

## Example texts

The Conference of the Parties to the FCCC has regularly met since 1995. The “Berlin Mandate” negotiating process concentrated on strengthening emission reduction commitments of Annex 1 Parties in the period after 2000. It concluded in 1997 with the adoption of the Kyoto Protocol. The Protocol established legally binding emission constraints for Annex 1 Parties including New Zealand. Different percentage reductions relative to 1990 emissions were set for different countries such that total global emissions are reduced by more than 5 %. New Zealand’s commitment is not to exceed 1990 emission levels, on average, during 2008-2012. Demonstrable progress towards meeting commitments is to be achieved by 2005. Forest sinks can be included as changes in forest carbon storage since January 1990. Countries may participate in emissions trading to meet their commitments as well as undertaking joint implementation to reduce emissions or planting forests. The “clean development” mechanism provides for a system of credits for projects in developing countries to reduce emissions and can lead to increase in the overall level of allowed emissions.

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Wang Q, Leichtman MD, White SH Childhood memory and self-description in young Chinese adults: the impact of growing up an only child. *Cognition* 1998 Nov;69(1):73-103. This study examined the relationship between self-description and childhood memory in 255 Chinese young adults. Ninety-nine participants were from only child families and 156 had siblings. All participants completed

two questionnaires: a version of the Twenty Statements Test of Kuhn and McPartland (Kuhn, M.H., McPartland, T.S., 1954. An empirical investigation of self-attitudes. *American Sociological Review* 19, 68-76) eliciting self-descriptions, and an instrument asking for earliest and other childhood memories. Based on theories positing a relationship between autobiography and the organization of the self, we predicted differences on both measures between only- and sibling-child participants. Findings indicated that compared with sibling children, only children had more private and fewer collective self-descriptions, earlier first memories, more specific and more self-focused memories. In addition, autobiographical measures were influenced by cohort, gender, preschool attendance, and urban/rural family effects. Findings are discussed in terms of literature on autobiography, the self and childhood in China.

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BENJAMIN FRANKLIN was born in Milk Street, Boston, on January 6, 1706. His father, Josiah Franklin, was a tallow chandler who married twice, and of his seventeen children Benjamin was the youngest son. His schooling ended at ten, and at twelve he was bound apprentice to his brother James, a printer, who published the "New England Courant." To this journal he became a contributor, and later was for a time its nominal editor. But the brothers quarreled, and Benjamin ran away, going first to New York, and thence to Philadelphia, where he arrived in October, 1723. He soon obtained work as a printer, but after a few months he was induced by Governor Keith to go to London, where, finding Keith's promises empty, he again worked as a compositor till he was brought back to Philadelphia by a merchant named Denman, who gave him a position in his business. On Denman's death he returned to his former trade, and shortly set up a printing house of his own from which he

published "The Pennsylvania Gazette," to which he contributed many essays, and which he made a medium for agitating a variety of local reforms. In 1732 he began to issue his famous "Poor Richard's Almanac" for the enrichment of which he borrowed or composed those pithy utterances of worldly wisdom which are the basis of a large part of his popular reputation. In 1758, the year in which he ceases writing for the Almanac, he printed in it "Father Abraham's Sermon," now regarded as the most famous piece of literature produced in Colonial America.

Meantime Franklin was concerning himself more and more with public affairs. He set forth a scheme for an Academy, which was taken up later and finally developed into the University of Pennsylvania; and he founded an "American Philosophical Society" for the purpose of enabling scientific men to communicate their discoveries to one another. He himself had already begun his electrical researches, which, with other scientific inquiries, he called on in the intervals of money-making and politics to the end of his life. In 1748 he sold his business in order to get leisure for study, having now acquired comparative wealth; and in a few years he had made discoveries that gave him a reputation with the learned throughout Europe. In politics he proved very able both as an administrator and as a controversialist; but his record as an office-holder is stained by the use he made of his position to advance his relatives. His most notable service in home politics was his reform of the postal system; but his fame as a statesman rests chiefly on his services in connection with the relations of the Colonies with Great Britain, and later with France. In 1757 he was sent to England to protest against the influence of the Penns in the government of the colony, and for five years he remained there, striving to enlighten the people and the ministry of England as to Colonial conditions. On his return to America he played an honorable part in the Paxton affair, through which he lost his seat in the Assembly; but in 1764 he was again despatched to England as agent for the colony, this time to petition the King to resume the government from the hands of the proprietors. In London he actively opposed the proposed Stamp Act, but lost the credit for this and much of his popularity through his securing for a friend the office of stamp agent in America. Even his effective work in helping to obtain the repeal of the act left him still a suspect; but he continued his efforts to present the case for the Colonies as the troubles thickened toward the crisis of the Revolution. In 1767 he crossed to France, where he was received with honor; but before his return home in 1775 he lost his position as postmaster through his share in divulging to Massachusetts the famous letter of Hutchinson and Oliver. On his arrival in Philadelphia he was chosen a member of the Continental Congress and in 1777 he was despatched to France as commissioner for the United States. Here he remained till 1785, the favorite of French society; and with such success did he conduct the affairs of his country that when he finally returned he received a place only second to that of Washington as the champion of American independence. He died on April 17, 1790.

Eliot, C.W. 1909. The Autobiography Of Benjamin Franklin. New York: P F Collier & Son Company. <http://www.infomotions.com/etexts/literature/american/1700-1799/franklin-autobiography-244.txt>

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## Human Gene Affects Memory

NIH scientists have shown that a common gene variant influences memory for events in humans by altering a growth factor in the brain's memory hub. On average, people with a particular version of the gene that codes for brain derived neurotrophic factor (BDNF) performed worse on tests of episodic memory - tasks like recalling what happened yesterday. They also showed differences in activation of the hippocampus, a brain area known to mediate memory, and signs of decreased neuronal health and interconnections. These effects are likely traceable to limited movement and secretion of BDNF within cells, according to the study, which reveals how a gene affects the normal range of human memory, and confirms that BDNF affects human hippocampal function much as it does animals'.

Long known to be critical for the growth and survival of neurons, BDNF has also recently been shown to play a key role in memory and hippocampal function in animals. To find out if it works similarly in humans, the researchers explored the consequences of a tiny variance in the human BDNF gene, where its molecular makeup differs slightly across individuals. People inherit two copies of the BDNF gene - one from each parent - in either of two versions. Slightly more than a third inherit at least one copy of a version nicknamed "met," which the researchers have now linked to poorer memory. It's called "met" because its chemical sequence contains the amino acid methionine in a location where the more common version, "val," contains valine.

"We are finding that this one amino acid substitution exerts a substantial influence on human memory, presumably because of its effects on the biology of the hippocampus," said Weinberger.

Despite its negative effect on memory, the "met" version's survival in the human genome suggests that it "may confer some compensatory advantage in other biological processes," note the researchers. Although they found that it does not confer increased susceptibility to schizophrenia, they suggest that the "met" variant might contribute to risk for - or increase functional impairment in - other disorders involving hippocampal dysfunction, such as Alzheimer's disease or mood disorders.

Drawing on participants in the NIMH intramural sibling study of schizophrenia, Egan and colleagues first assessed their hippocampal function and related it to their BDNF gene types.

Among 641 normal controls, schizophrenia patients, and their unaffected siblings, those who had inherited two copies of the "met" variant scored significantly lower than their matched peers on tests of verbal episodic (event) memory. Most notably, normal controls with two copies of "met" scored 40 percent on delayed recall, compared to 70 percent for those

with two copies of "val." BDNF gene type had no significant effect on tests of other types of memory, such as semantic or working memory.

The researchers then measured brain activity in two separate groups of healthy subjects while they were performing a working memory task that normally turns off hippocampus activity. Functional magnetic resonance imaging (fMRI) scans revealed that those with one copy of "met" showed a pattern of activation along the sides of the hippocampus, in contrast to lack of activation among those with two copies of "val."

Next, an MRI scanner was used to measure levels of a marker inside neurons indicating the cell's health and abundance of synapses - tiny junctions through which neurons communicate with each other. Again, subjects with one copy of "met" had lower levels of the marker, N-acetyl-aspartate (NAA), than matched individuals with two copies of "val." Analysis showed that NAA levels dropped as the number of inherited "met" variants increased, suggesting a possible "dose effect."

Unlike other growth factors, hippocampal BDNF is secreted, in part, in response to neuronal activity, making it a likely candidate for a key role in synaptic plasticity, learning and memory. To explore possible mechanisms underlying the observed "met"-related memory effect, the researchers examined the distribution, processing and secretion of the BDNF proteins expressed by the two different gene variants within hippocampal cells. When they tagged the gene variants with green fluorescent protein and introduced them into cultured neurons, they discovered that "val" BDNF spreads throughout the cell and into the branch-like dendrites that form synapses, while "met" BDNF mostly clumps inside the cell body without being transported to the synapses. To regulate memory function, BDNF must be secreted near the synapses.

"We were surprised to see that 'met' BDNF secretion can't be properly regulated by neural activity," said Lu.

The observed memory decrements are likely traceable to the failure of "met" BDNF to reach the synapses, as well as its inability to secrete in response to neuronal activity, say the researchers.

"Our study provides direct in vivo data that the molecular mechanisms related to activity dependent BDNF secretion and signaling, such as synaptic plasticity, may underlie humans' greatly expanded verbally-mediated memory system, just as it does for more rudimentary forms of memory in animals," said Egan.

In following-up their leads, the researchers are searching for a possible BDNF connection with the memory problems and hippocampal changes of Alzheimer's disease, depression and normal aging.

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### **BDNF plays a key role in memory**

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### **Two versions of BDNF gene**

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### **Met variant**

Despite its negative effect on memory, the "met" version's survival in the human genome suggests that it "may confer some compensatory advantage in other biological processes," note the researchers. Although they found that it does not confer increased susceptibility to schizophrenia, they suggest that the "met" variant might contribute to risk for - or increase functional impairment in - other disorders involving hippocampal dysfunction, such as Alzheimer's disease or mood disorders.

### **Schizophrenia study**

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#### **How BDNF works**

Unlike other growth factors, hippocampal BDNF is secreted, in part, in response to neuronal activity, making it a likely candidate for a key role in synaptic plasticity, learning and memory. To explore possible mechanisms underlying the observed "met"- related memory effect, the researchers examined the distribution, processing and secretion of the BDNF proteins expressed by the two different gene variants within hippocampal cells. When they tagged the gene variants with green fluorescent protein and introduced them into cultured neurons, they discovered that "val" BDNF spreads throughout the cell and into the branch-like dendrites that form synapses, while "met" BDNF mostly clumps inside the cell body without being transported to the synapses. To regulate memory function, BDNF must be secreted near the synapses.

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#### **Possible connection with memory problems in old age**

In following-up their leads, the researchers are searching for a possible BDNF connection with the memory problems and hippocampal changes of Alzheimer's disease, depression and normal aging.

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### **Human gene affects memory for events**

#### **People with a particular variant of the gene controlling the brain factor BDNF have reduced hippocampal function**

NIH scientists have shown that a common gene variant influences memory for events in humans by altering a growth factor in the brain's memory hub. On average, people with a particular version of the gene that codes for brain derived neurotrophic factor (BDNF) performed worse on tests of episodic memory - tasks like recalling what happened yesterday. They also showed differences in activation of the hippocampus, a brain area known to mediate memory, and signs of decreased neuronal health and interconnections. These effects

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#### **Two variants: "Met" variant linked to poorer episodic memory; "Val" variant more common**

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#### **"Met" variant might increase risk of Alzheimer's and other disorders involving the hippocampus**

Despite its negative effect on memory, the "met" version's survival in the human genome suggests that it "may confer some compensatory advantage in other biological processes," note the researchers. Although they found that it does not confer increased susceptibility to schizophrenia, they suggest that the "met" variant might contribute to risk for - or increase functional impairment in - other disorders involving hippocampal dysfunction, such as Alzheimer's disease or mood disorders.

#### **Study finds those with two copies of "met" perform dramatically worse on tests of episodic memory but not on other memory tests**

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#### **Two copies of "met" worse than one, but any "met" variant is associated with hippocampal dysfunction**

The researchers then measured brain activity in two separate groups of healthy subjects while they were performing a working memory task that normally turns off hippocampus activity. Functional magnetic resonance imaging (fMRI) scans revealed that those with one copy of "met" showed a pattern of activation along the sides of the hippocampus, in contrast to lack of activation among those with two copies of "val."

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#### **"Met" variant less successful in distributing BDNF proteins to the synapses**

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### **Text 1**

"Consolidation" is a term that is bandied about a lot in recent memory research. Here's my take on what it means.

Initially, information is thought to be encoded as patterns of neural activity - cells "talking" to each other. Later, the information is coded in more persistent molecular or structural formats (e.g., the formation of new synapses). It has been assumed that once this occurs, the memory is "fixed" — a permanent, unchanging, representation.

With new techniques, it has indeed become possible to observe these changes. Researchers found that the changes to a cell that occurred in response to an initial stimulation lasted some three to five minutes and disappeared within five to 10 minutes. If the cell was stimulated four times over the course of an hour, however, the synapse would actually split and new synapses would form, producing a (presumably) permanent change.

The hypothesis that new memories consolidate slowly over time was proposed 100 years ago, and continues to guide memory research. In modern consolidation theory, it is assumed that new memories are initially 'labile' and sensitive to disruption before undergoing a series of processes (e.g., glutamate release, protein synthesis, neural growth and rearrangement) that render the memory representations progressively more stable. It is these processes that are generally referred to as “consolidation”.

Recently, however, the idea has been gaining support that stable representations can revert to a labile state on reactivation.

In a way, this is not surprising. We already have ample evidence that retrieval is a dynamic process during which new information merges with and modifies the existing representation — memory is now seen as reconstructive, rather than a simple replaying of stored information

Researchers who have found evidence that supposedly stable representations have become labile again after reactivation, have called the process “reconsolidation”, and suggest that consolidation, rather than being a one-time event, occurs repeatedly every time the representation is activated.

This raises the question: does reconsolidation involve replacing the previously stable representation, or the establishment of a new representation, that coexists with the old?

Whether reconsolidation is the creating of a new representation, or the modifying of an old, is this something other than the reconstruction of memories as they are retrieved? In other words, is this recent research telling us something about consolidation (part of the encoding process), or something about reconstruction (part of the retrieval process)?

The principal player in memory consolidation research, in terms of brain regions, is the hippocampus. The hippocampus is involved in the recognition of place and the consolidation of contextual memories, and is part of a region called the medial temporal lobe (MTL), that also includes the perirhinal, parahippocampal, and entorhinal cortices. Lesions in the medial temporal lobe typically produce amnesia characterized by the disproportionate loss of recently acquired memories. This has been interpreted as evidence for a memory consolidation process.

Some research suggests that the hippocampus may participate only in consolidation processes lasting a few years. The entorhinal cortex, on the other hand, gives evidence of temporally graded changes extending up to 20 years, suggesting that it is this region that participates in memory consolidation over decades. The entorhinal cortex is damaged in the early stages of Alzheimer's disease.

There is, however, some evidence that the hippocampus can be involved in older memories — perhaps when they are particularly vivid.

A recent idea that has been floated suggests that the entorhinal cortex, through which all information passes on its way to the hippocampus, handles “incremental learning” — learning that requires repeated experiences. “Episodic learning” — memories that are stored after only one occurrence — might be mainly stored in the hippocampus.

This may help explain the persistence of some vivid memories in the hippocampus. Memories of emotionally arousing events tend to be more vivid and to persist longer than do memories of neutral or trivial events, and are, moreover, more likely to require only a single experience.

Whether or not the hippocampus may retain some older memories, the evidence that some memories might be held in the hippocampus for several years, only to move on, as it were, to another region, is another challenge to a simple consolidation theory.

So where does all this leave us? What is consolidation? Do memories reach a fixed state?

My own feeling is that, no, memories don't reach this fabled "cast in stone" state. Memories are subject to change every time they are activated (such activation doesn't have to bring the memory to your conscious awareness). But consolidation traditionally (and logically) refers to encoding processes. It is reasonable, and useful, to distinguish between:

- the initial encoding, the "working memory" state, when new information is held precariously in shifting patterns of neural activity,
- the later encoding processes, when the information is consolidated into a more permanent form with the growth of new connections between nerve cells,
- the (possibly much) later retrieval processes, when the information is retrieved in, most probably, a new context, and is activated anew

I think that "reconsolidation" is a retrieval process rather than part of the encoding processes, but of course, if you admit retrieval as involving a return to the active state and a modification of the original representation in line with new associations, then the differences between retrieval and encoding become less evident.

When you add to this the possibility that memories might "move" from one area of the brain to another after a certain period of time (although it is likely that the triggering factor is not time per se), then you cast into disarray the whole concept of memories becoming stable.

Perhaps our best approach is to see memory as a series of processes, and consolidation as an agreed-upon (and possibly arbitrary) subset of those processes.

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## Text 2

At the height of the Ice Age, between 34,000 and 30,000 B.C., much of the world's water was locked up in vast continental ice sheets. As a result, the Bering Sea was hundreds of meters below its current level, and a land bridge, known as Beringia, emerged between Asia and North America. At its peak, Beringia is thought to have been some 1,500 kilometers

wide. A moist and treeless tundra, it was covered with grasses and plant life, attracting the large animals that early humans hunted for their survival.

The first people to reach North America almost certainly did so without knowing they had crossed into a new continent. They would have been following game, as their ancestors had for thousands of years, along the Siberian coast and then across the land bridge.

Once in Alaska, it would take these first North Americans thousands of years more to work their way through the openings in great glaciers south to what is now the United States. Evidence of early life in North America continues to be found. Little of it, however, can be reliably dated before 12,000 B.C.; a recent discovery of a hunting lookout in northern Alaska, for example, may date from almost that time. So too may the finely crafted spear points and items found near Clovis, New Mexico.

Similar artifacts have been found at sites throughout North and South America, indicating that life was probably already well established in much of the Western Hemisphere by some time prior to 10,000 B.C. Around that time the mammoth began to die out and the bison took its place as a principal source of food and hides for these early North Americans. Over time, as more and more species of large game vanished whether from overhunting or natural causes plants, berries, and seeds became an increasingly important part of the early American diet. Gradually, foraging and the first attempts at primitive agriculture appeared. Native Americans in what is now central Mexico led the way, cultivating corn, squash, and beans, perhaps as early as 8,000 B.C. Slowly, this knowledge spread northward.

By 3,000 B.C., a primitive type of corn was being grown in the river valleys of New Mexico and Arizona. Then the first signs of irrigation began to appear, and, by 300 B.C., signs of early village life.

By the first centuries A.D., the Hohokam were living in settlements near what is now Phoenix, Arizona, where they built ball courts and pyramid like mounds reminiscent of those found in Mexico, as well as a canal and irrigation system.

The first Native-American group to build mounds in what is now the United States often are called the Adenans. They began constructing earthen burial sites and fortifications around 600 B.C. Some mounds from that era are in the shape of birds or serpents; they probably served religious purposes not yet fully understood.

The Adenans appear to have been absorbed or displaced by various groups collectively known as Hopewellians. One of the most important centers of their culture was found in southern Ohio, where the remains of several thousand of these mounds still can be seen. Believed to be great traders, the Hopewellians used and exchanged tools and materials across a wide region of hundreds of kilometers.

By around 500 A.D., the Hopewellians disappeared, too, gradually giving way to a broad group of tribes generally known as the Mississippians or Temple Mound culture. One city, Cahokia, near Collinsville, Illinois, is thought to have had a population of about 20,000 at its peak in the early 12th century. At the center of the city stood a huge earthen mound, flattened at the top, that was 30 meters high and 37 hectares at the base. Eighty other mounds have been found nearby.

Cities such as Cahokia depended on a combination of hunting, foraging, trading, and agriculture for their food and supplies. Influenced by the thriving societies to the south, they evolved into complex hierarchical societies that took slaves and practised human sacrifice.

In what is now the southwest United States, the Anasazi, ancestors of the modern Hopi Indians, began building stone and adobe pueblos around the year 900. These unique and amazing apartment-like structures were often built along cliff faces; the most famous, the "cliff palace" of Mesa Verde, Colorado, had more than 200 rooms. Another site, the Pueblo Bonito ruins along New Mexico's Chaco River, once contained more than 800 rooms.

Perhaps the most affluent of the pre-Columbian Native Americans lived in the Pacific Northwest, where the natural abundance of fish and raw materials made food supplies plentiful and permanent villages possible as early as 1,000 B.C. The opulence of their "potlatch" gatherings remains a standard for extravagance and festivity probably unmatched in early American history.

The America that greeted the first Europeans was, thus, far from an empty wilderness. It is now thought that as many people lived in the Western Hemisphere as in Western Europe at that time -- about 40 million. Estimates of the number of Native Americans living in what is now the United States at the onset of European colonization range from two to 18 million, with most historians tending toward the lower figure. What is certain is the devastating effect that European disease had on the indigenous population practically from the time of initial contact. Smallpox, in particular, ravaged whole communities and is thought to have been a much more direct cause of the precipitous decline in the Indian population in the 1600s than the numerous wars and skirmishes with European settlers.

Indian customs and culture at the time were extraordinarily diverse, as could be expected, given the expanse of the land and the many different environments to which they had adapted. Some generalizations, however, are possible. Most tribes, particularly in the wooded eastern region and the Midwest, combined aspects of hunting, gathering, and the cultivation of maize and other products for their food supplies. In many cases, the women were responsible for farming and the distribution of food, while the men hunted and participated in war.

By all accounts, Native-American society in North America was closely tied to the land. Identification with nature and the elements was integral to religious beliefs. Their life was essentially clan-oriented and communal, with children allowed more freedom and tolerance than was the European custom of the day.

Although some North American tribes developed a type of hieroglyphics to preserve certain texts, Native-American culture was primarily oral, with a high value placed on the recounting of tales and dreams. Clearly, there was a good deal of trade among various groups and strong evidence exists that neighboring tribes maintained extensive and formal relations — both friendly and hostile.

The first Europeans to arrive in North America — at least the first for whom there is solid evidence — were Norse, traveling west from Greenland, where Erik the Red had founded a settlement around the year 985. In 1001 his son Leif is thought to have explored the northeast coast of what is now Canada and spent at least one winter there.

While Norse sagas suggest that Viking sailors explored the Atlantic coast of North America down as far as the Bahamas, such claims remain unproven. In 1963, however, the ruins of

some Norse houses dating from that era were discovered at L'Anse-aux-Meadows in northern Newfoundland, thus supporting at least some of the saga claims.

In 1497, just five years after Christopher Columbus landed in the Caribbean looking for a western route to Asia, a Venetian sailor named John Cabot arrived in Newfoundland on a mission for the British king. Although quickly forgotten, Cabot's journey was later to provide the basis for British claims to North America. It also opened the way to the rich fishing grounds off George's Banks, to which European fishermen, particularly the Portuguese, were soon making regular visits.

Columbus never saw the mainland of the future United States, but the first explorations of it were launched from the Spanish possessions that he helped establish. The first of these took place in 1513 when a group of men under Juan Ponce de León landed on the Florida coast near the present city of St. Augustine.

With the conquest of Mexico in 1522, the Spanish further solidified their position in the Western Hemisphere. The ensuing discoveries added to Europe's knowledge of what was now named America — after the Italian Amerigo Vespucci, who wrote a widely popular account of his voyages to a "New World." By 1529 reliable maps of the Atlantic coastline from Labrador to Tierra del Fuego had been drawn up, although it would take more than another century before hope of discovering a "Northwest Passage" to Asia would be completely abandoned.

Among the most significant early Spanish explorations was that of Hernando De Soto, a veteran conquistador who had accompanied Francisco Pizarro in the conquest of Peru. Leaving Havana in 1539, De Soto's expedition landed in Florida and ranged through the southeastern United States as far as the Mississippi River in search of riches.

Another Spaniard, Francisco Vázquez de Coronado, set out from Mexico in 1540 in search of the mythical Seven Cities of Cibola. Coronado's travels took him to the Grand Canyon and Kansas, but failed to reveal the gold or treasure his men sought. However, his party did leave the peoples of the region a remarkable, if unintended, gift: Enough of his horses escaped to transform life on the Great Plains. Within a few generations, the Plains Indians had become masters of horsemanship, greatly expanding the range and scope of their activities.



While the Spanish were pushing up from the south, the northern portion of the present-day United States was slowly being revealed through the journeys of men such as Giovanni da Verrazano. A Florentine who sailed for the French, Verrazano made landfall in North Carolina in 1524, then sailed north along the Atlantic Coast past what is now New York harbor.

A decade later, the Frenchman Jacques Cartier set sail with the hope — like the other Europeans before him — of finding a sea passage to Asia. Cartier's expeditions along the St. Lawrence River laid the foundation for the French claims to North America, which were to last until 1763.

Following the collapse of their first Quebec colony in the 1540s, French Huguenots attempted to settle the northern coast of Florida two decades later. The Spanish, viewing the French as a threat to their trade route along the Gulf Stream, destroyed the colony in 1565. Ironically, the leader of the Spanish forces, Pedro Menéndez, would soon establish a town not far away — St. Augustine. It was the first permanent European settlement in what would become the United States.

[taken from the *Outline of U.S. History*, published by the U.S. Department of State.  
<http://usinfo.state.gov/products/pubs/historyotln/index.htm>]

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### **Text 3**

In this section, we will explore what is ozone and what is ultraviolet radiation. We then will explore the relationship between ozone and ultraviolet radiation from the sun. It is here that ozone plays its essential role in shielding the surface from harmful ultraviolet radiation. By screening out genetically destructive ultraviolet radiation from the Sun, ozone protects life on the surface of Earth. It is for this reason that ozone acquires an enormous importance. It is why we study it so extensively.

About 90% of the ozone in our atmosphere is contained in the stratosphere, the region from about 10 to 50-km (32,000 to 164,000 feet) above Earth's surface. Ten percent of the ozone is contained in the troposphere, the lowest part of our atmosphere where all of our weather takes place. Measurements taken from instruments on the ground, flown on balloons, and operating in space show that ozone concentrations are greatest between about 15 and 30 km.

Although ozone concentrations are very small, typically only a few molecules  $O_3$  per million molecules of air, these ozone molecules are vitally important to life because they absorb the biologically harmful ultraviolet radiation from the Sun. There are three different types of ultraviolet (UV) radiation, based on the wavelength of the radiation. These are referred to as UV-a, UV-b, and UV-c. UV-c (red) is entirely screened out by ozone around 35 km altitude, while most UV-a (blue) reaches the surface, but it is not as genetically damaging, so we don't worry about it too much. It is the UV-b (green) radiation that can cause sunburn and that can also cause genetic damage, resulting in things like skin cancer, if exposure to it is prolonged. Ozone screens out most UV-b, but some reaches the surface. Were the ozone layer to decrease, more UV-b radiation would reach the surface, causing increased genetic damage to living things.

Because most of the ozone in our atmosphere is contained in the stratosphere, we refer to this region as the stratospheric ozone layer. In contrast to beneficial stratospheric ozone, tropospheric ozone is a pollutant found in high concentrations in smog. Though it too absorbs UV radiation, breathing it in high levels is unhealthy, even toxic. The high reactivity of ozone results in damage to the living tissue of plants and animals. This damage by heavy tropospheric ozone pollution is often manifested as eye and lung irritation. Tropospheric ozone is mainly produced during the daytime in polluted regions such as urban areas. Significant government efforts are underway to regulate the gases and emissions that lead to this harmful pollution, and smog alerts are regular occurrences in polluted urban areas.

To appreciate the importance of stratospheric ozone, we need to understand something of the Sun's output and how it impacts living systems. The Sun produces radiation at many different wavelengths. These are part of what is known as the electromagnetic (EM) spectrum. EM radiation includes everything from radio waves (very long wavelengths) to X-rays and gamma rays (very tiny wavelengths). EM radiation is classified by wavelength, which is a measure of how energetic is the radiation. The energy of a tiny piece or "packet" of radiation (which we call a photon) is inversely proportional to its wavelength.

The human eye can detect wavelengths in the region of the spectrum from about 400 nm (nanometers or billionths of a meter) to about 700 nm. Not surprisingly, this is called the visible region of the spectrum. All the colors of light (red, orange, yellow, green, blue, and violet) fall inside a small wavelength band. Whereas radio waves have wavelengths on the order of meters, visible light waves have wavelengths on the order of billionths of a meter. Such a tiny unit is called a nanometer ( $1 \text{ nm} = 10^{-9} \text{ m}$ ). At one end of the visible "color" spectrum is red light. Red light has a wavelength of about 630 nm. Near the opposite end of the color spectrum is blue light, and at the very opposite end is violet light. Blue light has a wavelength of about 430 nm. Violet light has a wavelength of about 410 nm. Therefore, blue light is more energetic than red light because of its shorter wavelength, but it is less energetic than violet light, which has an even shorter wavelength. Radiation with wavelengths shorter than those of violet light is called ultraviolet radiation.

The Sun produces radiation that is mainly in the visible part of the electromagnetic spectrum. However, the Sun also generates radiation in ultraviolet (UV) part of the spectrum. UV wavelengths range from 1 to 400 nm. We are concerned about ultraviolet radiation because these rays are energetic enough to break the bonds of DNA molecules (the molecular carriers of our genetic coding), and thereby damage cells. While most plants and animals are able to either repair or destroy damaged cells, on occasion, these damaged DNA molecules are not repaired, and can replicate, leading to dangerous forms of skin cancer (basal, squamous, and melanoma).

Solar flux refers to the amount of solar energy in watts falling perpendicularly on a surface one square centimeter, and the units are watts per  $\text{cm}^2$  per nm. Because of the strong absorption of UV radiation by ozone in the stratosphere, the intensity decreases at lower altitudes in the atmosphere. In addition, while the energy of an individual photon is greater if it has a shorter wavelength, there are fewer photons at the shorter wavelengths, so the Sun's total energy output is less at the shorter wavelengths. Because of ozone, it is virtually impossible for solar ultraviolet to penetrate to Earth's surface. For radiation with a wavelength of 290 nm, the intensity at Earth's surface is 350 million times weaker than at the top of the atmosphere. If our eyes detected light at less than 290 nm instead of in the visible range, the world would be very dark because of the ozone absorption!

To appreciate how important this ultraviolet radiation screening is, we can consider a characteristic of radiation damage called an action spectrum. An action spectrum gives us a measure of the relative effectiveness of radiation in generating a certain biological response over a range of wavelengths. This response might be erythema (sunburn), changes in plant growth, or changes in molecular DNA. Fortunately, where DNA is easily damaged (where

there is a high probability), ozone strongly absorbs UV. At the longer wavelengths where ozone absorbs weakly, DNA damage is less likely. If there was a 10% decrease in ozone, the amount of DNA damaging UV would increase by about 22%. Considering that DNA damage can lead to maladies like skin cancer, it is clear that this absorption of the Sun's ultraviolet radiation by ozone is critical for our well-being.

While most of the ultraviolet radiation is absorbed by ozone, some does make it to Earth's surface. Typically, we classify ultraviolet radiation into three parts, UV-a (320-400 nm), UV-b (280-320 nm), and UV-c (200-280 nm). Sunscreens have been developed by commercial manufacturers to protect human skin from UV radiation. The labels of these sunscreens usually note that they screen both UV-a and UV-b. Why not also screen for UV-c radiation? When UV-c encounters ozone in the mid-stratosphere, it is quickly absorbed so that none reaches Earth's surface. UV-b is partially absorbed and UV-a is barely absorbed by ozone. Ozone is so effective at absorbing the extremely harmful UV-c that sunscreen manufacturers don't need to worry about UV-c. Manufacturers only need to eliminate skin absorption of damaging UV-b and less damaging UV-a radiation.

The screening of ultraviolet radiation by ozone depends on other factors, such as time of day and season. The angle of the Sun in the sky has a large effect on the UV radiation. When the Sun is directly overhead, the UV radiation comes straight down through our atmosphere and is only absorbed by overhead ozone. When the Sun is just slightly above the horizon at dawn and dusk, the UV radiation must pass through the atmosphere at an angle. Because the UV passes through a longer distance in the atmosphere, it encounters more ozone molecules and there is greater absorption and, consequently, less UV radiation striking the surface.

[adapted from the Stratospheric Ozone Electronic Textbook, produced by NASA's Goddard Space Flight Center Atmospheric Chemistry and Dynamics Branch.

[http://www.ccpo.odu.edu/SEES/ozone/oz\\_class.htm](http://www.ccpo.odu.edu/SEES/ozone/oz_class.htm)]

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### **How blood flows**

We all know that blood flows through our body in continuous motion, and that our heart is the pump that drives this motion. But the circulatory system is best understood not as a single system but in terms of its three constituent parts — pulmonary circulation (involving the lungs), coronary circulation (involving the heart), and systemic circulation (involving the blood vessels).

Pulmonary circulation is the movement of blood from the heart to the lungs, and back to the heart again.

The heart has four chambers — the upper chambers are called atriums; the lower chambers are called ventricles. Blood, with all the waste products it has collected in its journey through the body (most particularly carbon dioxide), enters the heart through the right atrium, via two large veins — the inferior vena cava and the superior vena cava. The inferior vena cava carries the blood from the lower half of the body; the superior from the upper half.

When the right atrium is filled with blood, it contracts, pushing the blood into the right ventricle, which then likewise contracts, pushing the blood into the pulmonary artery. The pulmonary artery carries the blood to the lungs, where carbon dioxide and oxygen are exchanged. The blood, now cleaned of its waste and rich in oxygen (because the oxygen drawn into the lungs through breathing binds with blood), is then carried by the pulmonary veins back to the heart — to the left atrium this time. From whence, in the same process as before, it passes through to the left ventricle, and then leaves the heart through the main artery — the biggest artery in the body — the aorta. From there, it begins to circulate throughout the body.

Coronary circulation refers to the movement of blood *within* the heart. Heart tissue needs nourishment, and this nourishment comes through capillaries in the heart.

So the heart and lungs have their own systems; systemic circulation is the part of the circulatory system that supplies nourishment to the tissues throughout the rest of the body. It does this, of course, through the blood vessels — arteries, veins, and capillaries. Arteries carry blood *away* from the heart. Veins carry blood *to* the heart. Capillaries connect the arteries to veins.

Because the heart is a pump, blood comes out in spurts, causing the outflow to vary in volume and speed. This means that blood flow can occur at a high pressure, which is why the arteries need to have thick walls, and why they need to be able to expand and contract to accommodate the changes in pressure.

Veins, on the other hand, carry blood to the heart in a continuous, even flow, and are therefore thinner than arteries, and less elastic.

Systemic circulation begins with the aorta. The aorta branches into many smaller arteries (the smallest are called arterioles), which carry the fresh, oxygenated blood through the body. Finally, the blood reaches the capillaries, where the oxygen and nutrients carried by the blood are released. The de-oxygenated blood now enters the veins, to travel back to the heart and begin its journey once more.

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## Introducing brain cells

The brain contains two types of nerve cell: **neurons** and **glia**. There are, roughly, some 100 billion neurons in the human brain. There are ten times as many glia. Yet it is neurons we talk about all the time. Of course, it is neurons that are important! But glia are the “glue” that hold the neurons together, and the latest research suggests that glia are more important than we have thought.

But neurons, although only 10% of our brain cells, do perform most of the information processing, which is why they have always been the principal focus of research.

Neurons, like glia, are a broad class of cells. There are several different types of neuron, but they all have certain attributes in common. They all have a **cell body / soma**, containing the **cell nucleus**. They all have thin tubes radiating from the cell body. These are called **neurites**, and come in two flavors: **axons** and **dendrites**. There is usually only one axon, and it is of uniform thickness, and very long by comparison with dendrites (axons can be over a meter in length). A neuron contains many dendrites, which are very short (rarely more than 2mm) and usually taper to a fine point.

It is the axon that carries the output of the neuron. It is the dendrites, which come in contact with many axons, that receive the incoming signals.

The soma, the cell body, is roughly spherical and contains the same organelles contained in any animal cell. The most important are the nucleus, the rough endoplasmic reticulum, the smooth endoplasmic reticulum, the Golgi apparatus, and the mitochondria.

The nucleus holds the chromosomes, which contain the DNA.

Endoplasmic reticulum are, very basically, folded membranes. Rough endoplasmic reticulum exist in all cells, but are particularly abundant in neurons. Rough ER contains **ribosomes**, tiny balls vitally involved in protein synthesis. Rough ER contrasts with smooth ER, which is just the same except that the membranes don't contain ribosomes. The function of smooth ER depends on its location within the cell. Most smooth ER plays no role in protein synthesis.

Yet another type of folded membrane is the Golgi apparatus, where processing of the proteins, after their assembly, takes place. It is thought that, among other functions, the Golgi apparatus is involved in sorting proteins for delivery to different parts of the neuron.

The last vital structure within the neuron is the mitochondrion. The function of this type of cell is to supply the energy the cell needs to function.

The shape of a neuron is governed by its **cytoskeleton**. The cytoskeleton consists of three types of element: **microtubules**, **microfilaments**, and **neurofilaments**.

Of these, by far the biggest are the microtubules, which may be thought of as hollow tubes that run through neurites. Neurofilaments are between microtubules and microfilaments in size. Similar filaments are found in cells other than neurons — one such is keratin, which, bundled together, makes hair. Unlike microtubules and microfilaments, both of which are made up of polymers, neurofilaments are made from single long protein molecules. This makes them very strong, and also very stable. However, neurofilaments can cause problems — the neurofibrillary tangles characteristic of Alzheimer's disease are neurofilaments gone wild.

Microfilaments are only about as thick as the cell membrane. Although they're found throughout the neuron, they're particularly abundant in the neurites.

These structures — the soma, the organelles within, the membrane, the cytoskeleton — exist in all cells, but now we come to a part of the neuron that is unique to neurons: the axon. As was mentioned, the axon is the means by which the neuron can send its message on — it's the output mechanism. The important thing to remember is that neurons, unlike other cells in the body, aren't in physical contact with each other. To communicate with each other they need something to leap the gap between them.

The synapse is the point of contact between the neurons, and information flows from across the gap between neurons in a process called **synaptic transmission**, which involves the release of chemicals called **neurotransmitters**.

The other type of neurite is the dendrites, which derive their name from the Greek for tree. This is because the dendrites, as they branch out from the soma in profusion, resemble the branches of a tree.

Neurons can be categorized in various ways. They can be classified by the number of neurites they have. A neuron with one neurite is **unipolar**; one with two is **bipolar**; one with more is **multipolar**. Most neurons are multipolar.

They can also be classified according to the shape and size of the dendritic tree, (e.g., pyramidal cells, stellate cells), or according to whether their dendrites have spines (**spiny** vs **aspinous**). They can also be classified according to the connections they make: **primary sensory neurons** connect with sensory surfaces; **motor neurons** connect with muscles; **interneurons** connect with other neurons. Most neurons are interneurons.

Neurons can also be classified according to axon length: **projection neurons** (or **Golgi Type I**) have long axons that extend from one part of the brain to another; **local circuit neurons** (or **Golgi Type II**) have short axons.

And finally, they can be classified according to chemistry, that is, on the particular neurotransmitters they release.

But most of the brain is taken up by glia, the support cells. Most glia are a type called **astrocytes**. It's the astrocytes that fill the spaces between neurons. **Oligodendroglia** provide the insulation for axons in the brain and spinal cord. **Schwann cells** are similar to the oligodendroglia, fulfilling the same function outside the brain and spinal cord, in the peripheral nervous system.