found that brain size and complexity is related foremost to increased social complexity, recent research has found an evolutionary relationship with mating system complexity in particular. In this context, larger brains may have evolved as the result of sexual selection for individuals with greater social acuity.

Interestingly, more recent research shows a negative relationship between levels of sperm competition among males, as measure by relative testes mass, and the development of brain size. Scientists have suggested that this negative relationship represents an investment trade-off between two very metabolically costly tissues. Your brain is responsible for about one quarter of your body's consumption of oxygen and sugar. In humans, the human brain is about as big as it can be while still fitting through the birth canal, and researchers have noted that the pelvis had to widen during human evolution to accommodate a larger head. While the human brain is capable of amazing feats of ingenuity, it is not necessarily clear that the adaptive benefits of a larger brain could justify such extreme costs. After all, animals with smaller brains are capable of many sophisticated behaviors like tool use, foraging, and cooperative group living. Even organisms without brains can be perform advanced motor activities. For example, slime molds have been shown to solve mazes and puzzles, and some carnivorous plants can even count! However, the sheer costliness of the human brain may indeed be the exact hallmark of evolution by sexual selection. Just like the peacock's tail, such a costly trait would not evolve through natural selection alone. Researcher Geoffrey Miller has even suggested that things like creativity, music, art, and humor might have been used as signals during pre-historic human courtship, leading to runaway selection for these signals—thereby driving the evolution of larger and larger brains.

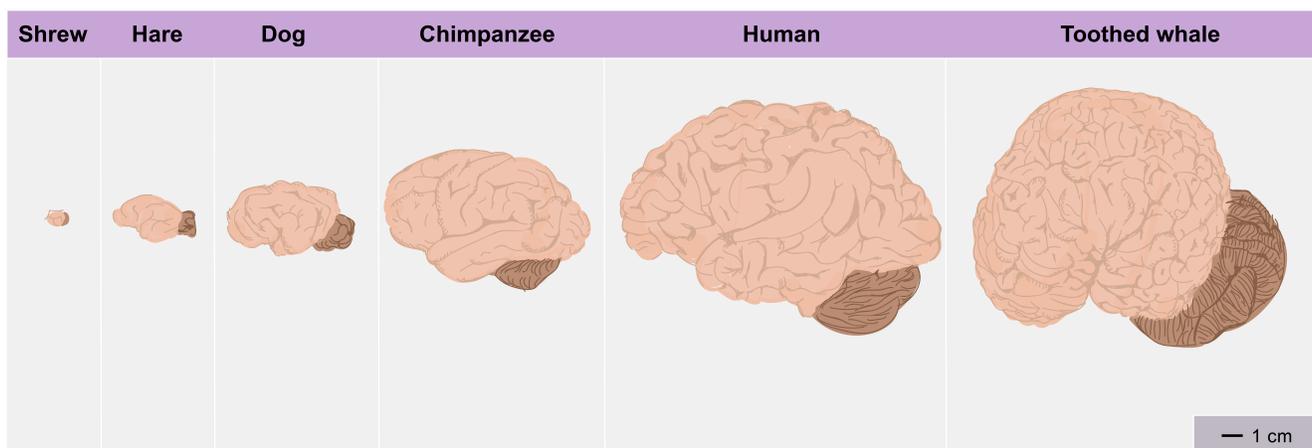
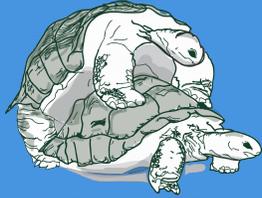


Figure 10.15 Mammalian Brain Size. Some scientists have proposed that large brains evolved as a result of sexual selection for greater cognitive abilities. While humans do have large brains, especially compared with other great apes, we do not have the largest brains of any mammal. Other mammals, like the whales and elephants, have much larger brains than humans, but do not exhibit similar behaviors. This raises the question, what is the purpose of having a larger brain?

10.12 Understanding human mating through language and culture



Biology is Sexy!

Researcher Geoffrey Miller has hypothesized that many human behaviors, like humor, music, visual art, altruism, verbal creativity, and extended vocabulary are the products of sexual selection. Miller proposed that the apparent redundancy of words in the human lexicon is the result of individuals using vocabulary to demonstrate their intelligence and consequently their fitness to potential mates. In experiments, researchers have demonstrated that men make greater use of more unusual words when in a romantic mindset, compared with men in a non-romantic mindset. This suggests that at least one use of an extended vocabulary is likely to gain access to mates. Some scientists go a step further and have hypothesized that human language as a whole evolved through sexual selection.

Geoffrey Miller has also suggested that human culture evolved through sexual selection for more creative traits in humans. Under this hypothesis, many human artifacts may have actually started as attempts at mate attraction. For example, clothing may have been used to enhance sexually desirable traits. Some scientists suggest that human intelligence, like our exceptional capacity for abstract reasoning, musicality, artistry, language and social guile, are examples of the handicap principle. Just like the peacock's tail, only those individuals in good health and with good genes should be able to produce such signals like high intelligence and musicality. An extension of the handicap hypothesis in humans posits that human intelligence is a courtship indicator of health, specifically resistance to parasites and pathogens. The logic follows that parasites and pathogens are often very deleterious to human cognitive capabilities, and therefore an individual with high intelligence is likely to be free of such infections. For example, Lyme disease, contracted most often through a tick bite, can severely impact the memory of victims of the disease, even years after the disease has been cured.

Read More

Here is the link to Geoff Miller's web page: <https://www.primalpoly.com/>

10.13 Understanding the naturalistic fallacy

Many things in our world are natural, but are not necessarily good. For example, arsenic is naturally occurring, but if you ingest this substance you will gravely suffer and you might die. In the same vein, many animals pose a threat to human survival and should not be approached. Assuming that something that is natural is “right” or “good” is referred to as “the naturalistic fallacy”.

We discuss the naturalistic fallacy here because it is important in our discussion of human evolution, specifically when we discuss sexual coercion in humans. The biggest problems with discussing human evolution arises when we begin to think that explaining why a particular behavior evolved amounts to justifying that behavior. For example, a person researching cancer wants to gain a greater understanding of the illness, in an effort to stop the disease progression, and ultimately prevent the disease altogether. No person researching cancer is thereby justifying or promoting cancer.

It is important however, that we as evolutionary biologists, take special care of topics related to human behavior. It is critical that we do not use our science to confirm our pre-existing biases, as we have seen this play out many times in history, in which ideas about evolution have been co-opted to justify inhumane practices like eugenics, racism, sexism, and xenophobia.



Check Yourself



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<https://open.lib.umn.edu/evolutionbiology/?p=1787#h5p-57>

10.14 Wrapping Up: Understanding the silent crickets



Understanding the silent crickets

Just like most animals, males of the Pacific field cricket, *Teleogryllus oceanicus*, signal to attract females. This song can grab the attention of female crickets from very far away. However, as you can imagine, the song is loud enough that it attracts predators as well. On the islands of Hawaii, this song attracts females of a parasitoid fly that lay eggs inside of singing males. These eggs then develop into larvae within the male cricket, and essentially eat the male from the inside out-killing him in the process. The larvae then emerge from the dead male cricket as flies that continue the same process as their mothers.

In this system, the presence of an alternative mating strategy, called satellite behavior, allows males to balance the relative costs and benefits associated with calling to mates. As you might imagine, any mechanism that would allow males to avoid the costs of this parasite would give these males a huge evolutionary advantage. So how do males attract females without attracting these deadly flies? Because of the relative risks of singing to attract females, male *T. oceanicus* will sometimes choose not to signal and instead will hang out near other males that are singing. Then when females approach that are attracted to the song that the singing male is producing, the non-signaler will intercept them. It's almost as if the signaling male is acting as a "wing-man" to the non-signaling male. However, the signaling males do not get any benefit from having these non-signalers hang around them- they lose out on opportunities to mate with approaching females and they are more likely to be infected by fly larvae.

As a result of these behaviors, the Pacific field crickets in Hawaii have evolved remarkable morphological and behavioral novelties. For example, pre-existing flexibility in this satellite behavior has made way for a novel wing mutation, "flatwing", that has caused individuals to lose all of the necessary wing structures for producing the songs used for attracting females. Subsequent selection on satellite behavior has led flatwings to gain greater success by targeting signaling individuals who are most likely to attract females. These mutant flatwing males now comprise 90% of the population on Kauai and 50% of the population on the island of Oahu.

Read More

Read more about this fascinating working taking place at the University of Minnesota [here](#).

As we conclude this chapter and prepare for in-class discussion, be sure to return to the chapter's [goals and objectives](#).

Chapter 11: Mating Systems

This chapter is under development!

Chapter 12: Sexual Orientation and the Evolution of Homosexuality



[12.0 Introduction](#)

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[12.2 What do we mean by “Sexual Orientation?”](#)

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[12.7 Fraternal birth order and the uterine environment](#)

[12.8 Why is homosexuality an evolutionary “problem?”](#)

[12.9 How did homosexuality evolve?](#)

[12.10 Testing some of the hypotheses about the evolution and occurrence of homosexuality](#)

[12.11 Understanding homophobia](#)

[12.12 Wrapping up: And tango makes three](#)

12.0 Introduction

Which one of these penguins is male?



Figure 12.1 There are known to be gay penguins couples all over the world.

1

Both of the penguins are male. In fact, they are a relatively famous couple of zoo penguins; in their desire to become fathers, they actually have attempted to steal eggs from other penguin couples, apparently going so far as to attempt deceit by leaving rocks in the place of the stolen eggs.

Read More

A related story involves the internationally renowned penguin dads, Jumbs and Kermit. If you're not familiar with their story, check out: <http://www.bbc.com/news/uk-england-kent-27405652>

Similarly, a same-sex penguin couple at the Central Park Zoo raised the now-famous Tango, star of the children's book, *And Tango Makes Three*.



1. <http://www.telegraph.co.uk/news/newsttopics/howaboutthat/3530723/Gay-penguins-steal-eggs-from-straight-couples.html>



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<https://open.lib.umn.edu/evolutionbiology/?p=1684#oembed-1>

Clearly, there is something compelling about same-sex penguin couples. Our attraction (or aversion) to these stories is itself interesting, and leads to a lot of biologically relevant questions about sexual attraction.



Consider the following questions

In this discussion of Sexual Orientation and the Evolution of Homosexuality, we'll address the following questions:

1. What is "sexual orientation"?
2. How widespread are same-sex mating preferences?
3. What factors contribute to the development of homosexuality?
4. Is homosexuality innate? Or influenced by environmental variables?
5. Why is homosexuality an evolutionary "problem?"
6. How can we understand the evolution of homosexuality?
7. What is homophobia?
8. How pervasive is homophobia in human cultures? How can we understand the widespread occurrence of homophobia?

12.1 Chapter Objectives



Learning Objectives

In this discussion of Sexual Orientation and the Evolution of Homosexuality, we'll address the following questions:

1. What is “sexual orientation”?
2. How widespread are same-sex mating preferences?
3. What factors contribute to the development of homosexuality? Is homosexuality innate? Or influenced by environmental variables?
4. Why is homosexuality an evolutionary “problem”?
5. How can we understand the evolution of homosexuality?
6. What is homophobia? How pervasive is homophobia in human cultures? How can we understand the widespread occurrence of homophobia?

But before we continue, let's learn a bit about your thought on the subject. Click T/F/IDK on the following:

- Homosexuality is widespread in non-human animals
- Homosexuality is widespread in human cultures
- Bisexuality is more common than strict, lifelong homosexuality
- Bisexuality is extremely uncommon
- There is no compelling evidence that homosexuality is genetically influenced
- Birth order may be associated with the occurrence of homosexuality
- Homosexuality is the product of evolution
- Homosexuality may be the product of natural selection
- Homophobia is only seen in humans
- Homophobia may be the product of natural selection

Lastly, what specific questions do you have about homosexuality? Your questions will be anonymous, but your classmates will be able to “like” specific questions. Hopefully this process will help us focus our discussion.

In the following discussion our goals are for you to:

- realize that sexual orientation governs many observable natural phenomena, that themselves can be studied scientifically
- understand that homosexuality is widespread in nature—in human and non-human animals
- realize that homosexuality and homophobia may have arisen as a result of natural selection
- appreciate that our understanding of same-sex sexual preferences is part of an emerging field of study, thus, many of the scientific studies we'll mention are relatively recent and, like all science, subject to revision

By the end of your reading and our in-class discussion, you should be able to meet the following objectives:

- define sexual orientation, heterosexuality, homosexuality, asexuality, bisexuality, and pansexuality
- explain what scientists mean when they say “sexual orientation is not binary”
- defend, using examples, the statement: “homosexuality is widespread in nature”
- cite evidence for a genetic basis of homosexuality, as well as evidence that homosexuality is environmentally influenced
- explain why some biologists call homosexuality “an evolutionary problem”
- explain why many biologists think that homosexuality is an adaptation that has been selected for
- describe a few of the hypotheses for the evolution of homosexuality
- explain how homophobia could be an adaptation (how could homophobia increase an individual's fitness?)

12.2 What do we mean by “Sexual Orientation?”

Sexual Orientation is an umbrella term that is used to refer to patterns of attraction—sexual, romantic, or both. Under this umbrella, individuals may assort themselves into categories such as homosexual, heterosexual, bisexual, pansexual, and asexual.

Read More

You’ll find an excellent overview of terms at: <https://www.plannedparenthood.org/learn/sexual-orientation-gender/sexual-orientation>

Disclaimer: Planned Parenthood is seen by many in a political light. Our intent with the above link is not to be political, rather Planned Parenthood’s discussion of terms was superior to that of other sources. Our intent is to share the best available content with our students.

You’ll note from the definitions the use of qualifiers such as “may,” and “often.” This pattern should serve as a clue that sexual orientation is COMPLICATED, and our understanding of the diversity of presentations is quickly changing.

Test yourself. Match each term with its correct definition.

12.3 Sexual preference is not binary

You may have heard things like “most people are bisexual,” and “sexual preferences exist on a continuum,” but are such claims scientific? That is, do we have evidence to justify such statements?

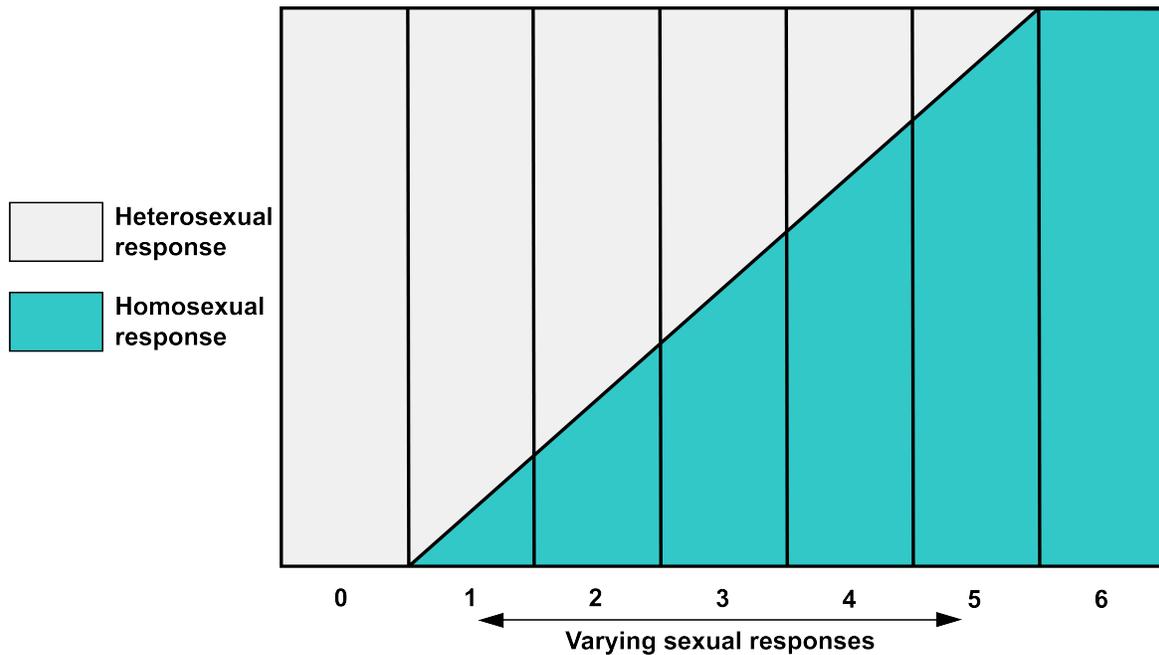


Figure 12.2 The Kinsey Scale

Some key work on sexuality was conducted in the 1940's and 1950's by the biologist Alfred Kinsey. Alfred Kinsey pioneered research in human sexuality through thousands of interviews and the development of “The Kinsey Scale” of human sexual preferences. The Kinsey Scale is a 7-point metric that categorizes individuals from 0 (exclusively heterosexual) to 6 (exclusively homosexual), and includes the midpoint 3 (equally homosexual and heterosexual).



Kinsey's main contributions were to (1) reveal that many people have preferences that aren't "0" or "6"—in other words, sexual preferences *do* exist on a continuum; and (2) revolutionize how we view female sexuality—that is, women are not just recipients of sex, women have sexual desires, and women cheat, fantasize, and masturbate. For many people, these ideas may be obvious, but at the time they were shocking and revolutionary.

Figure 12.3 Alfred Charles Kinsey

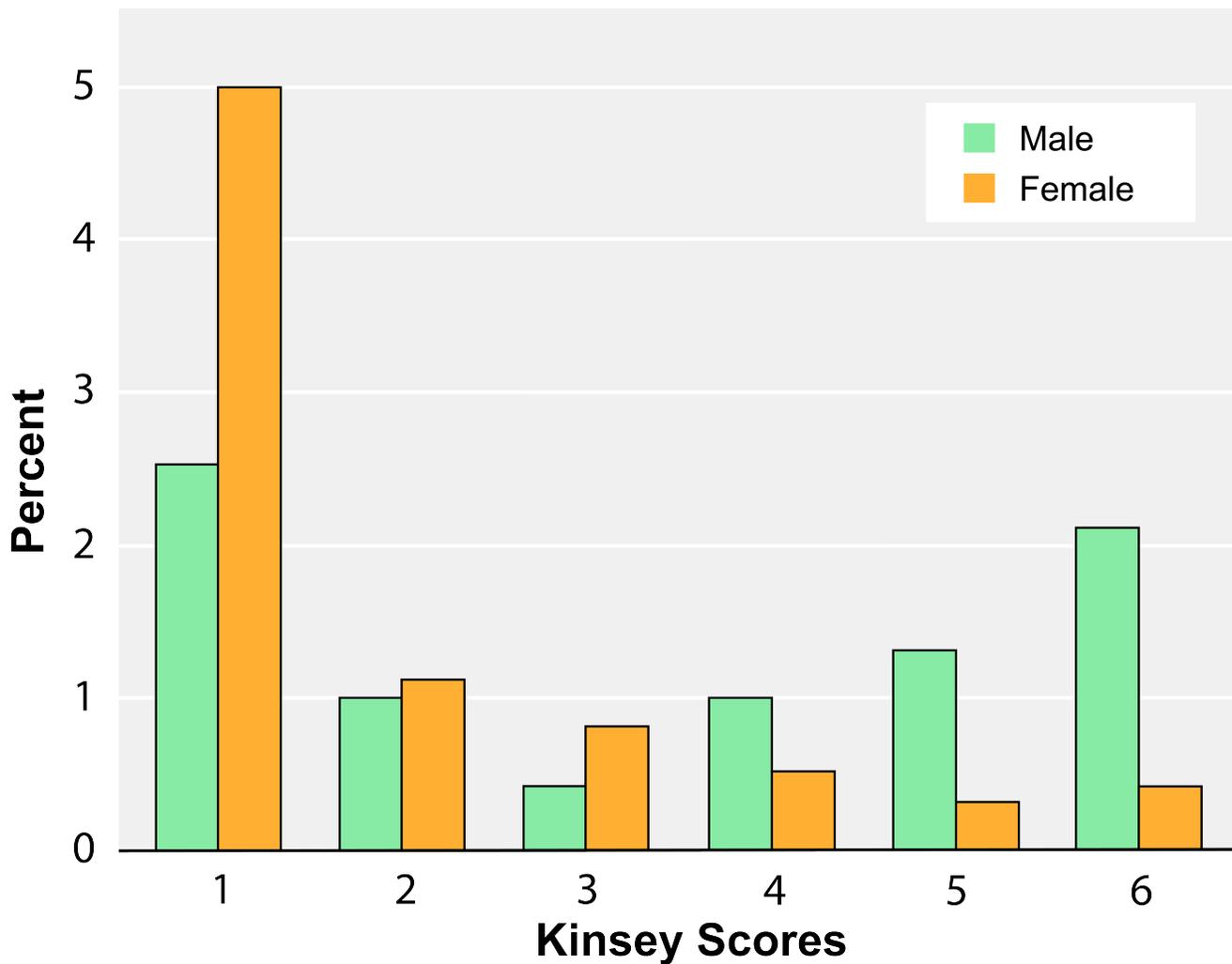


Figure 12.4 Above: distribution of Kinsey scores for 147 men and 238 women (who were not exclusively heterosexual) in an Australian sample from 2000.

More recent work has investigated the “continuum” concept of sexuality, with a focus on the prevalence of bisexuality. For example, an analysis of several reports revealed the presence of bisexuality in from ~2% to ~6% of individuals who identified as heterosexual, and from ~18% to ~88% in self-identified homosexuals. In the latter example, far more women, on average, expressed bisexual tendencies than did male homosexuals. In sum, bisexuality is fairly common, and sexual preference is not binary.

Diamond, L. M. Sexual Fluidity in Male and Females. Curr. Sex. Heal. Reports 8, 249–256 (2016).

Read More

- “Biography” feature on Alfred Kinsey: <https://www.biography.com/video/alfred-kinsey-full-episode-2071931808>
- Learn more about Alfred Kinsey at The Kinsey Institute: <https://kinseyinstitute.org/about/history/alfred-kinsey.php>



Check Yourself



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12.4 Homosexuality is widespread in nature

Same-sex sexual behavior is well documented in non-human animals, far beyond the penguin examples above. And, as with the penguin examples, it can be difficult for humans to detect same-sex couples in animals, especially when it is difficult to tell males and females apart. For example, the Laysan albatross is another “sexually monomorphic” (i.e., males and females look alike) bird species in which same-sex pair-bonds—in this case, in females—are common.

These birds are well known for their intricate courtship displays:



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<https://open.lib.umn.edu/evolutionbiology/?p=1399#oembed-1>

And, until recently, biologists didn't realize that same-sex partnerships were so common—in one location, 31% of the pairs studied were female-female. Upon further investigation, it became evident that these female-only birds were successfully hatching eggs and raising chicks.

Similarly, same-sex sexual behavior, of either a long-term or transient nature, has been documented in examples such as (but by no means limited to):

- Mammals such as gorillas, chimpanzees, bats, dolphins, bison, and humans
- Birds such as penguins, albatross, zebra finches, and acorn woodpeckers
- Reptiles such as garter snakes
- Amphibians such as the American toad
- Fish such as guppies and cichlids
- And many, many types of invertebrates such as squid, snails, fruit flies, and roundworms.

In some cases (such as Japanese beetles), the incidence of homosexuality is low, and not a large part of the animal's social structure. But in some animals, homosexuality is common enough to have significant impacts at the community level. For example, in bottlenose dolphins, ~50% of male sexual interactions are with other males.



a)



b)



c)



d)

Figure 12.5 a) dolphin; b) garter snake; c) Japanese beetle; and d) American toad

1 2 3 4

In addition, homosexuality is pervasive in human cultures, from around the world and throughout history. Recently, biologists at the University of Montpellier (in France) conducted a review of 107 societies from around the world, finding evidence of male homosexual preference in the majority of societies sampled. Thus, it seems fair to conclude that homosexuality is widespread in nature—in humans and non-human animals.

1. Barthes, J., Crochet, P. A. & Raymond, M. Male homosexual preference: Where, when, why? *PLoS One* **10**, 1–15 (2015).

1. from: https://en.wikipedia.org/wiki/Bottlenose_dolphin#/media/File:Tursiops_truncatus_01.jpg

2. From: https://commons.wikimedia.org/wiki/Popillia_japonica#/media/File:Popillia_japonica_-_japanese_beetle_-_mating_pair_on_filbert_tree_leaf.jpg

3. From: https://commons.wikimedia.org/wiki/File:Coast_Garter_Snake.jpg

4. From: https://commons.wikimedia.org/wiki/File:Bufo_americanus_PJC1.jpg



Figure 12.6 Locations studied by the University of Montpellier where male homosexual preference is dominant.

12.5 Is sexual orientation genetic?

Asking this question is a bit like asking, “are we born gay? Or straight?” This question can be problematic for some, because the motivation for asking the question may not be scientific. For example, individuals who have a social problem with homosexuals may be motivated to see sexual orientation as a choice, making homosexuality a characteristic one could choose *not* to exhibit. And in recent history, eugenicists (individuals who promote selective reproduction among “favored” types of humans) used a presumed genetic basis for homosexuality as an argument in favor of sterilizing gay people. The question can also be problematic because the stated or implied focus is typically on the cause of homosexuality, rather than heterosexuality. (We’ll say more about that in a bit.)

But, for now, let’s focus on the biology of homosexuality’s origins. While no serious scientist is claiming that same-sex mating preferences arise in a simple Mendelian fashion, or that there is a single “gay gene,” many have found evidence of a possible genetic basis. Some intriguing data are from the literature on twins. For example, X and Y discovered that *identical* twins (who arise from the same sperm and egg, and have nearly 100% identical genetics) are more alike with respect to sexual orientation than are *non-identical* twins (who arise from different eggs and sperm). However, identical twins don’t overlap completely in sexual preferences, a finding that suggests other factors—besides genetics—may be at work.

12.6 Is sexual orientation influenced by the environment?

Several studies have found correlations between same-sex sexual preferences and environmental conditions. In this case the “environment” can be the uterine environment, and refer to conditions during fetal development, or the environment can refer to conditions after birth.

The literature on post-birth experiences, and their impacts on sexual orientation, is challenging for many reasons, but largely because it is so difficult to disentangle the impact of a tolerant environment on a homosexual’s inclination to express their homosexuality. For example, there has been work suggesting that children of gay parents are more likely to grow up expressing same-sex sexual preferences. Is this because growing up in a gay family actually influences an individual’s sexuality, or because a family that is accepting of homosexuality creates a safe space for a homosexual or bisexual individual to express their sexuality?

Similarly, work in Denmark has shown that growing up in an urban environment is associated with the choice to marry a person of the same sex later in life. Diverse metropolitan areas are typically associated with greater tolerance towards gays and lesbians, so is it simply that this tolerance supports the expression of an existing characteristic, or is there something else about cities that promotes homosexuality? A summary from the Danish study includes the following statements: “For men, homosexual marriage was associated with having older mothers, divorced parents, absent fathers, and being the youngest child. For women, maternal death during adolescence and being the only or youngest child or the only girl in the family increased the likelihood of homosexual marriage.”¹

Somewhat more compelling is the work on the prenatal environment and homosexuality. According to many of these studies, differential exposure to prenatal hormones, specifically testosterone, influences sexuality later in life.

Several studies have found evidence, through the development of certain body parts (e.g., fingers, ears) that lesbians were exposed to more testosterone *in utero* than were straight women. For example, finger (or “digit”) lengths, especially the ratio between the second (2D) and fourth (4D) fingers, seems to vary as a function of exposure to testosterone in the womb. The result is that, *on average*, the 2D:4D ratio is larger in women than men. Note that these differences are rarely noticeable without doing precise measurements of an individual’s finger lengths.

1. Frisch, M., Hviid, A., Frisch, M. & Hviid, A. Childhood Family Correlates of Heterosexual and Homosexual Marriages : A National Cohort Study of Two Million Danes Childhood Family Correlates of Heterosexual and Homosexual Marriages : A National Cohort Study of Two Million Danes. (2006). doi:10.1007/s10508-006-9062-2

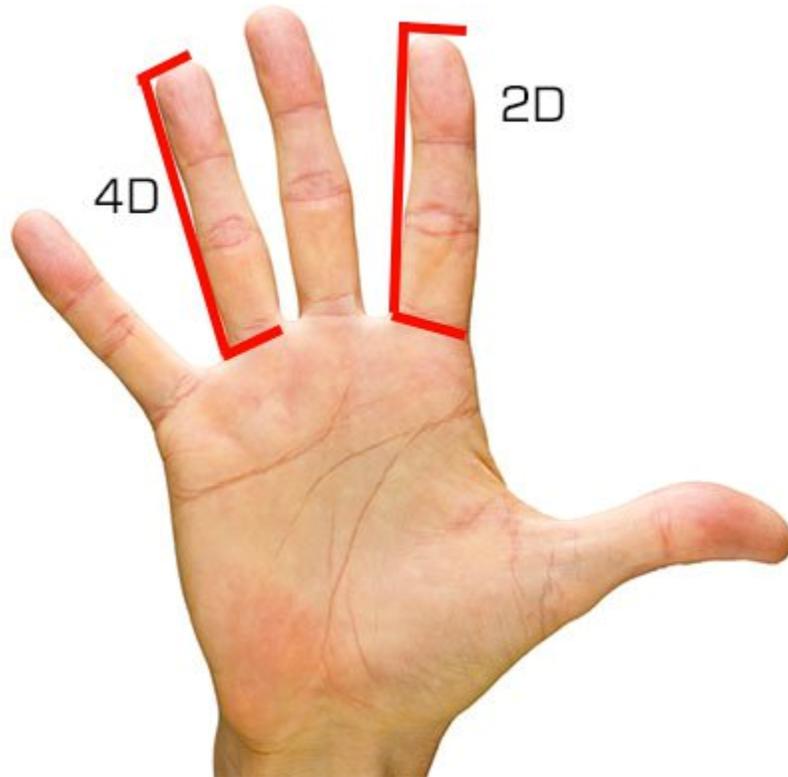


Figure 12.7 a) Visual representation of 2D and 4D

Check out the data represented in the following figure.²



Check Yourself

Figure 12.7b) Comparison of 2D:4D ratio



An interactive H5P element has been excluded from this version of the text. You can view it online here:
<https://open.lib.umn.edu/evolutionbiology/?p=1406#h5p-81>

Before we continue, consider the data represented in the following two figures.³ **What do they mean?**

2. from: Williams, T. J. et al. Finger-length ratios and sexual orientation. 404, 455–456 (2000).

3. from review of data in: Blanchard, R., Dickey, R. & Klassen, P. The relation of birth order to sexual orientation in men and women. (1998). doi:10.1017/S0021932098005112

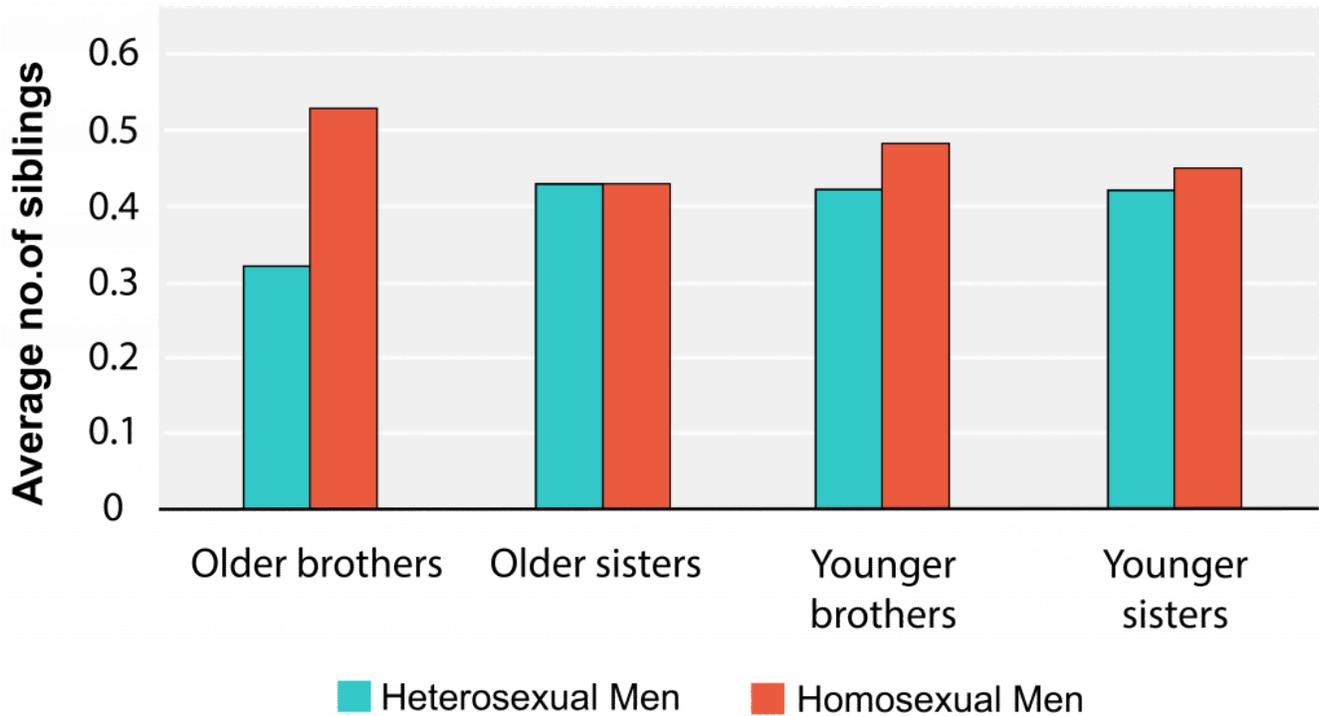


Figure 12.8 Siblings and Sexual Orientation: Males. The average number of siblings of each type, for both heterosexual (in blue; $n=225$) and homosexual (in orange; $n=385$) men.

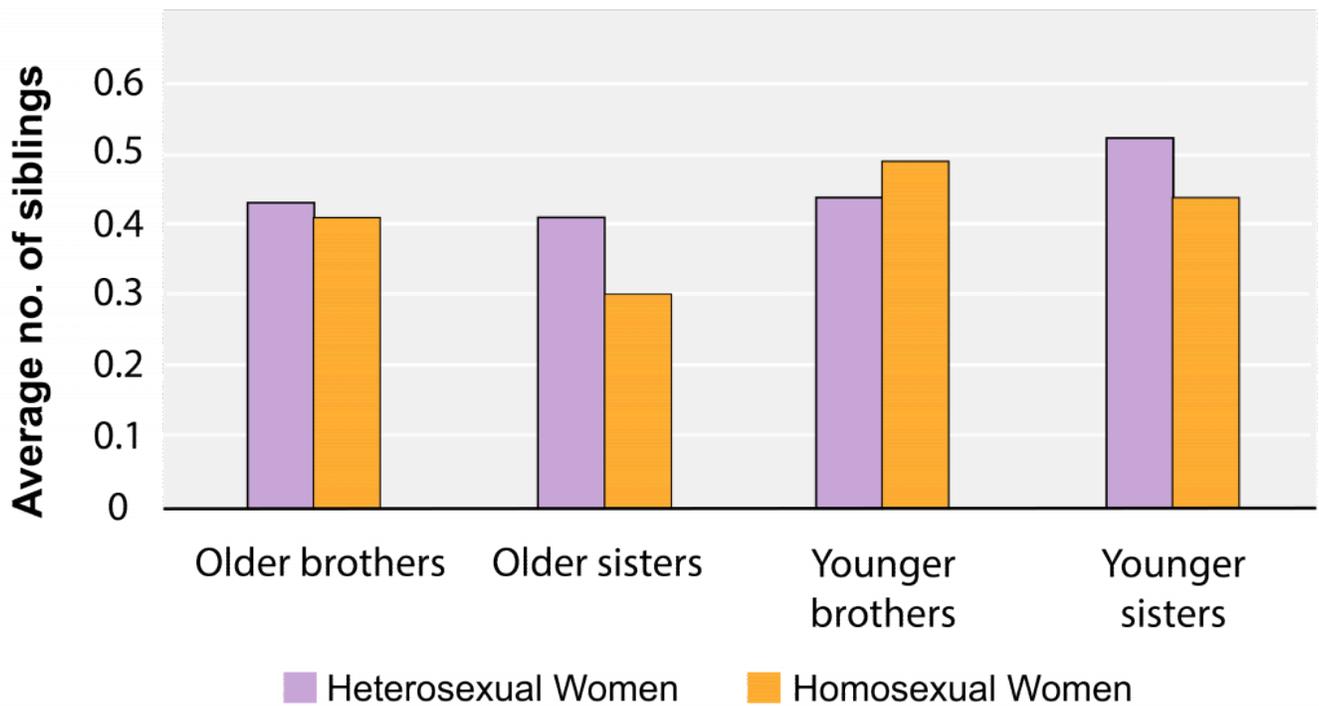


Figure 12.9 Siblings and Sexual Orientation: Females. The average number of siblings of each type, for both heterosexual (in blue; $n=192$) and homosexual (in orange; $n=162$) women.

12.7 Fraternal birth order and the uterine environment

What is the fraternal birth order effect?

In males, it appears to be that number of older brothers alters the likelihood of same-sex preferences later in life. Specifically, more older brothers (not sisters) is associated with homosexuality in men (not women). This is called the fraternal birth-order (FBO) effect in the scientific literature, and the evidence for the FBO effect is compelling. Simply, homosexual men, on average, have more older brothers than do heterosexual men, a difference that is not seen in homosexual versus heterosexual women.

A logical response to this finding would be to wonder whether growing up with older brothers somehow led more men to develop with same-sex sexual preferences, or if there was something about the uterine environment that favored homosexuality in successive male offspring.

Reflection Question: How would you address these differences scientifically?

Anthony Bogaert was interested in the FBO effect and whether it was due to exposures in the uterus during fetal development, or somehow due to the impact of growing up with older brothers. He tested this by analyzing data on sexual preferences in several groups of men, including one sample of men raised in step- or adoptive families. That is, he was able to compare homosexuality in men raised with their older brothers, and those raised apart from their older, biological brothers. He found that only biological older brothers were associated with male homosexuality, regardless of the amount of time spent with those older brothers. Bogaert used these data to suggest that it is uterine conditions, not how a person is raised, that is associated with same-sex sexual preferences in men.



Read More

For an accessible summary of the FBO effect, read: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1502267/pdf/zpq10531.pdf>

Caution: These differences in uterine influences on male and female homosexuality also illustrate a key point: male homosexuality and female homosexuality appear to have different causes, thus we should be careful not to transfer the findings of research on men to the reality of sexuality in women. Further, male and female homosexuality are likely influenced by multiple factors.

12.8 Why is homosexuality an evolutionary “problem?”

Some biologists have referred to the *evolutionary* problem with homosexuality, because same-sex sexual behavior is non-reproductive, yet homosexuality occurs in relatively high numbers—enough to support an adaptive function for homosexuality. So the big question is: **how can natural selection work on a trait that seems unable to increase an individual’s fitness?**



Points to Ponder

What do you think? How can a trait that is associated with non-reproductive sex be selected for? How could homosexual preferences increase an individual’s fitness? Answer in full sentences.

12.9 How did homosexuality evolve?

The literature on the evolution and occurrence of homosexuality has focused on several hypotheses, including some that are adaptive (fitness-enhancing) explanations and several that are non-adaptive explanations.

Suggested adaptive explanations include (but are not limited to),

1. **Social glue:** according to the social glue hypothesis, same-sex sexual interactions help to form bonds, reduce tension, repair relationships after conflict, and prevent future conflicts from occurring
2. **Kin selection:** this hypothesis centers on the idea that individuals can increase their fitness either by direct mechanisms (having their own offspring) or by indirect mechanisms (investing in, or somehow providing a benefit to, the offspring of their relatives. A homosexual individual might forego having his or her own direct offspring, but could benefit the family (and help get their own genes into the next generation) by investing in siblings, nieces, nephews, etc.
3. **Alliance formation:** similar to social glue, the alliance formation hypothesis posits that bonds forged during sex lead individuals to greater acts of bravery or sacrifice, to benefit those with whom they've been intimate. If same-sex sexual relationships lead to stronger alliances, and these alliances make better warriors or soldiers who are more likely to survive conflicts, that would lend support for the alliance formation.
4. **Practice:** according to the practice hypothesis, same-sex activities during immature stages make an individual more adept at courtship and copulation, with opposite-sex partners, as an adult.
5. **Enhanced family fertility:** according to the enhanced fertility hypothesis, some of the genetic components that can lead to homosexuality are also associated with enhanced fertility or success in getting mates. From this hypothesis we would predict that individuals who share genetic information with homosexual individuals would have greater reproductive success than those who do not.

Non-adaptive explanations include,

1. **Mistaken identity:** as the name suggests, some homosexual interactions may simply be the result of mistaken identity.
2. **Prison effect:** according to this explanation for the occurrence of homosexuality, individuals deprived of the opposite sex may resort to same-sex copulations to meet a biological urge to copulate.

You can probably imagine ways to test all of the above explanations, as well as potential problems associated with each suggestion. We'll consider a couple of these hypotheses, but keep in mind that this list is not exhaustive, and a thorough treatment of each hypothesis is not practical here.

12.10 Testing some of the hypotheses about the evolution and occurrence of homosexuality

Belgian biologists conducted a series of experiments with the damselfly *Ischnura elegans*. They began by allowing individual males the opportunity to choose between a single male and a single female, and then recorded the sex of the damselflies chosen by the males for copulation. The majority of the males (49/60) chose females in these initial choice experiments. Then, each male was marked and these individual, “focal” males were, one by one, placed in an enclosure for two days with 20 other males. After two days, each focal male was given a choice between another individual male and an individual female, and the focal male’s copulation choices were recorded. In these second choice experiments, the majority of the males (40/53) chose to attempt copulation with other males.



Figure 12.10 Damselfly.

From: Gossum, H. Van, Bruyn, L. De & Stoks, R. Reversible switches between male-male and male-female mating behaviour by male damselflies (2005). doi:10.1098/rsbl.2005.0315

These findings best support which of the following hypotheses for the evolution or occurrence of homosexuality?

Kin selection

Practice

Enhanced family fertility

Mistaken identity

Prison effect

1

1. From: [https://commons.wikimedia.org/wiki/Ischnura_elegans#/media/File:Thomas_Bresson_-_Agrion_%C3%A9l%C3%A9gant_\(by\).jpg](https://commons.wikimedia.org/wiki/Ischnura_elegans#/media/File:Thomas_Bresson_-_Agrion_%C3%A9l%C3%A9gant_(by).jpg)



Figure 12.11 Fruit fly, male.

In their study of the impact of male-male courtship in the fruit fly (*Drosophila melanogaster*), Scott McRobert and Laurie Tompkins note that “we have shown that *D. melanogaster* males that are courted [by males] when they are young subsequently copulate more quickly with females than do males that never elicited courtship [by other males]. The ability to copulate quickly is advantageous in these animals, and can lead to greater reproductive success. These findings lend support for which of the following hypotheses?

From: McRobert, S.P. and Tompkins, L. Two Consequences of Homosexual Courtship Performed by Drosophila melanogaster and Drosophila affinis Males Evolution 42, 1093–1097 (2017)

2

One study looked at family fertility data from homosexual and heterosexual males in Spain, Italy, and France. Consider the data in the following figure:

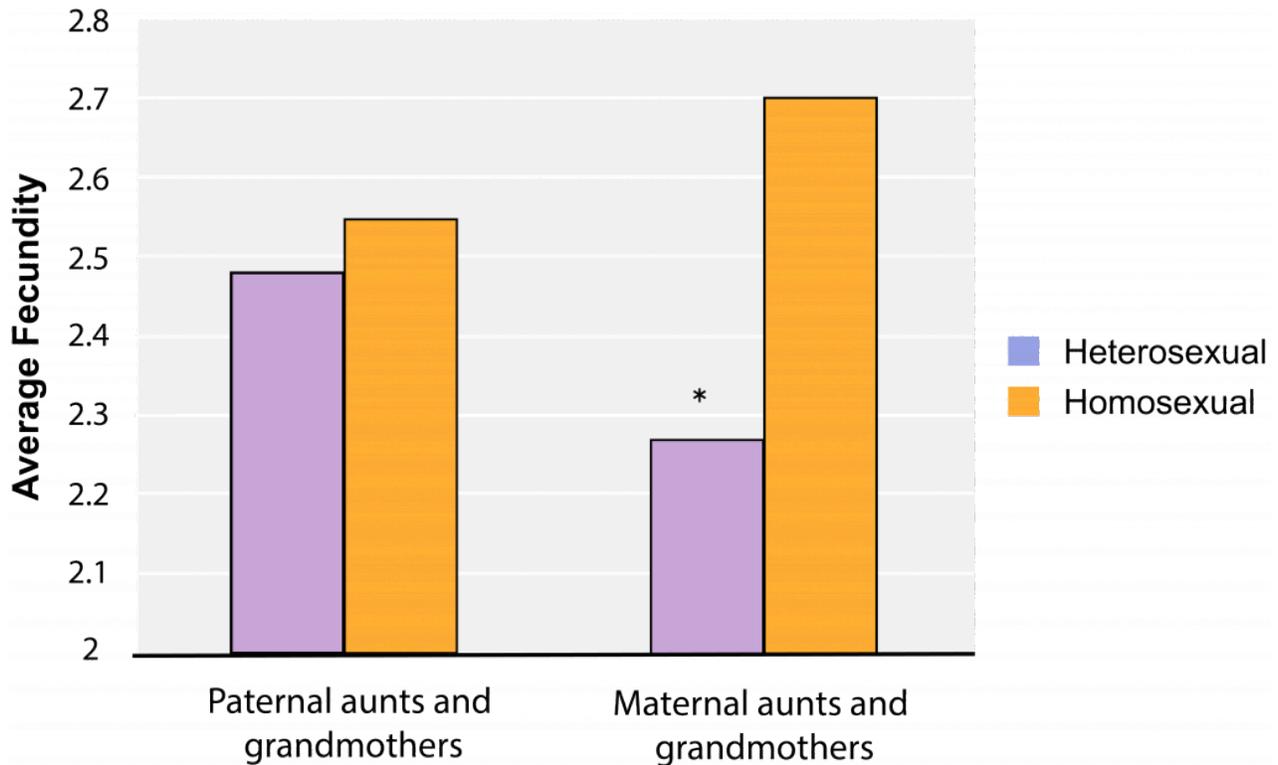


Figure 12.12 Average fecundity (number of offspring) of female relatives on mother’s and father’s side of homosexual and heterosexual males. * Indicates a statistically significant difference between homosexual and heterosexual males. From (Camperio Ciani and Pellizzari 2012)

Hopefully it is clear, from the examples above, that attributing “causality” to a single factor as a way to understand homosexuality is complicated, and probably misguided. Rather, we may come to realize that sexual orientation is influenced by many factors that are themselves context-dependent. No doubt our understanding of the origins of sexuality will develop rapidly in the near future.

12.11 Understanding homophobia



Read More

Hate Crimes

- Anti-gay hate crimes: <https://www.splcenter.org/fighting-hate/intelligence-report/2011/gays-remain-minority-most-targeted-hate-crimes>
- Data: <https://www.splcenter.org/fighting-hate/intelligence-report/2011/anti-gay-hate-crimes-doing-math>

Gay Rights Around the World

- <https://www.washingtonpost.com/graphics/world/gay-rights/>

Given that homosexual behavior is so widespread, and given the evidence in support of homosexuality as an adaptation, why is [homophobia](#) so common?

Does homophobia exist in non-human animals?

We were unable to find any clear evidence of homophobia in other animals, but that doesn't mean it doesn't exist. However, what would homophobia look like, in, for example, penguins? As a side note, please let us know if you find any compelling evidence of homophobia beyond humans!

Could homophobia be adaptive?

The question of whether or not homophobia is adaptive is one that has been scarcely addressed in the scientific literature. In this [Scientific American blog post](#), author Jesse Bering discusses several papers published in the mid 1980's through the mid 1990's, in which two authors present conflicting ideas about whether a child's sexual orientation is malleable (can be influenced by environment, early sexual experiences, or interpersonal connections), and if such malleability could influence the adaptability of homophobia. Gordon Gallop argues that if homophobia were adaptive, you would predict to see it expressed most as it concerns contact of homosexual individuals with children. Likewise, if parental influence can affect sexual decision making, one could argue that the parent who does not express acceptance for homosexuality may influence their offspring to have more heterosexual sex, resulting in more descendants. Interestingly, as Bering points out, there has not been much follow-up research to further address the question of the adaptive nature of homophobia. Furthermore, it is important to remember that just because something might be adaptive does not inform any moral stance for that issue (being adaptive does not make it "right").



Consider the following questions

1. What makes some individuals homophobic, and others not?
2. What is the cost to the individual, if any, of being homophobic?
3. Is homophobia genetic?
4. Finally, is it possible that homophobia is an adaptation?

12.12 Wrapping Up: And Tango Makes Three



And Tango Makes Three

Remember “And Tango Makes Three”, the children’s book about two male penguins, Roy and Silo, who adopted an egg? That book became notorious, making the American Library Associations top-ten list of “most challenged [or banned] books” during the most recent decade: <http://www.ala.org/bbooks/top-100-bannedchallenged-books-2000-2009>

Soon after the book’s publication, and after a six-year partnership, Silo left Roy for a female penguin named Scrappy. Reactions to the split were mixed but, as Roberta Sklar, a spokeswoman for the National Gay and Lesbian Task Force, said: “There’s almost an obsession with questions such as, ‘Is sexual orientation a birthright or a choice?’ And looking at the behavior of two penguins in captivity is not a way to answer that question.” She continued by noting that the public outcry (over the book, the penguin pair, and then their split) “is a little ridiculous. Or maybe a lot ridiculous.”



Figure 12.13 Chinstrap

1

However, as we discussed on pages 8.3 and 8.4, sexual orientation is complicated, non-binary, and often fluid. In humans and in penguins. http://www.nytimes.com/2005/09/24/nyregion/new-love-breaks-up-a-6year-relationship-at-the-zoo.html?_r=0

As we conclude this chapter and prepare for in-class discussion, be sure to return to the chapter's [goals and objectives](#).

12.13 Read More

Want to learn more?

In addition to the links and citations in the reading, the following peer-reviewed papers were useful in developing this chapter.

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Chapter 13: Sex Machine: Reproductive Anatomy and Physiology



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[13.3 Commonalities between male and female reproductive anatomy](#)

[13.4 Male reproductive anatomy](#)

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[13.9 Wrapping up: Revisiting circumcisions](#)

13.0 Introduction

Circumcision

In 2018 there was a bill introduced in Iceland to outlaw routine, parent-elected circumcision on infant boys. People who argued for this legislation argued that infants could not consent to this body alteration, that it lowers sensitivity of the penis, affecting sexual function, and that, in all but a few rare cases, the procedure is not medically necessary and presents risks to the child. Those arguing against the ballot proposal said that the procedure was minor, safe, and had both cultural and religious significance and some medical value. The measure was later dropped by Iceland's parliament, but a similar prohibition was previously proposed in the state of California and the legality of the practice has been challenged in many other countries. Routine male circumcision is still hotly debated in some circles. In the United States male infant circumcision rates vary widely by region and race, with the highest rates in white infants in the Midwest and the overall rate at just under 60%. What is male circumcision? What part of the body does it affect? Is it a good idea? How does male circumcision compare to female circumcision/female genital cutting? Some of these questions can be answered with knowledge of reproductive anatomy. Some of these questions are not able to be answered by science, but opinions on the topic can be informed by science. At the end of this chapter we will revisit these questions with our knowledge of reproductive anatomy, and look at some data about circumcision and female genital cutting.

13.1 Chapter Objectives



Learning Objectives

The goals of this chapter are to give you a foundational understanding of human reproductive anatomy and how our anatomical structures, working in concert with the endocrine system, function to make baby humans.

1. Define the following terms:
 - **Sexual dimorphism**
 - **Secondary sex characteristics**
 - **Endocrine system**
 - **Menstruation**
 - **Ovulation**
 - **Follicular phase**
 - **Luteal phase**
2. Describe and give examples of positive and negative feedback loops in reproductive signaling
3. Compare and contrast male and female reproductive signaling
4. List the major parts of the male and female reproductive systems and their functions:
 - Hypothalamus
 - Pituitary gland
 - Penis
 - Testis
 - Scrotum
 - Seminiferous tubules
 - Epididymis
 - Seminal vesicle:

- Bulbourethral (or Cowper's) gland
- Prostate gland
- Ductus (or vas) deferens
- Ejaculatory duct
- Penis
- Urethra
- Follicle
- Corpus luteum
- Vagina
- Clitoris
- Labia minor
- Labia major
- Cervix
- Uterus
- Oviducts
- Ovaries

5. List the important signaling hormones and what they do in male and female reproductive systems
6. Describe the three phases of the female reproductive cycle and what happens during each phase in the uterus, the ovary, and the hormone levels
7. List some ways that medications can impact reproductive functions

13.2 Every body is different.

Every body is different. But many things about our bodies are the same.

Humans come in a variety of shades, sizes and proportions, yet in total, our bodies are more similar to each other than they are different. In fact the human body shares similarities with bodies across the diversity of life. We share aspects of our reproductive system with all mammals, aspects of our basic physiology with all vertebrates, and aspects of our cell structure, biochemistry and genetics with all living things. In this chapter we will look at the human body specifically, not because the human body in terms of reproduction is very distinct from that of a 3-toed sloth, or because the basic structure is very different from a Galápagos tortoise, or because our cellular biology varies much from that of the fungus that inhabits bleu cheese. Instead we focus on the human body because the authors and readers of this text presumably each have a human body, and reading about ourselves is interesting.

Humans are sexually dimorphic

Like lions, peafowl, and marine iguanas, male and female humans are often superficially different in appearance (e.g. male hair patterns, female breasts), sounds (men typically have deeper voices), and smells (males and females have characteristic odors); this phenomenon is called **sexual dimorphism** and is observed in contrast to sexually monomorphic species (e.g. Laysan albatrosses, and emperor penguins; see chapter 7 for further discussion of sexual dimorphism) in which males and females are difficult for humans to distinguish.

Further, there are generalizable differences between males and females in terms of reproductive organs, circulating hormones, and **secondary sex characteristics**. Secondary sex characteristics are traits that become more pronounced during puberty; they are generally distinct between males and females, but they are not directly reproductive (i.e., they are not primary sex characteristics). Examples of secondary sex characteristics include breast development in females and thickening of vocal chords in males. While these generalizable traits are different on average between the sexes, there is wide variation in male and female anatomy and physiology, such that there is significant overlap in many of the traits. For example human males, on average, are taller than females. However there is quite a bit of overlap between the height ranges of males and female (see figure below).

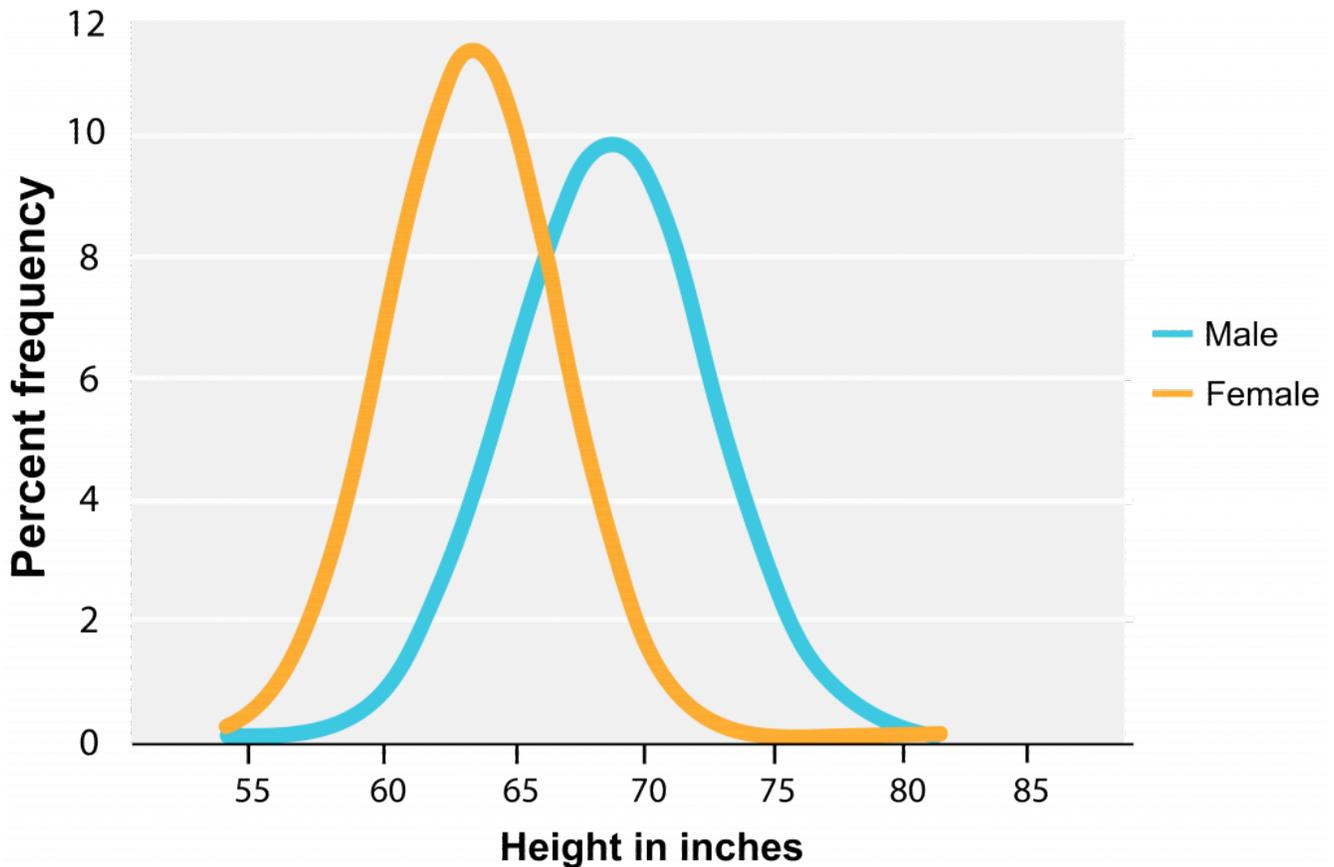


Figure 13.1 Average height for males and females in the United States.

Humans have specialized organs that aid in reproduction (primary sex characteristics). Some of these are located between the ears, some of these are located in the abdomen and some of these are located between the legs. The body orchestrates the functions of reproductive organs through small signaling molecules called hormones that circulate in the bloodstream. The **endocrine system** involves hormones that are secreted from glands in one area of the body and have an effect at one or more distant locations. The development of secondary sex characteristics and the maturation of the reproductive organs happen in response to increases in circulating hormones (primarily estrogen, progesterone and testosterone) that occurs during adolescence.



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13.3 Commonalities between male and female reproductive anatomy

During embryonic development the male and female fetus are indistinguishable before about 10 weeks of pregnancy. Fetal tissues begin in an undifferentiated state, and based on genetic signals and the interuterine environment the reproductive organs usually differentiate into structures typical of males and females (for illustrations see [chapter 8.6](#)). This means that for most of the reproductive parts there is an analogous part in the other sex that arose from the same original tissues. For example, testes and ovaries develop from the same tissue – originally located in the abdomen. In males the testes move down and outside the abdomen as they develop; in female they remain internal. Some structures (such as the oviducts) have a structure that was common in early development, but completely or partially disappears in later development; other structures (such as the uterus) have analogues that are very subtle structures in the male. See the following table for a list of analogous structures in male and female anatomy.

Structure in male	Structure in female
Glans (head) of the penis	Clitoral head
Penis shaft	V-shaped internal structure of clitoris
Foreskin	Clitoral hood
Penis skin	Labia minora
Testicle	Ovary
Scrotum	Labia majora

Figure 13.2 Some analogous structures in male and female anatomy

Commonalities between male and female reproductive signaling

Much of the reproductive physiology we will address is regulated by hormonal signals that arise in the brain and much of this signaling is shared between males and females.

Within the brain is a region called the hypothalamus (see figure 2). This portion of the brain sends signals to the pituitary gland located beneath it. In particular, the hypothalamus sends a hormonal signal called gonadotropin-releasing hormone (GRH) to the pituitary gland. In response to the GRH signal, the pituitary gland releases two hormones that circulate in the blood: luteinizing hormone (LH) and follicle stimulating hormone (FSH). These hormones travel throughout the body, triggering further hormone releases and physiological changes (discussed further below). There are feedback loops that tightly regulate the levels of circulating hormones. In addition to GRH, LH, and FSH, the hormones testosterone, estrogen and progesterone are important in reproductive

signaling. While we will focus on the effects of testosterone in males and estrogen and progesterone in females, all of these hormones are present and important in both males and females.

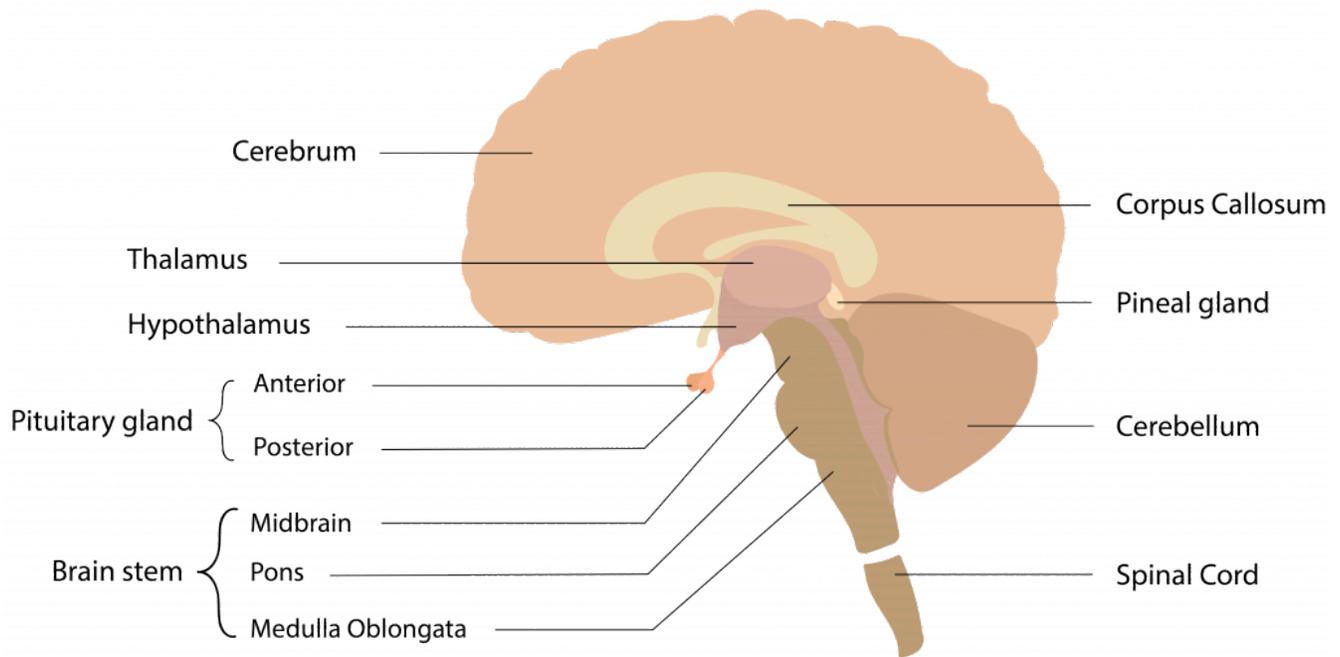


Figure 13.3 Side view of the human brain

In the following pages the reproductive anatomy and physiology of male and female reproduction are described. We begin with reproductive anatomy, which describes the organs and tissues involved in reproduction, and then go on to describe the physiology, or how these structures function together and respond to hormonal signals

13.4 Male reproductive anatomy

Some of the organs involved with male reproduction are diagrammed below.

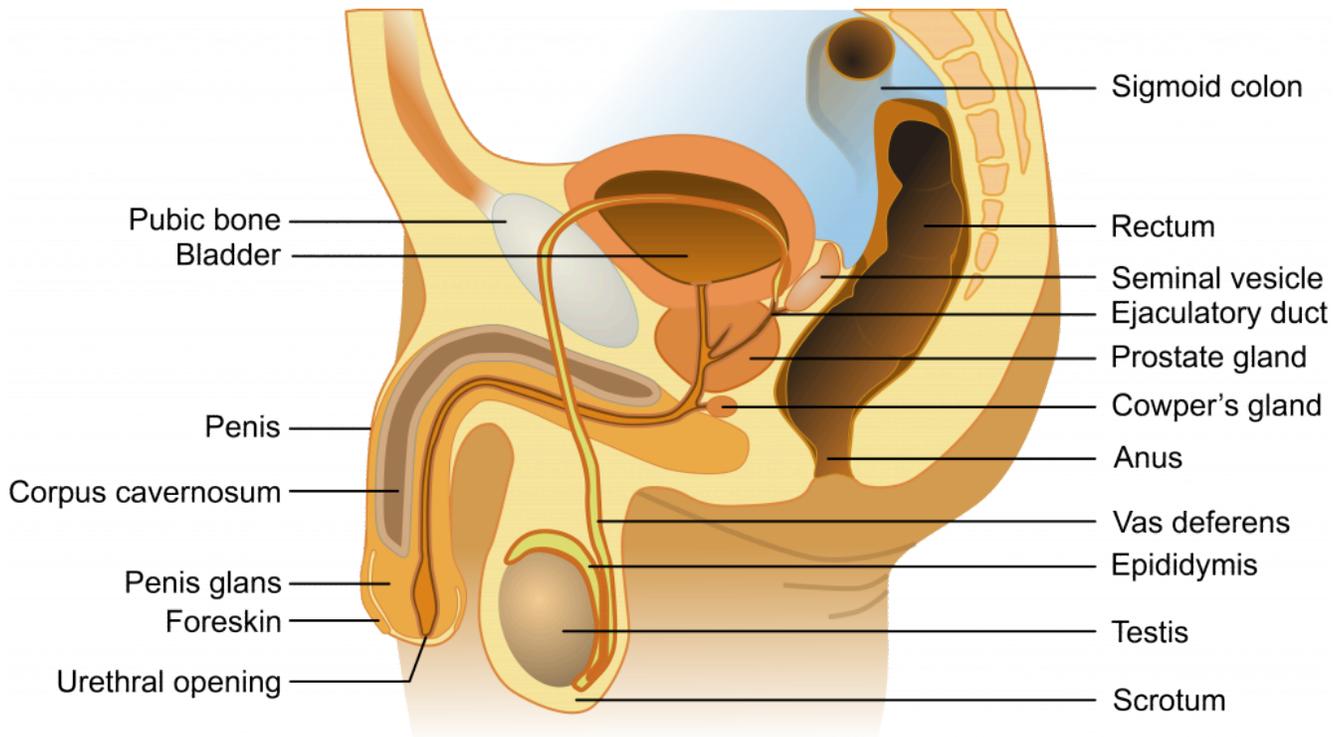


Figure 13.4 Male reproductive anatomy

Male reproductive anatomy involves the organs and glands that produce sperm, create semen to transport sperm, and conduct this liquid semen out of the body. Semen production involves the work of accessory glands, each responsible for the production of one or more key ingredients of semen. Male anatomical structures can be broken down into the following:

Sperm production

- **Testis:** males typically have two testes (also called testicles), which, in humans, descend from the abdomen during fetal development and are enclosed outside the abdomen in the scrotum. Each testis houses many tube-like structures (the seminiferous tubules) in which sperm are made. Specialized cells (the Leydig cells) in the testes produce testosterone.
- **Scrotum:** a pouch of skin that holds the testes that contracts or expands to adjust the distance the testes are from the body to regulate their temperature.
- **Seminiferous tubules:** these structures within the testes are the actual sites of sperm production (discussed further below)
- **Epididymis:** this rubbery device sits astride the testis. Sperm mature here and are stored prior to ejaculation (when sperm-bearing semen leaves the body, typically during orgasm)

Semen production

- **Seminal vesicles:** these two glands produce an alkaline (basic) fluid that can neutralize the acidity of the vagina. This fluid contains fructose and other nutrients to provide energy for the sperm.
- **Bulbourethral (or Cowper's) glands:** these two glands provide a mucus-rich alkaline fluid that lubricates the inside of the urethra to allow for easier passage of sperm and neutralizes the urethra (urine residue is acidic). Some of this fluid exits the penis prior to ejaculation (this pre-ejaculate fluid can also contain sperm). The remainder of the fluid combines with the semen ejaculate.
- **Prostate gland:** this organ wraps around the urethra and provides muscular contractions that help propel semen during ejaculation and block urine flow from the bladder during ejaculation. It also provides fluid in the ejaculate that contains enzymes and zinc that aid in sperm motility.

Sperm/semen transport:

- **Ductus (or vas) deferens:** this pair of muscle-lined tubes carry sperm from the epididymis of each testis into the abdominal cavity where they loop over the bladder and join with the ducts from the seminal vesicles to form the ejaculatory ducts. The muscles that line the ductus deferens contract to propel semen during ejaculation.
- **Ejaculatory ducts:** these ducts are formed by the joining of the vas deference with the duct from the seminal vesicle. Each ejaculatory duct empties into the urethra.
- **Penis:** the organ that encircles the urethra as the urethra exits the abdomen. This organ changes from flaccid (soft and limp) to erect (rigid and standing away from the body) during sexual arousal or spontaneously. In uncircumcised men the penis has a fold of skin called a foreskin that during the flaccid state, covers the head of the penis, and during the erect state retracts behind the glans (or head) of the penis.
- **Urethra:** the tube that runs from the bladder through the penis through which urine and semen exit the body.



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13.5 Male reproductive physiology

The organs and structures described on the previous page need to work together under hormonal signaling to release semen with mature and motile sperm out of the penis. As described earlier, in both males and females hormonal signaling begins in the brain.

When LH is released by the pituitary gland, it travels through the body in the bloodstream, and targets the testes. LH stimulates the testes to produce testosterone and several other hormones. Testosterone then circulates through the bloodstream and has effects all over the body and the brain (See table 1 for some effects of testosterone).

Location	Effect
Face and body	Hair growth
Vocal chords	Thickening (resulting in a lower voice)
Brain	Increases libido (sex drive), can increase aggression and competitive drive
Seminiferous tubules	Sperm production
Testes and penis	Triggers maturation during puberty
Muscles	Growth

Figure 13.5 Some of the effects circulating testosterone has on the body

The production of testosterone by the testes is regulated by a negative feedback loop (Figure 4). In short, negative feedback is when the effect of an action decreases the subsequent action. In this case the testosterone signals the hypothalamus to decrease the amount of GRH produced, therefore reducing the secretion of LH and FSH, and, in turn, decreasing testosterone production. If testosterone levels are low, the hypothalamus releases more GRH which signals the pituitary to secrete LH and FSH, again stimulating testosterone production from the testes. In this way the level of testosterone in the male's body is kept within a relatively narrow range.

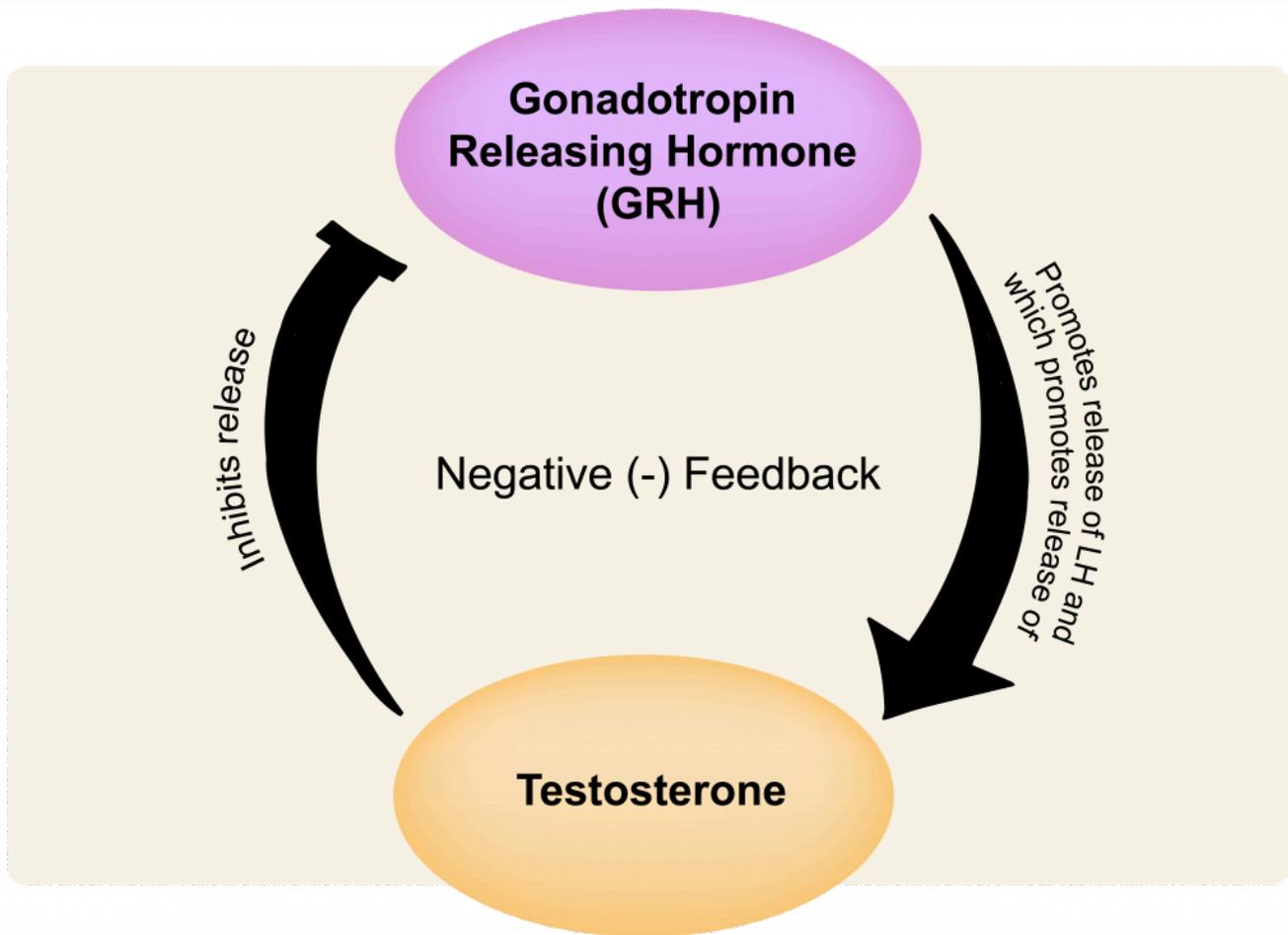


Figure 13.6 Negative feedback: When testosterone (T) levels are low, Gonadotropin Releasing Hormone (GRH) triggers the secretion of Luteinizing Hormone (LH) and Follicle Stimulating Hormone (FSH) from the pituitary. LH stimulates the testes to produce T. High T inhibits GRH production by the hypothalamus, reducing LH and FSH signaling from the pituitary.



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13.6 Sperm are produced in the testes

When the testes are exposed to FSH from the pituitary, Sertoli cells within seminiferous tubules in the testes produce sperm through meiosis. Immature sperm leave the testes and enter the epididymis where they mature (see Figure 13.7 below). The process of sperm production takes 2-3 months. Mature sperm are stored in the tail portion of the epididymis. Upon sexual arousal, the penis becomes engorged with blood, causing the penis to grow in size and become rigid. A penis in this state is termed “erect”.

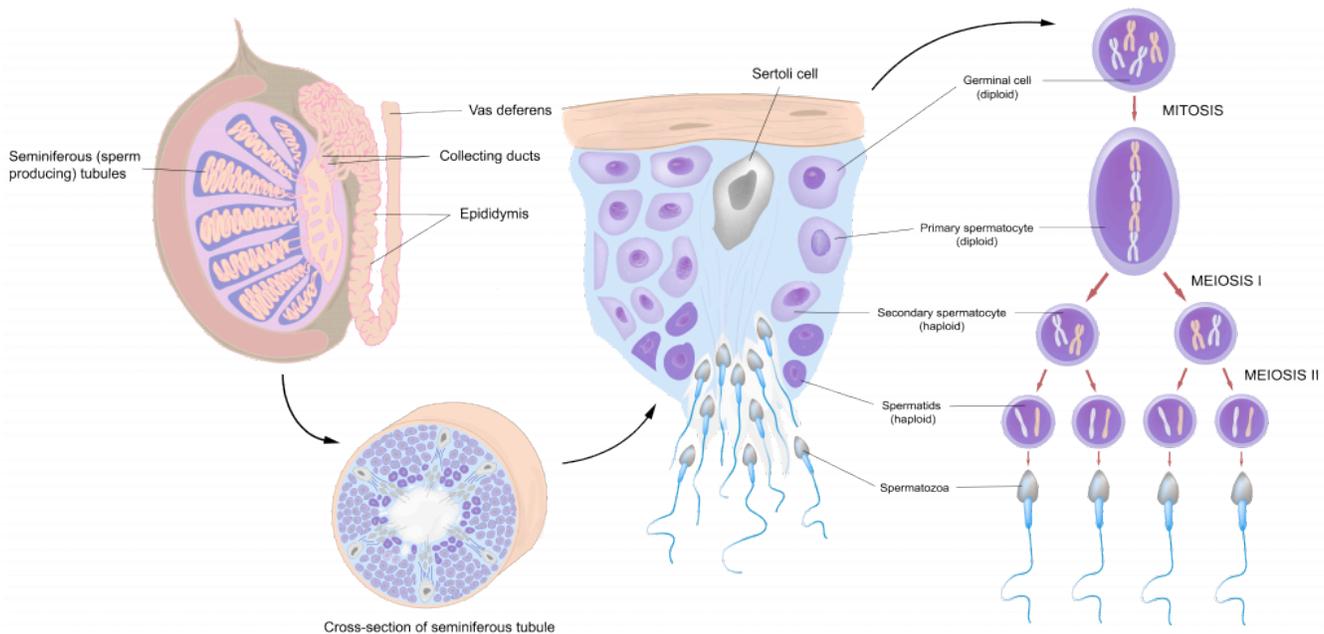


Figure 13.7 Anatomy of the testis and cross-section of seminiferous tubule. The “dividing cells that produce sperm” are called Sertoli cells. The chromosomes in mitosis are in post replication stage.

Erection and further sexual arousal and stimulation may lead to orgasm and ejaculation. During ejaculation, sperm leave the epididymis in testicular fluid through the vas deferens; this sperm-containing fluid combines with secretions from the seminal vesicle, prostate and the bulbourethral gland. The fluid (now called semen) leaves the penis through the urethra, propelled by repeated contractile muscle spasms that occur during orgasm.

Watch the video below for more details of how male reproductive anatomy and physiology work.



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13.7 Female reproductive anatomy

Some of the organs involved in female reproduction are diagrammed in Figure 13.8 below.

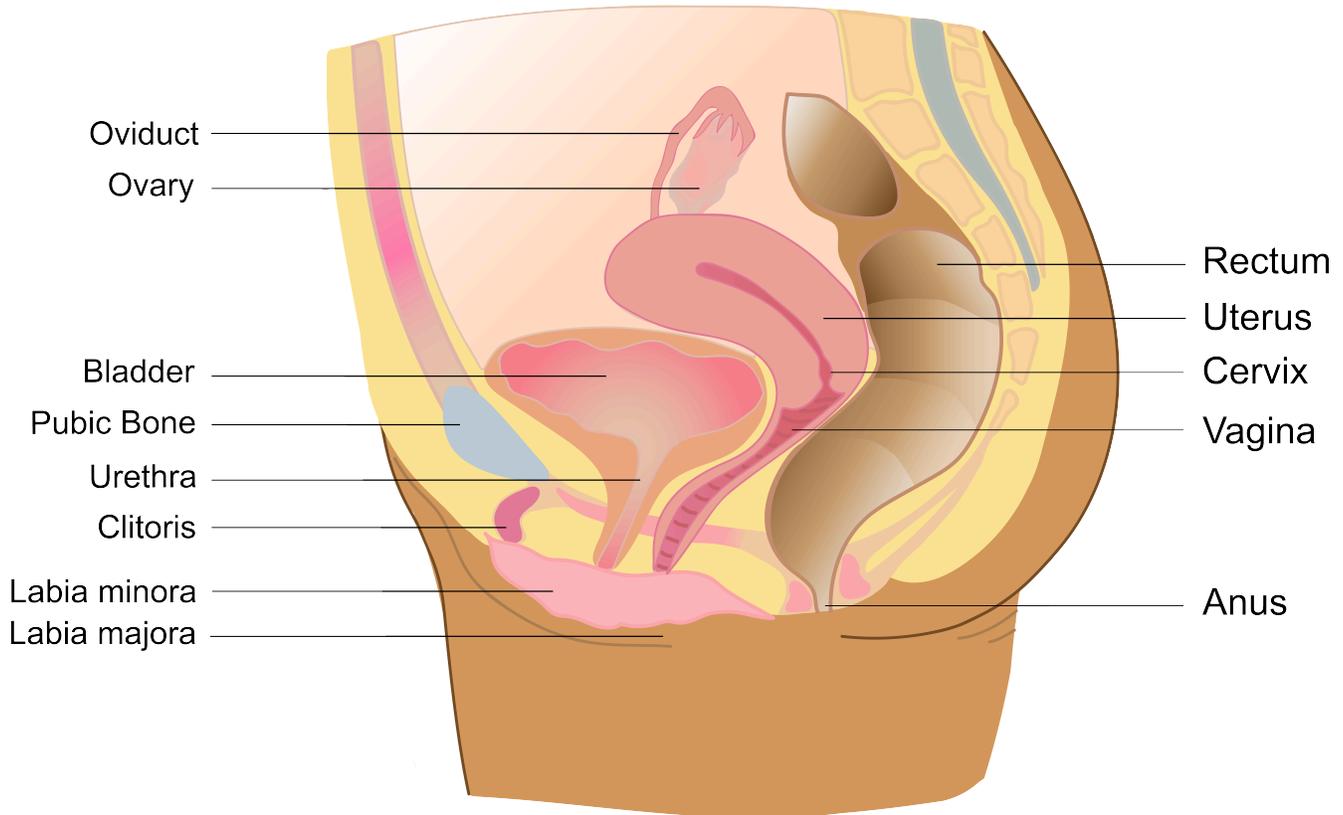


Figure 13.8 Female reproductive system

Female reproductive anatomy includes external structures (the clitoris and vulva), structures involved in the production of eggs and in fetal development (ovaries corpus luteum, and uterus), and structures involved in the transport of sperm, eggs, and babies (vagina, cervix and oviduct).

Egg production and fetal development

- **Ovary:** females have two ovaries that are the site of egg production, and, if an egg is fertilized, the site of the corpus luteum. The ovary produces hormones estrogen and progesterone and testosterone.
- **Corpus luteum:** the site of egg maturation within the ovary. After ovulation (release of the egg) the corpus luteum produces progesterone to maintain a possible pregnancy.
- **Uterus:** this muscle-lined, triangular organ is where a fertilized egg implants and develops. This organ develops a thick blood lining and sheds this lining on a monthly cycle.

Transport of eggs, sperm, and babies

- **Vagina:** a highly expandable pouch structure that serves as the opening of the female reproductive tract

to outside the body. The vagina is the point of sperm entry, and the point of exit for unfertilized eggs, menstrual discharge and for babies.

- **Cervix:** the opening between the vagina and the uterus. The size of this opening varies from tightly closed – to open for the passage of sperm, to open enough for a baby to pass through.
- **Oviducts (sometimes called fallopian tubes):** these ducts transport mature eggs from the ovary toward the uterus. If a sperm and egg are in the oviduct at the same time, the egg can be fertilized by a sperm.

Exterior structures

- **Vulva:** a general term for the exterior parts surrounding the vagina, including the labia majora and labia minora, which are the folds of skin on either side of the clitoris, urethra, and vagina.
- **Clitoris:** the sensitive nerve-rich organ that is analogous to the head of the penis. The part of the clitoris that is visible outside the body is dorsal to (closer to the belly) the urethra and the vagina). The interior part of the clitoris extends internally along either side of the vagina.



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13.8 Female reproductive physiology

Female reproductive anatomy and physiology has many similarities to that of the male. As described earlier, females also use LH and FSH secretion from the pituitary triggered by GRH from the hypothalamus to stimulate hormone production by the gonads. There is also negative feedback to regulate hormone production. However, in females, the interplay among the hormonal signals is more complicated than in males. While male hormonal feedbacks and signaling provide a relatively steady level of the sex hormone testosterone, for females there is a monthly cycle over which the circulating hormone levels go up and down at the same time as changes occur in the ovaries and the uterus. This surging of hormones along with the changes in the ovaries and uterus require the more complicated physiological controls described below.

The monthly female reproductive cycle can be divided into three phases, the follicular phase, ovulation, and the luteal phase. For each of these phases, there are concurrent changes happening in the uterus and in the ovaries. See the figure below for a diagram of the phases of the female reproductive cycle and what is happening in the ovary, the uterus, and circulating hormone levels.

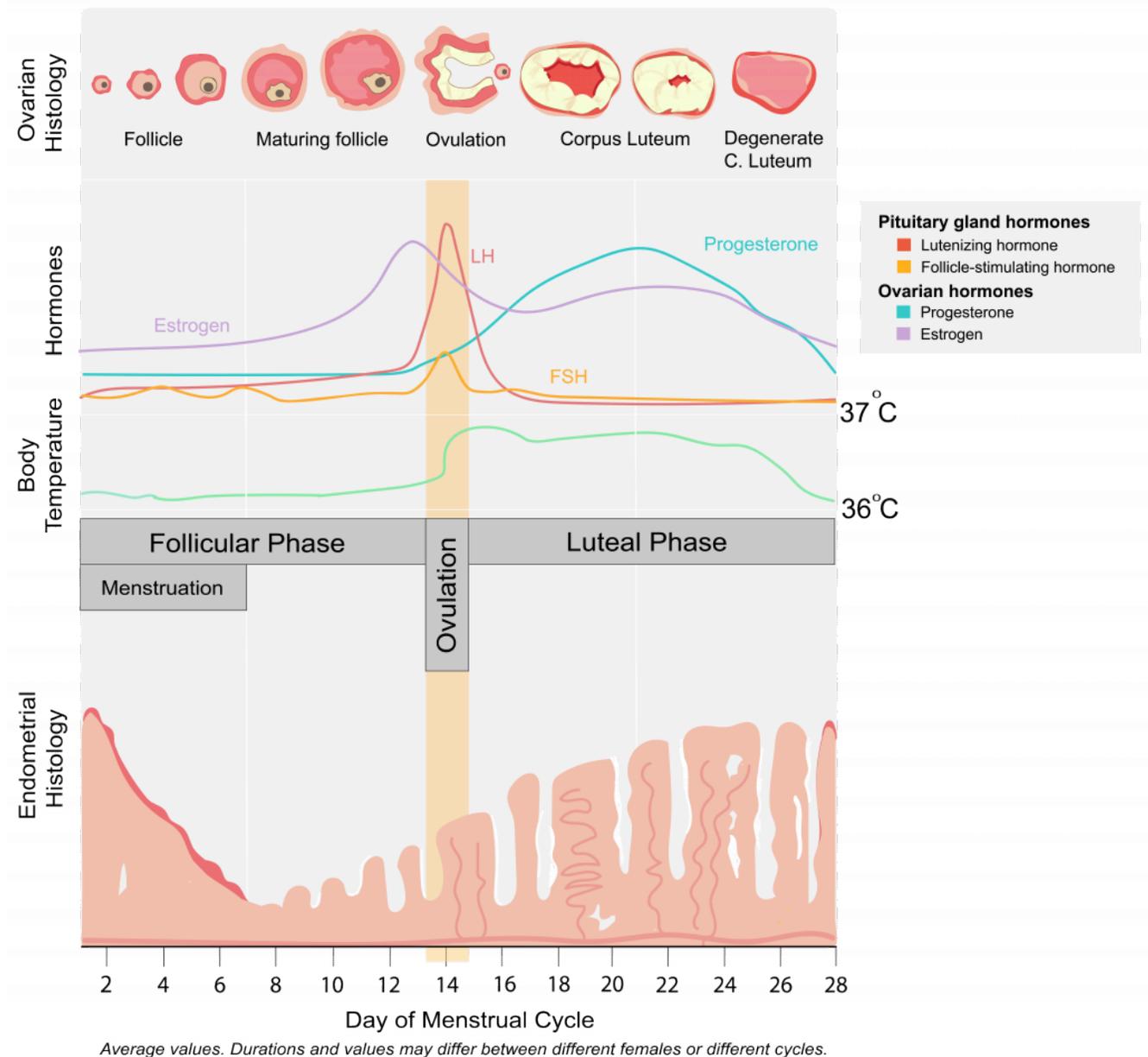


Figure 13.9 Female hormone cycle.

The Follicular Phase

The name “follicular phase” is in reference to the egg-containing follicle in the ovary that matures during this phase (note the ovarian histology shown in Figure 7). This phase begins on day one of a female’s reproductive cycle. Day one is defined as the first day of menstruation (the first day of a period). Menstruation occurs for about the first 5 days of the follicular phase. During these days, if a female is not pregnant, low circulating levels of the hormone progesterone trigger the breakdown of the endometrium (the lining of the uterus). This blood-rich tissue exits the uterus through the cervix and then leaves the body out the vagina. During menstruation, low circulating levels of estrogen and progesterone stimulate GRH production (from the hypothalamus in the brain), which leads to LH and FSH secretion by the pituitary gland. FSH signals the maturation of several **follicles** within the ovaries. These follicle cells produce a steadily increasing amount of estrogen (note the estradiol (a type of

estrogen) increases from day 1-12 in Figure 13.7). In the early follicular phase, the estrogen provides negative feedback for LH and FSH production. About 5 days into the follicular phase, after menstruation is done, estrogen production causes the endometrial layer of the uterus to begin to thicken, preparing the uterus for a fertilized egg. While several follicles in the ovary begin the maturation process, most times only one follicle in one ovary becomes dominant and continues to mature. At around the 12th day of the follicular phase, estrogen levels reach a high enough level to trigger a switch from estrogen providing negative feedback on GRH and therefore LH and FSH secretion by the pituitary, to estrogen providing a positive feedback (diagrammed in Figure 8). At this point the estrogen triggers increased GRH release, causing more LH and FSH release, causing a spike in circulating LH and FSH levels (note the LH and FSH spike between day 12 and 15). The spike in these hormones causes ovulation.

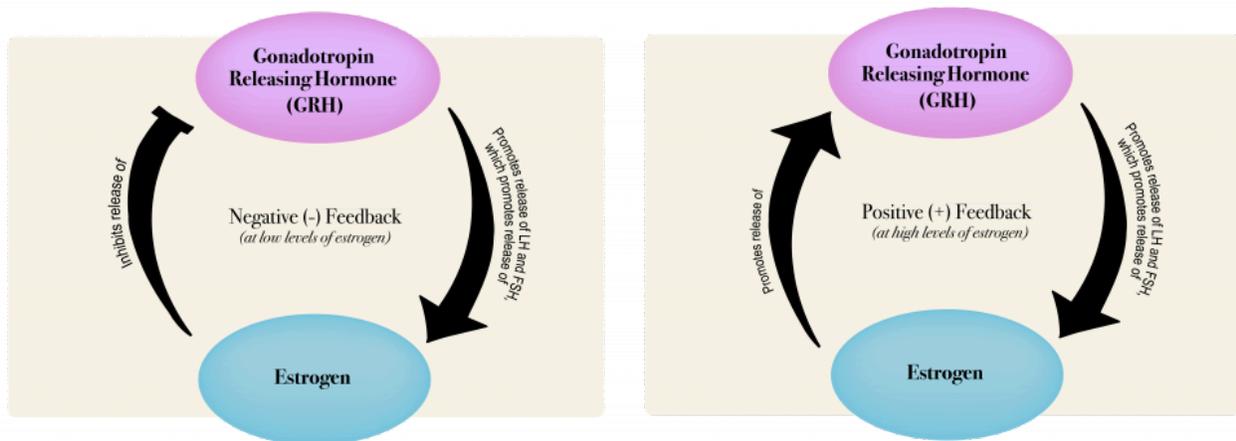


Figure 13.10 Positive feedback: At low levels, estrogen is regulated as a negative feedback (similar to T regulation). As the ovarian follicle develops, estrogen levels increase. At these higher estrogen levels, regulation of GRH switches to a positive feedback, in which estrogen cause increased release of GRH from the hypothalamus, which trigger a spike in LH and FSH secretion by the pituitary.



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Ovulation

Ovulation refers to the rupture of a mature follicle within the ovary; this ruptured follicle releases an oocyte (an unfertilized egg) into the abdominal space. Because this rupture is an actual breakage, some females will feel a twinge or slight pain during ovulation. Ovulation generally happens around day 14 of the reproductive cycle in one ovary. The oocyte travels into the oviducts and there completes meiosis I, yielding an oocyte and a smaller polar body. (Meiosis I begins when a female fetus is in utero, and it pauses for years until that female fetus is born, develops, and begins ovulation). At ovulation the oocyte begins meiosis II, but will only complete meiosis II if the oocyte is fertilized.

Luteal phase

During the luteal phase, the now empty follicle within the ovary collapses. This collapsed mass of cells is called a corpus luteum. The corpus luteum produces progesterone that enters the blood circulation. Progesterone signals the hypothalamus to signal the pituitary to reduce FSH and LH production, which prevents other follicles from maturing. If the oocyte in the oviduct is not fertilized, the corpus luteum degrades, causing a drop in progesterone, which triggers the beginning of menstruation and the return to the follicular phase of the reproductive cycle (back to day 1 after about a 28-day cycle).

If the oocyte is fertilized by a sperm in the oviduct, the oocyte completes meiosis II, forming a fertilized egg (now called a zygote) and another polar body. The zygote travels through the oviduct, going through several cell divisions until it is called a blastocyst. Then, the blastocyst implants into the uterine lining. At this point the blastocyst begins producing the hormone human chorionic gonadotropin (HCG), which signals the corpus luteum in the ovary to continue to produce progesterone. (HCG is the hormone detected by home pregnancy tests.) This continued elevated progesterone in the blood prevents the shedding of the uterine lining so that a pregnancy can be maintained. Eventually, after about 12 weeks of embryonic development, the placenta (the organ that connects the developing fetus to the mother) takes over the production of progesterone and the corpus luteum degrades. At this point, the embryo is referred to as a fetus, and will continue fetal development assisted by the placenta.



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In this chapter you have had a brief introduction to the general reproductive anatomy and physiology of humans. Often when biologists discuss evolution it seems as if we argue two contradictory points. On one hand, we revel in the diversity and variability that we see among individuals and species. On the other hand, we delight in the similarities among species that are close evolutionary relatives. Here we use the same approach to reproductive anatomy and physiology. On one hand, we argue that the hormonal signaling pathways and basic anatomy are quite similar in most humans and in most mammals. On the other hand, while the descriptions above are how most people's reproductive anatomy and physiology operate, there are individual differences (that arise from genetic, environmental, or unknown causes) in both anatomy and physiology that make the representations here overly simplistic and generalized – and so may not perfectly describe each person.

13.9 Wrapping Up: Revisiting Circumcisions



Revisiting Circumcisions

At the beginning of the chapter we asked some questions regarding circumcision. What is male circumcision? What part of the body does it affect? Is it a good idea? How does male circumcision compare to female circumcision/female genital cutting?

Male circumcision involves the removal of the foreskin of the penis. As diagrammed in Figure 3 (in 13.4), the foreskin is a thin flap of skin that covers the head (glans) of the penis during the flaccid state and retracts when the penis is erect. Risks associated with this procedure include pain, bleeding, infection, possible damage to the penis, and loss of sensitivity of the penis. Benefits of the procedure include a reduced occurrence of urinary tract infections, reduced risk of sexually transmitted infections, and reduced risk of penile cancer. The medical perspective on circumcision has shifted over recent years, from it being declared completely elective and medically unnecessary by the American Academy of Pediatrics in 1999 and 2005, to a revision of this stance in 2012 in light of new evidence. The academy's current position states:

“American Academy of Pediatrics found the health benefits of newborn male circumcision outweigh the risks, but the benefits are not great enough to recommend universal newborn circumcision. The AAP policy statement published Monday, August 27th, says the final decision should still be left to parents to make in the context of their religious, ethical and cultural beliefs.”

There is a procedure of “female genital cutting”, also known as “female genital mutilation”, or sometimes “female circumcision”. This process involves one or more of the following: the removal of the clitoral head, removal of the labia minora, and the stitching together of the labia majora, leaving only a small hole through which urine and menstrual fluid can leave the body. This process is distinct from male circumcision because there are no medical benefits to the procedure, there are frequent complications and dangerous side effects including extreme pain, hemorrhaging, possibly increased transmission of sexually transmitted infections, chronic urinary tract and genital tract infections, painful complications with sexual intercourse, complications with childbirth (including risk of death to the infant). Many of these risks can result in death.



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Science alone cannot answer whether male circumcision or female genital cutting are morally or ethically correct, but the data of the effects of these procedures and an understanding of the reproductive anatomy can certainly inform discussions of the morals and ethics of the procedures. As seen in the case of male circumcision, the position of physicians and medical organizations may change as new evidence comes to light.

As we conclude this chapter and prepare for in-class discussion, be sure to return to the chapter [goals and objectives](#).

Chapter 14: Copulation and Fertilization (Baby-making 101)



[14.0 Introduction](#)

[14.1 Chapter objectives](#)

[14.2 Diversity of sexual intimacy](#)

[14.3 Plant sex](#)

[14.4 Animal sex, from fish to birds](#)

[14.5 Mammal sex](#)

[14.6 Human procreative copulation](#)

[14.7 Human fertilization: from gametes to a zygote](#)

[14.8 Contraception](#)

[14.9 Fertility treatments](#)

[14.10 Wrapping up: Returning to sex education](#)

14.0 Introduction

Where do babies come from?

The question of where babies come from is a topic that has inspired many myths and fables—from finding babies under cabbage leaves, to babies being flown by storks from caves, to would-be parents who lure the storks to their homes with candy.

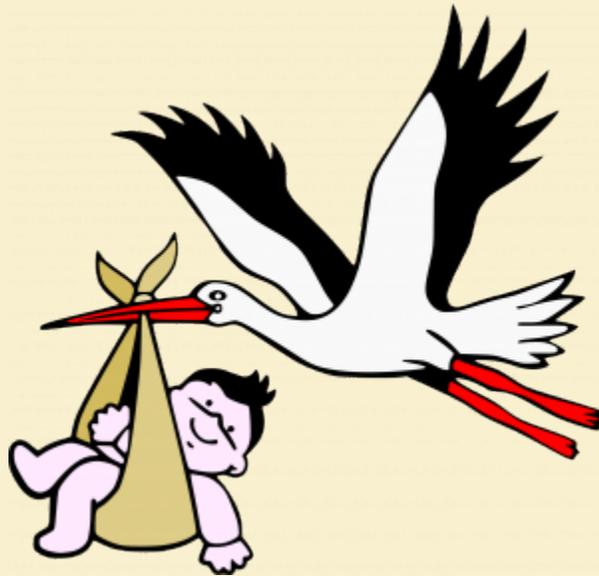


Figure 14.1 Illustration of the European myth of where babies come from; a stork carrying a baby.

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Of course all these stories are made up, by adults, to avoid telling children how babies are *actually* made. Why are adults afraid of telling children about sex? And what are the consequences of not knowing how reproduction works? How does comprehensive sex education—sex education that includes information about birth control and safe and healthy sexual relationships—affect endpoints like the frequency of children having sex, incidences of sexually transmitted infections, teen pregnancies, and abortions? We will revisit these questions after we tell you about copulation and fertilization, typically the first steps in making babies.

14.1 Chapter Objectives



Learning Objectives

Our goal for this chapter and the next is to provide you with a biological understanding of how babies are made, from fertilization through the post-partum (or post-birth) period. While our emphasis is on *human* reproduction, and we assume human reproduction is eminently interesting to most readers, we'll refer to other organisms as well. All organisms reproduce, and there is a dizzying array of fascinating reproductive stories beyond that of humans. In this chapter we will explore copulation and fertilization. In the next chapter, we will follow the fertilized embryo through its development.

By the end of your reading and our in-class discussion, you should be able to do the following.

1. Define the following terms:
 - **Fertilization**
 - **Internal fertilization**
 - **External fertilization**
 - **Pollinators/pollination**
 - **Pollen**
 - **Monoecious**
 - **Dioecious**
 - **Anther/filament**
 - **Stigma/style**
 - **Spermatophore**
 - **Cloaca**
 - **Monotremes**
 - **Marsupials**
 - **Placental mammals**
 - **Placenta**
 - **Zygote**
2. Describe how fertilization occurs in a variety of animals.
3. Describe common forms of contraception and how they work.
4. Evaluate the pros and cons of various types of contraception.

In this chapter we will dive into how reproduction occurs. We will start by looking at the act of copulation, then the event of **fertilization** (or the joining of sperm and egg) and look briefly at how this task is accomplished across a range

of organisms. In the following chapter we will look more closely at mammalian, then specifically human, embryonic and fetal development.

14.2 Diversity of sexual intimacy

Often acts of sexual intimacy (or copulation, or intercourse) are described as “having sex.” Humans and other animals have sex in a variety of ways including:

Oral sex: generally describes mouth-to-genital contact

Anal sex: generally describes contact between the genitals and/or the mouth and the anus

Vaginal sex: contact between genitals and the vagina

This is not an exhaustive list of human sexual practices. Other body parts—besides the genitals, mouth, and anus—can be involved, as can non-human devices such as artificial phalluses (dildos) and vibrators. Listing the full diversity of human sexual expression (if even possible) is beyond the scope of this textbook. The only type of human sexual intimacy that can result in a baby involves the entry of sperm-bearing semen into a vagina. If conditions are favorable, the semen can pass through the cervix and uterus and meet with a mature oocyte in the oviduct, causing fertilization of the oocyte. Under certain conditions a fertilization event can result in a baby.

The fact that the types of sexual intimacy that lead to the creation of a baby are limited does not mean that other forms of sexual intimacy are not legitimate and natural expressions of sexual desire. Much of the diversity of sexual expression mentioned here has also been observed in non-human animals. For creative and scientifically accurate descriptions of some of these non-human examples of sexual expression, see the series of short films by [Isabella Rossellini](#).

Diversity of Fertilization

In sexually reproducing organisms the fertilization of an oocyte (or egg cell) with a sperm cell is necessary to achieve reproduction. Across living organisms there are a variety of strategies in which sperm cells meet egg cells. Some fertilization events are *internal*, meaning that fertilization happens inside the body of the female. Others are *external*, meaning that unfertilized eggs are deposited outside the body and sperm is placed on them, and then the embryo develops outside the female’s body.

14.3 Plant Sex

The sexual organs of flowering plants are located in their flowers (think about that the next time you are smelling roses). Plants are rooted to the ground or to other structures; this fact makes finding a mate a challenge. Specifically, male gametes, or sperm, often cluster into **pollen** grains; these pollen grains must get close enough to female gametes (eggs) to enable fertilization. Flowering plants use various strategies to deal with the challenge of immobility. Some plants rely on water (in the case of some aquatic plants) or wind to achieve fertilization. In these plants, pollen from male flowers is specialized to be highly mobile. Other plants rely on the movement of animals (called pollinators) to move pollen to another plant. Pollinator animals include many insects, birds, mammals, and even some lizards. If you can move pollen from one plant to another, you can help plants have sex.

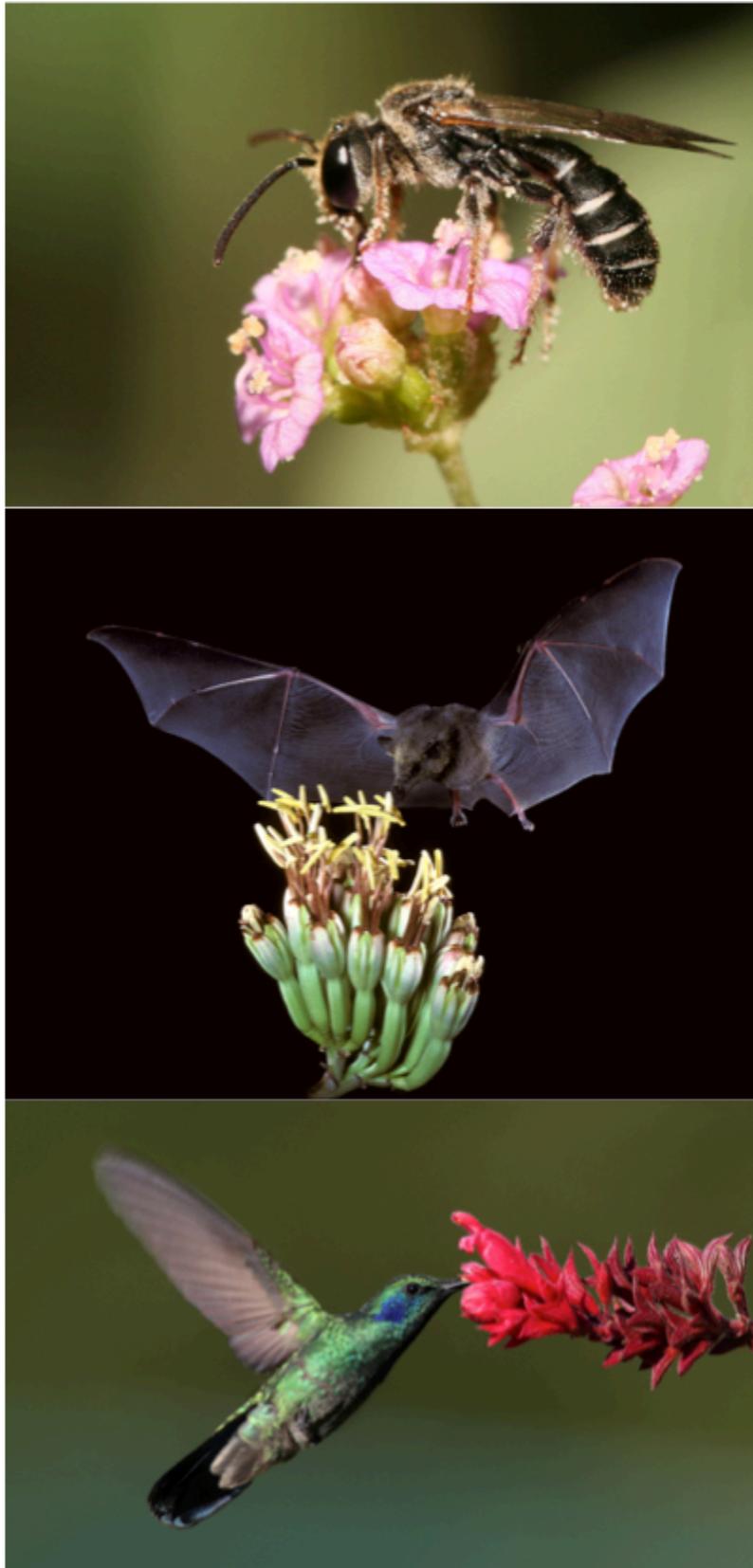


Figure 14.2 A sweat bee pollinating a gathering pollen and nectar from a flower, in the process pollinating it.

A note about plant sex

Some plants (like asparagus, plum trees and holly bushes) have completely separate sexes (all the flowers on an individual plant each produce only sperm or eggs). These plants are dioecious. Some plants (like zucchini) have both male and female flowers that reside on the same plant. These plants are monoecious. Some plants have both male and female parts in the same flower. These flowers, such as the lily flower pictured here, are perfect flowers. Plants that have both male and female parts on the same flower may employ one of several adaptations to prevent self-fertilizing (a type of extreme inbreeding). For more discussion of the differences in sexes see Chapter 7 on “Sex and Gender”. For more discussion on the evolutionary pros and cons of self-fertilizing, see Chapter 6 on “Why Sex?”

1. By Muhammad Mahdi Karim - Own work, GFDL 1.2, <https://commons.wikimedia.org/w/index.php?curid=6575681> Bat feeding at a cactus flower By U.S. Fish and Wildlife Service Headquarters - *Choeronycteris mexicana*, Mexican long-tongued bat Uploaded by Dolovis, Public Domain, <https://commons.wikimedia.org/w/index.php?curid=31315207> Hummingbird (the lesser violetear) feeding at a flower By Mdf, CC BY-SA 3.0, <https://commons.wikimedia.org/w/index.php?curid=3872140>

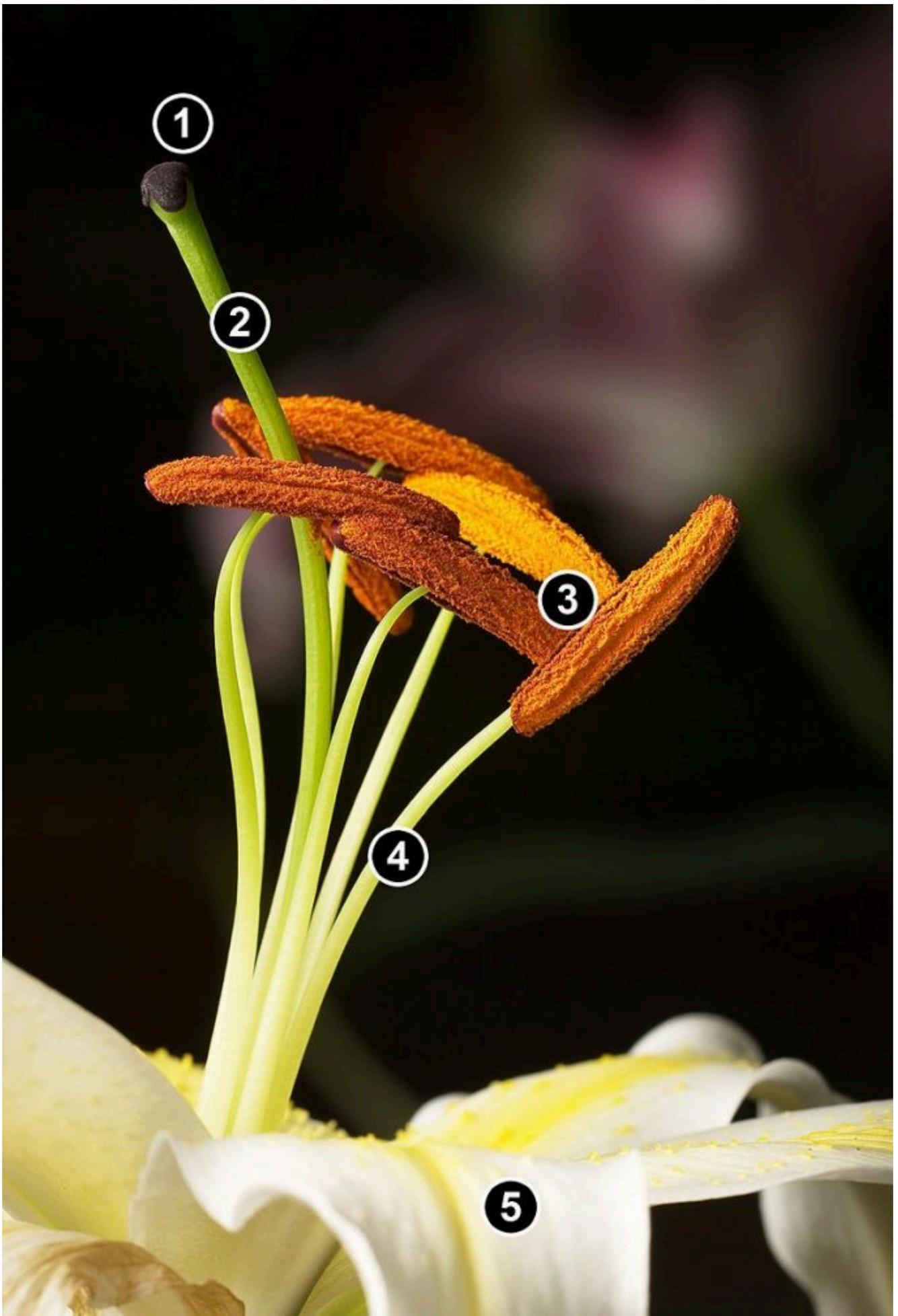


Figure 14.3 *Reproductive parts of Easter Lily (Lilium longiflorum). 1&2 Stigma & Style (lead to the egg producing ovary), 3. Stamens (produce pollen/sperm), 4. Filament, 5. Petal*

2

Once the pollen from the male portion of a flower (specifically, the **anther**) meets a **stigma** on the female portion of a flower, the pollen grain forms a pollen tube, through the female **style**, to the flower's ovary. Once the ovary is reached the pollen grain releases two sperm cells. One fertilizes the egg cell, while the other fertilizes a diploid cell to create a nutrient-rich structure called an **endosperm**. This endosperm provides a source of energy to the developing plant embryo (As an aside, in plants that humans use as grain-food sources, these endosperms are the part we rely on for food. For example, in wheat plants the process of creating white flour involves isolating the endosperm from the rest of the grain).

14.4 Animal sex, from fish to birds

Most fish have external fertilization, but many have highly ritualized courtship routines that they use to choose a mate. The male will often perform courtship demonstrations of dancing, nest building or territory defense. (See examples of these with [puffer fish](#), [clown fish](#), and [trumpet fish](#).) The male will follow the female, and once she deposits eggs on a surface, he will follow and deposit milt (the fish semen that contains sperm) over top.

A few fish (like guppies) have internal fertilization, in which the male inserts a specialized tubular fin into the female reproductive opening and deposits sperm into her reproductive tract.

Some fish are *mouthbrooders*, meaning that one fish puts the eggs in its mouth for incubation. Many cichlids are maternal mouth brooders. For these fish, the female lays the eggs and then picks them up in her mouth. Male fish will then encourage the female fish to open her mouth and will fertilize the eggs while in her mouth. For paternal mouth-brooding fish, the male puts the eggs in his mouth to incubate after he has fertilized them externally. Recall the discussion of anal-fin egg mimicry in Chapter 4...cichlid fish are fascinating!

Amphibians

Like fish, many amphibians have external fertilization. Many frogs and toads, for example, have a courting ritual in which the male frog rides on the female's back and places specialized digits (essentially frog thumbs) on either side of the female in specialized, so-called nuptial pads. This grip helps keep the male frog from falling off as the female hops or swims around. The female eventually deposits her eggs and the male is in an ideal position to fertilize them.



Figure 14.4 Frog thumbs

A few salamanders perform external fertilization similar to that of frogs and toads, however most salamanders have internal fertilization. Male salamanders do not have penises to deposit sperm inside the female. Rather, they deposit an encased capsule of sperm and nutrients, a **spermatophore**, on the ground as part of a mating ritual. A female can pick up the spermatophore with her **cloaca** (a combined urinary and genital opening) and will use these sperm to fertilize her eggs internally. Most salamanders will then lay the fertilized eggs, however in a few species (such as the fire salamander) the eggs hatch inside the female and the female [gives birth to larval salamanders](#).

Reptiles

Reptiles (e.g., lizards, turtles, snakes, and crocodiles) have internal fertilization. Reptiles have a great diversity of penises; some have a penis that is branched at the end (each end is called a *hemipenis*; reptiles only use one at a time), and some tortoises have umbrella-shaped penises. Some reptiles give birth to live young (called *viviparity*) and some lay eggs.

Birds

Most birds do not have penises, but achieve internal fertilization via cloacal contact (or “cloaca kiss”). In these birds, males and females contact their cloacas together, typically briefly, and transfer sperm to the female. Interestingly, water fowl such as ducks and geese have penises and use them for internal fertilization. Why would some birds use penises for fertilization, while others do not?

14.5 Mammal Sex

Mammals fall into three reproductive categories: monotremes, marsupials, and placental mammals. We will focus on one type of placental mammal (humans) for most of the remainder of this chapter, but the reproduction of monotremes and marsupials is interesting and worthy of mention.

Monotremes are clearly mammals because they have fur and they nurse their young with milk. However, monotremes are distinct from other mammals because they lay eggs. The only extant (or, not extinct) monotremes are platypuses and [echidnas](#). Similar to that of other mammals, monotreme fertilization is internal. However, the genitals of monotremes differ from those of other mammals in that the females have a cloaca for urination, defecation, and copulation. Echidna males have a penis with four heads, two of which are active at a time. The males do not urinate from the penis, instead they have a cloaca similar to that of the female. Platypuses have two-headed penises, however the left penis is more developed. Female platypuses have two ovaries, but only the left one functions. In echidnas the eggs are incubated in a specialized pouch, while platypuses curl up around the eggs to incubate them. And like other mammals, all monotreme babies nurse to obtain nutrients from the mother, but they do so by lapping at specialized pores on the mother that secrete milk.



Figure 14.5 Platypuses

1

Marsupials are mammals that include opossum, kangaroos, koalas, wombats, and wallabies. Marsupials have internal fertilization that involves a *bifurcated* (split) penis in males, and two vaginas and two uteri in females. These animals give birth to very undeveloped young. The newborn animals make their way to a pouch in the

1. By Klaus - Flickr: Wild Platypus 4, CC BY-SA 2.0, <https://commons.wikimedia.org/w/index.php?curid=32551315>

mother. In the pouch they nurse for several weeks or months as they develop further, and become less dependent on the mother.

Placental mammals (including humans) have internal gestation, in which the offspring develops further than in marsupials, and nutrient exchange between mother and fetus relies on a well-developed **placenta**. The placenta is an organ formed during embryonic development. The placenta allows for nutrient and waste exchange between the mother and the developing offspring. While marsupial mammals also have placentas, the structure in the placental mammals is much larger to allow these animals to develop further internally. Like monotremes and marsupials, after birth, infant placental mammals obtain nourishment by nursing.

For the rest of the chapter we will go into specifics about how human babies are formed, but keep in mind that humans share many aspects of the details of our development with other placental mammals.

14.6 Human procreative copulation

While human copulation can take many forms, we'll focus here on the basics of procreative sex, whereby male ejaculate is used to fertilize a female egg. Human copulation typically begins with arousal. For females, this is accompanied by the production and secretion of vaginal fluids that can signal arousal and enable easier penetration. For males, arousal is accompanied by an *erection* (or “hard on,” “boner,” or any number of other euphemisms), which is itself the result of rapid movement of blood into the spongy tissues of the penis. This blood causes the spongy area to swell, and makes the entire penis stiff.

An erect penis can penetrate a lubricated vagina. Humans (and many other animals) typically follow penetration by repeated thrusting of the penis into the vagina, an activity that is often accompanied by involuntary vocalizations (e.g., grunting, moaning), heavy breathing, and an accelerated heart rate. This period of copulation is sometimes termed the *plateau*, and it precedes ejaculation.

The thrusting, moaning, and panting typically culminate, especially for the male, in *orgasm*. During orgasm, the male usually ejaculates, releasing semen from the urethra. Orgasm for both partners can also involve whole-body muscle spasms, more involuntary vocalizations, and a feeling of euphoria. While multiple orgasms are unusual for males, females may experience several orgasmic pulses, either with or without the aid of manual stimulation (e.g., hands or a vibrator).

Read More

Read more about the enigma of the human female orgasm [here](#).

Orgasm is followed by a refractory period, during which normal breathing and heart rate resume. This period is often marked by intense sensations of wellbeing and calm. In males, the end of the refractory period is delimited by the ability to have another erection.

14.7 Human fertilization: from gametes to a zygote

As we discussed in chapter 13, fertilization in humans happens in the oviducts. For this to happen, the sperm need to arrive in the oviducts when there is an egg there. Sperm can stay alive in the female reproductive tract for 3-5 days. An egg needs to be fertilized within about 12 hours of ovulation; and while some fast-swimming sperm can reach the egg within an hour, many will take a day or more to swim that far. Based on these sperm swimming and egg survival times, the most likely timing for vaginal sex to occur to achieve fertilization is from 1-3 days prior to ovulation.

Note: while there are birth control methods that take advantage of this timing, there are a LOT of babies conceived by people thinking that it is a “safe” time to have sex. Keep in mind that the drive to have sex is increased for both males and females during times of high fertility. So if you are trying to convince yourself you are “safe” from pregnancy, remember there are evolutionary drivers for reproduction that may be greater than your ability to calculate pregnancy risk.

In a typical ejaculate there are about 100 million sperm. When these sperm are ejaculated in semen into a vagina, they begin swimming toward the cervix, through the cervix, through the uterus and into the oviducts. This is a perilous journey for the sperm. Many never make it through the cervix, some are attacked by immune cells in the uterus, and roughly half of those that remain enter the empty oviduct (remember, in general only one follicle in one ovary matures per menstrual cycle). Out of the 100 million-plus contenders only several dozen sperm actually reach the egg. When sperm and egg meet in the oviduct, the head of the sperm (or **acrosome**) secretes an enzyme that helps the sperm swim through the jelly-like coating of the egg. Once through this layer, the sperm fuses with the cell membrane of the egg; the membrane then undergoes chemical changes, blocking other sperm. Only the genetic material from the sperm enters the egg (mitochondria and all other parts of the sperm remain outside the egg). At this point the egg completes meiosis II. The genetic material from the sperm fuses with the genetic material from the egg and a fertilized egg, or **zygote**, is formed.

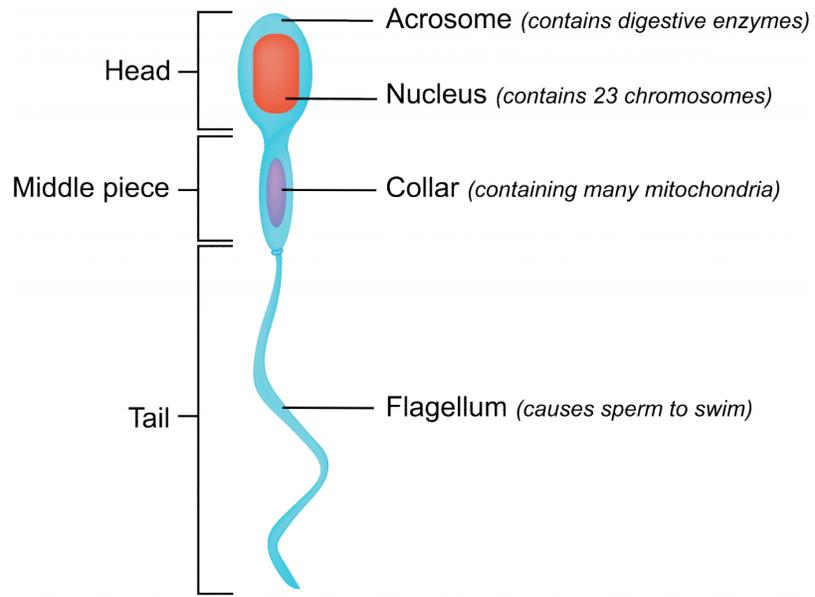


Figure 14.6 A zygote

14.8 Contraception

Our understanding of fertilization and pregnancy has been used to increase and decrease the odds of pregnancy. Technologies that are used to reduce the chance of pregnancy are called *birth control* or *contraception*. Technologies that are used to increase the chance of pregnancy can be broadly categorized as *fertility treatments*.

Below are some of the common methods of contraception and a bit of information about each. For more detailed and more frequently updated information ask a medical health professional. Some hormonal methods of birth control carry some serious—but rare—side-effect risks, such as potentially fatal blood clots, so medical consultation is important when selecting appropriate contraception. Estimations of failure rates of these methods (what percentage of people using the method for a year will have a pregnancy) vary widely between “typical use,” or how the contraception is often used, and “perfect use,” or how the contraception *should* be used. Examples of the difference between typical use and perfect use are things like taking birth control pills at different times of the day or missing a pill, not putting a condom on right away during sex, or not putting a condom on correctly. Finally, note that a few contraception methods decrease the chances of contracting a sexually transmitted infection (STI).

Read More

For more information on birth control methods, Planned Parenthood has good information.
<https://www.plannedparenthood.org/learn/birth-control>

Type	Description	Failure Rate	Other Information
Abstinence	Refraining from sexual activity broadly, or refraining from sexual activity that involves penis-vagina contact in order to prevent pregnancy	Effectiveness close to 100% if adhered, however high failure rate because of lack of compliance with abstinence	There is a small possibility of pregnancy if semen is ejaculated near the vagina (say in the rectum) or if semen gets on hands or other objects that come in contact with the vagina. Chance of contracting STIs is reduced with abstinence (there is still some risk of STI if there is sexual contact that is not intercourse)
Periodic Abstinence or Fertility Awareness Methods	Refraining from vaginal intercourse during times of high fertility	24% failure rate under "typical use". "Perfect use" of more detailed indicators range in failure rate from 0.4 -5%	Use of fertility awareness indicators beyond the calendar days (including temperature and cervical mucus) dramatically improve the effectiveness of this method (for more information see https://www.plannedparenthood.org/learn/birth-control/fertility-awareness)
Barrier methods			
Male Condom	A thin material sheath that fits over the penis during sexual activity to contain ejaculate. Often made from latex, polyurethane, or others.	18% failure rate under typical use, 2% failure rate for "perfect use"	Condoms are one of the few methods of contraception that decrease the odds of contracting a STDs
Female Condom	A tube of thin material with rings at both ends that can contain semen within a woman's body	21% failure rate "typical use" 5% failure rate for "perfect use"	Use of female condoms decrease the risk of contracting STDs
Cervical cap	A cup that is put in place over the cervix prior to sex that blocks the entry of sperm into the cervix. Works best with spermicide	14% failure rate if you haven't given birth, 29% if you have given birth	The cervix changes shape from pregnancy making a cervical cap less effective
Diaphragm	A silicone shallow dish with spermicide inserted into the vagina prior to sex. The disk covers the cervix and blocks the entry of sperm into cervix	6-12% failure rate	
Hormone methods			
Hormonal methods usually contain estrogen and/or a progesterone mimic called progestin. There are some possible side-effects and risk associated (blood clots, libido effects, mood changes, depression, etc.). There are also some possible side effects (acne reduction, reduction of menstrual cramps, etc.).			
Birth control pill	Pills taken daily that contain estrogen and/or progestin	9% failure rate for "typical use", 1% for "perfect use"	Some medications like certain antibiotics interfere with the effectiveness
Hormonal rings/ patches	Hormonal birth control in which hormones (estrogen and/or progestin) are delivered across skin (rings and patches)	9% failure rate for "typical use", 1% for "perfect use"	Some medications like certain antibiotics interfere with the effectiveness
Hormonal injections	Progestin injections once every 3 months	6% failure rate for "typical use", 1% for "perfect use"	Prevents ovulation
Hormonal implant	A thin rod that is put under the skin in the upper arm that releases hormones that prevent pregnancy	<1% failure rate	Lasts up to 4 years
Interuterine devices (progesterone)	A small device placed in the uterus coated with a slow release formulation of progestin	<1% failure rate	Ovulation is prevented. Many women do not menstruate when they have this type of IUD
Emergency contraception	Pills taken after unprotected sex to prevent pregnancy	<5% effectiveness depends on type and how soon after sex EC is taken	Emergency contraceptive pills appear to interfere with ovulation, some may also reduce the odds of implantation (see copper IUD for another method of emergency contraception). This method of contraception should not be confused with medications that disrupt established pregnancies (see abortion below).
Non-hormonal IUD			

Figure 14.7 Various birth control methods. Information provided by Planned Parenthood

14.9 Fertility Treatments

Fertility treatments (sometimes called *assisted reproductive technology*, or ART) can increase the odds of a person getting pregnant. Some of the common methods for increasing fertility are listed in the table below.

Fertility Treatments (sometimes called assisted reproductive technology (ART)) can increase the odds of a person getting pregnant. Some of the common methods for increasing fertility are listed in the table below.		
Type	Description	Other Information
Fertility Awareness methods	Becoming aware of fertility and timing vaginal sex to coincide with most fertile times	The same techniques can be used to increase the chance of pregnancy were used to decrease the chance of pregnancy. You can have vaginal sex during the high fertility time.
Inter-uterine insemination	In this procedure sperm in a semen collection are separated from the other fluid and washed. When a female is found to be ovulation (often detected by ultrasound), this concentrated sperm solution is injected by catheter through the cervix and into the uterus. The sperm are given a head start toward the egg.	This technique is often done in combination with medications given to the female to increase the odds of ovulation.
Hormonal interventions	Oral or injectable medications are taken at various times during the menstrual cycle to increase the odds of ovulation.	Most of these medications increase the odds of multiple ovulations that can result in dizygotic twins and higher order multiple pregnancies (triplets and beyond).
In vitro fertilization (IVF)	The female is induced through hormonal treatments to multiple ovulate. Mature oocytes are harvested by needle (inserted through vagina into ovary.) Eggs are placed in a petri dish with sperm. Sometimes sperm are individually injected into eggs. Fertilized embryos are allowed to develop in an incubator for 3-5 days until the morula or blastula stage. Under some protocols the embryos are assisted through the "hatching" stage by technicians manually or chemically poking a hole in the zona pellucida. 1 or more embryos are then transferred to the uterus.	Multiple births (twins or higher-order) are also quite common in patients undergoing IVF. The decision of how many embryos to transfer at one time is influenced by parental preference and maternal age. The chances of pregnancy are higher with the transfer of multiple embryos, however pregnancy risks to mother and child are also increased with twins and higher order multiple pregnancies.
IVF with pre-implantation diagnostics	IVF is carried out as above, however before implantation, one cell is removed from the embryo (remember the cells are pluripotent -so they can each become an entire human - and you can remove one cell without affecting the embryo). The rest of the embryo is frozen. The cell undergoes genetic testing. In this way embryos can be screened for genetic diseases and even for sex. Embryos that do not have disorders are thawed and implanted in the uterus.	This procedure is controversial for several reasons. For some people who have serious genetic diseases in their family, it is a way of having children who will not have to suffer from these diseases. However, the fact that the embryos that test positive for genetic disease are disposed of is problematic for some people. Additionally, while some people are fine with this procedure for serious genetic diseases, some people are uncomfortable with the use of the procedure to screen for sex, or other non-disease genetic features.

Figure 14.8 Various fertility treatments. Information provided by Planned Parenthood

14.10 Wrapping Up: Returning to Sex Education



What do you know about sex education?

What happens when children are taught about copulation and sexual intimacy? What are the consequences to not teaching sex education? What are the impacts of so-called “comprehensive sex education” compared to so-called “abstinence-only” sex education? Comprehensive sex education involves age-appropriate instruction that explains that abstinence from sex is the least risky sexual behavior, but also includes information on birth control, sexually transmitted infections, healthy relationships, and other topics. “Abstinence-only” programs limit information on contraception and sexually transmitted infections and promote the expectation of abstaining from sex until marriage. So what are the outcomes of these two approaches to teaching sex education?

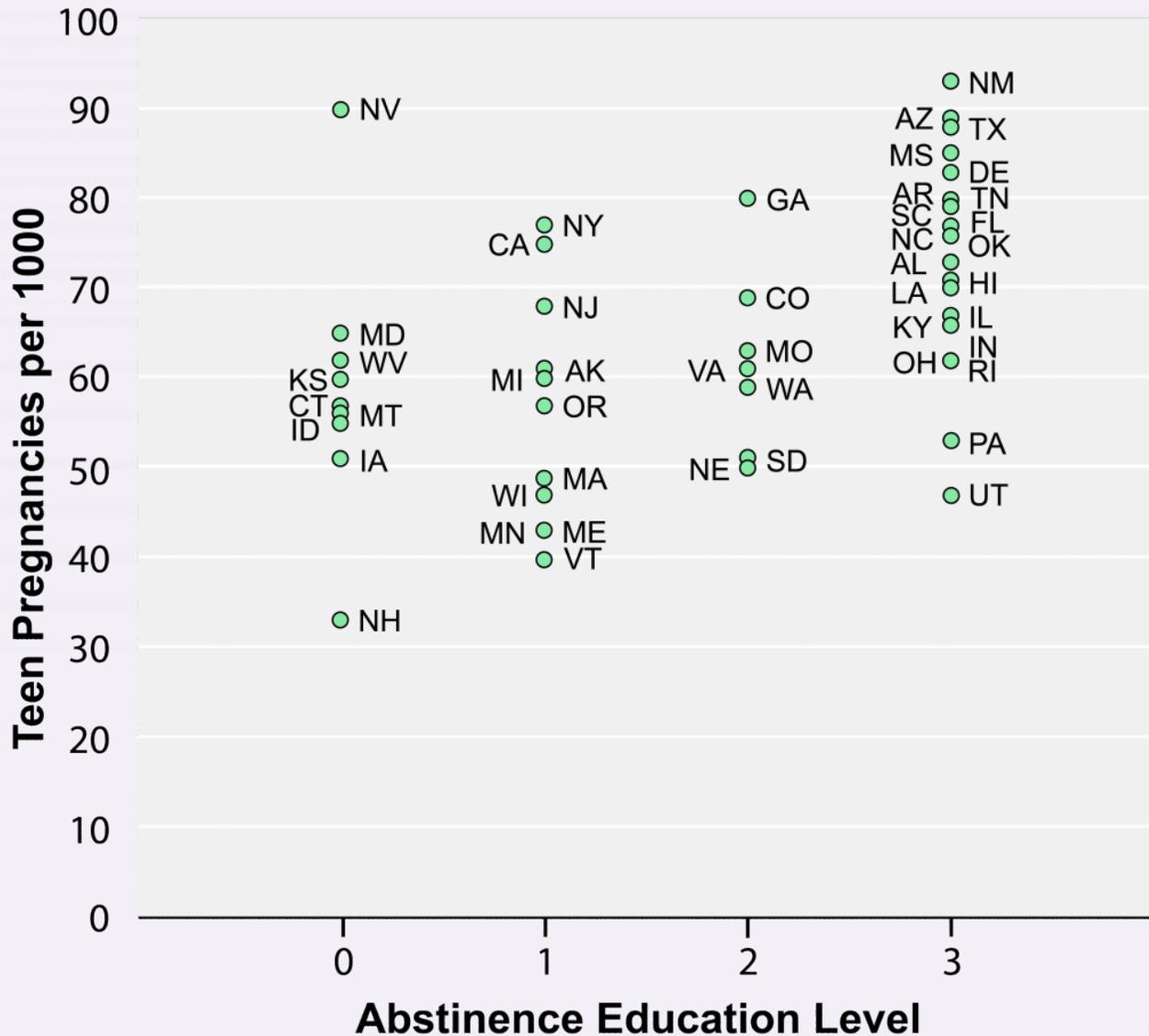
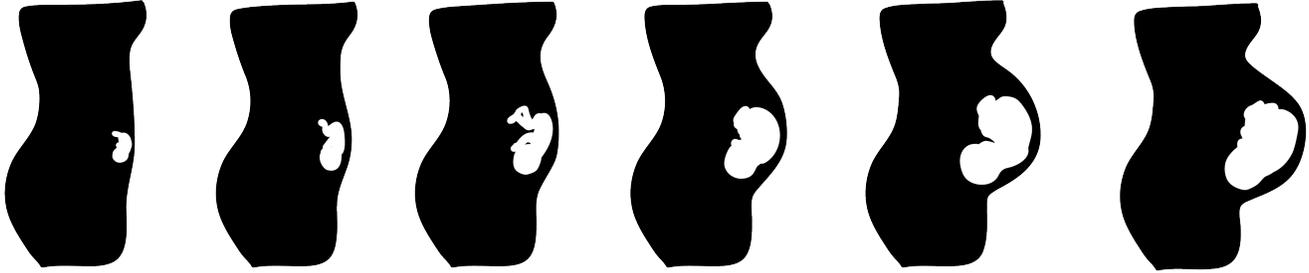


Figure 14.9 The relationship between teen pregnancy rates (per 1000 individuals) on a state-by-state basis in the USA. From the article (by Stanger-Hall and Hall, 2011): “Among the 48 states in this analysis (all U.S. states except North Dakota and Wyoming), 21 states stressed abstinence-only education in their 2005 state laws and/or policies (level 3), 7 states emphasized abstinence education (level 2), 11 states covered abstinence in the context of comprehensive sex education (level 1), and 9 states did not mention abstinence (level 0) in their state laws or policies.”

In an analysis of published studies, researchers (Chin et al., 2012) found that children who received comprehensive sex education compared to those who received no sex education had lower sexual activity, lower numbers of sex partners, lower incidences of unprotected sex, lower pregnancy rates, and increased use of contraceptives. Children who received abstinence-only education had lower sexual activity, but no difference in number of sex partners, unprotected sexual activity, frequency of contraceptives, or pregnancy when compared to those who received no sex education. In other words, abstinence-only sex education has only slightly different outcomes than no sex education at all.

Storks delivering babies and finding infants under cabbage leaves make for adorable imagery. However, you will probably not be surprised that the authors of this text are convinced (by the above data and numerous other studies) that children need more information than these fables to make healthy decisions about their sex lives, to protect themselves, and to engage in healthy relationships.

Chapter 15: Gestation & Birth



[15.0 Introduction](#)

[15.1 Chapter objectives](#)

[15.2 The first two weeks](#)

[15.3 Gastrulation, neurulation and beyond](#)

[15.4 Gestation](#)

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[15.7 Abortion](#)

[15.8 Wrapping Up: Revisiting the missing mother case](#)

15.0 Introduction

The case of the missing mother

In 2002 a 52-year-old woman needed a kidney transplant. She and her family underwent genetic testing to see if one of her children could serve as a kidney donor (healthy adults can donate one kidney with few side effects). Genetic tests suggested that this woman was not the biological mother¹ of her children. Her husband was genetically identified as the biological father, she had clearly given birth to the children, and the couple has not had medical interventions to get pregnant. So how could someone give birth to children who did not seem to be biologically hers? Clearly this story is unusual, but it brings to mind a number of questions about how babies are usually made, and how our understanding of the typical biological process of baby-making could help us understand this scientific puzzle.

1. Note about the term “mother”: Not all females who have babies identify with the gendered term mother. For clarity and brevity, this term is used in this chapter in reference to females who are producing offspring. We will also use the term “egg-parent” to be more biologically accurate and inclusive.

15.1 Chapter Objectives



Learning Objectives

Our goal for this chapter is to provide you with a biological understanding of how babies are made, from fertilization through the post-partum (or post-birth) period. While our emphasis is on human reproduction, and we assume human reproduction is eminently interesting to most readers, we'll refer to other organisms as well. All organisms reproduce, and there is a dizzying array of fascinating reproductive stories beyond that of humans. By the end of your reading, you should be able to do the following:

1. Define the following terms:
 - **placenta**
 - **dizygotic twins**
 - **monozygotic twins**
 - **pluripotent**
 - **stem cells**
 - **chimerism**
 - **oxytocin**
2. Identify the stages of embryonic development and describe the general characteristics of each stage
 - **Zygote**
 - **Embryo**
 - **Morula**
 - **Blastocyst**
 - **Gastrulation**
 - **Ectoderm**
 - **Mesoderm**
 - **Endoderm**
 - **Neurulation**

- **Fetus**

15.2 The first two weeks

As we discussed in [chapter 14](#), once the genetic material from a sperm fuses with the genetic material from an egg, a zygote is formed. This zygote will now begin to divide from one cell to two, from two cells to 4, etc. During this time the embryo¹ begins to move down the oviduct toward the uterus. Around day 2-3 post-fertilization the developing embryo is a ball of cells called a **morula**. Around day 4-5 after fertilization the egg becomes a **blastula**, which is a hollow ball of cells. At this stage it enters the uterus and will “hatch” out of a tough outer covering called the zona pellucida. Once hatched (1-2 weeks post-fertilization) the embryo will interact with the uterine lining and may implant, or imbed itself in the uterine lining. Only about 50% of embryos successfully implant in the uterine lining (the rest are carried out of the body with the next onset of menstruation).

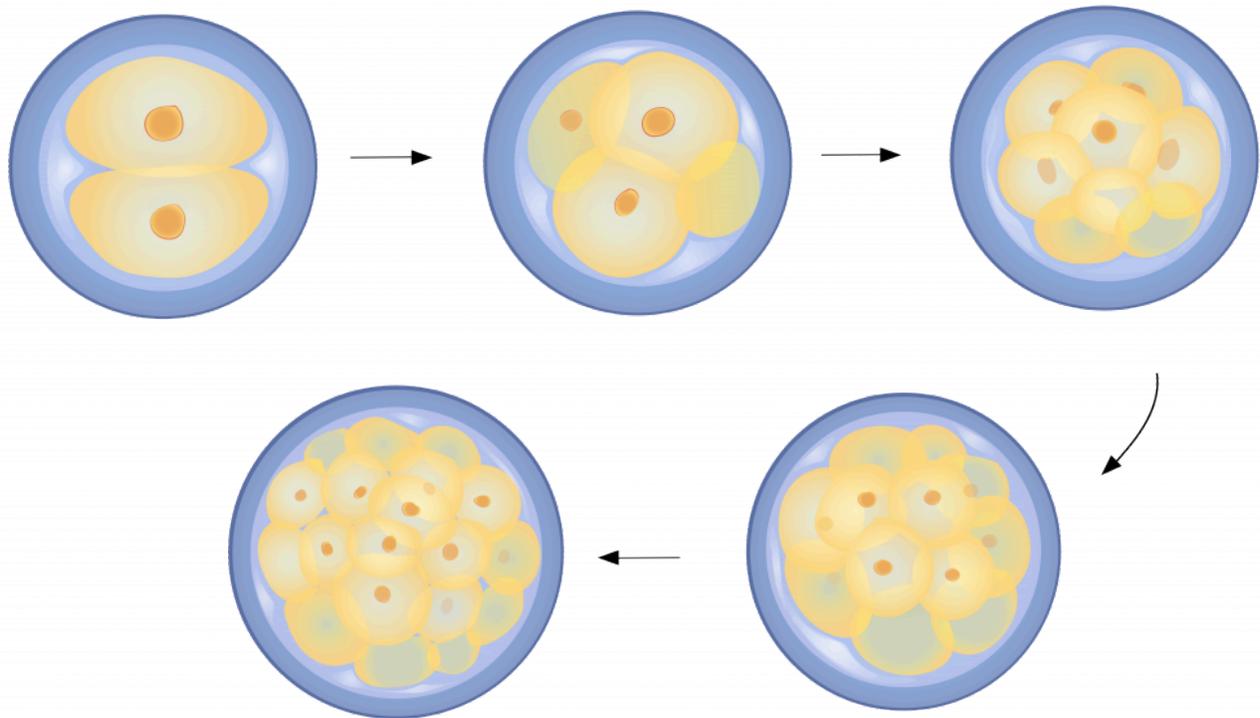


Figure 15.1 Stages of Embryo Development

1. Embryo is a general term for the early stages of the developing organism. The terms zygote, morula, and blastocyst refer to more specific developmental stages. Once a developing human reaches around 12 weeks it is referred to as a fetus.

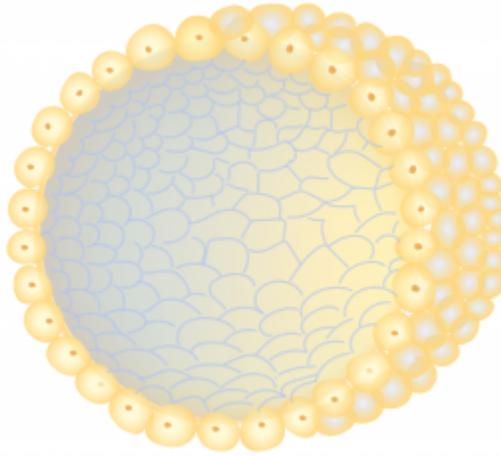


Figure 15.2 A blastula

Once implantation has occurred, a pregnancy is established and circulating levels of human chorionic gonadotropin (HCG) increase. HCG is the hormone that is detected in urine by over-the-counter pregnancy tests. This hormone also (as discussed in chapter 13.8) signals the corpus luteum (the collapsed group of cells from which an oocyte emerged) to continue to produce progesterone. Progesterone prevents the shedding of the uterine lining that would normally happen during menstruation. The corpus luteum continues to produce progesterone for the first 7-10 weeks of pregnancy, after which the developing placenta takes over. What is often confusing when talking about pregnancy stages is that, while implantation is happening only about 2 weeks post-fertilization, the calculation of stage of pregnancy is done from the date of the first day of the last menstrual cycle (first day of the period). Thus, a physician would describe a person who has a newly established pregnancy as 4 weeks pregnant (even though the first two weeks of the “pregnancy” were before fertilization occurred!).

Figure 15.3 Animation of a zygote cycle

The outer cells of the blastocyst that are imbedded in the uterine wall will differentiate into the placenta, and the inner cells will eventually develop into the fetus. At this stage the cells of the developing embryo are identical and **pluripotent** (i.e., from those cells, any of the parts of the human body can develop). In fact, if an embryo splits in two prior to this time, the result can be two fully formed babies that are genetically identical (these are **monozygotic** or **identical twins**).



Check Yourself



An interactive H5P element has been excluded from this version of the text. You can view it online here:
<https://open.lib.umn.edu/evolutionbiology/?p=2658#h5p-76>

Read More

For detailed explanation of the stages of embryonic and fetal development, see: https://embryology.med.unsw.edu.au/embryology/index.php/Main_Page

15.3 Gastrulation, neurulation, and beyond

Gastrulation, the formation of a **gastrula**, occurs around 3 weeks post-fertilization (week 5 of pregnancy). At this stage, the embryonic cells are characterized by three distinct layers: the **ectoderm**, the **mesoderm**, and the **endoderm**. The cells are said to have **differentiated** and will give rise to one of several specific tissue types.

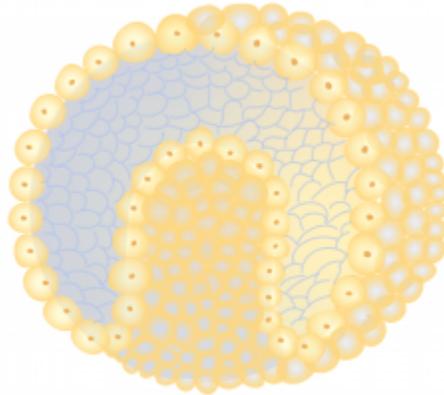


Figure 15.4 A gastrula

Ectoderm	Mesoderm	Endoderm
Skin	Muscle	Lung
Neurons	Kidney	Thyroid
Hair	Red blood cells	Pancreas
Nails	Gonads	Digestive tract
Eye lens	Heart	Bladder
Tooth enamel	Spleen	

Figure 15.5 Tissue types that arise from each of the three layers of the gastrula

Neurulation, the formulation of **neurula**, occurs around week 4 post-fertilization (week 6 of pregnancy). At this stage the neural tube (which becomes the spinal cord and brain) closes and the tissues that will eventually comprise the brain begin to fold inward. The embryo at this stage is about 3 mm in length (or about half the length of a grain of rice).

After neurulation, the limb buds begin to form and the embryo elongates. At 7 weeks post-fertilization (week 9

of pregnancy) the embryo is about 15 mm in length and has limbs and digital rays that will eventually become fingers and toes.

At around the 12th week after fertilization (week 14 of pregnancy) the embryo becomes classified as a fetus and the pregnancy enters the second trimester. The fetus is about 10 cm long at this point. Somewhere around the 24th week of pregnancy, the fetus, if delivered early, may be viable with medical intervention; current survival rates for babies born at 24 weeks, and that receive extensive medical care, are around 50%. The third trimester begins around the 27th week. Pregnancy continues until roughly 40 weeks after the date of last menstruation. For detailed explanation of the stages of embryonic and fetal development, see https://embryology.med.unsw.edu.au/embryology/index.php/Main_Page

Pluripotency

Cells that can differentiate into any cell type are **pluripotent**. They are often also called **stem cells**. Stem cells are of interest to scientists for basic research and for their potential for use in the treatment of certain human diseases such as blood cancers and neurodegenerative diseases. Embryonic stem cells are derived from the inner cells of a blastula. There are adult stem cells in places like bone marrow that are not completely pluripotent, but these cells can also differentiate into a more limited number of cell types. The use of human embryonic stem cells in research has been controversial because it involves the destruction of a human embryo at the blastula stage. The source for these embryonic stem cells has generally been extra embryos that were created by in-vitro fertilization (IVF; see description in the fertility treatments table in chapter 14).

As researchers have learned more about pluripotency, they have learned to induce adult non-stem cells into pluripotency. These types of cells may someday replace embryonic stem cells in research and therapeutic applications.



Check Yourself



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<https://open.lib.umn.edu/evolutionbiology/?p=2662#h5p-77>

15.4 Gestation

During pregnancy the female body goes through a lot of changes. See here <https://www.sciencealert.com/this-gif-shows-how-women-s-organs-shift-during-pregnancy> for an animation of some of the changes and how organs shift during pregnancy. There are many other physiological changes that happen in a female body when pregnant. Many of pregnancy's side effects are surprising. For example, very early in pregnancy the parent may find themselves out of breath after relatively mild physical exertion. One of the reasons for this is that increasing progesterone levels in the blood cause the body to respond differently to carbon dioxide in the bloodstream. Also, throughout pregnancy, hemoglobin (the oxygen-binding protein in blood) in the fetus binds oxygen much more tightly than does the mother's hemoglobin. Therefore, the fetus is taking oxygen away from the parent's blood, causing the them to breathe more deeply to replenish oxygen.

A newly discovered side-effect of pregnancy is that some of the fetal cells will wind up in the pregnant parent's tissues. In a recent study researchers looked for Y chromosome-containing cells in mothers who had given birth to sons –and found them in every tissue-type they investigated. The cells were present in low frequency, but the fact that they were there indicated that these cells had migrated from the fetus into the parent's tissues. It is unknown how long after pregnancy the cells persist in the parent's body and what effects these cells may have.



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<https://open.lib.umn.edu/evolutionbiology/?p=2672#h5p-78>

15.5 How are twins made?

Monozygotic twins (often called identical twins) occur when a zygote splits in two in early development and each of the resulting zygotes develop into a complete baby. Remember that the cells at this point are pluripotent, so the cells that split off are quickly replaced without any effect on the fully developed baby. The rate of monozygotic twins is around 4 per 1000 pregnancies that result in live birth. Monozygotic twins share virtually all the same genetic information (except for any new mutations that occur after separation).



Figure 15.6 Expedition 45/46 Commander, Astronaut Scott Kelly along with his brother, former Astronaut Mark Kelly speak to news media outlets about Scott Kelly's 1-year mission aboard the International Space Station.

1

Conjoined twins are twins that are monozygotic (they originate from the same embryo) but are joined together by shared tissues and organs. Conjoined twins are extremely rare (about 1 in 100,000 births). There are two proposed mechanisms by which conjoined twins are thought to occur. Either an early embryo splits incompletely, or two early embryos that have split fuse back together later in development. Conjoined twins can be conjoined in different locations. Depending on where they are conjoined and what structures they share, some conjoined twins can be surgically separated.

Dizygotic twins (often called fraternal twins) occur when two separate eggs are ovulated and fertilized (by different sperm), and then implant in the uterus. This can happen spontaneously if a female has two eggs that are ovulated in one cycle. The occurrence of dizygotic twins occurring without medical intervention varies and appears to be influenced by both genetic and environmental factors. Central Africa has the highest twinning rate

1. Photo Date: January 19, 2015. Location: Building 2. Photographer: Robert Markowitz

in the world with the country of Benin having close to 3% of babies born as twins. In other populations the rate is 10-fold lower. The possibility of dizygotic twins is greater if the egg-parent is taking certain types of fertility drugs that can induce multiple ovulation. Additionally, patients undergoing *in vitro* fertilization sometimes choose to have two or more embryos transferred to the uterus – making twins or higher-order multiples (e.g., triplets, quadruplets, etc.) more likely. Dizygotic twins are as genetically related to each other as any other sibling pair.

The frequency of higher-order multiple births (triplets, quadruplets and higher) is very low (around 5 for every 100,000 live births). Higher order multiples can occur spontaneously (without fertility treatments) and can occur through monozygotic, dizygotic, or a combination of twinning events.

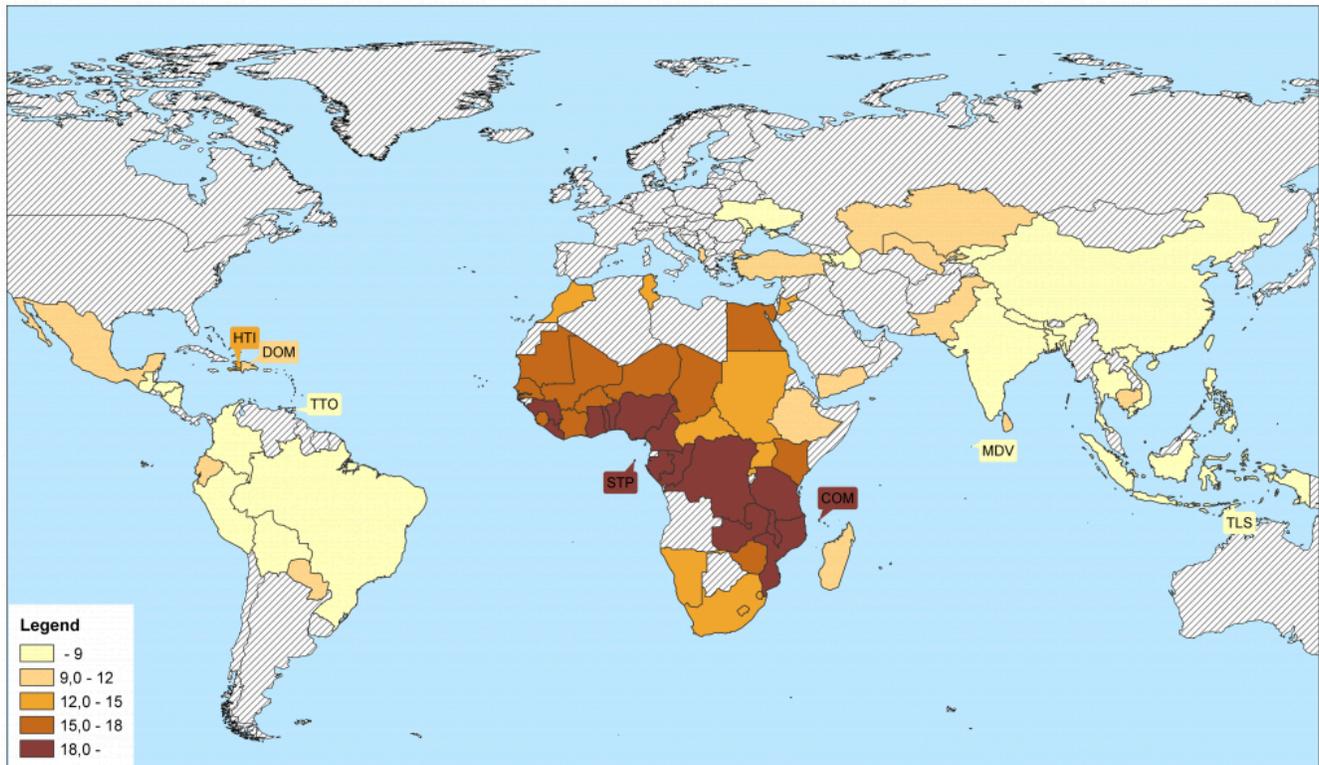


Figure 15.7 Twins per 1000 births.

2

In recent decades the frequency of dizygotic twins and higher-order multiple births has increased with the increased use of fertility drugs and procedures such as *in vitro* fertilization. The risks to both the pregnant parent and babies associated with twin pregnancies are much higher than those of single pregnancies. In fact, more than half of all twins are born preterm (before 37 weeks). While not all preterm births have lasting effects, preterm birth is associated with a suite of possible complications from short-term challenges to breathing and temperature regulation, to long-term physical or cognitive effects. For example, the risk of cerebral palsy is about 0.2% in singleton births, 1.4% for twins, and 4.5% for triplets. Multiple pregnancies are also associated with higher rates of *preeclampsia*, a condition that is associated with high maternal blood pressure and decreased organ function and, in severe cases, can be fatal to both the mother and baby.

15.6 Labor, delivery, and lactation

At about 40 weeks of gestation, a series of signals begins to ready the fetus for delivery and the pregnant body for labor. While the exact timing and sequence of events of labor can vary, labor and delivery is a classic positive feedback loop in that the early portions of labor amplify and further the onset of labor (see chapter 13 for a description of a positive feedback loop). Early labor can last between hours and weeks. In the early stages of labor there are mild uterine *contractions* that will move the baby's head against the cervix. The pressure of the baby's head stretch and begin to open the cervix. Nerve cells in the cervix detect the stretching and send signals to the hypothalamus in the brain to secrete the hormone **oxytocin** into the parent's circulation. Oxytocin triggers the uterus to contract more forcefully, pushing the baby's head further against the cervix and causing it to open further and signal for more oxytocin release. Uterine contractions eventually push the baby out through the open cervix and vagina.

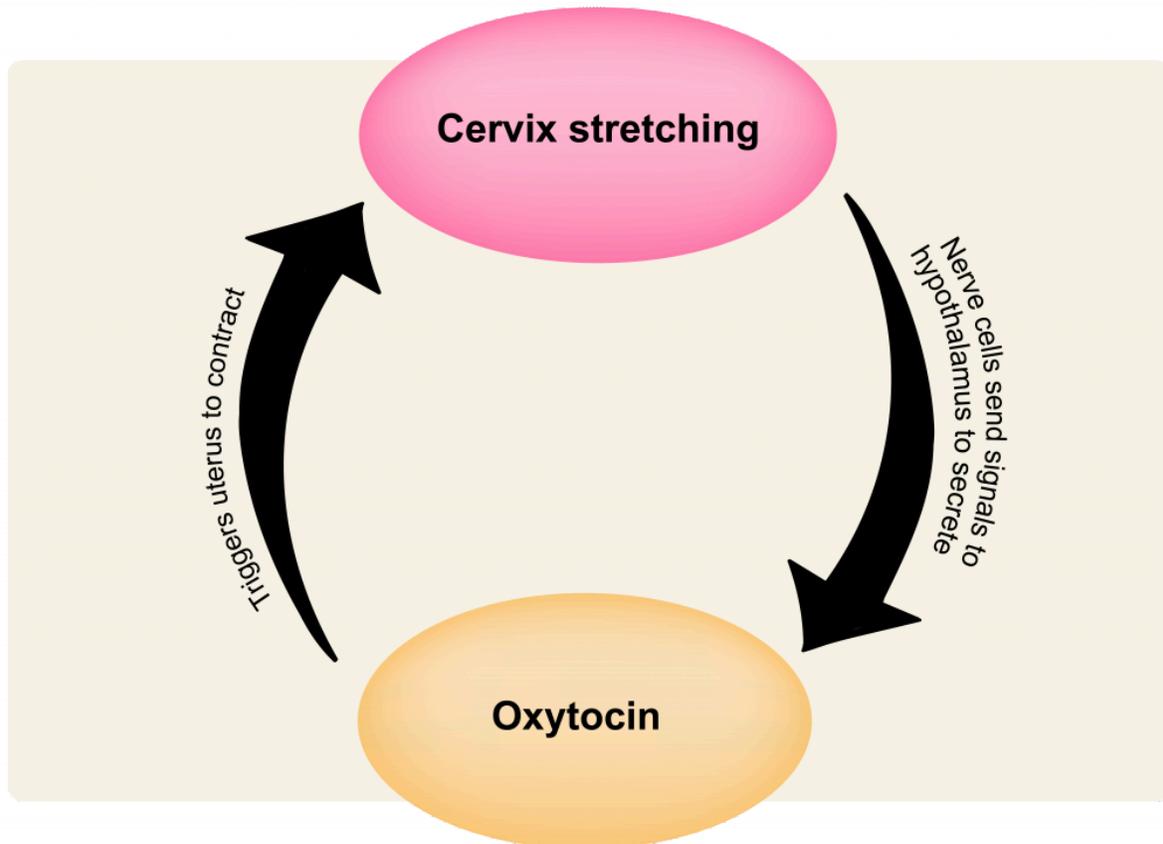


Figure 15.8 Positive feedback loop of labor. As the baby grows, its weight pushes on the cervix causing the cervix to stretch. This triggers the release of oxytocin, which causes the uterine contractions. The contractions push the baby against the cervix causing further cervical stretching.

Once the baby is delivered, additional uterine contractions push the placenta out as well. The expelled placenta is referred to as “afterbirth”. Here is a 3D imaging animation on the anatomical process of birth.



One or more interactive elements has been excluded from this version of the text. You can view them online here:
<https://open.lib.umn.edu/evolutionbiology/?p=2679#oembed-1>

Lactation

Oxytocin, the same hormone that stimulated uterine contractions, also triggers the “let down” of milk from the breasts. Nipple stimulation increases oxytocin release. When a baby can nurse immediately after delivery it can help stimulate uterine contractions that will expel the placenta (or afterbirth). The early milk that a newborn receives is called **colostrum**. Colostrum contains high levels of antibodies and white blood cells from the parent; these can help the baby avoid infections. In addition to these immunity benefits, lactation is important in forging parent-infant bonds. The hormone oxytocin, which was involved in the initiation of lactation, is also involved in establishing this early attachment.



Check Yourself



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<https://open.lib.umn.edu/evolutionbiology/?p=2679#h5p-80>

15.7 Abortion

It is difficult to have a thorough discussion of baby making without a discussion of abortion. Few topics are more divisive than abortion, especially in countries such as the United States. The controversies surrounding abortion encompass legal, ethical, and moral issues that are beyond the scope of this text (and beyond the expertise of its authors). Thus, our discussion of abortion will be restricted to current abortion methods and then the legal standing of abortion in the United States. This information can help inform both one's opinions on whether and under what conditions abortions should be legal and whether one considers an abortion a personally acceptable choice. However, this information alone cannot address the ethical and moral questions.

Abortion is defined as the termination of an established pregnancy. Somewhat confusingly, medical personnel sometimes refer to miscarriages (loss of a pregnancy by non-deliberate means) as “spontaneous abortions.” Abortions that are done to purposely end a pregnancy are sometimes called “**induced abortions.**” In this text when we use the term abortion we are referring to induced abortions.

The vast majority (over 90%) of abortions occur in the first trimester. Abortions within the first trimester can be conducted by the administration of medicine or by physical extraction. Medicinal abortions are done in the U.S. until the 9th or 10th week of pregnancy. The most common medication for this purpose is a combination of pills commonly called RU486. One pill, *mifepristone*, functions by binding to progesterone receptors. As discussed earlier, progesterone is the hormone that maintains the lining of the uterus during a pregnancy. When the progesterone receptors are bound by this drug, progesterone cannot signal to maintain the uterine lining. The second drug, called *misoprostol*, causes the cervix to soften and the uterus to contract. Together these drugs cause the uterine lining to shed along with the implanted embryo.

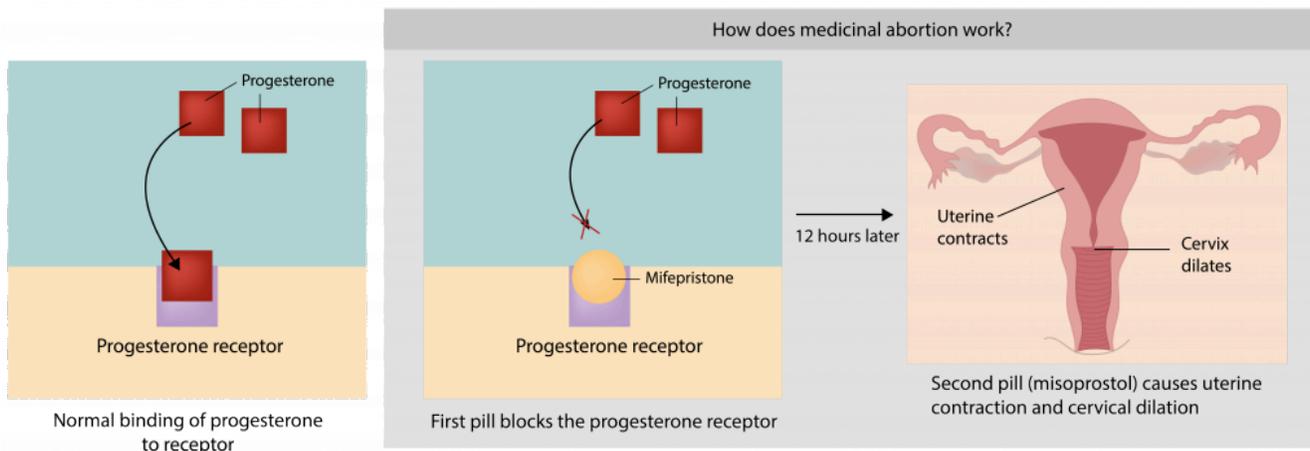


Figure 15.9 Mechanism of medicinal abortion (RU486).

Note: This abortion-pill method disrupts an established pregnancy (an embryo that is implanted in the uterine lining). This is **not** the same as emergency contraception, in which the establishment of a pregnancy is prevented

(see more details about emergency contraception in [Chapter 14](#)).

An abortion by physical extraction involves either manual or drug-induced opening of the cervix and the placement of a small tube into the uterus. The tube suctions out the contents of the uterus with suction from a syringe or with a mechanical vacuum pump. Early term abortions that are overseen by qualified medical personnel are relatively safe for the pregnant person (that is, safer than carrying a pregnancy to term).

Second trimester abortions are much less common, and involve a more complicated procedure that involves the scraping of the lining of the uterus. Third trimester abortions are extremely rare and are often the result of life-threatening conditions to the pregnant individual, or discovery of fetal abnormalities that indicate the baby will not survive. The risks of abortion to the pregnant person increase with increasing stages of pregnancy.

In the United States prior to 1973, the legality of induced abortion was determined on a state-by-state basis. However, in 1973 the case of patient “Roe” went to the U.S. Supreme Court after she was denied an abortion in Texas. The lawyers for Roe argued that an abortion fell under medical privacy. The Supreme Court agreed, with the caveat that this right was balanced by the government’s interests in maternal health and potential human life. They determined that the state’s ability to interfere with pregnancy termination was influenced by fetal viability (the potential for the fetus to survive outside the uterus). However, the date at which a fetus is viable is somewhat of a moving metric, depending on medical advances, medical care available, and individual development. Since 1973, many states have instituted some barriers to abortion including waiting periods, mandatory ultrasounds, and counseling prior to abortion. However, the court system has repeatedly struck down bans of abortion during the first trimester of pregnancy or limitations on abortion when the life or health of the pregnant person is compromised. In 2018 and 2019, numerous states (8 as of the publishing of this text, with other states moving in this direction) have passed laws restricting first trimester abortions, including a complete ban on almost all abortions in Alabama. These laws are likely to spend a long time in the court system to determine if they will eventually take effect.

Aside from the legal restrictions on abortion, there are many states that have very few abortion providers, meaning that abortion is not effectively available to people without means to travel. For example, in Mississippi 99% of parishes (counties) do not have an abortion provider. Other states in which more than 95% of counties do not have abortion providers include Nebraska, the Dakotas, Kansas, West Virginia, Wisconsin and Wyoming.

15.8 Wrapping Up: Revisiting the missing mother case



Revisiting the missing mother case

Recall the woman who did not seem to be the genetic mother of her children. Upon further testing, cells from different parts of her body were found to be genetically distinct from the cells her doctors had originally tested. The DNA in these other cells matched what one would expect from the mother of the tested children. Scientists determined that this woman is an example of a rare biological phenomenon of **chimerism**. When her mother was pregnant, there were two fertilized embryos (as you would have in the case of dizygotic twins), however these two embryos implanted very close to one another in the uterus. During the very early stages of development, the embryos merged and the early cells differentiated into endoderm, mesoderm, and ectoderm. Those cells from one embryo differentiated into some portions of the woman's body, those from the other embryo differentiated into other portions. Because most people never undergo this kind of genetic testing, the frequency of chimerism is not known.

As this case of chimerism became nationally known, it saved another woman who was mired in an uphill legal and custody battle. This second woman had DNA testing done as a routine measure when pursuing child support. The results came back that the children's father was the biological father, but that she did not seem to be the children's biological mother. She was accused of fraud and threatened with losing custody of her children. The woman was pregnant at the time and was forced to have a legal witness and immediate genetic testing during the birth of her next child who was also found to not be a match. At this point she was suspected of running some sort of surrogacy scam, and was threatened with further legal action. It wasn't until news coverage of the first woman's chimerism that the second woman was more thoroughly tested and found to have chimerism also. Her children were her biological offspring, but she was a chimera between two sibling zygotes that fused in the womb. The judge dismissed all accusations against her. And now this woman can accurately claim to be both biological mother and aunt to her children!

As we conclude this chapter and prepare for in-class discussion, be sure to return to the chapter's [goals and objectives](#).

Chapter 16: Sex & Violence

This is a planned chapter.

Chapter 17: The Biology of Love

This is a planned chapter.

Icon Key

When you approach any icon in the textbook,		
Icon	Description	What you should focus on
	Goals & Objectives	The goals and objectives for each chapter are meant to serve 2 functions: 1. To prepare you for the upcoming reading 2. To convey our expectations for what you should be able to do at the end of the reading
	Points to Ponder	Some questions in each chapter either require deeper thought or do not have obvious answers, but rather are simply points to ponder.
	Biology is Sexy	Sometimes the content may not seem overtly related to sex, however, everything we discuss is related to sex.
	Read More	We want to share a lot of interesting material with you, but some of the content is not directly related to the chapter's goals and objectives.
	Check Yourself	Throughout each chapter there will be opportunities for you to test your knowledge before proceeding to new content.
	Wrapping It Up	We begin each chapter with a biological problem and return to the problem at the end of each chapter in an attempt to wrap it up.