PHYSIOLOGICAL PSYCHOLOGY

Complementary Course

of

B. SC COUNSELLING PSYCHOLOGY

IV semester

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STUDY MATERIAL

PHYSIOLOGICAL PSYCHOLOGY

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IV semester

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Module 1
PHYSIOLOGICAL BASIS OF MOTIVATION
EATING

Module 2
Physiological Basis of Emotion

Module 3
Learning, Memory and Localization of Higher Order Function
EATING

Eating is a complex event that involves psychological, sociological, economic, environmental, cultural, and internal physiologic components. All of these influences are expressed in the final food that is chosen, handled, and eaten by the individual. It is ingesting food to provide for all humans and animals nutritional needs, particularly for energy and growth. All creatures must eat in order to survive: carnivores eat other animals, herbivores eat plants, and omnivores consume a mixture of both. Eating is an activity of daily living.

"Eating is the action of taking solid foods in the mouth in order to nourish oneself: this action is carried out by insertion [of the foodstuff] in the mouth, followed by mastication, swallowing, and digestion." This is the definition of "eating" proposed by Diderot.

Eating can be divided into the following processes: eating proper, or ingestion, whereby food enters into the body; and digestion, the process through which nutrients from food are extracted in the gastrointestinal tract. Digestion is followed by absorption, the process through which nutrients are passed through into the blood stream; and by excretion, through which indigestible and unabsorbable products from food are eliminated.

The ability to eat and digest food hinges on an intricate, complex, and coordinated system known as the digestive system, all under control both of the central nervous system (brain and spinal cord) and of digestive system’s own intrinsic nervous system, which is sometimes called the body’s "second brain." The digestive system comprises two main groups of organs: the organs of the alimentary canal, also known as the gastrointestinal (GI) tract, and the accessory digestive organs.

The GI tract is a continuous tube that runs from the mouth to the anus. The organs of the gastrointestinal tract include the mouth, pharynx, esophagus, stomach, small intestine (consisting of duodenum, jejunum, and ileum), and large intestine. It is within the GI tract that food is chewed or masticated, then broken down into still smaller fragments, and absorbed into the blood.

The accessory organs of the digestive system are the teeth, tongue, salivary glands, liver, gallbladder, and pancreas. The teeth and tongue allow for chewing, tasting, and rasping of food. The other accessory organs of the digestive system produce secretions that aid in digestion. In embryonic life, these organs develop as outpouchings from the primitive GI tract, and their secretions travel into the GI tract via ducts.

In order to understand eating and digestion, it is important to imagine what the body needs to do when you think about food, eat food, swallow food, when food lands in your stomach, and when food makes it way through the small and large intestines. The digestive system is designed to prepare the body for eating and digestion before the first piece of food passes our lips. Once food is ingested, this system is designed to efficiently extract and absorb nutrients while it rids the body of waste products.

Preparation for Eating

In order to understand how the body prepares for eating, it is important to realize that eating and digestion require that our body maximizes blood flow to the digestive organs, in order to both supply oxygen and energy to these organs, and to carry away the nutrients that have been absorbed.
Blood flow to the digestive system is controlled primarily by the autonomic nervous system (ANS). The ANS has two anatomically and functionally different subdivisions, the sympathetic nervous system and the parasympathetic nervous system. The sympathetic nervous system is designed to stimulate the body to prepare for and engage in activities and behaviors that are highly arousing, for example, "fight or flight reactions," while the parasympathetic nervous system is designed to prepare the body to engage in activities and behaviors that are relaxing.

Eating and digestion require the body to be relaxed, to allow for blood to be shunted away from the muscles to the digestive system. In fact, from an evolutionary point of view, the process of eating requires us to stand still or (preferably) to sit or lie down, and concentrate on taking apart the food item and ingesting it, rather than running around. Thus the processes involved in eating are antithetical to moving about, either to get somewhere or to escape danger. As a consequence of this organization, the body cannot appropriately engage in relaxing behaviors if the sympathetic nervous system is activated, and it cannot engage in arousing behaviors if the parasympathetic nervous system is activated. In other words, if you feel stressed, or you are engaged in physical activity, or you must flee from danger, you will not be able to eat and digest food, and vice versa.

Both mental stress and aerobic exercise involve activation of the sympathetic nervous system. You may have noticed that if you try to eat while you have been highly stressed, or while you are "on the go," or after you have exercised aerobically, your mouth may have been dry, making it difficult to moisten, taste, and swallow food. You may have also experienced stomach cramping and pain upon ingesting food. These responses occur because your sympathetic nervous system is stimulated. Your body is worried about maximizing its ability to fight or run; it is not ready to eat a meal.

If, however, you are in a relaxed state, the thought of food, the sight of food, or simply making a mental association with food, sets into motion a series of events that prepares the GI tract for incoming food. Upon sensing that eating is imminent, the parasympathetic nervous system prepares the GI tract via signals sent through three cranial nerves that exit from the brainstem: the vagus nerve (cranial nerve X), the trigeminal nerve (cranial nerve V), and the glossopharyngeal nerve (cranial nerve IX).

Hunger and satiety are sensations. Hunger represents the physiological need to eat food. Satiety is the absence of hunger; it is the sensation of feeling full.

Appetite is another sensation experienced with eating; it is the desire to eat food. There are several theories about how the feeling of hunger arises. A healthy, well-nourished individual can survive for weeks without food intake, with claims ranging from three to ten weeks. The sensation of hunger typically manifests after only a few hours without eating and is generally considered to be unpleasant.

Hunger is also the most commonly used term to describe the condition of people who suffer from a chronic lack of sufficient food and constantly or frequently experience the sensation of hunger.

Biological mechanisms

The fluctuation of leptin and ghrelin hormone levels results in the motivation of an organism to consume food. When an organism eats, adipocytes trigger the release of leptin into the body. Increasing levels of leptin result in a reduction of one's motivation to eat. After hours of non-consumption, leptin levels drop significantly. These low levels of leptin cause the release of a secondary hormone, ghrelin, which in turn reinitiates the feeling of hunger.

Some studies have suggested that an increased production of ghrelin may enhance appetite evoked by the sight of food, while an increase in stress may also influence the hormone's production. These findings may help to explain why hunger can prevail even in stressful situations.
achieved when the need for food has been satisfied is called satiety. The satiety center in animals is located in the ventromedial nucleus of the hypothalamus.

**Short-term regulation of hunger and food intake**

Short-term regulation of hunger and food intake involves neural signals from the GI tract, blood levels of nutrients, GI tract hormones, and psychological factors.

**Neural signals from the GI tract**

One method that the brain uses to evaluate the contents of the gut is through vagal nerve fibers that carry signals between the brain and the gastrointestinal tract (GI tract). Studies have shown that through these vagal nerve fibers, the brain can sense a difference between different macronutrients. Stretch receptors work to inhibit appetite upon distention of the GI tract by sending signals along the vagus nerve afferent pathway and inhibiting the hunger center.

**Nutrient signals**

Blood levels of glucose, amino acids, and fatty acids provide a constant flow of information to the brain that may be linked to regulating hunger and energy intake. Nutrient signals that indicate fullness, and therefore inhibit hunger include the following:

- Rising blood glucose levels
- Elevated blood levels of amino acids
- Blood concentrations of fatty acids

**Hormone signals**

The hormones insulin and cholecystokinin (CCK) are released from the GI tract during food absorption and act to suppress feeling of hunger. CCK is key in suppressing hunger because of its role in inhibiting neuropeptide Y. Glucagon and epinephrin levels rise during fasting and stimulate hunger. Gnrelin, a hormone produced by the stomach, is a hunger stimulant.

**Psychological factors**

Psychological states appear to play a role in short-term food intake. Merely repeatedly imagining the consumption of a food, for example, can reduce the subsequent actual consumption of that food by reducing the motivation to consume it. Two psychological processes appear to be involved in regulating short-term food intake: liking and wanting. Liking refers to the palatability or taste of the food, which is reduced by repeated consumption. Wanting is the motivation to consume the food, which is also reduced by repeated consumption of a food and may be due to change in memory-related processes. Wanting can be triggered by a variety of psychological processes. Thoughts of a food may intrude on consciousness and be elaborated on, for instance, as when one sees a commercial or smells a desirable food. Eating one food can induce a craving for its complements, foods that are perceived to add pleasure to the consumption of that food, by priming a goal to consume those foods. Participants who drank a sip of cola, for example, were subsequently willing to pay more for a voucher for a cheeseburger that they could redeem later than controls who did not drink the cola.

**Long-term regulation of hunger and food intake**

**Physiological factors**

Leptin, a hormone secreted exclusively by adipose cells in response to an increase in body fat mass, is an important component in the regulation of long term hunger and food intake. Leptinserves as the brain's indicator of the body's total energy stores. When leptin levels rise in the bloodstream they bind to receptors in ARC. The functions of leptin are to:

- Suppress the release of neuropeptide Y (NPY), which in turn prevents the release of appetite enhancing orexins from the lateral hypothalamus. This decreases appetite and food intake, promoting weight loss.
• Stimulate the expression of cocaine and amphetamine regulated transcript (CART).

Though rising blood levels of leptin do promote weight loss to some extent, its main role is to protect the body against weight loss in times of nutritional deprivation. Other factors also have been shown to effect long-term hunger and food intake regulation including insulin.[7]

**Set-point theories of hunger and eating**

The set-point theories of hunger and eating are a group of theories developed in the 1940s and 1950s that operate under the assumption that hunger is the result of an energy deficit and that eating is a means by which energy resources are returned to their optimal level, or energy set-point. According to this assumption, a person's energy resources are thought to be at or near their set-point soon after eating, and are thought to decline after that. Once the person's energy levels fall below a certain threshold, the sensation of hunger is experienced, which is the body's way of motivating the person to eat again. The set-point assumption is a negative feedback mechanism. Two popular set-point theories include the glucostatic set-point theory and the lipostatic set-point theory.

• The set-point theories of hunger and eating present a number of weaknesses.
• The current epidemic of obesity and other eating disorders undermines these theories.
• The set-point theories of hunger and eating are inconsistent with basic evolutionary pressures related to hunger and eating as they are currently understood.
• Major predictions of the set-point theories of hunger and eating have not been confirmed.
• They fail to recognize other psychological and social influences on hunger and eating.

**Positive-incentive perspective**

The positive-incentive perspective is an umbrella term for a set of theories presented as an alternative to the set-point theories of hunger and eating. The central assertion to the positive-incentive perspective is the idea that humans and other animals are not normally motivated to eat by energy deficits, but are instead motivated to eat by the anticipated pleasure of eating, or the positive-incentive value. According to this perspective, eating is controlled in much the same way as sexual behavior. Humans engage in sexual behavior, not because of an internal deficit, but instead because they have evolved to crave it. Similarly, the evolutionary pressures of unexpected food shortages have shaped humans and all other warm blooded animals to take advantage of food when it is present. It is the presence of good food, or the mere anticipation of it that makes one hungry.

**Premeal hunger**

Prior to consuming a meal, the body's energy reserves are in reasonable homeostatic balance. However, when a meal is consumed, there is a homeostasis-disturbing influx of fuels into the bloodstream. When the usual mealtime approaches, the body takes steps to soften the impact of the homeostasis-disturbing influx of fuels by releasing insulin into the blood, and lowering the blood glucose levels. It is this lowering of blood glucose levels that causes premeal hunger, and not necessarily an energy deficit.

**Behavioral response**

Hunger appears to increase activity and movement in many animals - for example, an experiment on spiders showed increased activity and predation in starved spiders, resulting in larger weight gain. This pattern is seen in many animals, including humans while sleeping. It even occurs in rats with their cerebral cortex or stomachs completely removed. Increased activity on hamster wheels occurred when rats were deprived not only of food, but also water or B vitamins such as thiamine. This response may increase the animal's chance of finding food, though it has also been speculated the
reaction relieves pressure on the home population. There is also a difference between the neurological responses in human males and females in response to hunger and satiety.

**Disorders**

Physiologically, eating is generally triggered by hunger, but there are numerous physical and psychological conditions that can affect appetite and disrupt normal eating patterns. These include depression, food allergies, ingestion of certain chemicals, bulimia, anorexia nervosa, pituitary gland misfunction and other endocrine problems, and numerous other illnesses and eating disorders. A chronic lack of nutritious food can cause various illnesses, and will eventually lead to starvation. When this happens in a locality on a massive scale it is considered a famine. If eating and drinking is not possible, as is often the case when recovering from surgery, alternatives are enteral nutrition and parenteral nutrition.

**FEEDING CENTERS IN THE BRAIN**

Feeding center is a group of cells in the lateral hypothalamus that when stimulated cause a sensation of hunger. The lateral hypothalamus or lateral hypothalamic area is a part of the hypothalamus. It is concerned with hunger. Damage to this area can cause reduced food intake. Stimulating the lateral hypothalamus causes a desire to eat, while stimulating the ventromedial hypothalamus causes a desire to stop eating.

**Function**

The glucostatic explanation is based on the homeostatic theory which indicates that the body has balanced states of equilibrium for each system. When out of balance, the body will be pushed to restore balance. Therefore, when the blood sugar levels drop, the glucostatic receptors in the blood take a message to the lateral hypothalamus, which is the feeding center of the brain. This causes certain neurons in the brain to fire in unison, creating the sensation of hunger. Now the person wants to eat.

When the glucose level increases because the person is eating or has eaten, the glucostatic receptors in the blood then send a message to the Ventro-medial Hypothalamus (the satiety or satisfaction center) and the sensation of fullness occurs. Damage to the lateral hypothalamus may lead to a condition known as Fröhlich's syndrome.

**EATING SIGNALS - METABOLIC SIGNALS**

Eating requires at least two basic decisions: what to eat, which is a decision about food choice, and how much to eat, which is a decision about food intake. This distinction is important because food choice and intake involve different behaviors, different controlling signals and different physiological mechanisms. Feeding behavior is controlled by a variety of signals. ‘Cephalic’ signals, such as the taste, smell, sound and sight of food, control food choice and can influence the amount of food consumed in the short-term. Gastrointestinal signals resulting from changes in distention or the release of gut peptides may play a role in the control of short-term intake within a meal or across several meals. Metabolic signals generated by the supply and utilization of metabolic fuels not only influence food choice, but also how much food is consumed in the short-term. Metabolic signals also determine food intake in the long-term and are important in maintaining energy balance over nutritionally significant intervals.

**Receptor site**
Russek (1963) first proposed the liver as a site where changes in metabolism are detected to control feeding behavior. Although his hypothesis was initially ignored for many years, it is now generally accepted that information about hepatic metabolism is communicated to the brain and contributes to the control of food intake.

Work in our laboratory on the role of the liver in feeding behavior has taken a number of different directions, although perhaps the most compelling evidence stems from comparing the effects on food intake of hepatic portal and jugular vein infusion of nutrients and metabolic inhibitors. In one series of experiments, taking acue from Russek’s studies, we compared the effects of hepatic portal and jugular (i.e. systemic) infusions of glucose on satiety in rats. These studies showed that, under relatively normal feeding conditions, glucose infusions within the physiological range suppressed food intake more effectively when delivered into the hepatic portal vein than when given by a jugular route (see Friedman et al., 1996). In other experiments, we studied the role of the liver in hunger by comparing hepatic portal and jugular infusions of the fructose analogue, 2,5-anhydro-D-mannitol (2,5-AM), which we had shown triggered feeding in rats when given by gastric gavage or an intraperitoneal route. The results showed clearly that portal infusion of 2,5-AM elicited food intake more rapidly and at lower doses than did infusions into the jugular vein (see Tordoff et al., 1991).

Nature of the stimulus
Since studies in our laboratory first demonstrated an inverse relationship between hepatocyte ATP concentration and food intake, we have been focused on testing the role of changes in hepatic energy status as a stimulus for hunger and satiety. Under a variety of conditions, eating behavior triggered by injection of 2-5-AM was associated with the analogue’s effect of reducing liver ATP (e.g. Friedman et al., 2002). The decrease in ATP was due largely to trapping of phosphate in phosphorylated forms of 2-5-AM (Rawson et al., 1994). Most telling therefore was the observation that preventing the decrease in ATP by administration of exogenous phosphate also prevented the eating response (Rawson and Friedman, 1994). Subsequently, we found that eating behavior stimulated by administration of other metabolic inhibitors, including fatty acid oxidation inhibitors, was also associated with lowered hepatic ATP levels (e.g. Ji et al., 2000).

Transduction mechanism
Little is known about how changes in hepatocyte energy metabolism are transduced into a signal the nervous system can interpret. We have begun to investigate this question along two tracks. In one set of studies (Rawson et al., 2003), we studied the effects of 2,5-AM on intracellular Ca2+ concentration in hepatocytes as such changes are well known to be involved in cellular signaling in a variety of tissues. The results showed that 2,5-AM produced an increase in intracellular Ca2+ in ~50% of hepatocytes and that the rise was due to release of intracellular calcium stores. In another set of experiments (Friedman et al., 2003), we tested a hypothesis that changes in hepatocyte ATP levels generate a signal by lowering activity of the sodium pump, causing cellular depolarization (Langhans and Scharrer, 1987). Using nuclear magnetic spectroscopy, we showed that 2,5-AM increased intracellular sodium with a latency consistent with that of the eating response, a finding supporting Langhans and Scharrer’s conjecture. These intriguing results require considerable follow-up before the effects of 2,5-AM seen in vitro can be understood and directly related to the behavioral response seen in vivo.

Transmission of the signal
Theoretically, changes in hepatic energy status could be transmitted to the brain via a neural or humoral route; at present, however, there is evidence only for a neural connection, specifically via vagal afferent neurons. Evidence that vagal sensory fibers carry the metabolic signals from liver that control food intake stem from a variety of studies (see Langhans, 1998; Horn et al., 2001) showing that interruption of vagal afferent transmission can alter ad libitum food intake and prevent the eating response to metabolic inhibitors that act in liver. Other studies using the immunocytohistochemical expression of Fos as a marker for neural activity have demonstrated that metabolic inhibitors that stimulate feeding behavior activate areas in the brain known to receive and process vagal afferent input. Electrophysiological experiments provide the most direct demonstration that metabolic perturbations trigger hepatic vagal sensory neurons. Niijima (this volume) was the first to show that infusion of glucose can decrease activity in the hepatic branch of the vagus. Subsequently, Niijima and his colleagues reported that these fibers respond to a range of nutrients, hormones and other agents. Using techniques that allow for measurement of single unit activity in the hepatic branch of the vagus, we recently found (Horn and Friedman, 2004) afferent responses to infusion of serotonin (5-HT) and cholecystokinin (CCK). By comparing the effects of hepatic portal and jugular infusions of these agents it was possible to identify ‘portal’ and ‘jugular’ responsive units. In keeping with the anatomical observation that fibers in the hepatic branch also innervate the stomach and intestine, we found that cutting the gastroduodenal sub-branch (GDB) of the hepatic vagus eliminated ~75% of the spontaneous activity in the hepatic branch as well as most of the response to 5-HT and CCK. These and other findings indicate that only a small proportion of afferents in the hepatic branch innervate the liver and that afferents of hepatic origin have a different pharmacology than those from the gastrointestinal tract.

HYPOTHALAMIC REGULATIONS

Hypothalamic Regions Important in Appetite Regulation

Arcuate Nucleus

The ARC is a key hypothalamic nucleus in the regulation of appetite. In mice, lesions of the ARC using monosodium glutamate produce obesity and hyperphagia. Anatomically related to the median eminence, the ARC is not fully insulated from the circulation by the blood-brain barrier and, hence, is strategically positioned to integrate a number of peripheral signals controlling food intake. Two major neuronal populations in the ARC are prominently implicated in the regulation of feeding. One population, localized more medially in the ARC, increases food intake and coexpresses neuropeptide Y (NPY) and Agouti-related protein (AgRP). The second population of neurons, coexpressing cocaine- and amphetamine-related transcript (CART) and pro-opiomelanocortin (POMC), inhibits food intake and tends to cluster more laterally in the ARC. Neuronal projections from these two populations then communicate with other hypothalamic areas involved in appetite regulation, such as the PVN, DMN and LHA. This network of neuronal circuitry can be modulated by hyperpheral signals, such as leptin and insulin.

Paraventricular Nucleus

The PVN lies to either side of the roof of the third ventricle and it is thought to play an important role in the control of both appetite and endocrine function. The PVN is particularly important in the detection and integration of NPY, AgRP and melanocortin signals. Microinjection of almost all known orexigenic peptides into the PVN, including NPY and AgRP, stimulate feeding. NPY/AgRP and POMC neurons from the ARC communicate with PVN neurons containing corticotrophin-releasing hormone (CRH) and thyrotrophin-releasing hormone (TRH). Both CRH and...
TRH have been implicated in the control of energy balance, by contributions to both food intake and energy expenditure. Therefore, in energy balance, a key role for the PVN is to convey information from the ARC to other brain areas involved in appetite regulation.

**Lateral Hypothalamic Area**

The LHA is another key downstream target of neuronal projections from the ARC and contains the orexigenic neuropeptides melanin-concentrating hormone (MCH) and orexins. NPY, AgRP, and α-MSH immunoreactive terminals are extensive in the LHA and are in contact with MCH and orexin-expressing cells. MCH immunoreactive fibers project to the cortex and spinal cord, consistent with a potential role in appetite control and energy expenditure. Interestingly, work by another group found that a subpopulation of MCH neurons express CART and mainly project to the brainstem. By contrast, MCH fibers lacking CART have been found to project to the forebrain, suggesting MCH may modulate food intake and energy expenditure through two separate neuronal projections depending on the presence of CART. Lesioning of the LHA reduces bodyweight. The severity of the LHA syndrome and near-normal recovery of food intake and bodyweight depend on the location and size of the lesion. These observations led to the conception that the LHA was a 'feeding center' under restraint by signals from the VMN.

**Dorsomedial Nucleus**

Destruction of the DMN results in hyperphagia and obesity, though less dramatically than VMN lesioning. The DMN contains a high level of NPY terminals and α-MSH terminals originating in the ARC. α-MSH fibers also project from the DMN to the PVN, terminating on TRH-containing neurons. In the DMN, α-MSH fibers are in close apposition to NPY neurons. α-MSH may suppress NPY gene expression in the DMN indirectly via separate inhibitory interneurons, possibly through GABAergic pathways. It is proposed that decreased POMC input from the ARC to the DMN causes a reduction in MC4-R signaling, leading to decreased GABAergic inhibition of DMN NPY neurons and, hence, increased NPY mRNA expression. In diet-induced obesity, obese Agouti mice and MC4-R-knockout mice, NPY mRNA expression is increased in the DMN, whereas it is reduced in the ARC. This difference in NPY response is again highlighted by the finding that NPY levels in the DMN, in contrast to the ARC and PVN, are not elevated during fasting. It is thought that lack of leptin signaling on NPY neurons in the DMN may partly account for this since leptin-deficient ob/ob mice show increased NPY mRNA in the ARC but not in the DMN.

**Ventromedial Nucleus**

Lesions of the VMN result in rapid-onset hyperphagia and obesity, leading to the hypothesis that the VMN is a satiety center, acting as a restraint on feeding. Consistent with this, neuroimaging studies in humans have shown increased signaling in the area of the VMN following an oral glucose load. The VMN has a large population of glucose-responsive neurons that respond to blood glucose levels and numerous histamine, dopamine, serotonin, and GABA neurons that respond to feeding-related stimuli. The VMN receives NPY, AgRP, and POMC neuronal projections from the ARC. Brain-derived neurotrophic factor (BDNF) is highly expressed in the VMN and is important during development for neuronal survival. It is a member of the neurotrophin family, which binds to the TrkB receptor, a human mutation of which has been described, resulting in severe obesity. Lateral ventricle administration of BDNF reduces food intake and bodyweight. Recent work implicates the role of ARC POMC neurons in activating VMN BDNF neurons to decrease food intake. The VMN has also recently been described as the site of a novel hypothalamic appetite-regulatory circuit involving triiodothyronine (T3).
OBESITY

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health, leading to reduced life expectancy and/or increased health problems. Body mass index (BMI), a measurement which compares weight and height, defines people as overweight (pre-obese) when their BMI is between 25 kg/m² and 30 kg/m², and obese when it is greater than 30 kg/m². Obesity increases the likelihood of various diseases, particularly heart disease, type 2 diabetes, breathing difficulties during sleep, certain types of cancer, and osteoarthritis. Obesity is most commonly caused by a combination of excess caloric intake, lack of physical activity, and genetic susceptibility, although a few cases are caused primarily by genes, endocrine disorders, medications or psychiatric illness. Evidence to support the view that some obese people eat little yet gain weight due to a slow metabolism is limited; on average obese people have a greater energy expenditure than their thin counterparts due to the energy required to maintain an increased body mass.

The primary treatment for obesity is dieting and physical exercise. To supplement this, or in case of failure, anti-obesity drugs may be taken to reduce appetite or inhibit fat absorption. In severe cases, surgery is performed or an intragastric balloon is placed to reduce stomach volume and/or bowel length, leading to earlier satiation and reduced ability to absorb nutrients from food. Obesity is a leading preventable cause of death worldwide, with increasing prevalence in adults and children, and authorities view it as one of the most serious public health problems of the 21st century. Obesity is stigmatized in the modern Western world, though it has been perceived as a symbol of wealth and fertility at other times in history, and still is in many parts of Africa.

Classification

Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health. It is defined by body mass index (BMI) and further evaluated in terms of fat distribution via the waist–hip ratio and total cardiovascular risk factors. BMI is closely related to both percentage body fat and total body fat. Some modifications to the WHO definitions have been made by particular bodies. The surgical literature breaks down "class III" obesity into further categories whose exact values are still disputed.

- Any BMI ≥ 35 or 40 is severe obesity
- A BMI of ≥ 35 or 40–44.9 or 49.9 is morbid obesity
- A BMI of ≥ 45 or 50 is super obese

As Asian populations develop negative health consequences at a lower BMI than Caucasians, some nations have redefined obesity; the Japanese have defined obesity as any BMI greater than 25 while China uses a BMI of greater than 28.

Effects on health

Excessive body weight is associated with various diseases, particularly cardiovascular diseases, diabetes mellitus type 2, obstructive sleep apnea, certain types of cancer, and osteoarthritis. As a result, obesity has been found to reduce life expectancy. Obesity is one of the leading preventable causes of death worldwide. Large-scale American and European studies have found that mortality risk is lowest at a BMI of 22.5–25 kg/m² in non-smokers and at 24–27 kg/m² in current smokers, with risk increasing along with changes in either direction. A BMI above 32 has been associated with a doubled mortality rate among women over a 16-year period. In the United States obesity is estimated to cause an excess 111,909 to 365,000 deaths per year, while 1 million (7.7%) of deaths in the European Union are attributed to excess weight. On average, obesity reduces life expectancy by six to seven years: a BMI
of 30–35 reduces life expectancy by two to four years, while severe obesity (BMI > 40) reduces life expectancy by 10 years.

**Causes of Obesity**

At an individual level, a combination of excessive caloric intake and a lack of physical activity is thought to explain most cases of obesity. A limited number of cases are due primarily to genetics, medical reasons, or psychiatric illness. In contrast, increasing rates of obesity at a societal level are felt to be due to an easily accessible and palatable diet, increased reliance on cars, and mechanized manufacturing.

A 2006 review identified ten other possible contributors to the recent increase of obesity: (1) insufficient sleep, (2) endocrine disruptors (environmental pollutants that interfere with lipid metabolism), (3) decreased variability in ambient temperature, (4) decreased rates of smoking, because smoking suppresses appetite, (5) increased use of medications that can cause weight gain (e.g., atypical antipsychotics), (6) proportional increases in ethnic and age groups that tend to be heavier, (7) pregnancy at a later age (which may cause susceptibility to obesity in children), (8) epigenetic risk factors passed on generationally, (9) natural selection for higher BMI, and (10) assortative mating leading to increased concentration of obesity risk factors (this would not necessarily increase the number of obese people, but would increase the average population weight). While there is substantial evidence supporting the influence of these mechanisms on the increased prevalence of obesity, the evidence is still inconclusive, and the authors state that these are probably less influential than the ones discussed in the previous paragraph.

**Genetics**

Like many other medical conditions, obesity is the result of an interplay between genetic and environmental factors. Polymorphisms in various genes controlling appetite and metabolism predispose to obesity when sufficient calories are present. As of 2006 more than 41 of these sites have been linked to the development of obesity when a favorable environment is present. The percentage of obesity that can be attributed to genetics varies, depending on the population examined, from 6% to 85%. Obesity is a major feature in several syndromes, such as Prader-Willi syndrome, Bardet-Biedl syndrome, Cohen syndrome, and MOMO syndrome. (The term "nonsyndromic obesity" is sometimes used to exclude these conditions.) In people with early-onset severe obesity (defined by an onset before 10 years of age and body mass index over three standard deviations above normal), 7% harbor a single point DNA mutation.

**Management**

The main treatment for obesity consists of dieting and physical exercise. Diet programs may produce weight loss over the short term, but keeping this weight off can be a problem and often requires making exercise and a lower calorie diet a permanent part of a person's lifestyle. Success rates of long-term weight loss maintenance are low and range from 2–20%. In a more structured setting, however, 67% of people who lost greater than 10% of their body mass maintained or continued to lose weight one year later. An average maintained weight loss of more than 3 kg (6.6 lb) or 3% of total body mass could be sustained for five years. Some studies have found significant benefits in mortality in certain populations with weight loss. In a prospective study of obese women with weight related diseases, intentional weight loss of any amount was associated with a 20% reduction in mortality. In obese women without obesity related illnesses a weight loss of greater than 9 kg (20 lb) was associated with a 25% reduction in mortality. A recent review concluded that certain subgroups such as those with
type 2 diabetes and women show long term benefits in all cause mortality, while outcomes for men do not seem to be improved with weight loss. A subsequent study has found benefits in mortality from intentional weight loss in those who have severe obesity.

The most effective treatment for obesity is bariatric surgery; however, due to its cost and the risk of complications, researchers are searching for other effective yet less invasive treatments.

**Dieting**

Diet to promote weight loss are generally divided into four categories: low-fat, low-carbohydrate, low-calorie, and very low calorie. A meta-analysis of six randomized controlled trials found no difference between three of the main diet types (low calorie, low carbohydrate, and low fat), with a 2–4 kilogram (4.4–8.8 lb) weight loss in all studies. At two years these three methods resulted in similar weight loss irrespective of the macronutrients emphasized.

Very low calorie diets provide 200–800 kcal/day, maintaining protein intake but limiting calories from both fat and carbohydrates. They subject the body to starvation and produce an average weekly weight loss of 1.5–2.5 kilograms (3.3–5.5 lb). These diets are not recommended for general use as they are associated with adverse side effects such as loss of lean muscle mass, increased risks of gout, and electrolyte imbalances. People attempting these diets must be monitored closely by a physician to prevent complications.

**Exercise**

With use, muscles consume energy derived from both fat and glycogen. Due to the large size of leg muscles, walking, running, and cycling are the most effective means of exercise to reduce body fat. Exercise affects macronutrient balance. During moderate exercise, equivalent to a brisk walk, there is a shift to greater use of fat as fuel.

**Weight loss programs**

Weight loss programs often promote lifestyle changes and diet modification. This may involve eating smaller meals, cutting down on certain types of food, and making a conscious effort to exercise more. These programs also enable people to connect with a group of others who are attempting to lose weight, in the hopes that participants will form mutually motivating and encouraging relationships.

**Medication**

The two most commonly used medications to treat obesity: orlistat (Xenical) and sibutramine (Meridia)

**BASES OF SPECIFIC HUNGER**

A type of hunger that is satisfied by specific dietary requirements, such as vitamins and minerals. Many animals vary their food intake according to the nutritive value of the products of digestion. A variety of mechanisms are involved in this type of regulation. The simplest mechanism is the direct detection of the substance in the food, as is the case with sodium. Animals can detect sodium in the diet in two main ways. First, sodium salt (NaCl) is a primary aspect of taste in most vertebrates. Secondly, sodium has profound effects upon the body fluids, and its presence there can be directly detected. Sodium appetite appears to be innate, but many animals are adept at learning and remembering the location of sources of sodium. There are many vitamins and minerals that animals are not able to detect, either by taste or by their levels in the blood. Nevertheless, deficient animals develop...
strong preferences for foods containing the missing substances. Rats (Rattusnorvegicus) deficient in thiamine show an immediate marked preference for a novel food, even when that food is thiamine deficient. The preference is short lived. If consumption of a novel food is followed by recovery from the dietary deficiency, however, then the rat rapidly learns to prefer the novel food. Such rapid learning on the basis of the physiological consequences of ingestion enables the rat to exploit new sources of food, and to find out which contains the required ingredients.

The effects of a vitamin-deficient diet have much in common with poison avoidance. Vitamin-deficient rats are reluctant to eat familiar food, and show a more than normal interest in novel foods. The aversion to previously familiar food persists even after the animals have recovered from the deficiency. Rats that become sick after eating poisoned food also show an aversion to familiar food and an interest in novel foods.

THIRST

Thirst is often viewed by physiologists and physicians as a central nervous system mechanism that regulates the body's water and minerals. The significance of the thirst drive is emphasized by three facts: 50 to 70 percent of adult body weight is water, the average adult ingests and loses 2.5 liters of water each day, and body weight is regulated within 0.2 percent from one day to the next. Clearly, water is essential to life and the body responds in a manner that ensures survival.

Thirst is the craving for fluids, resulting in the basic instinct of animals to drink. It is an essential mechanism involved in fluid balance. It arises from a lack of fluid sand/or an increase in the concentration of certain osmolites, such as salt. If the water volume of the body falls below a certain threshold or the osmolite concentration becomes too high, the brain signals thirst. Continuous dehydration can cause many problems, but is most often associated with neurological problems such as seizures and renal problems. Excessive thirst, known as polydipsia, along with excessive urination, known as polyuria, may be an indication of diabetes.

There are receptors and other systems in the body that detect a decreased volume or an increased osmolite concentration. They signal to the central nervous system, where central processing succeeds. Some sources therefore distinguish "extracellular thirst" from "intracellular thirst", where extracellular thirst is thirst generated by decreased volume and intracellular thirst is thirst generated by increased osmolite concentration. Nevertheless, the craving itself is something generated from central processing in the brain, no matter how it is detected.

Detection

There are many different receptors for sensing decreased volume or an increased osmolite concentration.

Decreased volume

- Renin-angiotensin system
  
  Hypovolemia leads to activation of the renin angiotensin system (RAS) and a decrease in atrial natriuretic peptide. These mechanisms, along with their other functions, contribute to elicit thirst, by affecting the subfornical organ. For instance, angiotensin II, activated in RAS, is a powerful dipsogen (i.e., it stimulates thirst) which acts via the subfornical organ.

- Other
  o Arterial baroreceptors sense a decreased arterial pressure, and signals to the central nervous system in the area postrema and nucleus tractus solitarius.
  o Cardiopulmonary receptors sense a decreased blood volume, and signal to the area postrema and nucleus tractus solitarius[2] as well.
Increased osmolite concentration

An increase in osmotic pressure, e.g. after eating a salty meal activates osmoreceptors. There are osmoreceptors already in the central nervous system, more specifically in the hypothalamus, notably in two circumventricular organs that lack an effective blood-brain barrier, the organum vasculosum of the lamina terminalis (OVLT) and the subfornical organ (SFO). However, although located in the same parts of the brain, these osmoreceptors that evoke thirst are distinct from the neighbouring osmoreceptors in the OVLT and SFO that evoke arginine vasopressin release to decrease fluid output. In addition, there are visceral osmoreceptors.

Salt craving

Because sodium is also lost from the plasma in hypovolemia, the body's need for salt proportionately increases in addition to thirst in such cases. This is also a result of the renin-angiotensin system activation.

Elderly

For adults over age 50, the body's thirst sensation diminishes and continues diminishing with age, causing many to suffer symptoms of dehydration.

Central processing

The area postrema and nucleus tractus solitarius signal, by 5-HT, to lateral parabrachial nucleus, which in turn signal to median preoptic nucleus. In addition, the area postrema and nucleus tractus solitarius also signal directly to subfornical organ. Thus, the median preoptic nucleus and subfornical organ receive signals of both decreased volume and increased osmolite concentration. They signal to higher integrative centers, where ultimately the conscious craving arises. However, the true neuroscience of this conscious craving is not fully clear.

OSMOTIC AND HYPOVOLEMIC THIRST

There are mainly 2 kinds of thirsts reported, Osmotic thirst and Hypovolemic thirst.

Osmotic Thirst

It's the thirst resulting from eating salty foods. Eating salty food causes sodium ions to spread through the blood and extracellular fluid of the cell. The higher concentration of solutes outside the cell results in osmotic pressure, drawing water from the cell to the extracellular fluid. Certain neurons detect the loss of water and trigger osmotic thirst to help restore the body to the normal state.

The brain detects osmotic pressure from:

- Receptors around the third ventricle. The OVLT (organum vasculosum laminae terminalis) and the subfornical organ (detect osmotic pressure and salt content). Receptors in the periphery, including the stomach, which detect high levels of sodium. Receptors in the OVLT, subfornical organ, stomach and elsewhere relay information to areas of the hypothalamus including: the supraoptic nucleus and paraventricular nucleus. Both control the rate at which the posterior pituitary releases vasopressin. Receptors also relay information to the lateral preoptic area which controls drinking. When osmotic thirst is triggered, water that you drink has to be absorbed through the digestive system. To inhibit thirst, the body monitors swallowing and detects the water contents of the stomach and intestines.

Hypovolemic thirst

It's the thirst resulting from loss of fluids due to bleeding or sweating. Thirst is thirst associated with low volume of body fluids. Triggered by the release of the hormones vasopressin and angiotensin II, which constrict blood vessels to compensate for a drop in blood pressure. Angiotensin II stimulates

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neurons in areas adjoining the third ventricle. Neurons in the third ventricle send axons to the hypothalamus where angiotensin II is also released as a neurotransmitter. Animals with osmotic thirst have a preference for pure water. Animals with hypovolemic thirst have a preference for slightly salty water as pure water dilutes body fluids and changes osmotic pressure.

REGULATING DRINKING BEHAVIOUR

Thirst is a conscious sensation that results in a desire to drink. Although all normal humans experience thirst, science can offer no precise definition of this phenomenon because it involves numerous physiological responses to a change in internal fluid status, complex patterns of central nervous system function, and psychological motivation. Three factors are typically recognized as components of thirst: a body water deficit, brain integration of central and peripheral nerve messages relating to the need for water, and an urge to drink. In laboratory experiments, thirst is measured empirically with subjective perceptual scales (for example, ranging from "not thirsty at all" to "very, very thirsty") and drinking behavior is quantified by observing the timing and volume of fluid consumed.

Psychologists classify thirst as a drive, a basic compelling urge that motivates action. Other human drives involve a lack of nutrients (for example, glucose, sodium), oxygen, or sleep; these are satiated by eating, breathing, and sleeping. Clark Hull published a major, relevant theory describing the nature of human drives in 1943. He observed that learned habits, in addition to the thirst drive, influence drinking strongly. If a behavior reduces thirst, that behavior is reinforced and learned as a habit. Irrelevant behaviors (for example, sneezing, grooming) provide no reinforcement, have no effect on drinking, and do not become habits. Further, Hull realized that external incentives, such as the qualities or quantity of a fluid, also influence fluid consumption. On a hot summer day, for example, a cold beverage is more attractive than a cup of hot tea. Yet when chilled to a very low temperature, a cold beverage becomes an aversive stimulus to drinking behavior. Physiologists have popularized the term alliesthesia (from Greek root words referring to altered sensation) to describe the fact that the sensation of thirst may have either pleasant or unpleasant qualities, depending on the intensity of the stimulus and the state of the person.

Numerous investigations have verified that thirst and drinking behavior are complex entities. For example, drinking behavior (that is, the timing and the amount of fluid consumed) is not linearly related to the intensity of perceived thirst. Nor should we infer that individuals experience thirst simply because they drink. These facts indicate that thirst and drinking behavior are distinct entities that influence each other and are influenced by numerous internal and external factors.

Physiological Components of Thirst

In 1954, Edward Adolph and colleagues proposed a multiple-factor theory of thirst that has not been refuted to date. This theory states that no single mechanism can account for all drinking behavior and that multiple mechanisms, sometimes with identical functions, act concurrently. Because water is essential to life, the existence of redundant mechanisms has great survival value. Among these, thirst appears to be regulated primarily by evaluation of changes in the concentration of extracellular fluid, measured as the osmolality of blood plasma. (Osmolality is a measurement that describes the concentration of all dissolved solids in a solution, that is, dissolved substances per unit of solvent. In research and clinical laboratories, the unit for osmolality of blood is mOsm/kg or milliosmoles per kilogram of water.) Below a certain threshold level of plasma osmolality, thirst is absent. Above this threshold, a strong desire to drink appears in response to an increase of 2 to 3 percent in the level of dissolved substances in blood. The brain's thirst center lies deep within the brain, in an area known as
the hypothalamus. This anatomical site contains cells that respond to changes in the concentration of body fluids. When the thirst center is stimulated by an increased concentration of blood (that is, dehydration), thirst and fluid consumption increase. As the brain senses the concentration of blood, it allows a minor loss of body water before stimulating the drive to drink. This phenomenon has been named voluntary dehydration. Specifically, several research studies since the 1930s have observed that adults and children replace only 34 to 87 percent of the water lost as sweat, by drinking during exercise or labor in hot environments. The resulting dehydration is due to the fact that thirst is not perceived until a 1 to 2 percent body weight loss occurs. Inter-individual differences, resulting in great voluntary dehydration in some individuals, have caused them to be named reluctant drinkers.

Reduced extracellular fluid volume, including blood volume, also increases thirst. Experiments (for example, reducing blood volume without altering blood concentration) have demonstrated that volume-sensitive receptors in the heart and blood vessels likely regulate drinking behavior by increasing the secretion of hormones. This effect is relatively minor, however. Animal research suggests that a change in extracellular fluid concentration accounts for most (for example, 70 percent) of the increased fluid consumption that follows moderate whole-body dehydration, whereas a decrease of fluid volume per se plays a secondary role. Thus, thirst is extinguished when body fluid concentration decreases and fluid volume increases. Osmolality-sensitive nerves in the mouth, throat, and stomach also play a role in abating thirst. As fluid passes through the mouth and upper gastrointestinal tract, the sense of dryness decreases. When this fluid fills the stomach, stretch receptors sense an increase in gastric fullness and the thirst drive diminishes.

As dehydration causes the body's extracellular fluid to become more concentrated, the fluid inside cells moves outward, resulting in intracellular dehydration and cell shrinkage, and the hormone arginine vasopressin (AVP, also known as the antidiuretic hormone) is released from the brain. AVP serves two purposes: to reduce urine output at the kidneys and to enhance thirst; both serve to restore normal fluid balance. Other hormones influence fluid-mineral balance directly and indirectly. Renin, angiotensin II, and aldosterone are noteworthy examples. As dehydration reduces circulating blood volume, blood pressure decreases and renin is secreted from blood vessels inside the kidneys. Renin activates the hormone angiotensin II, which subsequently stimulates the release of aldosterone from the adrenal glands. Both angiotensin II and aldosterone increase blood pressure and enhance the retention of sodium and water; these effects indirectly reduce the intensity of thirst. Angiotensin II also affects thirst directly. When injected into sensitive areas of the brain, it causes a rapid increase in fluid consumption that is followed by a slower increase in sodium chloride consumption and water retention by the kidneys.

**Host Factors**

Repeated training sessions in cool or hot environments alter fluid consumption in four ways. First, physical training increases the secretion of the hormone AVP, which stimulates drinking and body water retention. Second, exercise-heat acclimation (that is, adaptations due to exercise in a hot environment over eight days) increases the volume of fluid consumed and the number of times that adults drink during exercise. Third, frequent rest periods, in the midst of labor or exercise, will increase fluid replacement time and enhance fluid consumption. Humans tend to drink less when they are preoccupied or are performing physical or mental tasks. Fourth, learned behaviors can enhance fluid consumption when thirst is absent. This phenomenon is widely appreciated among military personnel and athletes who are trained to consume water at regular intervals, whether they are thirsty or not.

Several research groups have reported that chronological age influences thirst and drinking behavior. Elderly men experience a blunted thirst drive and reduced fluid intake, perhaps due to their

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brains' reduced ability to sense changes in plasmaosmolality or blood volume. Further, elderly individuals experience a decrease in the ability of their kidneys to conserve water. This suggests that the elderly are predisposed to dehydration when illness increases water loss (that is, vomiting, diarrhea) or when physical incapacity prevents access to water.

**Fluid and Environmental Characteristics**

Many fluid characteristics stimulate or enhance drinking, during or after exposure to a hot environment. Fluid temperature (consumption is greatest at 14 to 16°C, reduced above 37°C), turbidity, sweetness, fruit flavorings (for example, cherry, grape, orange, lemon), addition of citric acid which imparts a citrus flavor, and addition of sodium chloride or other minerals are examples. These components enhance palatability and increase fluid consumption. The addition of a small amount of salt (sodium chloride), besides enhancing palatability, may result in thirst and increased drinking, due to the specific action of sodium on fluid movements. An increased sodium concentration outside of cells causes water to leave cells via osmosis. The resulting cellular dehydration is an important stimulus for drinking. Increased beverage carbonation tends to reduce the palatability of a fluid as well as the volume of fluid consumed, without an increase in thirst. In addition, intakes of food and water are closely related. During 24-hour observations of fluid intake, most studies report that the majority of fluid (69 to 78 percent) is consumed during meals. The foregoing characteristics, therefore, tend to reduce the magnitude of voluntary dehydration. Conversely, fluid characteristics may influence drinking behavior negatively, regardless of the intensity of thirst. Experiments conducted during mild prolonged exercise have shown that the following qualities are perceived as undesirable: nausea, bloating, an objectionable feeling in the mouth, excessive viscosity, and excessive sweetness (see Passe, 1996).

Exercise and high ambient temperature may independently alter an individual's perception of fluid palatability. For example, drinking behavior increases when air temperature exceeds 25°C. Fluid consumption can also be enhanced by changing the shape of a fluid container, proximity of fluid containers to the drinker, volume of fluid that is available, and time allowed for drinking. Societal customs may influence fluid consumption, as evidenced by cross-cultural differences in beverage preferences. Even rituals, such as accepting the friendly offer of a beverage in a social setting, may enhance fluid intake beyond that driven by physiological cues. These factors usually involve learned habits. Similarly, when people repeatedly drink fluids with initially unfamiliar flavors, the palatability of the fluids is enhanced.

Although a comprehensive theory of thirst and fluid balance eludes description, it is likely that the thirst drive increases and diminishes because multiple factors (for example, oral dryness, gastric distension, osmolality, volume, fluid qualities) are re-integrated concurrently by the brain's thirst center.

**Factors That Alter Thirst**

**Increase Thirst**
- increased concentration of blood
- decreased blood volume
- decreased blood pressure
- mouth and throat dryness
- increased angiotensin II

**Decrease Thirst**
- decreased concentration of blood
• increased blood volume
• increased blood pressure
• increased stomach fullness
• decreased angiotensin II

SLEEP

Sleep is associated with a state of muscle relaxation and reduced perception of environmental stimuli. Sleep is a naturally recurring state of mind characterized by altered consciousness, relatively inhibited sensory activity, inhibition of nearly all voluntary muscles, and reduced interactions with surroundings. It is distinguished from wakefulness by a decreased ability to react to stimuli, but is more easily reversed than the state of hibernation or of being comatose. Mammalian sleep occurs in repeating periods, in which the body alternates between two highly distinct modes known as non-REM and REM sleep. REM stands for "rapid eye movement" but involves many other aspects including virtual paralysis of the body.

The diverse purposes and mechanisms of sleep are the subject of substantial ongoing research. Sleep seems to assist animals with improvements in the body and mind. A well-known feature of sleep in humans is the dream, an experience typically recounted in narrative form, which resembles waking life while in progress, but which usually can later be distinguished as fantasy. Sleep is sometimes confused with unconsciousness, but is quite different in terms of thought process.

STAGES OF SLEEP

In mammals and birds, sleeping is divided into two broad types: rapid eyemovement (REM) and non-rapid eye movement (NREM or non-REM) sleep. Each type has a distinct set of associated physiological, neurological, and psychological features. The American Academy of Sleep Medicine (AASM) further divides NREM into three stages: N1, N2, and N3, the last of which is also called delta sleep or slow wave sleep (SWS).

Sleep proceeds in cycles of REM and NREM, the order normally being N1 → N2 → N3 → N2 → REM. There is a greater amount of deep sleep (stage N3) early in the night, while the proportion of REM sleep increases later in the night and just before natural awakening.

In humans, each sleep cycle lasts from 90 to 110 minutes on average, and each stage may have a distinct physiological function. This can result in sleep that exhibits loss of consciousness but does not fulfill its physiological functions (i.e., one may still feel tired after apparently sufficient sleep).

NREM sleep

According to the 2007 AASM standards, NREM consists of three stages. There is relatively little dreaming in NREM. Stage N1 refers to the transition of the brain from alpha waves having a frequency of 8 to 13 Hz (common in the awake state) to theta waves having a frequency of 4 to 7 Hz. This stage is sometimes referred to as somnolence or drowsy sleep. Sudden twitches and hypnic jerks, also known as positive myoclonus, may be associated with the onset of sleep during N1. Some people may also experience hypnagogic hallucinations during this stage, which can be troublesome to them. During N1, the subject loses some muscle tone and most conscious awareness of the external environment.

Stage N2 is characterized by sleep spindles ranging from 11 to 16 Hz (most commonly 12–14 Hz) and K-complexes. During this stage, muscular activity as measured by EMG decreases, and conscious awareness of the external environment disappears. This stage occupies 45% to 55% of total sleep in adults. Stage N3 (deep or slow-wave sleep) is characterized by the presence of a minimum of
20% delta waves ranging from 0.5 to 2 Hz and having a peak-to-peak amplitude $>75$ μV. (EEG standards define delta waves to be from 0 – 4 Hz, but sleep standards in both the original R&K, as well as the new 2007 AASM guidelines have a range of 0.5 – 2 Hz.) This is the stage in which parasomnias such as night terrors, nocturnal enuresis, sleepwalking, and somniloquy occur. Many illustrations and descriptions still show a stage N3 with 20%-50% delta waves and a stage N4 with greater than 50% delta waves; these have been combined as stage N3.

**REM sleep**
Rapid eye movement sleep, or REM sleep, accounts for 20%-25% of total sleep time in most human adults. The criteria for REM sleep include rapid eye movements as well as a rapid low-voltage EEG. Most memorable dreaming occurs in this stage. At least in mammals, a descending muscular atonia is seen. Such paralysis may be necessary to protect organisms from self-damage through physically acting out scenes from the often-vivid dreams that occur during this stage.

**Timing**

**The human biological clock**
Sleep timing is controlled by the circadian clock, sleep-wake homeostasis, and in humans, within certain bounds, willed behavior. The circadian clock—an innertimekeeping, temperature-fluctuating, enzyme-controlling device—works in tandem with adenosine, a neurotransmitter that inhibits many of the bodily processes associated with wakefulness. Adenosine is created over the course of the day; high levels of adenosine lead to sleepiness. In diurnal animals, sleepiness occurs as the circadian element causes the release of the hormone melatonin and a gradual decrease in core body temperature. The timing is affected by one's chronotype. It is the circadian rhythm that determines the ideal timing of a correctly structured and restorative sleep episode. Homeostatic sleep propensity (the need for sleep as a function of the amount of time elapsed since the last adequate sleep episode) must be balanced against the circadian element for satisfactory sleep. Along with corresponding messages from the circadian clock, this tells the body it needs to sleep. Sleep offset (awakening) is primarily determined by circadian rhythm. A person who regularly awakens at an early hour will generally not be able to sleep much later than his or her normal waking time, even if moderately sleep-deprived.

**Functions**
The multiple theories proposed to explain the function of sleep reflect the as-yet-incomplete understanding of the subject. It is likely that sleep evolved to fulfill some primeval function and took on multiple functions over time. (As an analogy, the larynx in all mammals controls the passage of food and air, but may have descended in humans to take on speech capabilities in addition.) It has been pointed out that, if sleep were not essential, one would expect to find 1) animal species that do not sleep at all, 2) animals that do not need recovery sleep when they stay awake longer than usual, and 3) animals that suffer no serious consequences as a result of lack of sleep. No animals have been found to date that satisfy any of these criteria.

**NEURAL MECHANISM OF SLEEP**
Sleep process would start with the activation of sleep-promoting neurons located in the preoptic area of the anterior hypothalamus. This activation leads to the inhibition of wake-promoting neurons located in the posterior hypothalamus, basal forebrain and mesopontine tegmentum, which, in turn, removes inhibition from the sleep-promoting structures, thereby enhancing the sleep process. Sleep-promoting neurons are supposed to contain γ-aminobutyric acid and inhibit cholinergic, noradrenergic, and serotonergic or histaminergic wake-promoting neurons at sleep onset during sleep.
Chemicals in brain
Compared to slow-wave sleep, both waking and paradoxical sleep involve higher use of the neurotransmitter acetylcholine, which may cause the faster brainwaves. The monoamine neurotransmitters norepinephrine, serotonin and histamine are completely unavailable. Injections of acetylcholinesterase inhibitor, which effectively increases available acetylcholine, have been found to induce paradoxical sleep in humans and other animals already in slow-wave sleep. Carbachol, which mimics the effect of acetylcholine on neurons, has a similar influence. In waking humans, the same injections produce paradoxical sleep only if the monoamine neurotransmitters have already been depleted. Two other neurotransmitters, orexin and gamma-Aminobutyric acid (GABA), seem to promote wakefulness, diminish during deep sleep, and inhibit paradoxical sleep. Unlike the abrupt transitions in electrical patterns, the chemical changes in the brain show continuous periodic oscillation.

Role of brain stem
Neural activity during REM sleep seems to originate in the brain stem, especially the pontinetegmentum and locus coeruleus. According to the activation-synthesis hypothesis proposed by Robert McCarley and Allan Hobson in 1975–1977, control over REM sleep involves pathways of "REM-on" and "REM-off" neurons in the brain stem. REM-on neurons are primarily cholinergic (i.e., involve acetylcholine); REM-off neurons activate serotonin and noradrenaline, which among other functions suppress the REM-on neurons. McCarley and Hobson suggested that the REM-on neurons actually stimulate REM-off neurons, thereby serving as the mechanism for the cycling between REM and non-REM sleep. They used Lotka–Volterra equations to describe this cyclical inverse relationship. Kayuza Sakai and Michel Jouvet advanced a similar model in 1981. Whereas acetylcholine manifests in the cortex equally during wakefulness and REM, it appears in higher concentrations in the brain stem during REM. The withdrawal of orexin and GABA may cause the absence of the other excitatory neurotransmitters.

Research in the 1990s using positron emission tomography confirmed the role of the brain stem. It also suggested that, within the forebrain, the limbic and paralimbic systems, generally connected with emotion showed more activation than other areas. The areas activated during REM sleep are approximately inverse to those activated during non-REM sleep.

Eye movements
Most of the eye movements in “rapid eye movement” sleep are in fact less rapid than those normally exhibited by waking humans. They are also shorter in duration and more likely to loop back to their starting point. About seven of such loops take place over one minute of REM sleep. Whereas in slow-wave sleep the eyes can drift apart, the eyes of the paradoxical sleeper move in tandem. These eye movements follow the ponto-geniculo-occipital waves originating in the brain stem. The eye movements themselves may relate to the sense of vision experienced in the dream, but a direct relationship remains to be clearly established. It does happen that congenitally blind people, who do not typically have visual imagery in their dreams, still move their eyes in REM sleep.

Circulation, respiration, and thermoregulation
Generally speaking, the body suspends homeostasis during paradoxical sleep. Heart rate, cardiac pressure, cardiac output, arterial pressure, and breathing rate quickly become irregular when the body moves into REM sleep. In general, respiratory reflexes such as response to hypoxia diminish. Overall, the brain exerts less control over breathing; electrical stimulation of respiration-linked brain areas does not influence the lungs, as it does during non-REM sleep and in waking. The fluctuations of heart rate and arterial pressure tend to coincide with PGO waves and rapid eye movements, twitches, or
sudden changes in breathing. Erections of the penis (nocturnal penile tumescence or NPT) normally accompany REM sleep in rats and humans. If a male has erectile dysfunction (ED) while awake, but has NPT episodes during REM, it would suggest that the ED is from a psychological rather than a physiological cause. In females, erection of the clitoris (nocturnal clitoral tumescence or NCT) causes enlargement, with accompanying vaginal blood flow and transudation (i.e. lubrication). During a normal night of sleep the penis and clitoris may be erect for a total time of from one hour to as long as three and a half hours during REM.

Body temperature is not well regulated during REM sleep, and thus organisms become more sensitive to temperatures outside their thermoneutral zone. Cats and other small furry mammals will shiver and breathe faster to regulate temperature during NREMS but not during REMS. With the loss of muscle tone, animals lose the ability to regulate temperature through body movement. (However, even cats with pontine lesions preventing muscle atonia during REM did not regulate their temperature by shivering.) Neurons which typically activate in response to cold temperatures—triggers for neural thermoregulation—simply do not fire during REM sleep, as they do in NREM sleep and waking.

Consequently, hot or cold environmental temperatures can reduce the proportion of REM sleep, as well as amount of total sleep. In other words, if at the end of a phase of deep sleep, the organism's thermal indicators fall outside of a certain range, it will not enter paradoxical sleep lest deregulation allow temperature to drift further from the desirable value. This mechanism can be 'fooled' by artificially warming the brain.

Muscle

REM atonia, an almost complete paralysis of the body, is accomplished through the inhibition of motor neurons. When the body shifts into REM sleep, motor neurons throughout the body undergo a process called hyperpolarization: their already-negative membrane potential decreases by another 2–10 millivolts, thereby raising the threshold which a stimulus must overcome to excite them. Muscle inhibition may result from unavailability of monoamine neurotransmitters, the abundance of acetylcholine in the brainstem, and perhaps from mechanisms used in waking muscle inhibition. The medulla oblongata, located between pons and spine, seems to have the capacity for organism-wide muscle inhibition. Some localized twitching and reflexes can still occur.

Lack of REM atonia causes REM behavior disorder, sufferers of which physically act out their dreams. (An alternative explanation of this relationship is that the sleeper "dreams out the act": that the muscle impulse precedes the mental image. This explanation could also apply to normal sleepers whose commands to their muscles are suppressed.) (Note that conventional sleepwalking takes place during slow-wave sleep.) Narcolepsy by contrast seems to involve excessive and unwanted REM atonia—i.e., cataplexy and excessive daytime sleepiness while awake, hypnagogic hallucinations before entering slow-wave sleep, or sleep paralysis while waking. Other psychiatric disorders including depression have been linked to disproportionate REM sleep. Patients with suspected sleep disorders are typically evaluated by polysomnogram.

Lesions of the pons to prevent atonia have induced functional “REM behavior disorder” in animals.

Psychology of Dreaming

Rapid eye movement sleep has since its discovery been closely associated with dreaming. Waking up sleepers during a REM phase is a common experimental method for obtaining dream reports; 80% of neurotypical people can give some kind of dream report under these circumstances. Sleepers awakened from REM tend to give longer more narrative descriptions of the dreams they were
experiencing, and to estimate the duration of their dreams as longer. Lucid dreams are reported far more often in REM sleep. (In fact these could be considered a hybrid state combining essential elements of REM sleep and waking consciousness.) The mental events which occur during REM most commonly have dream hallmarks including narrative structure, convincingsness (experiential resemblance to waking life), and incorporation of instinctual themes.

**Paradoxical Sleep**

This is one of the two basic states of sleep and is notable for a presence of rapid eye movement (REM). It is a deep stage of sleep with intense brain activity in the forebrain and midbrain. It is characterized by dreaming and the absence of motor function with the exception of the eye muscles and the diaphragm. It occurs cyclically several times during sleep, but it comprises the smallest portion of the sleep cycle.

**Sleep Disorders**

Humans may suffer from a number of sleep disorders. A sleep disorder (somnipathy) is a medical disorder of the sleep patterns of a person or animal. Some sleep disorders are serious enough to interfere with normal physical, mental and emotional functioning. A test commonly ordered for some sleep disorders is the polysomnography.

**Common disorders**

- Primary insomnia: Chronic difficulty in falling asleep and/or maintaining sleep when no other cause is found for these symptoms.
- Bruxism: Involuntary grinding or clenching of the teeth while sleeping.
- Delayed sleep phase syndrome (DSPS): inability to awaken and fall asleep at socially acceptable times but no problem with sleep maintenance, a disorder of circadian rhythms. Other such disorders are advanced sleep phase syndrome (ASPS) and Non-24-hour sleep-wake syndrome (Non-24), both much less common than DSPS.
- Hypopnea syndrome: Abnormally shallow breathing or slow respiratory rate while sleeping.
- Narcolepsy: Excessive daytime sleepiness (EDS) often culminating in falling asleep spontaneously but unwillingly at inappropriate times.
- Cataplexy: a sudden weakness in the motor muscles that can result in collapsing to the floor.
- Night terror: Pavornocturnus, sleep terror disorder: abrupt awakening from sleep with behavior consistent with terror.
- Parasomnias: Disruptive sleep-related events involving inappropriate actions during sleep stages - sleep walking and night-terrors are examples.
- Periodic limb movement disorder (PLMD): Sudden involuntary movement of arms and/or legs during sleep, for example kicking the legs. Also known as nocturnal.
- Rapid eye movement behavior disorder (RBD): Acting out violent or dramatic dreams while in REM sleep.
- Restless legs syndrome (RLS): An irresistible urge to move legs. RLS sufferers often also have PLMD.
- Situational circadian rhythm sleep disorders: shift work sleep disorder (SWSD) and jet lag.
- Obstructive sleep apnea: Obstruction of the airway during sleep, causing lack of sufficient deep sleep; often accompanied by snoring. Other forms of sleep apnea are less common.
- Sleep paralysis: is characterized by temporary paralysis of the body shortly before or after sleep. Sleep paralysis may be accompanied by visual, auditory or tactile hallucinations. Not a disorder unless severe. Often seen as part of Narcolepsy.
• Sleepwalking or somnambulism: Engaging in activities that are normally associated with wakefulness (such as eating or dressing), which may includewalking, without the conscious knowledge of the subject.
• Nocturia: A frequent need to get up and go to the bathroom to urinate at night. It differs from Enuresis, or bed-wetting, in which the person does not arouse from sleep, but the bladder nevertheless empties.
• Somniphobia: a dread of sleep.

Classifications
  ✤ Dyssomnias - A broad category of sleep disorders characterized by either hypersonmolence or insomnia. The three major subcategories include intrinsic (i.e., arising from within the body), extrinsic (secondary to environmental conditions or various pathologic conditions), and disturbances of circadian rhythm. MeSH
  • Insomnia
  • Narcolepsy
  • Obstructive sleep apnea
  • Restless leg syndrome
  • Periodic limb movement disorder
  • Hypersomnia
  ▪ Recurrent hypersomnia - including Kleine-Levin syndrome
  ▪ Posttraumatic hypersomnia
  ▪ "Healthy" hypersomnia
    o Circadian rhythm sleep disorders
  ▪ Delayed sleep phase syndrome
  ▪ Advanced sleep phase syndrome
  ▪ Non-24-hour sleep-wake syndrome
  • Parasomnias - A category of sleep disorders that involve abnormal and unnatural movements, behaviors, emotions, perceptions, and dreams in connection with sleep.
    ▪ REM sleep behaviour disorder
    ▪ Sleep terror
    ▪ Sleepwalking (or somnambulism)
    ▪ Bruxism (Tooth-grinding)
    ▪ Bedwetting or sleep enuresis.
    ▪ Sleep talking (or somniloquy)
    ▪ Sleep sex (or sexsomnia)
    ▪ Exploding head syndrome - Waking up in the night hearing loud noises.
  ✤ Medical or Psychiatric Conditions that may produce sleep disorders
    o Psychoses (such as Schizophrenia)
    o Mood disorders
      ✤ Depression
      ✤ Anxiety
    o Panic
    o Alcoholism
      ✤ Sleeping sickness - a parasitic disease which can be transmitted by the Tsetsefly.
      ✤ Snoring - Not a disorder in and of itself, but it can be a symptom of deeper problems.
Insomnia

Insomnia is a symptom which can accompany several sleep, medical and psychiatric disorders, characterized by persistent difficulty falling asleep and/or difficulty staying asleep. Insomnia is typically followed by functional impairment while awake. Both organic and non-organic insomnia without other cause constitute a sleep disorder, primary insomnia. One definition of insomnia is “difficulties initiating and/or maintaining sleep, or non-restorative sleep, associated with impairments of daytime functioning or marked distress for more than 1 month.” According to the United States Department of Health and Human Services in the year 2007, approximately 64 million Americans regularly suffer from insomnia each year. Insomnia is 41% more common in women than in men.

Classification / Types of insomnia

Although there are several different degrees of insomnia, three types of insomnia have been clearly identified: transient, acute, and chronic.

1. **Transient insomnia** lasts for less than a week. It can be caused by another disorder, by changes in the sleep environment, by the timing of sleep, severe depression, or by stress. Its consequences - sleepiness and impaired psychomotor performance - are similar to those of sleep deprivation.

2. **Acute insomnia** is the inability to consistently sleep well for a period of less than a month.

3. **Chronic insomnia** lasts for approximately a month. It can be caused by another disorder, or it can be a primary disorder. Its effects can vary according to its causes. They might include being unable to sleep, muscular fatigue, hallucinations, and/or mental fatigue; but people with chronic insomnia often show increased alertness. Some people that live with this disorder see things as if they are happening in slow motion, wherein moving objects seem to blend together. Can cause double vision.

Causes

Insomnia can be caused by:

- Psychoactive drugs or stimulants, including certain medications, herbs, caffeine, nicotine, cocaine, amphetamines, methylphenidate, MDMA and modafinil
- Fluoroquinolone antibiotic drugs, see Fluoroquinolone toxicity, associated with more severe and chronic types of insomnia
- Restless Legs Syndrome can cause insomnia due to the discomforting sensations felt and need to move the legs or other body parts to relieve these sensations. It is difficult if not impossible to fall asleep while moving.
- Pain—any injury or condition that causes pain. Pain can preclude an individual from finding a comfortable position in which to fall asleep, and in addition can cause awakening if, during sleep, the person rolls over and puts pressure on the injured or painful area of the body.
- Hormone shifts such as those that precede menstruation and those during menopause
- Life problems like fear, stress, anxiety, emotional or mental tension, work problems, financial stress.

Narcolepsy

Narcolepsy is a chronic sleep disorder, or dyssomnia, characterized by excessive daytime sleepiness (EDS) in which a person experiences extreme fatigue and possibly falls asleep at inappropriate times, such as while at work or at school. Narcoleptics usually experience disturbed nocturnal sleep and an abnormal daytime sleep pattern, which is often confused with insomnia. When a narcoleptic falls asleep they generally experience the REM stage of sleep within 10 minutes; whereas most people do not experience REM sleep until after 30 minutes. There is little evidence to suggest that narcoleptics tend to have a shorter life span. Another problem that some narcoleptics experience is
cataplexy, a sudden muscular weakness brought on by strong emotions (though many people experience cataplexy without having an emotional trigger. It often manifests as muscular weaknesses ranging from a barely perceptible slackening of the facial muscles to the dropping of the jaw or head, weakness at the knees, or a total collapse. Usually only speech is slurred, vision is impaired (double vision, inability to focus), but hearing and awareness remain normal. In some rare cases, an individual's body becomes paralyzed and muscles become stiff.

Narcolepsy is a neurological sleep disorder. It is not caused by mental illness or psychological problems. It is most likely affected by a number of genetic abnormalities that affect specific biological factors in the brain, combined with a set of environments, such as a virus.

The term narcolepsy derives from the French word narcolepsie created by the French physician Jean-Baptiste-Édouard Gélineau by combining the Greek (narkē, "numbness" or "stupor"), and (lepsis), "attack" or "seizure").

Sex

In biology, sex is a process of combining and mixing genetic traits, often resulting in the specialization of organisms into a male or female variety (known as a sex). Sexual reproduction involves combining specialized cells (gametes) to form offspring that inherit traits from both parents. Gametes can be identical in form and function (known as isogametes), but in many cases an asymmetry has evolved such that two sex-specific types of gametes (heterogametes) exist: male gametes are small, motile, and optimized to transport their genetic information over a distance, while female gametes are large, non-motile and contain the nutrients necessary for the early development of the young organism. An organism's sex is defined by the gametes it produces: males produce male gametes (spermatozoa, or sperm) while females produce female gametes (ova, or egg cells); individual organisms which produce both male and female gametes termed hermaphroditic. Frequently, physical differences are associated with the different sexes of an organism; these sexual dimorphisms can reflect the different reproductive pressures the sexes experience.

Sexual reproduction

Sexual reproduction is a process where organisms form offspring that combine genetic traits from both parents. Chromosomes are passed on from one parent to another in this process. Each cell has half the chromosomes of the mother and half of the father. Genetic traits are contained within the deoxyribonucleic acid (DNA) of chromosomes — by combining one of each type of chromosomes from each parent, an organism is formed containing a doubled set of chromosomes. The life cycle of sexually reproducing organisms cycles through haploid and diploid stages.

This double-chromosome stage is called "diploid", while the single-chromosome stage is "haploid". Diploid organisms can, in turn, form haploid cells (gametes) that randomly contain one of each of the chromosome pairs, via a process called meiosis. Meiosis also involves a stage of chromosomal crossover, in which regions of DNA are exchanged between matched types of chromosomes, to form a new pair of mixed chromosomes. Crossing over and fertilization (the recombining of single sets of chromosomes to make a new diploid) result in the new organism containing a different set of genetic traits from either parent.

In many organisms, the haploid stage has been reduced to just gametes specialized to recombine and form a new diploid organism; in others, the gametes are capable of undergoing cell division to produce multicellular haploid organisms. In either case, gametes may be externally similar, particularly...
in size (isogamy), or may have evolved an asymmetry such that the gametes are different in size and other aspects (anisogamy). By convention, the larger gamete (called an ovum, or egg cell) is considered female, while the smaller gamete (called a spermatozoon, or sperm cell) is considered male. An individual that produces exclusively large gametes is female, produces both types of gametes is a hermaphrodite; in some cases hermaphrodites are able to self-fertilize and produce offspring on their own, without a second organism.

**Animal**

Most sexually reproducing animals spend their lives as diploid organisms, with the haploid stage reduced to single cell gametes. The gametes of animals have male and female forms—spermatozoa and egg cells. These gametes combine to form embryos which develop into a new organism. The male gamete, a spermatozoon (produced within a testicle), is a small cell containing a single long flagellum which propels it. Spermatozoa are extremely reduced cells, lacking many cellular components that would be necessary forembryonic development. They are specialized for motility, seeking out an egg cell and fusing with it in a process called fertilization. Female gametes are egg cells (produced within ovaries), large immobile cells that contain the nutrients and cellular components necessary for a developing embryo.

Egg cells are often associated with other cells which support the development of the embryo, forming an egg. In mammals, the fertilized embryo instead develops within the female, receiving nutrition directly from its mother. Animals are usually mobile and seek out a partner of the opposite sex for mating. Animals which live in the water can mate using external fertilization, where the eggs and sperm are released into and combine within the surrounding water. Most animals that live outside of water, however, must transfer sperm from male to female to achieve internal fertilization.

In most birds, both excretion and reproduction is done through a single posterior opening, called the cloaca—male and female birds touch cloaca to transfer sperm, a process called "cloacal kissing". In many other terrestrial animals, males use specialized sex organs to assist the transport of sperm—these male sex organs are called intromittent organs. In humans and other mammals this male organ is the penis, which enters the female reproductive tract (called the vagina) to achieve insemination—a process called sexual intercourse. The penis contains a tube through which semen (a fluid containing sperm) travels. In female mammals the vagina connects with the uterus, an organ which directly supports the development of a fertilized embryo within (a process called gestation). Because of their motility, animal sexual behavior can involve coercive sex. Traumatic insemination, for example, is used by some insect species to inseminate females through a wound in the abdominal cavity—a process detrimental to the female's health.

**Evolution**

Sexual reproduction first appeared about a billion years ago, evolved within ancestral single-celled eukaryotes. The reason for the initial evolution of sex, and the reason(s) it has survived to the present, are still matters of debate. Some of the many plausible theories include: that sex creates variation among offspring, sex helps in the spread of advantageous traits, and that sex helps in the removal of disadvantageous traits. Sexual reproduction is a process specific to eukaryotes, organisms whose cells contain a nucleus and mitochondria. In addition to animals, plants, and fungi, other eukaryotes (e.g. the malaria parasite) also engage in sexual reproduction. Some bacteria use conjugation to transfer genetic material between bacteria; while not the same as sexual reproduction, this also results in the mixture of genetic traits. What is considered defining of sexual reproduction is the difference between the gametes and the binary nature of fertilization. Multiplicity of gamete types
within a species would still be considered a form of sexual reproduction. However, no third gamete is known in multicellular animals.

Sex determination

The most basic sexual system is one in which all organisms are hermaphrodites, producing both male and female gametes—this is true of some animals (e.g., snails) and the majority of flowering plants. In many cases, however, specialization of sex has evolved such that some organisms produce only male or only female gametes. The biological cause for an organism developing into one sex or the other is called sex determination. In the majority of species with sex specialization organisms are either male (producing only male gametes) or female (producing only female gametes). Exceptions are common—for example, in the roundworm *C. elegans* the two sexes are hermaphrodite and male (a system called androdioecy). Sometimes an organism's development is intermediate between male and female, a condition called intersex. Sometimes intersex individuals are called "hermaphrodite"; but, unlike biological hermaphrodites, intersex individuals are unusual cases and are not typically fertile in both male and female aspects.

Genetic

In genetic sex determination systems, an organism's sex is determined by the genome it inherits. Genetic sex determination usually depends on asymmetrically inherited sex chromosomes which carry genetic features that influence development; sex may be determined either by the presence of a sex chromosome or by how many the organism has. Genetic sex determination, because it is determined by chromosome assortment, usually results in a 1:1 ratio of male and female offspring. Humans and other mammals have an XY sex determination system: the Y chromosome carries factors responsible for triggering male development. The default sex, in the absence of a Y chromosome, is female. Thus, XX mammals are female and XY are male. XY sex determination is found in other organisms, including the common fruit fly and some plants. In some cases, including in the fruit fly, it is the number of X chromosomes that determines sex rather than the presence of a Y chromosome. In birds, which have a ZW sex-determination system, the opposite is true: the W chromosome carries factors responsible for female development, and default development is male. In this case ZZ individuals are male and ZW are female. The majority of butterflies and moths also have a ZW sex-determination system. In both XY and ZW sex determination systems the sex chromosome carrying the critical factors is often significantly smaller, carrying little more than the genes necessary for triggering the development of a given sex. Like humans and other mammals, the common fruit fly has an XY sex determination system.

Many insects use a sex determination system based on the number of sex chromosomes. This is called XX/XO sex determination—the O indicates the absence of the sex chromosome. All other chromosomes in these organisms are diploid, but organisms may inherit one or two X chromosomes. In field crickets, for example, insects with a single X chromosome develop as male, while those with two develop as female. In the nematode *C. elegans* most worms are self-fertilizing XX hermaphrodites, but occasionally abnormalities in chromosome inheritance regularly give rise to individuals with only one X chromosome; these XO individuals are fertile males (and half their offspring are male). Other insects, including honey bees and ants, use a haplodiploid sex-determination system. In this case diploid individuals are generally female, and haploid individuals (which develop from unfertilized eggs) are male. This sex-determination system results in highly biased sex ratios, as the sex of offspring is determined by fertilization rather than the assortment of chromosomes during meiosis.

Nongenetic
For many species sex is not determined by inherited traits, but instead by environmental factors experienced during development or later in life. Many reptiles have temperature-dependent sex determination: the temperature embryosexperience during their development determines the sex of the organism. In someturtles, for example, males are produced at lower incubation temperatures than females; this difference in critical temperatures can be as little as 1-2°C.

Many fish change sex over the course of their lifespan, a phenomenon called sequential hermaphroditism. In clownfish, smaller fish are male, and the dominant and largest fish in a group becomes female. In many wrasses the opposite is true—most fish are initially female and become male when they reach a certain size. Sequential hermaphrodites may produce both types of gametes over the course of their lifetime, but at any given point they are either female or male. In some ferns the default sex is hermaphrodite, but ferns which grow in soil that has previously supported hermaphrodites are influenced by residual hormones to instead develop as male.

**Sexual dimorphism**

Many animals have differences between the male and female sexes in size and appearance, a phenomenon called sexual dimorphism. Sexual dimorphisms are often associated with sexual selection—the competition between individuals of one sex to mate with the opposite sex. Antlers in male deer, for example, are used in combat between males to win reproductive access to female deer. In many cases the male of a species is larger in size; in mammals species with high sexual size dimorphism tend to have highly polygynous mating systems—presumably due to selection for success in competition with other males. Other animals, including most insects and many fish, have larger females. This may be associated with the cost of producing egg cells, which requires more nutrition than producing sperm—larger females are able to produce more eggs. Occasionally this dimorphism is extreme, with males reduced to living as parasites dependent on the female.

In birds, males often have a more colourful appearance and may have features (like the long tail of male peacocks) that would seem to put the organism at a disadvantage (e.g., bright colors would seem to make a bird more visible to predators). One proposed explanation for this is the handicap principle. This hypothesis says that, by demonstrating he can survive with such handicaps, the male is advertising his genetic fitness to females—traits that will benefit daughters as well, who will not be encumbered with such handicaps.

Sex differences in humans include, generally, a larger size and more body hair in men; women have breasts, wider hips, and a higher body fat percentage.

**DYNAMICS OF SEXUAL BEHAVIOUR**

In lower animals we speak about sexual motivation as a "drive." That is, we state that some internal, innate force pushes the animal to engage in reproductive behavior. Humans don't simply give in to an internal push towards sexual behavior. Instead, human motivation to engage in sexual behavior is due to a complex relationship among several factors. Most theorists refer to motivation as an inferred need, desire or impulse which initiates, directs and sustains behavior (e.g., Coon, 1997; Wood & Wood, 1996). One group of psychologists calls motivation a factor which explains the relations between stimuli and behavior (Bernstein, Clarke-Stewart, Roy, & Wickens, 1997). By combining these two definitions and applying them to human sexual behavior we could say that sexual motivation is an inferred, internal state influenced by several factors which determines engagement in sexual activity.

**Factors in Human Sexual Motivation**

Sexual motivation is influenced by hormones such as testosterone, estrogen, progesterone, oxytocin, and vasopressin. In most mammalian species, sex hormones control the ability to engage in sexual behaviours. However, sex hormones do not directly regulate the ability to copulate in primates.
(including humans). Rather, sex hormones in primates are only one influence on the motivation to engage in sexual behaviours.

Physical pleasure has both a physiological component (the physical sensations associated with touch) and a subjective psychological component. Where does something subjective like pleasure fit in our breakdown into physiological and environmental components? Pleasure is an emotion (Cofer, 1972), which, according to the Schacter-Singer theory, is a subjective feeling based upon physiological arousal and interpretations of the stimuli that are linked to the arousal (Cornelius, 1996). Thus emotions are both physiologically- and cognitively-based. This indicates that another category exists into which we might place sexual motivators, but to state this would be to miss the larger issue. The larger issue is that pleasure is influenced by both our cognitions and our physiological functioning. As a factor involved in sexual motivation, it is not unusual to be associated with motivation and to simultaneously be associated with other variables that are themselves identified as related to sexual motivation and which may or may not belong to the same category. Thus, identifying categories and then placing the elements of sexual motivation into discrete categories is a difficult, if not impossible, task. Rather than attempting to do so, the current author will identify the variables that have been linked to sexual motivation and identify, where possible, any mediating variables.

**Physiological Correlates** - An analysis of human sexual motivation couldn't proceed without first discussing physiological factors, in particular, hormones. The influence of hormones in sexual behavior is well-supported by research. Both men and women produce estrogens, progestins and androgens, though women produce far more estrogens and progestin and men more androgens (Hokanson, 1969; Leger, 1992). In lower species, hormone levels are almost directly correlated with sexual behavior, however, as one moves up the phylogenetic scale, other elements become involved (Fisher, 1993; Hokanson, 1969). In humans, hormones are also related to sexual desire, but are not the entire story. In males, a minimum level of testosterone is necessary to maintain normal sexual motivation in males (Leger, 1992). If males' testosterone levels fall below the threshold, sexual motivation is greatly reduced. However, once the threshold level is reached, it no longer predicts sexual behavior. Women's studies also show correlations between hormones and sexual desire (Leger, 1992; Sherwin & Gelfan, 1987; Sherwin, Gelfan, & Brender, 1985), however, the results are inconsistent (Leger, 1992). Since neither increases nor decreases in hormones in either males or females are perfectly correlated with sexual desire, it stands to reason that there must be other factors involved. As Hokanson (1969) concludes, hormones serve the primary purpose of readying the individual for action, but other factors determine whether the individual actually engages in sexual activity. Another physiological factor in sexual motivation may well be odor and sense of smell. Of all the elements researched, odor and sense of smell have received the least attention, probably because, as Kohl and Francoeur (1995) state, their influence on sexual behavior is difficult to ascertain. However, body odor (i.e., airborne hormones) definitely influences our behaviors. In their review of numerous studies such as synchronization of menstrual cycles of women who live together, and the influence of hormone-scented masks on individuals' ratings of others, Kohl and Francoeur (1995) state that odor must be involved in our sexual behaviors also.

Helen Fisher (1993) also agrees that odors may influence sexual behavior and cites that some men in Greece swear by body-odor scented handkerchiefs which they use to lure women into relationships.

**Sexual Orientation** - Our desire to engage in sexual behavior with someone is also influenced by sexual orientation. Sexual orientation refers to the direction of an individual's sexual attraction (Wood, et al., 1996). Most individuals are heterosexual (Laumann, 1994; Wellings, et al., 1994) which means they are primarily attracted to the opposite sex. Homosexuals are individuals who are attracted to the same sex and bisexuals are attracted to both sexes. Why are individuals attracted to
one sex rather than another? LeVay (1995) believes that most researchers of the topic agree it is a combination of multiple factors including genetic makeup, hormones and social experiences. He further believes that newer studies (e.g., Bailey & Pillard, 1991; Bailey, Pillard, Neale, & Agyei, 1993) indicate that genes are perhaps more influential than the other factors. Studies indicate that the percentage of individuals who call themselves homosexual is quite small, ranging from about .5% to 2.8% (Laumann, 1994; Wellings, et al., 1994). This estimate is significantly lower than the rates given in the problematic Kinsey Reports (1948; 1953). In his review of several studies on the prevalence of homosexuality, LeVay (1995) states that it is best to keep an open mind towards reviewing new evidence since changing attitudes and beliefs appear to be linked to self-stated homosexuality.

What he was referring to was the indication that individuals are more likely to express their gay behavior within their own culture as that culture becomes more accepting of homosexuality. Thus it is apparent that culture influences the expression of one's sexual orientation which in turn influences sexual motivation.

Pleasure - As mentioned earlier, pursuit of erotic pleasure is a primary reason to engage in sexual behavior (Abramson et al., 1995; Hatfield et al., 1993). Kinsey and colleagues (1948; 1953) found that children between the ages of 2 and 5 years of age spontaneously touch their genitals. At this age, one could not argue that this sexual behavior is learned or designed to contribute to reproduction. Abramson and Pinkerton (1995) point out that the pleasure of sexual behavior is physiologically and psychologically-based and that the sex organs do not exist merely to guarantee reproductive behavior. As an example, they cite the female orgasm, uncommon during vaginal penetration, but very common by more direct means of clitoral stimulation. In other words, sexual pleasure does not occur merely to ensure procreation. We engage in sexual behavior because it is enjoyable. However, as will be reviewed later, what is considered pleasurable, may well be influenced by one's interpretation of the stimuli.

Cognitions - How a stimulus is interpreted influences how individuals respond to that stimulus. Zellman and Goodchild (1983) surveyed 400 teenagers and found that the behaviors girls felt conveyed romantic interest were the same actions boys considered invitations to sex. Since societies create very different gender roles for men and women, differences in interpretation of the same data are bound to occur (Wade, et al., 1996). Wade's comments indicate that culture influences sexual behaviors, not only through performance of behaviors that are considered appropriate, but also through interpretation of those behaviors.

Cognitions and arousal - Based upon the results of surveys such as the Kinsey studies (1948; 1953), men have been considered to be more sexually responsive than women. Early studies comparing men and women's subjective responses to erotic films supported that theory. However, when studies were conducted comparing male and female physiological responses to male-produced, male-intended erotic films, researchers found that men and women actually experienced the same physiological arousal (Laan, Everaerd, Van Bellen, & Hanewald; 1994). When participants were asked to express their feelings about the stimuli, men reported sexual arousal and positive affect, yet women reported disgust and lack of arousal. In other words, both men and women experienced the same physiological arousal but different subjective arousal. When women viewed an erotic film produced by women for women, the female participants showed the same physiologic arousal as they did to male-produced films, but reported significantly greater sexual arousal, interest and positive affect. As interpreted by the researchers, the difference was due to how women interpreted the content of the films. Essentially, this study indicated that interpretation of the stimuli is of great importance in subjective feelings of sexual arousal.

Cognitions affect sexual arousal in another fashion. According to Kalat (1996), inhibition of arousal can occur in individuals who believe that sex is shameful. These individuals experience sexual
arousal, but have difficulties achieving sexual orgasm because of their thoughts. Palace and Gorzalka (1992) studied sexually functional and dysfunctional women and found that cognitions and physiological arousal were simultaneously important in sexual arousal. They hypothesized that cognitions and physiological arousal comprise a feedback loop to determine overall sexual arousal. These many studies indicate that the thoughts individuals have regarding various stimuli impact individuals' sexual motivation through influencing their arousal or their interpretations of behavior.

**Attraction** - Numerous elements have been identified as playing a role in attraction. For example, attraction is a function of proximity (how frequently you cross paths with someone), familiarity and similarity (e.g. in looks, or attitudes) (Kalat, 1996). This has been supported both with studies of attraction to friends and to romantic partners. Playing hard-to-get also contributes to human's attraction to one another (Hatfield, Walster, Piliavin & Schmidt, 1988). Apparently individuals make attributions about potential significant others based upon how quickly that person returns a show of interest. Those who are easily attained are less attractive than those who are more difficult to attain due to the traits the relationship-seeker attributes to her. For example, relationship seekers fear that easy-to-get women might display inappropriate behaviors in public. However, a hard-to-get woman who indicates interest in the relationship-seeker has positive traits attributed to her such as warmth and friendliness.

Another overwhelmingly important element in attraction is physical attractiveness. As stated previously, research between attitudes and behaviors are not always consistent. Research on what individuals find attractive in potential dates provides further evidence for this inconsistency in human sexual behavior. Although subjects stated that physical attractiveness was one of the least important elements in their attraction to someone else, in actual experiments using blind dates, the only factor which predicted whether subjects desired a second date with the same person was the attractiveness of the blind date (Walster, Aronson, Abrahams, & Rottman, 1966). This was true for both male and female participants of the study. In a study on physical attractiveness and relationship length, the factor which best predicted whether couples would remain together nine months after they began dating was the similarity in their physical attractiveness (White, 1980). This "matching" phenomenon in which people tend to select mates that match them in terms of physical attractiveness, has been replicated and expanded upon with consistent results (Feingold, 1988). It might seem that we learn to appreciate beauty from the culture that we are born into, yet studies of pre-school children indicate that they too, prefer attractive classmates and also make attributions based on classmates' physical characteristics (Dion & Berscheid, 1971).

Attraction to others is yet another element of sexual motivation that has its roots in both nature and nurture -- it is obviously innate to seek out attractive others, yet we still lean towards mates who are more similar to us, an apparent influence of culture and learning in addition to an inherited predisposition.

**Learning** - Learning is, of course, highly influential in sexual motivation. We copy the behaviors of those we respect and admire. We learn to repeat behaviors that are rewarded (and sexual behavior is rewarding for most) and we learn to discontinue behaviors that have negative outcomes. Conditioning is believed to influence sexual motivation. Certain stimuli may increase sexual arousal. For example, one might become sexually aroused by candlelight due to the learned association with sexual pre-encounters such as a romantic, candlelight dinner. It has also been proposed that conditioning accounts for sexually dysfunctional behaviors and sexual deviance (O'Donohue & Plaud, 1994). For example, a pedophile (person sexually aroused by children) might have been accidentally sexually aroused in the presence of a child. Principles of conditioning indicate he
would seek this same combination of factors in the future inorder to achieve the same pleasurable circumstances again. In her study of sexual motivators, Barbara Leigh (1989) states that fear of rejection, a learned component, isindeed the reason most often given by single men for not engaging in sex. Matching theory (Carli, Ganley, & Pierce-Otay, 1991), which states that individuals within couples are frequently very similar in attractiveness ratings, is easily understood using the principles of conditioning. For example, an average-looking man who is rebuffed whenever he approaches beautiful females should reduce his attempts to interact with beautiful women. Similarly, he should rebuff less-attractive women if he could interact with more attractive women. Who he ultimately couples with should be very similar in looks due to the conditioning of each person's partner-choosing behaviors.

Conditioning as a theory to explain sexual deviance and dysfunction is not without its critics. O'Donohue and Plaud (1994) examined several studies which used behavioral and aversion therapy to change sexual behaviors. Due to methodological problems in the studies they examined, they believe that conditioning plays a much smaller role in sexual motivation than previously believed. Thus conditioning mayplay some role in the sexual motivation, but how much of a role it plays is not clear. Culture - As mentioned throughout this essay, culture determines what behaviors are gender appropriate, what behaviors may or may not be performed in public, and what behaviors are considered sexually arousing. Yet culture and learning areinextricably tied together. An individual could not acquire his or her culture's norms without learning taking place. Conversely, there is very little one could learn which is not influenced by culture. For example, when we model the behaviors of individuals from our own society, we are copying behaviors that are more than likely already societal-influenced. If we view behaviors performed by individuals from another culture, we do so through lenses already colored by our society's influence. Hence any learning we might acquire from a culturally-different person is mediated by our own culture first.

**Attitudes and Culture** - Attitudes are defined as relatively stable evaluations of a person, object, situation or issue (Wood et al., 1996). Studies have shown that behaviors normally considered proper in one culture, may be improper or arousing in another. In other words, attitudes towards sexual behaviors are culturally learned. For example, some cultures find kissing repulsive (Tiefer, 1995) while other cultures insist on same-gender sex as a rite of passage into adulthood (Herdt, 1984). It is still noted, even in newer surveys in the United States (e.g., Laumann et al., 1994), that men and women have different attitudes toward sexual behaviors. For example, men are more interested in a variety of sexual behaviors, such as group sex, than are women. These divergences are undoubtedly, as mentioned earlier, a function of the gender roles each society impresses upon its members. A comparison of Swedish and American college students sought to examine if indeed the difference in men's and women's attitudes could be definitively tied to culture, rather than inherent gender differences (Weinberg, Lottes, Shaver, 1995). Specifically, it was believed that men and women in Sweden would have more convergent and relaxed attitudes toward sexual behaviors than the American participants. Sweden is generally known to have more relaxed sexual standards. It is believed that this is due, in part, to several years of mandatory sex education and the relatively equal power that women have in society. The study indeed showed that Swedish men and women had very similar attitudes towards sexual behaviors. Americans, as expected, had very different attitudes about what constituted appropriate sexual behaviors. While the current author cautioned earlier against drawing causal conclusions from a descriptive study such as this, the information further indicates that culture is associated with differences in sexual attitudes. The influence of learning on sexual motivation is quite profound. Attraction, cognitions, and sexual orientation, variables mentioned previously, are also influenced by learning. Thus a key component which determines the level of our sexual motivation is learning.
HORMONES AND SEXUAL BEHAVIOUR

Sex hormones are steroids (fat soluble compounds) that control sexual maturity and reproduction. These hormones are produced mainly by the endocrine glands. The endocrine glands in females are ovaries and those in males are testes. While both males and females have all types of hormones present in their bodies, females produce the majority of two types of hormones, estrogens and progesterone, while males produce mainly androgens such as testosterone. Most androgens produced by females are converted to estrogens and some androgens in males are also converted to estrogens. Sex hormones are synthesized from cholesterol (a fatty acid) and other compounds and secreted throughout a person's lifetime at different levels. Their production increases at puberty and normally decreases in old age.

Hormone Production

The production of hormones is a complex process. At puberty, the brain's hypothalamus gland produces increased amounts of gonadotropin-releasing hormone. This hormone stimulates the nearby pituitary gland to release two other hormones: follicle-stimulating hormone (FSH) and luteinizing hormone (LH). Finally, these two hormones signal the sex glands (gonads) to produce the sex hormones.

Female Reproductive Cycle

Females produce three estrogens: estradiol, estriol, and estrone. These estrogens stimulate growth of the ovaries and begin preparing the uterus for pregnancy. Estrogens also control the body's secondary sex characteristics, including breast and pelvic development and the distribution of fat and muscle. Progesterone maintains uterine conditions during pregnancy. It also acts on the central nervous system in a way that isn't yet understood. During the monthly reproductive cycle, FSH stimulates growth of an ovarian body called the graafian follicle. The follicle encloses the egg. LH aids in the rupture of the follicle, sending the egg to the fallopian tubes. LH also promotes growth of the corpus luteum (a yellow, progesterone-secreting mass of cells that forms from an ovarian follicle after the release of a mature egg) as the ovary prepares to release the egg into the uterus. If no pregnancy occurs within 10-12 days, the corpus luteum withers and the uterus sheds the blood supply that was formed to nourish a fetus. This shedding of the uterine lining and blood supply is called menstruation (the period). The production of estrogens and progesterone drops dramatically, and the cycle begins again.

Male Reproduction

In males, LH stimulates the development of the testes. The testes produce the androgens testosterone and androsterone. When FSH activates the testes' spermforming cells, testosterone maintains the process of forming sperm. This is the tenweek process results in sperm constantly ready for release by ejaculation from the penis. The androgens also promote the secondary sex characteristics of muscle growth, lowered voice range, the Adam's apple, and increased body hair.

THE DEVELOPMENTAL ASPECTS OF SEXUAL BEHAVIOR

Adult human sexual behavior results from a long, complex, and often hazardous development. Until about the beginning of our century, sex was believed to be largely instinctive, i.e., the result of biological heredity. Most people simply assumed that, at some time after puberty, sexual desire and sexual activity "came naturally" to every male and female, and that no social conditioning was involved. Sexuality was a "force of nature" which appeared suddenly and then, all by itself, found its full "natural" expression. Society could suppress this force, but had no part in shaping it. The first serious challenge of this traditional view came from Sigmund Freud (1856-1939) and his followers. In his practice as a physician, Freud encountered many patients suffering from what was then called hysteria, i.e., a severe
disability, such as paralysis or blindness, for which no physical cause could be found. Indeed, according to all standard medical tests, the patients should have been able to function normally. After interviewing these men and women over long periods of time, Freud noticed that their disabilities seemed somehow related to painful or disturbing childhood experiences. He further discovered that these early experiences, of which the patients were no longer consciously aware, were of asexual nature. Finally, he found that once the experiences were again clearly remembered and understood by the suffering adults, their mysterious disabilities disappeared.

On the basis of these and other findings, Freud gradually developed his psychoanalytic theory which since then has had a profound influence on European and American thought. However, when it was first proposed the theory was greeted with outcries of public indignation. It was plainly inconceivable to most people that a long forgotten childhood experience should continue to have any decisive influence on a person's adult life, and they were positively outraged at the suggestion that such experiences were sexual. In their view, children were "innocent" and "by nature" utterly incapable of sexual feelings or responses. For Freud, on the other hand, the sexuality of children and, indeed, infants was an indisputable fact of utmost importance.

According to psychoanalytic thinking, there is a basic sexual instinct or drive present universally in all human beings from the moment of birth. This instinct, which strives for sensual pleasure, is at first diffuse and attains its eventual proper direction and focus only through a process of "psychosexual maturation". Human infants first seek their gratification in a direct, unhindered, and undiscriminating way, until they learn to modify and control their instinctual urges through social conditioning. Human sexuality thus unfolds under the influence of two opposing forces: the "pleasure principle" and the "reality principle". In other words, a child's personality development can be described as a contest between biological drive and cultural constraint. This contest proceeds in three major steps, which are coordinated with the child's physiological maturation: the oral, anal, and phallic phases.

In the oral phase (from Latin os: mouth), the chief source of pleasure is the mouth. As it sucks the mother's breast, the infant finds not only nourishment, but deep physical and psychological satisfaction. In this phase, the mouth also serves as an organ of exploration. The infant puts everything in its mouth in order to get to know it. "Taking in" the world is the first attempt at mastering it. In the following anal phase (from Latin anus: the rectal opening), the main source of sensual gratification shifts from the mouth to the anal area. The child now begins to gain control over the bowel movements and thereby, indirectly, over the attending adults, whom it can now please or displease by eliminating or withholding feces. At the same time, the child learns to grant or withhold affection, say yes or no, in short, to master the world by "holding back" and "letting go." While the oral and anal phases, which extend roughly through the first three years of life, are the same for both sexes, the now following phallic phase (from Greek phallos: penis) brings an increasing awareness of sexual differences and of the male and female sex organs. The most pleasurable zones of the body are no longer the mouth or the anus, but the penis (for boys) and the clitoris (for girls).

This is the phase in which children become actively curious about their surroundings, poke their fingers into things, look inside their toys by taking them apart, and also investigate their own and each other's bodies. The most important aspect of this phase, however, is the development of the so-called Oedipus complex, i.e., the child's erotic attachment to the parent of the opposite sex and a feeling of rivalry toward the parent of the same sex. (The term "Oedipus complex" alludes to the legendary Greek king Oedipus who unknowingly killed his father and married his mother.) For example, it is the rule for a four-year-old boy to be deeply in love with his mother. She is, for him, the only woman he knows and cares to know. However, this woman already has a husband—the father. The boy is jealous of him and

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would like to push him aside in order to assume his position. This desire is usually expressed openly and spontaneously, as for instance when the boy climbs into his mother's bed announcing: "When I grow up, I'll marry you". Obviously, this situation can be compared to that of King Oedipus, although there is one important difference: Oedipus actually did remove his father forever from his mother's side, and he did marry her. The normal development of a child takes another course. The boy replaces his desire to marry his mother with the wish to marry a woman like his mother, and his urge to take the place of his father turns into the determination to become a man like his father. The boy can make this transition easily, if the father provides an attractive model to follow, and if he actively encourages his son to become a man. At the same time, it is the mother's task to help her son realize that she has already chosen and is no longer available as a sexual object. These parental attitudes will lead the boy to seek his sexual gratification elsewhere. (In the case of a girl, the development takes the opposite course: she loves her father and is jealous of her mother. The respective psychoanalytic term is "Electra complex", after Electra, a legendary Greek princess who, after the death of her beloved father, helped kill her mother who had murdered him. [It must be pointed out, however, that the notion of an Electra complex was advanced by some of Freud's followers, not by Freud himself, who did not subscribe to it.]) Freud believed that every child normally progressed from the oral to the anal and finally to the phallic phase, unless some negative influence interfered with this development. However, if the particular needs of any one of these phases were either unfulfilled or gratified to excess, the child could become "fixated" and thus hampered in its psychosexual growth. For example, a child's too rigid or overindulgent toilet training could lead to a fixation at the anal level of satisfaction.

As an adult, such a child would then turn into an "anal character", i.e., a person who is obsessed with discipline, order, and cleanliness, who hoards money (the unconscious equivalent of feces, which can be "withheld" from others) or who prefers anal stimulation to all other forms of sexual intercourse. An "oral character," on the other hand, would continue to depend mainly on his mouth even for sexual satisfaction, or he might become a compulsive eater, smoker, or drinker. Children who do not become fixated in this manner eventually reach "genital maturity." That is to say, after a so-called latency period, during which obvious sexual interests seem largely suspended, the sexual instinct reawakens with puberty and seeks satisfaction through genital intercourse. Oral and anal stimulation may still be enjoyed to a limited extent, but they now take second place to coitus which, for adults, is the one truly "mature" form of sexual expression.

As can be gathered from this brief and superficial sketch, Freud's concept of human sexuality is extraordinarily broad. Indeed, he stretches this concept to cover responses and activities that, before him, were considered to be completely nonsexual. Even today, the average layman may find it difficult to see any sexual implications in a baby's suckling on the mother's breast, or in an adult's compulsive eating habits. As a matter of fact, many scientists also continue to challenge the psychoanalytic view. For example, anthropologists who have studied various primitive cultures suggest that the Oedipal conflict may not be a universal human experience. Social psychologists have raised serious doubts as to whether an innate sexual drive or instinct even exists at all. Finally, many behaviorists and learning theorists maintain that Freud's whole theory is unnecessarily complex and that there are simpler (and therefore more convincing) explanations of human behavior. Moreover, the fact remains that this theory has never been scientifically tested on a sufficient scale to be proven or disproven.

It is therefore obvious that Freud's teachings cannot simply be accepted as dogma, but have to be studied and evaluated within the cultural context of his particular time. Eventually, such a critical evaluation may even lead to a better understanding of our own post-Freudian culture. Freud was one of history's most brilliant and uncompromising thinkers as well as a great writer, and his works (which
comprise 24 volumes in their English language edition) contain deep insights not only into human sexuality, but also into the history and character of Western civilization.

Some of Freud's disciples and followers, however, have shown little allegiance to his critical spirit, but instead have converted elements of his theory into convenient tools of social control. As a consequence, the liberating impulse of psychoanalytic thinking has often been obscured and perverted. This tendency has been particularly noticeable in America where, contrary to Freud's own intentions, some of his hypotheses have been used to justify the persecution and oppression of sexual minorities. The scope of the present book does not permit a detailed discussion of the various psychoanalytic schools or even of Freud's original theory. On the other hand, experience has shown that this theory does not lend itself to simplification and popularization. Where such simplifications have been attempted, they have all too often led to serious misunderstandings. It is true that Freudian terms have long since entered our everyday language, and that today we can read about the "Oedipus complex" and "the subconscious" in newspapers and popular magazines. We hear of "Freudian slips", "ego," "superego", "libido", and "sublimation" in movies, on radio, and on television. Nevertheless, when taken out of their theoretical context, these words can create considerable confusion, and, among laymen, they are usually misapplied.

Fortunately, in the meantime, it has become very well possible to describe the development of sexual behavior without any reference to psychoanalytic concepts. Recent empirical sex research has provided us with a great deal of new information as to how people learn to act the way they do. We have also gained some understanding of the statistical frequency of certain behaviors. This, in turn, has forced us to reexamine many traditional assumptions about the "nature" of human sexuality. As a result, we are now able to take another entirely fresh look at the subject.

Around the middle of our century, Alfred C. Kinsey and his associates of the Institute for Sex Research in Bloomington, Indiana, published two monumental studies of human sexual behavior which were based on personal interviews with thousands of individuals from all age groups and all walks of life. Previously, such studies had always been forced to rely on small samples of medical patients or sex offenders, and the full range of "normal" sexuality was therefore largely unknown. Kinsey's work provided the first reliable statistical data on the behavior of healthy, average men and women. (Sexual Behavior in the Human Male, 1948, and Sexual Behavior in the Human Female, 1953.) At about the same time, Clellan S. Ford and Frank A. Beach, an anthropologist and a psychologist, wrote a cross-cultural study in which they compared the patterns of sexual behavior in 191 different societies. (Patterns of Sexual Behavior, 1951.)

More recently, John Money of Johns Hopkins University and some fellow researchers have conducted extensive research into sexual malformations and the problems of gender identity. (Sex Errors of the Body, 1968; Man and Woman, Boy and Girl, 1973; and Sexual Signatures, 1975.) In addition, William H. Masters and Virginia E. Johnson of the Reproductive Biology Research Foundation in St. Louis, Missouri, have carried out a thorough scientific investigation of human sexual functioning and malfunctioning. (Human Sexual Response, 1966; Human Sexual Inadequacy, 1970; and The Pleasure Bond, 1975.)

These and many other new studies of human sexuality owe little or nothing to psychoanalytic theory, and on certain issues they sharply disagree with Freud. Nevertheless, they confirm at least some of his basic contentions. For example, it is today generally accepted that sexual behavior does not "come naturally" to human beings, but is, in fact, shaped by social conditioning. It is further quite obvious that this conditioning has different goals and produces different results in different societies. There is also no longer any doubt that children are capable of sexual responses, and that certain early childhood experiences can have a crucial influence on a person's later sexual development.
Unfortunately, it is less clear than ever what all this social conditioning really means. The physician Freud had been mainly concerned with helping his patients, and for him and his followers sexual childhood experiences could easily be defined as either beneficial or harmful according to a single criterion: they were beneficial if they furthered the individual's "genital maturity," and they were harmful if they hindered or prevented it. Sexual behavior was thus described in terms of maturity and immaturity, health and sickness, norm and deviation. In the meantime, however, sex researchers have become much more cautious. They now realize that sexual norms change a great deal from one time and place to another and that, in regard to human behavior, terms like "maturity" and "health" are value judgments rather than judgments of fact. In Freud's time, sexual health and maturity were believed to manifest themselves in a monogamous marriage devoted to the procreation of children.

Sex, love, marriage, and procreation were therefore seen as inseparable. Indeed, sexual activity without any of its "socially redeeming" features was considered evil: sex without love (masturbation and prostitution), sex without marriage (premarital and extramarital intercourse), sex without procreation (childhood sex play, sex after the menopause, homosexuality). Today, we know that this particular value system is far from universal, and that it was typical only of the Western middle classes during a certain historical period. Medieval farmers or feudal lords, for example, lived by an entirely different value system, and the same must be said for people in the traditional African and Asian cultures. Finally, we see that in our own society more and more men and women are breaking away from their inherited middle class morality and are searching for new values. Under these circumstances, we have to be very careful about establishing any specific goals, norms, or standards for sexual behavior. Our first obligation is simply to understand it, and we therefore need an objective description in morally neutral terms.

Objectivity is not the only requirement, however. The description also has to be clear and precise, and this is a difficult task in itself. Nowhere is the terminological confusion greater than in the area of human sexuality.

In fact, this confusion already begins with the very concept of sex. We know that the term "sex" somehow refers to the difference and the attraction between males and females, but the extent of this difference and the character of this attraction are still largely disputed. Nevertheless, modern research has done a great deal to clarify the issues, and particularly the study of childhood development has provided us with some very valuable clues. It has been observed, for instance, that hermaphroditic children (i.e., children who are "sexually unfinished") may be raised as either boys or girls and develop all the "appropriate" attitudes, including their choice of sexual partner. To put it another way, children whose sex is misdiagnosed at birth learn to identify with the sex that is assigned to them. Furthermore, once a certain critical period has passed, this identification is permanent. Even if the mistake is later discovered, it cannot be corrected. After a certain age, a boy raised as a girl will continue to consider himself female and, in most cases, feel sexually attracted to males, while a girl raised as a boy will continue to consider herself male and, in most cases, feel sexually attracted to females.

In other words, if "sex" has to do with the contrast between male and female, then a person's "sexual" development has at least three aspects:
1. The male or female characteristics of the body (physical sex),
2. The social role as male or female (gender role), and
3. The preference for male or female sexual partners (sexual orientation).
A great deal of confusion can be avoided if these three aspects of human sexuality are considered separately, and it seems useful, therefore, to keep the following definitions firmly in mind:

**Physical Sex**
Physical sex is defined as a person's maleness or femaleness. It is determined on the basis of five physical criteria: chromosomal sex, gonadal sex, hormonal sex, internal accessory reproductive structures, and external sex organs. People are male or female to the degree in which they meet the physical criteria for maleness or femaleness. Most individuals are clearly male or female by all five physical criteria. However, a minority fall somewhat short of this test, and their physical sex is therefore ambiguous (hermaphroditism).

**Gender Role**

Gender role is defined as a person's masculinity or femininity. It is determined on the basis of certain psychological qualities that are nurtured in one sex and discouraged in the other. People are masculine or feminine to the degree in which they conform to their gender roles. Most individuals clearly conform to the gender role appropriate to their biological sex. However, a minority partially assume a gender role that contradicts their biological sex (transvestism), and for an even smaller minority such a role inversion is complete (transsexualism).

**Sexual Orientation**

Sexual orientation is defined as a person's heterosexuality or homosexuality. It is determined on the basis of preference for sexual partners. People are heterosexual or homosexual to the degree in which they are erotically attracted to partners of the other or same sex. Most individuals develop a clear erotic preference for partners of the other sex (heterosexuality). However, a minority are erotically attracted to both men and women (ambisexuality), and an even smaller minority are attracted mainly to partners of their own sex (homosexuality). It is important to realize that not only physical sex but also gender role and sexual orientation are matters of degree, and that they may be independent of each other. Thus, they may appear in different combinations in different individuals. A few examples of physical males may illustrate the point:

- **Male-Masculine-Heterosexual**
  A person of male sex usually adopts the masculine gender role and develops a heterosexual orientation. Such an individual then conforms to our image of the "typical" male

- **Male-Masculine-Homosexual**
  A person of male sex who has adopted the masculine gender role may very well develop a homosexual orientation. Such an individual may then look and behave like any other "typical" male in all respects but one—his choice of sexual partner.

- **Male-Feminine-Heterosexual**
  A person of male sex may adopt the feminine gender role. Such an individual may then try everything possible (including a "sex change operation") to make the body conform to the feminine self-image. In this case, an erotic preference for males, would, of course, have to be considered heterosexual.

- **Male-Feminine-Homosexual**
  A person of male sex may adopt the feminine gender role and try everything possible to make the body conform to the feminine self-image. If such an individual then also developed an erotic preference for females this sexual orientation could only be called homosexual. Obviously, the last two examples represent rather extreme cases, and it should be remembered that even where a man identifies with the feminine gender role, his identification does not have to be complete. He may adopt that role only partially or occasionally, and he may not consider himself female at all. He may only cultivate feminine mannerisms and prefer feminine clothes or feminine occupations. It should further be noted that, in any or all of these cases, he may be heterosexual, ambisexual, or homosexual. In short, the four examples given here are not meant to establish new norms, classifications, or human stereotypes. They
should simply be taken as a hint at the wide range and astonishing variety of human life. We must never forget that each individual person is unique, that few people ever fall into tidy sexual categories, and that there are countless shades and gradations.

Indeed, the very distinction between physical sex, gender role, and sexual orientation can help us to avoid hasty judgments and unwarranted generalizations. It can remind us, for instance, that not every effeminate man is a homosexual, and that not all homosexuals are effeminate. It also makes clear why somebody can think of himself as less than a "real man" when he knows very well that he is male. Finally, it shows us the possible extent and the limitations of a "sex change".

Once we realize how social conditioning influences our development as males and females, we have taken the first step toward understanding the development of our "sexual" behavior. Moreover, we can now make another useful distinction. In the preceding text, we have used the term "sexual orientation" very broadly to indicate an erotic preference for male or female partners. However, most people know that erotic preferences are usually much more specific. For example, a "typical" male is by no means attracted to all females, but only to those of a certain age, height, weight, hair color, etc. In fact, he may prefer not only a special type of female, but a special type of sexual intercourse under special conditions. These particular preferences and tastes within the general framework of a person's sexual orientation are best described as personal sexual interests. They too are the result of conditioning.

It is, of course, true that all human beings are born with the capacity to respond to many kinds of sensual stimulation. We also know that erections of the penis, the lubrication of the vagina, muscular contractions, and rhythmic pelvic movements can be observed in very young infants. In short, nobody has to learn the physiological responses that lead to orgasm. Still, everybody does learn under which specific circumstances these responses may be triggered. From their first years of life, children learn to react positively to certain stimuli and negatively to certain others. As a result of their personal experiences, they then acquire their individual behavior patterns. Thus, as already mentioned, human beings learn to be masculine or feminine, heterosexual or homosexual. They also learn to masturbate, to engage in coitus, and to feel happy or guilty about sex. They learn to prefer younger or older partners, blondes or brunettes, Europeans, Africans, or Asians. Some persons develop a strong attachment to one particular partner and are unable to respond to anyone else; others change their partners frequently. Some like variety in their erotic techniques; others stick to a single approach throughout their lives. Some men and women depend on complete privacy for their sexual responsiveness; others find additional stimulation in the knowledge that they are being watched. There are people whose sexual advances are passionate, inconsiderate, and even brutal, and there are others who enjoy making love slowly, gently, and deliberately. Certain individuals may ever prefer solitary masturbation to any sexual intercourse, and certain others may seek sexual contact with animals.

Since these and many other personal sexual interests, choices, and preferences are developed through learning, they may appear natural, reasonable and, indeed, inevitable to the person involved. Even behavior which seems outrageous, fantastic, meaningless, or absurd to most people may be meaningful and rewarding to a certain individual because of the way in which he has been conditioned. A man who becomes sexually excited at the sight of a wooden horse may merely reflect some early experience in which sexual pleasure was associated with a merry-go-round, and his behavior may be no more difficult to explain than that of another man who becomes aroused while watching a striptease show. The latter response may have a certain advantage over the former, but neither of them should be of any social concern. A great number of people, however, seem to find comfort in the assumption that there is only one right way of doing anything. They take no joy in the infinite variety of human sexual

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behavior, but instead see it as an affront to their sense of stability and order. Such people are always tempted to set up their own preferences as universal norms, and to condemn everybody who disagrees with them.

On the other hand, it is clear that every society has a right to protect itself against sexual acts that involve force or violence, or which take place in front of unwilling witnesses. Such acts may be satisfying to the person who commits them, but since they obviously violate fundamental rights of others, they are socially unacceptable.

Traditionally, they have always been treated as serious crimes which deserved severe punishment. However, in modern times there has been a growing tendency to view such acts as symptoms of mental illness rather than crimes. By the 19th century, psychiatrists began to argue in court that certain sexual offenders should not be sent to prison but to a mental hospital, and that they should not be punished but cured. In support of this argument, numerous attempts were made to classify sexual acts as normal or abnormal, healthy or sick. The best known of these attempts is perhaps that of Richard von Krafft-Ebing, a Viennese psychiatrist. In his book Psychopathia Sexualis (1886), he presented a long list of supposedly pathological sexual interests, for which he invented a number of rather fanciful special terms. Since then many other psychiatrists have followed his example, the lists have grown longer, and the special terms have become even more outlandish and exotic.

Unfortunately, these lists usually do not restrict themselves to socially harmful acts, but include many types of behavior that are merely uncommon, unconventional, or disliked by the writer. Indeed, to this very day studies on "sexual psychopathology" have rarely been more than moralistic tracts in scientific disguise. They are important mainly as historical documents which reflect the sexual standards and moral obsessions of a particular time. (For further details, see "Conformity and Deviance.") Nevertheless, it cannot be denied that some people develop behavior patterns which are unacceptable even to themselves. For example, a man may realize that his sexual acts are harmful to others, but he may have great difficulty controlling himself. In another case, such compulsive behavior may not be antisocial, but since it creates a sense of helplessness in the individual, he may still find it highly disturbing. There are also some men and women who feel guilty and apprehensive about any kind of sexual activity, and some others are so self-conscious and inhibited that their sexual responses are inadequate.

It is fair to say that all of these people are sexually maladjusted. In other words, their particular learning experiences have rendered them incapable of full sexual communication. They either have become insensitive to the needs of others, or are unable to fulfill them. They cannot relate to their sexual partners as complete persons, or adapt their own desires to different circumstances and situations. Instead, they seem condemned to repeat the same frustrating and self-defeating acts. In short, they fail to achieve the full amount of physical and emotional satisfaction of which most human beings are capable.

Module 2

Physiological Basis of Emotion

Nature of Emotion
Joy, sorrow, hope, love, excitement, anger, hate, and many such feelings are experienced in the course of the day by all of us. The term emotion is often considered synonymous with the terms ‘feeling’ and ‘mood’. Feeling denotes the pleasure or pain dimension of emotion, which usually involves bodily functions. Mood is an affective state of long duration but of lesser intensity than emotion. Both these terms are narrower than the concept of emotion. Emotions are a complex pattern of arousal, subjective feeling, and cognitive interpretation. Emotions, as we experience them, move us internally, and this process involves physiological as well as psychological reactions.

Physiological aspects of emotion

‘Divya is desperate to get a job. She has prepared well for the interview and feels confident. As she enters the room and the interview begins, she becomes extremely tense. Her feet go cold, her heart starts pounding, and she is unable to answer appropriately’.

Why did this happen? Try thinking about a similar situation that you have faced sometime in your life. Can you describe probable reasons for this? As we will see, a great deal of physiological changes happen when we experience emotion. When we are excited, afraid or angry, these bodily changes might be relatively easy to note. All of you must have noted the increase in heart rate, throbbing temples, increased perspiration, and trembling in your limbs when you are angry or excited about something. Sophisticated equipment has made it possible to measure the exact physiological changes that accompany emotions. Both autonomic as well as somatic nervous system play important roles in the emotional process. The experience of emotions is a result of a series of neurophysiological activations in which thalamus, hypothalamus, limbic system, and the cerebral cortex are involved significantly. Individuals with extensive injury in these brain areas have been known to demonstrate impaired emotional abilities. Selective activation of different brain areas has been experimentally shown to arouse different emotions in infants and adults.

The neural basis of emotion
The nervous system, central as well as peripheral, plays a vital role in the regulation of emotion.

Role Of the Autonomic Nervous System in Emotion

The visceral system of the Peripheral Nervous System is known as the Autonomic Nervous System. The sensory (afferent) and motor (efferent) nerves connecting the surface of the body with the central nervous system constitute the peripheral nervous system. In other words, rest of the nervous system, other than the brain Autonomic Nervous system.

The nerve fibers of the Autonomic Nervous System are connected with the function of blood vessels, endocrine glands, heart, lungs, stomach, intestines, in and bladders etc. The Autonomic Nervous System is controlled by the old brain, and is not under the functional control of the cortex. The synapse of system is situated outside of both the spinal column and central nervous system. Physiologists have discovered two divisions of the autonomic nervous system, i.e., sympathetic division and parasympathetic division. These two divisions never function together. Either of the two functions at a time.

The Sympathetic Division
Structure:
The spinal nerves emerge from the middle portions of the spinal cord. These spinal nerves emerging on both the sides of the spinal cord run a series of ganglia. Ganglia are the nerve centers present in both the sides of the spinal cord. These nerve fibers run up and down the body synapse with the effect or neurons that go into muscles, glands, skin, and viscera. These fibers coming from thoracolumbar segments of the spinal column finally reach organs from the head to toes - all parts of the body. This part of the autonomic system is called sympathetic nervous division because they make the visceral organs function in 'sympathy' during emergency conditions of serious effort or exercise, states of fear and anger.

Functions:

The sympathetic division acts in three major events, such as during (a) excitement, emotion of fear, anger and elation, (b) violent exercise and bodily activities and (c) extreme cold when the life is endangered.

Owing to the function of the sympathetic division during emotion such as anger and rage, medulla of the adrenal gland pours excess amount of its “adrenaline” secretion to the blood stream. This secretion in the blood stream is associated with strong emotional experiences. This leads to release of stored sugar from the liver into the blood.

There are chemical changes in the blood as a result of which the blood clots easily and quickly. Blood pressure increases, pulse beats become rapid and vigorous. The passages of the lungs enlarge and more air is admitted due to heavy breathing. The pupils of the eyes are dilated and thus more light enters into the eyes. Heavy sweating occurs throughout the body. Palms and hands are full with sweating. The temperature of the skin sometimes rises and at times falls several degrees.

The adrenal medulla also secretes another hormone called "noradrenaline” which constricts the blood vessels at the surface of skin. Bloods are chanalised from stomach and sex organs to the motor organs, such as, muscles of and arms. The digestive functions come to stop. There is cessation of digestive juices due to inhibitive function of the sympathetic division. The blood from these is diverted to the muscles.

Hairs stand on their roots. The adrenaline secreted from adrenal glands expedites the actions and reinforces emergency-facing processes. There is evidence that the thyroids and pituitary glands also secrete hormones during emotion. During joy, the stomach maximum visceral changes, where as in fear and anger, the adrenal functions vigorously. During sorrow, the gall bladder becomes most active. These glandular responses in emotion are adaptive in nature, which means individual becomes able to cope physically with emergency situation.

The visceral activities as well as the neural activities are involving emotion. Almost the total nervous system is involved in emotional response. Electrical responses are also closely associated with the visceral and the activities during emotion. The electrical responses, such as galvanic responses and brain potentials undergo changes during emotion. The autonomic activities energizing sweat glands lead to perspiration, which produces changes in the electrical properties of the skin. The tissues –of the skin generate electromotive force and the electrical resistance of the skin is changed.

The Parasympathetic Division

Structure:
From the two end segments of the spinal cord, i.e., from the upper and the lower segments, the nerves of the parasympathetic division emerge on both the sides. The upper division of the spinal column is called cranial part and the lower segment is called the sacral part. These nerves then pass the rough series of ganglia and reach the visceral organs structures having synapses outside the central nervous system. Thus, parasympathetic division of the autonomic nervous system is situated from the above and below the sympathetic division. This division is thus known “cranio-sacral division' of the autonomic nervous system. Parasympathetic nerves like the sympathetic division reach almost all the organs of the body from head to toes. When sympathetic division is active, the parasympathetic division takes rest and vice-versa. Whether a particular division accelerates or inhabits a particular organ or system depends on the welfare of the organism at that moment depending on the situation.

Functions:

Parasympathetic division is involved in the ordinary vital ions of life. The parasympathetic division maintains the ordinary processes of life. Protection of the eyes from the bright light is the work of this division. The constrictions of the pupils of the eyes are done by this division for protection purpose. It adjusts the lens of the eye for new vision. The construction of food, its digestion and the excretion are done by parasympathetic on. During sexual union more blood supply to the sex organs are made is division. It meets the physiological demands of the body to maintain. It stores up energy in abundance for future use by the sympathetic division during emergency.

But owing to prolonged emotion, if both the divisions of the Autonomic nervous system become overactive that may lead to organic pathology, parasympathetic over activity may lead to peptic ulcer, backache, and headache etc. The sympathetic over activity may lead to psychosomatic diseases, such as asthma, tuberculosis, migraine etc. for which psychosomatic medicines are prescribed by the physicians.

THE LIMBIC SYSTEM

The limbic system is composed of structures in the brain that deal with emotions (such as anger, happiness and fear) as well as memories. This article will address the limbic system, its parts and their functions in the human body. The limbic system is a convenient way of describing several functionally and anatomically interconnected nuclei and cortical structures that are located in the telencephalon and diencephalon. These nuclei serve several functions, however most have to do with control of functions necessary for self preservation and species preservation. They regulate autonomic and endocrine function, particularly in response to emotional stimuli. They set the level of arousal and are involved in motivation and reinforcing behaviors. Additionally, many of these areas are critical to particular types of memory. Some of these regions are closely connected to the olfactory system, since this system is critical to survival of many species.

Areas that are typically included in the limbic system fall into two categories. Some of these are subcortical structures, while many are portions of the cerebral cortex. Cortical regions that are involved in the limbic system include the hippocampus as well as areas of neocortex including the insular cortex, orbital frontal cortex, subcallosal gyrus, cingulate gyrus and parahippocampal gyrus. This cortex has been termed the "limbic lobe" because it makes a rim surrounding the corpus callosum, following the lateral ventricle. Subcortical portions of the limbic system include the olfactory bulb, hypothalamus, amygdala, septal nuclei and some thalamic nuclei, including the anterior nucleus and possibly the dorsomedial nucleus.
One way in which the limbic system has been conceptualized is as the "feeling and reacting brain" that is interposed between the "thinking brain" and the output mechanisms of the nervous system. In this construct, the limbic system is usually under control of the "thinking brain" but obviously can react on its own. Additionally, the limbic system has its input and processing side (the limbic cortex, amygdala and hippocampus) and an output side (the septal nuclei and hypothalamus).

Limbic system commands certain behaviors that are necessary for the survival of all mammals. It gives rise and modulates specific functions that allow the animal to distinguish between the agreeable and the disagreeable. Here specific affective functions are developed, such as the one that induces the females to nurse and protect their toddlers, or the one which induces these animals to develop ludic behaviors (playful moods). Emotions and feelings, like wrath, fright, passion, love, hate, joy and sadness, are mammalian inventions, originated in the limbic system. This system is also responsible for some aspects of personal identity and for important functions related to memory. And, when the superior mammals arrived on the Earth, the third cerebral unit was finally developed: the neopallium or rational brain, a highly complex net of neural cells capable of producing a symbolic language, thus enabling man to exercise skillful intellectual tasks such as reading, writing and performing mathematical calculations. The neopallium is the great generator of ideas or, as expressed by Paul MacLean, "it is the mother of invention and the father of abstractive thought".

The Main Areas Involved with Emotions

It is important to stress that all these structures interconnect intensively and none of them is the sole responsible for any specific emotional state. However, some contribute more than others to this or that kind of emotion. We shall review now, one by one, the best known structures of the limbic system.

Amygdala

A little almond shaped structure, deep inside the antero-inferior region of the temporal lobe, connects with the hippocampus, the septal nuclei, the prefrontal area and the medial dorsal nucleus of the thalamus. These connections make it possible for the amygdala to play its important role on the mediation and control of major affective activities like friendship, love and affection, on the expression of mood and, mainly, on fear, rage and aggression. The amygdala, being the center for identification of danger, is fundamental for self preservation. When triggered, it gives rise to fear and anxiety which lead
the animal into a stage of alertness, getting ready to flight or fight. Experimental destruction of both amygdalas (there are two of them, one in each hemisphere) tames the animal, which becomes sexually non-discriminative, deprived of affection and indifferent to danger. The electrical stimulus of these structures elicits crises of violent aggressively. Humans with marked lesions of the amygdala, loose the affective meaning of the perception of an outside information, like the sight of a well known person. The subject knows, exactly, who the person is, but is not capable to decide whether he likes or dislikes him (or her).

**Hippocampus**

Is particularly involved with memory phenomena, specially with the formation of long-term memory (the one that, sometimes, lasts forever). When both hippocampi (right and left) are destroyed, nothing can be retained in the memory. The subject quickly forgets any recently received message. The intact hippocampus allows the animal to compare the conditions of a present threat with similar past experiences, thus enabling it to choose the best option, in order to guarantee its own survival.

**Thalamus**

Lesion or stimulation of the medial dorsal and anterior nuclei of the thalamus are associated with changes in emotional reactivity. However, the importance of these nuclei on the regulation of emotional behavior, is not due to the thalamus itself, but to the connections of these nuclei with other limbic system structures. The medial dorsal nucleus makes connections with cortical zones of the pre-frontal area and with the hypothalamus. The anterior nuclei connect with the mamillary bodies, and through them, via fornix, with the hippocampus and the cingulate gyrus, thus taking part in the Papez's circuit.

**Hypothalamus**

This structure has ample connections with the other prosencephalic areas and the mesencephalus. Lesions of the hypothalamic nuclei interfere with several vegetative functions and some of the so-called motivated behaviors, like thermal regulation, sexuality, combativeness, hunger and thirst. The hypothalamus is also believed to play a role in emotion. Specifically, its lateral parts seem to be involved with pleasure and rage, while the median part is like to be involved with aversion, displeasure and a tendency to uncontrollable and loud laughing. However, in general terms, the hypothalamus has more to do with the expression (symptomatic manifestations) of emotions than with the genesis of the affective states. When the physical symptoms of emotion appear, the threat they pose returns, via hypothalamus, to the limbic centers and, thence, to the pre-frontal nuclei, increasing anxiety. This negative feed-back mechanism can be so strong as to generate a situation of panic. As it will be seen later on, the knowledge of this phenomenon is very important, for clinical and therapeutic reasons.

**Cingulate gyrus**

It is located in the medial side of the brain between the cingulate sulcus and the corpus callosum (principal fiber bundle connecting the two cerebral hemispheres). There is still much to be learned about this gyrus, but it is already known that its frontal part coordinates smells and sights with pleasant memories of previous emotions. This region also participates in the emotional reaction to pain and in the regulation of aggressive behaviour. Wild animals, submitted to the ablation of the cingulate gyrus (cingulectomy), become totally tamed. The cutting of a single bundle of this gyrus (cingulotomy) reduces pre-existent depression and anxiety levels, by interrupting neural communication across the Papez's circuit.
Brainstem
The brainstem is the region responsible for the "emotional reactions", (indeed, they are just reflex answers) of inferior vertebrates, like reptiles and amphibians. The involved structures are the reticular formation, and the locus coeruleus, a concentrated mass of nor-epinephrine secreting neurons. It is important to stress that, even in humans, these primitive structures remain active, not only as alerting mechanisms, vital for survival, but in the maintenance of the sleep-awake cycle.

Ventral Tegmental Area
In the ventral tegmental area, located in the mesencephalic part of the brain stem, there is a compact group of dopamine-secreting neurons whose axons end in the nucleus accumbens (mesolimbic dopaminergic pathway). The spontaneous firing or the electrical stimulation of neurons belonging to that region produce pleasurable sensations, some of them similar to orgasm. Many people who, for a genetic error, have a reduction of D2 (dopamine) receptors in the accumbens nucleus, become, sooner or later, incapable to obtain gratification from the common pleasures of life. Thus, they seek atypical and noxious "pleasurable" alternatives, like alcoholism, cocaine addiction, impulsive gambling and compulsion for sweet foods. Certain brainstem structures, like the nuclei of the cranial nerves, stimulated by impulses coming from the cortex and the striatum (a subcortical formation), are responsible for the physiognomic: expressions of anger, joy, sadness, tenderness, etc.

Septum
The septal region lies anteriorly to the thalamus. Inside it, one finds the centers of orgasm (four for women and one for men). This area has been associated with different kinds of pleasant sensations, mainly those related to sexual experiences

Prefrontal area
This area comprises the entire non-motor anterior region of the frontal lobe. It underwent a great deal of development during the evolution of mammals. It is specially large in man and in some species of dolphins. It does not belong to the traditional limbic circuit, but its intense bi-directional connections with thalamus, amygdala and other subcortical structures, account for the important role it plays in the genesis and, specially, in the expression of affective states. When the pre-frontal cortex suffers a lesion, the subject loses his sense of social responsibility as well as the capacity for concentration and abstraction. In some cases, although consciousness and some cognitive functions, like speech, remain intact, the subject can no longer solve problems, even the most elementary ones. When pre-frontal lobotomy was used for treatment of certain psychiatric disturbances, the patients entered into a stage of "affective buffer", no longer showing any sign of joy, sadness, hope or despair. In their words or attitudes, no traces of affection could be detected

Related Areas
Besides the hypothalamus, hippocampus, and amygdala, there are other areas in the structures near to the limbic system that are intimately connected to it:

The cingulate gyrus is the part of the cerebrum that lies closest to the limbic system, just above the corpus colossum. It provides a pathway from the thalamus to the hippocampus, seems to be responsible for focusing attention on emotionally significant events, and for associating memories to smells and to pain.
The **ventral tegmental area** of the brain stem (just below the thalamus) consists of dopamine pathways that seem to be responsible for pleasure. People with damage here tend to have difficulty getting pleasure in life, and often turn to alcohol, drugs, sweets, and gambling.

The **basal ganglia** (including the caudate nucleus, the putamen, the globus pallidus, and the substantia nigra) lie over and to the sides of the limbic system, and are tightly connected with the cortex above them. They are responsible for repetitive behaviors, reward experiences, and focusing attention.

The **prefrontal cortex**, which is the part of the frontal lobe which lies in front of the motor area, is also closely linked to the limbic system. Besides apparently being involved in thinking about the future, making plans, and taking action, it also appears to be involved in the same dopamine pathways as the ventral tegmental area, and plays a part in pleasure and addiction.

**AGGRESSIVE BEHAVIOUR**

The most apparent type of aggression is that seen in the interaction between a predator and its prey. An animal defending itself against a predator becomes aggressive in order to survive and to ensure the survival of its offspring. Because aggression against a much larger enemy or group of enemies would lead to the death of an animal, animals have developed a good sense of when they are outnumbered. This ability to gauge the strength of other animals gives animals “fight or flight” response to predators; depending on how strong they gauge the predator to be, animals will either become aggressive or flee.

**Biology**

Aggression is directed to and often originates from outside stimuli, but has a very distinct internal character. Using various techniques and experiments, scientists have been able to explore the relationships between various parts of the body and aggression.

**In the brain**

Many researchers focus on the brain to explain aggression. The areas involved in aggression in mammals include the amygdala, hypothalamus, prefrontal cortex, cingulate cortex, hippocampus, septal nuclei, and periaqueductal gray of the midbrain. Because of the difficulties in determining the intentions of animals, aggression is defined in neuroscience research as behavior directed at an object or animal which results in damage or harm to that object or animal.

In many animals, aggression is encoded by pheromones. In mice, Major urinary proteins (Mups) have been demonstrated to promote innate aggressive behavior in males. Mups were demonstrated to activate olfactory sensory neurons in the vomeronasal organ (VNO), a subsystem of the nose known to detect pheromones via specific sensory receptors, of mice and rats. The hypothalamus and periaqueductal gray of the midbrain are the most critical areas controlling aggression in mammals, as shown in studies on cats, rats, and monkeys. These brain areas control the expression of all the behavioral and autonomic components of aggression in these species, including vocalization. They have direct connections with both the brainstem nuclei controlling these functions and areas such as the amygdala and prefrontal cortex.

Electrical stimulation of the hypothalamus causes aggressive behavior. The hypothalamus expresses receptors that help determine aggression levels based on their interactions with the neurotransmitters serotonin and vasopressin. The amygdala is also critically involved in aggression. Stimulation of the amygdala results in augmented aggressive behavior in hamsters, while lesions of
anevolutionarily homologous area in the lizard greatly reduce competitive drive and aggression (Bauman et al. 2006). Several experiments in attack-primed Syrian Golden Hamsters support the claim of the amygdala being involved in control of aggression. Using expression of c-fos as a neuroanatomically localized marker of activity, the neural circuitry involved in the state of “attack readiness” in attack primed hamsters was studied. The results showed that certain structures of the amygdala were involved in aggressiveness: the medial nucleus and the cortical nuclei showed distinct differences in involvement as compared to other structures such as the lateral and basolateral nuclei and central nucleus of the amygdala, which were not associated with any substantial changes in aggressiveness. In addition, c-fos expression was found most clearly in the most dorsal and caudal aspects of the corticomedial amygdala (CMA). In the same study, it was also shown that lesions of the CMA significantly reduced the number of aggressive behaviors. Eight of eleven subjects failed to attack. Also a correlation between lesion site and attack latency was determined: the more anterior the lesion, the longer mean elapsed time to the aggressive behavior.

The prefrontal cortex (PFC) has been implicated in aggressive psychopathology. Reduced activity of the prefrontal cortex, in particular its medial and orbitofrontal portions, has been associated with violent/antisocial aggression. Specifically, regulation of the levels of the neurotransmitter serotonin in the PFC has been connected with a particular type of pathological aggression, induced by subjecting genetically predisposed, aggressive, wild-type mice to repeated winning experience; the male mice selected from aggressive lines had lower serotonin tissue levels in the PFC than the low-aggressive lines in this study.

**Neurotransmitters and hormones**

Various neurotransmitters and hormones have been shown to correlate with aggressive behavior. The most often mentioned of these is the hormone testosterone. In one source, it was noted that concentration of testosterone most clearly correlated with aggressive responses involving provocation. In adulthood, it is clear that testosterone is not related to any consistent methods of measuring aggression on personality scales, but several studies of the concentration of blood testosterone of convicted male criminals who committed violent crimes compared to males without a criminal record or who committed non-aggressive crimes revealed in most cases that men who were judged aggressive/dominant had higher blood concentrations of testosterone than controls. However, a correlation between testosterone levels and aggression does not prove a causal role for testosterone. Studies of testosterone levels of male athletes before and after a competition revealed that testosterone levels rise shortly before their matches, as if in anticipation of the competition, and depend on the outcome of the event: testosterone levels of winners are higher relative to those of losers. Interestingly, testosterone levels in female criminals versus females without a criminal record mirror those of males: testosterone levels are higher in women who commit aggressive crimes or are deemed aggressive by their peers than non-aggressive females. However, no specific response of testosterone levels to competition was observed in female athletes, although a mood difference was noted. Testosterone has been shown to correlate with aggressive behavior in mice and in some humans, but in contrast to some long-standing theories, various experiments have failed to find a relationship between testosterone levels and aggression in humans. The possible correlation between testosterone and aggression could explain the “roid rage” that can result from anabolic steroid use, although an effect of abnormally high levels of steroids does not prove an effect at physiological levels.

Another line of research has focused more on the effects of circulating testosterone on the nervous system mediated by local metabolism within the brain. Testosterone can be metabolized to 17b-estradiol by the enzyme aromatase or to 5α-dihydrotestosterone by 5α-reductase. Aromatase is highly
expressed in regions involved in the regulation of aggressive behavior, such as the amygdala and hypothalamus. In studies using genetic knock out techniques in inbred mice, male mice that lacked a functional aromatase enzyme displayed a marked reduction in aggression. Long-term treatment of these mice with estradiol partially restored aggressive behavior, suggesting that the neural conversion of circulating testosterone to estradiol and its effect on estrogen receptors affects inter-male aggression. Also, two different estrogen receptors, ERα and ERβ, have been identified as having the ability to exert different effects on aggression. In studies using estrogen receptor knockout mice, individuals lacking a functional ERα displayed markedly reduced inter-male aggression while male mice that lacked a functional ERβ exhibited normal or slightly elevated levels of aggressive behavior. These results imply that ERα facilitates male-male aggression, whereas ERβ may inhibit aggression. However, different strains of mice show the opposite pattern in that aromatase activity is negatively correlated with aggressive behavior. Also, in a different strain of mice the behavioral effect of estradiol is dependent on day length: under long-days (16h of light) estradiol reduces aggression, and under short-days (8h of light) estradiol rapidly increases aggression.

**Glucocorticoids** also play an important role in regulating aggressive behavior. In adult rats, acute injections of corticosterone promote aggressive behavior and acute reduction of corticosterone decreases aggression; however, a chronic reduction of corticosterone levels can produce abnormally aggressive behavior. In addition, glucocorticoids affect development of aggression and establishment of social hierarchies. Adult mice with low baseline levels of corticosterone are more likely to become dominant than are mice with high baseline corticosterone levels.

**Dehydroepiandrosterone (DHEA)** is the most abundant circulating androgen and can be rapidly metabolized within target tissues into potent androgens and estrogens. Gonadal steroids generally regulate aggression during the breeding season, but non-gonadal steroids may regulate aggression during the non-breeding season. Castration of various species in the non-breeding season has no effect on territorial aggression. In several avian studies, circulating DHEA has been found to be elevated in birds during the non-breeding season. These data support the idea that non-breeding birds combine adrenal and/or gonadal DHEA metabolism to maintain territorial behavior when gonadal testosterone secretion is low. Similar results have been found in studies involving different strains of rats, mice, and hamsters. DHEA levels also have been studied in humans and may play a role in human aggression. Circulating DHEAS (its sulfated ester) levels rise during adrenarche (~7 years of age) while plasma testosterone levels are relatively low. This implies that aggression in pre-pubertal children with aggressive conduct disorder might be correlated with plasma DHEAS rather than plasma testosterone, suggesting an important link between DHEAS and human aggressive behavior.

Another chemical messenger with implications for aggression is the neurotransmitter serotonin. In various experiments, serotonin action was shown to be negatively correlated with aggression (Delville et al. 1997). This correlation with aggression helps to explain the aggression-reducing effects of selective serotonin reuptake inhibitors such as fluoxetine (Delville et al. 1997), aka prozac. While serotonin and testosterone have been the two most researched chemical messengers with regards to aggression, other neurotransmitters and hormones have been shown to relate to aggressive behavior as well. The neurotransmitter vasopressin causes an increase in aggressive behavior when present in large amounts in the anterior hypothalamus (Delville et al. 1997). The effects of norepinephrine, cortisol, and other neurotransmitters are still being studied.
In a nonmammilian example, the fruitless gene in Drosophila melanogaster is a critical determinant for how fruit flies fight. Patterns of aggression can be switched, with males using female patterns of aggression or females using male patterns, by manipulating either the fruitless or transformer genes in the brain. Candidate genes for differentiating aggression between the sexes are the Sry (sex determining region Y) gene, located on the Y chromosome and the Sts (steroid sulfatase) gene. The Sts gene encodes the steroid sulfatase enzyme, which is pivotal in the regulation of neurosteroid biosynthesis. It is expressed in both sexes, is correlated with levels of aggression among male mice, and increases dramatically in females after parturition and during lactation, corresponding to the onset of maternal aggression.

Gender

Gender is a factor that plays a role in both human and animal aggression. Males are historically believed to be generally more physically aggressive than females (Coie & Dodge 1997, Maccoby & Jacklin 1974), and men commit the vast majority of murders (Buss 2005). This is one of the most robust and reliable behavioral sex differences, and it has been found across many different age groups and cultures. There is evidence that males are quicker to aggression (Frey et al. 2003) and more likely than females to express their aggression physically (Bjorkqvist et al. 1994). When considering indirect forms of non-violent aggression, such as relational aggression and social rejection, some scientists argue that females can be quite aggressive although female aggression is rarely expressed physically (Archer, 2004; Card, Stucky, Sawalani, & Little, 2008).

Although females are less likely to initiate physical violence, they can express aggression by using a variety of non-physical means. Exactly which method women use to express aggression is something that varies from culture to culture. On Bellona Island, a culture based on male dominance and physical violence, women tend to get into conflicts with other women more frequently than with men. When in conflict with males, instead of using physical means, they make up songs mocking the man, which spread across the island and humiliate him. If a woman wanted to kill a man, she would either convince her male relatives to kill him or hire an assassin. Although these two methods involve physical violence, both are forms of indirect aggression, since the aggressor herself avoids getting directly involved or putting herself in immediate physical danger.
Module 3

Learning, Memory and Localization of Higher Order Function

NERVOUS SYSTEM

The nervous system is our processing system, and the system that keeps us in contact with the outside world. It tells us that we exist, and along with the muscles allows us to move and react to stimuli. Our consciousness resides in our nervous systems, as do our thoughts and emotions. In short, the roles of the nervous system are: responsible for coordination of movement, response to environmental stimuli, intelligence, self-awareness, thought, and emotion. It composed of nerve cells called neurons, which are specialized to carry nerve impulses.

Nervous system has two major divisions: (the division is arbitrary; the two systems work together and are connected to one another). The two systems are:

1. Central Nervous System: (CNS) - includes spinal cord and brain. In the "center" of the body.
2. Peripheral Nervous System: (PNS) - the rest of the nervous system: PNS is further divided into the Somatic Nervous System (connects to skeletal muscle) and Autonomic Nervous System (connects to smooth (involuntary) muscles). The Autonomic Nervous System is further divided into the Sympathetic Nervous System (usually causes effects associated with emergency situations) and the Parasympathetic Nervous System (promotes activities associated with a normal state).

![Diagram of the Human Nervous System]

Learning and Nervous System
Neuroscience, also known as Neural Science, is the study of how the nervous system develops, its structure, and what it does. Neuroscientists focus on the brain and its impact on behaviour and cognitive functions. Not only is neuroscience concerned with the normal functioning of the nervous system, but also what happens to the nervous system when people have neurological, psychiatric and neurodevelopmental disorders. The nervous system and the brain are the physical foundation of the human learning process. Neuroscience links our observations about cognitive behaviour with the actual physical processes that support such behaviour. This theory is still “young” and is undergoing rapid, controversial development.

Anatomy of the Brain

The brain consists of the brain stem and the cerebral hemispheres. The brain stem is divided into hind-brain, mid-brain and a ‘between-brain’ called the diencephalon. The hind-brain is an extension of the spinal cord. It contains networks of neurons that constitute centres for the control of vital functions such as breathing and blood pressure. Within these are networks of neurons whose activity controls these functions. Arising from the roof of the hind-brain is the cerebellum, which plays an absolutely central role in the control and timing of movements. The midbrain contains groups of neurons, each of which seems to use predominantly a particular type of chemical messenger, but all of which project up to cerebral hemispheres. It is thought that these can modulate the activity of neurons in the higher centres of the brain to mediate such functions as sleep, attention or reward. The diencephalon is divided into two very different areas called the thalamus and the hypothalamus: The thalamus relays impulses from all sensory systems to the cerebral cortex, which in turn sends messages back to the thalamus. This back-and-forward aspect of connectivity in the brain is intriguing - information doesn’t just travel one way. The hypothalamus controls functions such as eating and drinking, and it also regulates the release of hormones involved in sexual functions. The cerebral hemispheres consist of a core, the basal ganglia, and an extensive but thin surrounding sheet of neurons making up the grey matter of the cerebral cortex.

The basal ganglia play a central role in the initiation and control of movement. Packed into the limited space of the skull, the cerebral cortex is thrown into folds that weave in and out to enable a much larger surface area for the sheet of neurons than would otherwise be possible. This cortical tissue is the most highly developed area of the brain in humans - four times bigger than in gorillas. It is divided into a large number of discrete areas, each distinguishable in terms of its layers and connections. The functions of many of these areas are known - such as the visual, auditory, and olfactory areas, the sensory areas receiving from the skin (called the somaesthetic areas) and various motor areas. The pathways from the sensory receptors to the cortex and from cortex to the muscles cross over from one side to the other. Thus movements of the right side of the body are controlled by the left side of the cortex (and vice versa).

Similarly, the left half of the body sends sensory signals to the right hemisphere such that, for example, sounds in the left ear mainly reach the right cortex. However, the two halves of the brain do not work in isolation - for the left and right cerebral cortex are connected by a large fibre tract called the corpus callosum. The cerebral cortex is required for voluntary actions, language, speech and higher functions such as thinking and remembering. Many of these functions are carried out by both sides of the brain, but some are largely lateralised to one cerebral hemisphere or the other. Areas concerned with some of these higher functions, such as speech (which is lateralised in the left hemisphere in most people), have been identified.

Cortex and learning

PHYSIOLOGICAL PSYCHOLOGY
The cerebral cortex is the seat of our highest forms of intelligence, and its understanding is thus a goal for all students of mind and brain. Neocortex has an intricate design which exhibits a characteristic organization into six distinct cortical layers (Brodmann, 1909; Martin, 1989). Differences in the thickness of these layers and the sizes and shapes of neurons led the German anatomist Korbinian Brodmann to identify more than fifty divisions, or areas, of neocortex. This classification has been invaluable to later scientists as a basis for discerning different functional roles for different parts of the brain. On the other hand, why the neocortex has six layers, or indeed a laminar design, has remained a mystery from a functional point of view. The present article proposes a model that provides clear functional roles for these layers for purposes of visual perception, and suggests that similar functional roles may be at work in all sensory and cognitive processing.

Linking cortical anatomy to behaviour cannot be done without a sufficiently powerful method. This is true because cortical organization exhibits multiple scales of processing, including individual neurons within the various layers, neural circuits that link these neurons within and between these layers, functional columns that are defined through these interlaminar interactions, cortical maps that are defined by the global organization of these columns within a cortical area, and thalamocortical and corticocortical interactions that occur between different thalamic and cortical areas. These cortical interactions, moreover, occur both bottom-up, from more peripheral to more central areas, and top-down, from more central to more peripheral areas, and have a characteristic laminar organization of their own.

In order to make functional sense of such complex interactions, one needs to be able to link cortical organization to the behaviours that it controls; one needs to show how these designs lead to useful behavioural properties that have been selected and maintained through evolution.

Despite a need for rule learning in everyday life, the brain regions involved inexplicit rule induction remain undetermined. Here we use event-related functional magnetic resonance imaging to measure learning-dependent neuronal responses during an explicit categorization task. Subjects made category decisions, with feedback, to exemplar letter strings for which the rule governing category membership was periodically changed. Bilateral fronto-polar prefrontal cortices were selectively engaged following rule change. This activation pattern declined with improving task performance reflecting rule acquisition. The vocabulary of letters comprising the exemplars was also periodically changed, independently of rule changes. This exemplar change modulated activation in left anterior hippocampus. Our finding that fronto-polar cortex mediates rule learning supports a functional contribution of this region to generic reasoning and problem-solving behaviours.

**Hippocampus and Learning**

The hippocampus is a small organ located within the brain's medial temporal lobe and forms an important part of the limbic system, the region that regulates emotions. The hippocampus is associated mainly with memory, in particular long-term memory. The organ also plays an important role in spatial navigation. Damage to the hippocampus can lead to loss of memory and difficulty in establishing new memories. In Alzheimer's disease, the hippocampus is one of the first regions of the brain to be affected, leading to the confusion and loss of memory so commonly seen in the early stages of the disease.

The major functions of the hippocampus include:

**Memory**

Historically, the link between the hippocampus and long-term memory formation was first described by William Scoville and Brenda Milner who reported what happened to an epileptic individual who underwent surgery on the organ that was intended to relieve his seizures.
The patient had severe amnesia after the procedure as well as an inability to form new memories of events such as when or where a situation occurred (termed episodic memory). The only memories he did retain were those from many years earlier, as far back as childhood.

Experts generally agree that the hippocampus plays a role in the formation of new memories and in the detection of new surroundings, occurrences and stimuli. Some also believe the organ is involved in declarative memory; that is memories that can be stated verbally such as facts and figures. However, studies have shown that damage to the hippocampus does not affect a person's ability to learn a new skill such as playing a musical instrument or solving certain types of puzzles which suggests that the memories involved in learning a procedure are governed by brain areas other than the hippocampus.

**Spatial navigation and spatial memory**

Neuroscientist John O'Keefe and psychology professor Lynn Nadel studied the involvement of the hippocampus in memory formation and learning behaviors in the 1960's and 1970's. Together, they wrote the landmark 1978 book "The Hippocampus as a Cognitive map," which outlines the role of the hippocampus in learning and storing information referring to portions of space, in the form of cognitive maps.

**Behavioural inhibition**

Animal experiments investigating the effects of hippocampal damage have previously suggested that, firstly, the damage causes hyperactivity and, secondly, that it affects the ability to inhibit responses that have previously been learnt.

**Synaptic Plasticity**

Historically, it was generally thought that the role of the synapse was to simply transfer information between one neuron and another neuron or between a neuron and a muscle cell. In addition, it was thought that these connections, once established during development, were relatively fixed in their strength, much like a solder joint between two electronic components. One exciting development in neurobiology over the past forty years is the realization that most synapses are extremely plastic; they are able to change their strength as a result of either their own activity or through activity in another pathway. Many think that this synaptic plasticity is central to understanding the mechanisms of learning and memory.

There are two general forms of synaptic plasticity, intrinsic and extrinsic. Intrinsic mechanisms, also known as homosynaptic mechanisms, refer to changes in the strength of a synapse that are brought about by its own activity. (Homo from the Greek meaning the same.) Extrinsic plasticity, or heterosynaptic plasticity, is a change in the strength of a synapse brought about by activity in another pathway. Abstract models of synaptic plasticity demonstrate how the concept of synaptic plasticity can contribute to different forms of learning, memory and development and how this might contribute to machine learning. Biophysical models of synaptic plasticity are based on actual cellular and molecular mechanisms observed in neurons and demonstrate how synaptic plasticity can arise from real biological mechanisms.

**Hebbian theory**

It is a theory in neuroscience that proposes an explanation for the adaptation of neurons in the brain during the learning process. It describes a basic mechanism for synaptic plasticity, where an increase in synaptic efficacy arises from the presynaptic cell's repeated and persistent stimulation of the postsynaptic cell. Introduced by Donald Hebb in his 1949 book *The Organization of Behavior*, the theory is also called *Hebb's rule, Hebb's postulate, and cell assembly theory*. Hebb states it as follows:
“Let us assume that the persistence or repetition of a reverberatory activity (or "trace") tends to induce lasting cellular changes that add to its stability. When an axon of cell A is near enough to excite a cell B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A’s efficiency, as one of the cells firing B, is increased.”

The theory is often summarized as "cells that fire together, wire together", although this is an oversimplification of the nervous system not to be taken literally, as well as not accurately representing Hebb's original statement on cell connectivity strength changes. The theory is commonly evoked to explain some types of associative learning in which simultaneous activation of cells leads to pronounced increases in synaptic strength. Such learning is known as Hebbian learning.

**Hebbian engrams and cell assembly theory**

Hebbian theory concerns how neurons might connect themselves to become engrams. Hebb's theories on the form and function of cell assemblies can be understood from the following:

"The general idea is an old one, that any two cells or systems of cells that are repeatedly active at the same time will tend to become ‘associated’, so that activity in one facilitates activity in the other.""When one cell repeatedly assists in firing another, the axon of the first cell develops synaptic knobs."

Gordon Allport posits additional ideas regarding cell assembly theory and its role informing engrams, along the lines of the concept of auto-association, described as follows:

"If the inputs to a system cause the same pattern of activity to occur repeatedly, the set of active elements constituting that pattern will become increasingly strongly interassociated. That is, each element will tend to turn on every other element and (with negative weights) to turn off the elements that do not form the part of the pattern. To put it another way, the pattern as a whole will become ‘auto associated’ we may called a learned (auto associated) pattern an engram.

Hebbian theory has been the primary basis for the conventional view that when analysed from a holistic level, engrams are neuronal nets or neural networks. Work in the laboratory of Eric Kandel has provided evidence for the involvement of Hebbian learning mechanisms at synapses in the marine gastropod Aplysiacalifornica.

Experiments on Hebbian synapse modification mechanisms at the central nervous system synapses of vertebrates are much more difficult to control than are experiments with the relatively simple peripheral nervous system synapses studied in marine invertebrates. Much of the work on long-lasting synaptic changes between vertebrate neurons (such as long-term potentiation) involves the use of non-physiological experimental stimulation of brain cells. However, some of the physiologically relevant synapse modification mechanisms that have been studied in vertebrate brains do seem to be examples of Hebbian processes. One such study reviews results from experiments that indicate that long-lasting changes in synaptic strengths can be induced by physiologically relevant synaptic activity working through both Hebbian and non-Hebbian mechanisms.

**MEMORY CONSOLIDATION**

Memory consolidation refers to the process by which a temporary, labile memory is transformed into a more stable, long-lasting form. Memory consolidation was first proposed in 1900 (Muller and
Pilzecker 1900; Lechner et al. 1999) to account for the phenomenon of retroactive interference in humans, that is, the finding that learned material remains vulnerable to interference for a period of time after learning. Support for consolidation was already available in the facts of retrograde amnesia, especially as outlined in the earlier writings of Ribot (1881). The key observation was that recent memories are more vulnerable to injury or disease than remote memories, and the significance of this finding for consolidation was immediately appreciated.

In normal memory a process of organization is continually going on a physical process of organization and a psychological process of repetition and association. In order that ideas may become a part of permanent memory, time must elapse for these processes of organization to be completed.

Consolidation is the processes of stabilizing a memory trace after the initial acquisition. It may perhaps be thought of part of the process of encoding or of storage, or it may be considered as a memory process in its own right. It is usually considered to consist of two specific processes, synaptic consolidation (which occurs within the first few hours after learning or encoding) and system consolidation (where hippocampus-dependent memories become independent of the hippocampus over a period of weeks to years).

History

Memory consolidation was first referred to in the writings of the renowned Romanteacher of rhetoric Quintillian. He noted the “curious fact... that the interval of a single night will greatly increase the strength of the memory,” and presented the possibility that “… the power of recollection ... undergoes a process of ripening and maturing during the time which intervenes.” The process of consolidation was later proposed based on clinical data illustrated in 1882 by Ribot’s Law of Regression, “progressive destruction advances progressively from the unstable to the stable”. This idea was elaborated on by William H. Burnham a few years later in a paper on amnesia integrating findings from experimental psychology and neurology. Coining of the term “consolidation” is credited to the German researchers Müller and Alfons Pilzecker who rediscovered the concept that memory takes time to fixate or undergo “Konsolidierung” in their studies conducted between 1892 and 1900.

Systematic studies of retrograde amnesia started to emerge in the 1960s and 1970s. These were accompanied by the creation of animal models of human amnesia in an effort to identify brain substrates critical for slow consolidation. Meanwhile, neuropharmacological studies of selected brain areas began to shed light on the molecules possibly responsible for fast consolidation. In recent decades, advancements in cellular preparations, molecular biology, and neurogenetics have revolutionized the study of consolidation.

Synaptic Consolidation

Synaptic consolidation is one form of memory consolidation seen across all species and long-term memory tasks. Long-term memory, when discussed in the arena of synaptic consolidation, is memory that lasts for at least 24 hours. An exception to this 24-hour rule is long-term potentiation, or LTP, a model of synaptic plasticity related to learning, in which an hour is thought to be sufficient. Synaptic consolidation is achieved faster than systems consolidation, within only minutes to hours of learning. LTP, one of the best understood forms of synaptic plasticity, is thought to be a possible underlying process in synaptic consolidation.

Standard Model
The standard model of synaptic consolidation suggests that alterations of synaptic protein synthesis and changes in membrane potential are achieved through activating intracellular transduction cascades. These molecular cascades trigger transcription factors that lead to changes in gene expression. The result of the gene expression is the lasting alteration of synaptic proteins, as well as synaptic remodeling and growth. In a short time-frame immediately following learning, the molecular cascade, expression and process of both transcription factors and immediate early genes, are susceptible to disruptions. Disruptions caused by specific drugs, antibodies and gross physical trauma can block the effects of synaptic consolidation.

**Long-term Potentiation**

LTP can be thought of as the prolonged strengthening of synaptic transmission, and is known to produce increases in the neurotransmitter production and receptor sensitivity, lasting minutes to even days. The process of LTP is regarded as a contributing factor to synaptic plasticity and in the growth of synaptic strength, which are suggested to underlie memory formation. LTP is also considered to be an important mechanism in terms of maintaining memories within brain regions, and therefore is thought to be involved in learning. There is compelling evidence that LTP is critical for Pavlovian fear conditioning in rats suggesting that it mediates learning and memory in mammals. Specifically, NMDA-receptor antagonists appear to block the induction of both LTP and fear conditioning and that fear conditioning increases amygdaloidal synaptic transmission that would result in LTP.

**Timeline of Consolidation**

Synaptic consolidation, when compared to systems consolidation; which is said to take weeks to months to years to be accomplished, is considerably faster. There is evidence to suggest that synaptic consolidation takes place within minutes to hours of memory encoding or learning, and as such is considered the ‘fast’ type of consolidation. As soon as six hours after training, memories become impervious to interferences that disrupt synaptic consolidation and the formation of long-term memory.

**Spacing Effect**

Distributed learning has been found to enhance memory consolidation, specifically for relational memory. Experimental results suggest that distributing learning over the course of 24 hours decreases the rate of forgetting compared to massed learning, and enhances relational memory consolidation. When interpreted in the context of synaptic consolidation, mechanisms of synaptic strengthening may depend on the spacing of memory reactivation to allow sufficient time for protein synthesis to occur, and thereby strengthen long-term memory.

**Protein Synthesis**

Protein synthesis has been suggested to play a critical role in the formation of new memories. Studies have shown that protein synthesis inhibitors administered after learning, weaken memory, suggesting that protein synthesis is required for memory consolidation. Additionally, reports have suggested that the effects of proteinsynthesis inhibitors also inhibit LTP. However, it should be noted that other results have shown that protein synthesis may not in fact be necessary for memory consolidation, as it has been found that the formation of memories can withstand vast amounts of protein synthesis inhibition, suggesting that this criterion of proteinsynthesis as necessary for memory consolidation is not unconditional.
Dietary Flavanoids

There is evidence to suggest that dietary flavanoids have effects on encouraging LTP and synaptic plasticity, therefore affecting memory. Specifically, it was found that dietary-derived flavanoids might protect neurons, enhance neuronal function, and stimulate neuronal regeneration. Additionally, these dietary phytochemicals interact with several neuronal signaling cascade pathways that are responsible for alterations in LTP, and consequently, learning and human memory. Flavanoids may trigger certain events, including the activation of the CREB transcription factor, which is important to the enhancement of short-term and long-term memory. This activation then triggers the synthesis of important proteins related to LTP, ultimately leading to synapse growth and eventually long-term memory.

System Consolidation

System Consolidation is the second form of memory consolidation. It is a reorganization process in which memories from the hippocampal region where memories are first encoded are moved to the neo-cortex in a more permanent form of storage. System consolidation is a slow dynamic process that can take from one to two decades to be fully formed in humans, unlike synaptic consolidation that only takes minutes to hours for new information to stabilize into memories.

Standard Model

The Standard model of systems consolidation was first developed by Paul W. Frankland; it states that when novel information is originally encoded and registered, memory of these new stimuli becomes retained in both the hippocampus and cortical regions. Later the hippocampus’ representations of this information become active in explicit memory is retained in the hippocampus for up to one week after initial learning, representing the hippocampus-dependent stage. During this stage the hippocampus ‘teaching’ the cortex more and more about the information and when the information is recalled it strengthens the cortico-cortical connection thus making the memory hippocampus-independent. Therefore from one week and beyond their initial training experience, the memory is slowly transferred to the neo-cortex where it becomes permanently stored.

Semantic vs. Episodic Memory

Long Term Memory is often divided into two further main types: explicit (or declarative) memory and implicit (or procedural) memory. Declarative memory (“knowing what”) is memory of facts and events, and refers to those memories that can be consciously recalled (or "declared"). It is sometimes called explicit memory, since it consists of information that is explicitly stored and retrieved, although it is more properly a subset of explicit memory. Declarative memory can be further sub-divided into episodic memory and semantic memory.

Nadel and Moscovitch argued that when studying the structures and systems involved in memory consolidation, semantic memory and episodic memory need to be treated as different types of memory. This additional distinction expands the Standard Model by Frankland, which does not consider the types of memory as separate. Evidence from extensive neuro-imaging research on the different function of cortical and hippocampus memory traces have found that the hippocampus provides temporal and spatial context, whereas the cortical traces are primarily context-free. Episodic memory, no matter whether new or old, relies on hippocampus-cortical networks whereas remote semantic memories can be retrieved independent of the hippocampus.

Multiple trace theory (MTT)
It is a memory consolidation model advanced as an alternative model to strength theory. It posits that each time some information is presented to a person, it is neurally encoded in a unique memory trace composed of a combination of its attributes. Further support for this theory came in the 1960s from empirical findings that people could remember specific attributes about an object without remembering the object itself. The mode in which the information is presented and subsequently encoded can be flexibly incorporated into the model. This memory trace is unique from all others resembling it due to differences in some aspects of the item's attributes, and all memory traces incorporated since birth are combined into a multiple-trace representation in the brain. In memory research, a mathematical formulation of this theory can successfully explain empirical phenomena observed in recognition and recall tasks.

**REM sleep**

Rapid eye movement (REM) sleep has been implicated in the overnight learning in humans by the re-organization of novel information in the hippocampal and cortical regions of the brain. REM sleep elicits an increase in neuronal activity following an enriched or novel waking experience, thus increasing neuronal plasticity and therefore playing an essential role in the consolidation of memories. In particular studies have been done on sensory and motor-related tasks. In one study testing finger-tapping, people were split into two groups and tested post-training with or without intervening sleep; results concluded that sleep post-training increases both speed and accuracy in this particular task, while increasing the activation of both cortical and hippocampal regions; whereas the post-training awake group had no such improvements.

**Zif268 & REM Sleep**

Zif268 is an Immediate Early Gene (IEG) thought to be involved in neuroplasticity by an up-regulation of the transcription factor during REM sleep after pre-exposure to an enriched environment. Results from studies testing the effects of zif268 on mice brains postmortem, suggest that a waking experience prior to sleep can have a enduring effect in the brain, due to an increase of neuroplasticity.

**MEMORY RE-CONSOLIDATION**

Memory re-consolidation is the process of previously consolidated memories being recalled and then actively consolidated all over again, in order to maintain, strengthen and modify memories that are already stored in the long-term memory. Several retrievals of memory (either naturally through reflection, or through deliberate recall) may be needed for long-term memories to last for many years, depending on the depth of the initial processing. However, these individual retrievals can take place at increasing intervals, in accordance with the principle of spaced repetition (this is familiar to us in the way that “cramming” the night before an exam is not as effective as studying at intervals over a much longer span of time).

**Distinctions from Consolidation**

Questions arose if reconsolidation was a unique process or merely another phase of consolidation. Both consolidation and reconsolidation can be disrupted by pharmacological agents (e.g. the protein synthesis inhibitor anisomycin) and both require the transcription factor CREB. However, recent amygdala research suggests that BDNF is required for consolidation (but not reconsolidation) whereas the transcription factor and immediate early gene Zif268 is required for reconsolidation but not
consolidation. A similar double dissociation between Zif268 for reconsolidation and BDNF for consolidation was found in the hippocampus for fear conditioning. However not all memory tasks show this double dissociation, such as object recognition memory.

MEMORY IN BRAIN DAMAGED INDIVIDUALS

Why does brain damage impair memory? Object recognition is thought to be the canonical test of declarative memory, the type of memory putatively impaired after damage to the temporal lobes. Studies of object recognition memory have helped elucidate the specific anatomical structures involved in declarative memory implicating, in particular, the perirhinal cortex (Zola-Morgan et al., 1989b; Gaffan and Murray, 1992; Meunier et al., 1993; Mumby and Pinel, 1994; Aggleton et al., 1997; Baxter and Murray, 2001; Málková et al., 2001; Winters et al., 2004). Furthermore, electrophysiological data have identified properties of neurons that seem likely to form part of the mechanism underlying recognition memory (Brown and Aggleton, 2001). However, no full mechanistic account has been provided that explains why impairments after damage to perirhinal cortex should be exacerbated not only by lengthening the delay between presentation of to-be-remembered items and test (Meunier et al., 1993; Mumby and Pinel, 1994) but also by lengthening the list of to-be-remembered items (Meunier et al., 1993), or why such impairments are only revealed when stimuli are trial unique rather than repeatedly presented (Eacott et al., 1994).

Object recognition is the canonical test of declarative memory, the type of memory putatively impaired after damage to the temporal lobes. Studies of object recognition memory have helped elucidate the anatomical structures involved in declarative memory, indicating a critical role for perirhinal cortex. We offer a mechanistic account of the effects of perirhinal cortex damage on object recognition memory, based on the assumption that perirhinal cortex stores representations of the conjunctions of visual features possessed by complex objects. Such representations are proposed to play an important role in memory when it is difficult to solve a task using representations of only individual visual features of stimuli, thought to be stored in regions of the ventral visual stream caudal to perirhinal cortex. The account is instantiated in a connectionist model, in which development of object representations with visual experience provides a mechanism for judgment of previous occurrence. We present simulations addressing the following empirical findings: (1) that impairments after damage to perirhinal cortex (modeled by removing the "perirhinal cortex" layer of the network) are exacerbated by lengthening the delay between presentation of to-be-remembered items and test, (2) that such impairments are also exacerbated by lengthening the list of to-be-remembered items, and (3) that impairments are revealed only when stimuli are trial unique rather than repeatedly presented. This study shows that it may be possible to account for object recognition impairments after damage to perirhinal cortex within a hierarchical, representational framework, in which complex conjunctive representations in perirhinal cortex play a critical role.

Any brain function can be disrupted by brain trauma resulting in inattention, difficulty concentrating, excessive sleepiness, faulty judgment, depression, irritability, emotional outbursts, and slowed thinking. However, memory loss is one of the most common cognitive side effects of traumatic brain injury (TBI). Even in mild TBI, memory loss is still very common. The more severe the victim's memory loss after the TBI, the more significant the brain damage will most likely be.

Some TBI-related amnesia such as patients unable to recall what happened just before, during and after the head injury is temporary. Temporary memory loss is often caused by swelling of the brain in response to the damage it sustained. But because the brain is pressed against the skull, even parts that were not injured are still not able to work. The patient's memory typically returns as the swelling goes
down over a period of weeks or even months. Temporary memory loss may also be an emotional response to the stressful events surrounding a TBI.

Damage to the nerves and axons (connection between nerves) of the brain may also result in memory loss. The brain cannot heal itself like an arm or a leg, so any function that is damaged during a TBI is permanently impaired unless the brain learns how to perform that function differently. Fixed amnesia may include the loss of meanings of certain common, everyday objects or words, or a person may not remember skills he had before the TBI.

A different kind of memory loss is called anteretrograde amnesia, which is an inability to form memories of events that happened after the injury. Doctors are not sure, exactly, why this happens, but some research has shown that it may have something to do with the fact that TBI's reduce the levels of a protein in the brain that helps the brain balance its activity. Without enough of that particular protein, the brain can easily overload and memory formation is affected.

In general, symptoms of brain injury should lessen over time as the brain heals but sometimes the symptoms worsen because the patient's inability to adapt to the brain injury. It is not uncommon for psychological symptoms to arise and worsen after a brain injury.

At the current time, there is no treatment for memory loss following TBI; if the memory does not come back on its own, it will be lost permanently. There is a great deal of research in the field of TBI and memory loss, but, sadly, there are no cures for TBI-related amnesia at this time.

**Forgetting**

It is the apparent loss or modification of information already encoded and stored in an individual's long term memory. It is a spontaneous or gradual process in which old memories are unable to be recalled from memory storage. Forgetting also helps to reconcile the storage of new information with old knowledge. Problems with remembering, learning and retaining new information are a few of the most common complaints of older adults. Memory performance is usually related to the active functioning of three stages. These three stages are encoding, storage and retrieval. Many different factors influence the actual process of forgetting. An example of one of these factors could be the amount of time the new information is stored in the memory. Events involved with forgetting can happen either before or after the actual memory process. The amount of time the information is stored in the memory, depending on the minutes hours or even days, can increase or decrease depending on how well the information is encoded. Studies show that retention improves with increased rehearsal. This improvement occurs because rehearsal helps to transfer information into long term memory-practice makes perfect.

Forgetting information from short term memory (STM) can be explained using the theories of trace decay and displacement. Forgetting from long term memory (LTM) can be explained using the theories of interference and lack of consolidation.

**Trace Decay Theory of Forgetting**

This explanation of forgetting in short term memory assumes that memories leave a trace in the brain. A trace is some form of physical and/or chemical change in the nervous system. Trace decay theory states that forgetting occurs as a result of the automatic decay or fading of the memory trace. Trace decay theory focuses on time and the limited duration of short term memory. This theory suggests short term memory can only hold information for between 15 and 30 seconds unless it is rehearsed. After this time the information / trace decays and fades away.

**Displacement theory**

It provides a very simple explanation of forgetting. Because of its limited capacity, suggested by Miller to be 7+/− 2 items, STM can only hold small amounts of information. When STM is 'full', new
information displaces or ‘pushes out’ old information and takes its place. The old information which is
displaced is forgotten in STM. It was also assumed that the information that had been in the short-term
store for the longest was the first to be displaced by new information, similar to the way in which boxes
might fail off the end of a conveyor belt - as new boxes are put on one end, the boxes which have been
on the conveyor belt the longest drop off the end.

**Interference Theory**

If you had asked psychologists during the 1930s, 1940s, or 1950s what caused forgetting you
would probably have received the answer "Interference". It was assumed that memory can be disrupted
or interfered with by what we have previously learned or by what we will learn in the future. This idea
suggests that information in long term memory may become confused or combined with other
information during encoding thus distorting or disrupting memories. Interference theory states that
forgetting occurs because memories interfere with and disrupt one another, in other words forgetting
occurs because of interference from other memories (Baddeley, 1999). There are two ways in which
interference can cause forgetting:

1. **Proactive interference** (pro=forward) occurs when you cannot learn a new task because of an
   old task that had been learnt. When what we already know interferes with what we are
currently learning – where old memories disrupt new memories.

2. **Retroactive interference** (retro=backward) occurs when you forget a previously learnt task
due to the learning of a new task. In other words, later learning interferes with earlier
learning - where new memories disrupt old memories.

**Lack of Consolidation.**

The previous accounts of forgetting have focused primarily on psychological evidence, but
memory also relies on biological processes. For example, we can define a memory
trace as: 'some permanent alteration of the brain substrate in order to represent some aspect of a past experience'.

When we take in new information, a certain amount of time is necessary for changes to the
nervous system to take place – the consolidation process – so that it is properly recorded. During this
period information is moved from short term memory to the more permanent long term memory.

The brain consists of a vast number of cells called neurons, connected to each other by
synapses. Synapses enable chemicals to be passed from one neuron to another. These chemicals, called
neurotransmitters, can either inhibit or stimulate the performance of neurons.

So if you can imagine a network of neurons all connected via synapses, there will be a pattern
of stimulation and inhibition. It has been suggested that this pattern of inhibition and stimulation can
be used as a basis for storing information. This process of modifying neurons in order form new
permanent memories is referred to as consolidation (Parkin, 1993).

There is evidence that the consolidation process is impaired if there is damage to the
hippocampus (a region of the brain). In 1953, HM had brain surgery to treat his epilepsy, which had
become extremely severe. The surgery removed parts of his brain and destroyed the hippocampus, and
although it relieved his epilepsy, it left him with a range of memory problems. Although his STM
functioned well, he was unable to process information into LTM.

The main problem experienced by HM is his inability to remember and learn new things. This
inability to form new memories is referred to as anterograde amnesia. However, of interest in our
understanding of the duration of the process of consolidation is HM's memory for events before his
surgery. In general, his memory for events before the surgery remains intact, but he does have some memory loss for events which occurred in the two years leading up to surgery.

Pinel (1993) suggests that this challenges Hebb's (1949) idea that the process of consolidation takes approximately 30 minutes. The fact that HM's memory is disrupted for the two-year period leading up to the surgery indicates that the process of consolidation continues for a number of years. Finally, aging can also impair our ability to consolidate information.

Retrieval Failure Theory
Retrieval failure is where the information is in long term memory, but cannot be accessed. Such information is said to be available (i.e. it is still stored) but not accessible (i.e. it cannot be retrieved). It cannot be accessed because the retrieval cues are not present. When we store a new memory we also store information about the situation and these are known as retrieval cues. When we come into the same situation again, these retrieval cues can trigger the memory of the situation. Retrieval cues can be:
- External / Context - in the environment, e.g. smell, place etc.
- Internal / State - inside of us, e.g. physical, emotional, mood, drunk etc.

LOCALIZATION OF LANGUAGE
One of the oldest controversies in psychology and neurology concerns localization of function, the notion that different aspects of behaviour are mediated by different parts of the brain. The issue of mental localization was debated by classical thinkers from 400 bc to ad 200 and Aristotle even argued that the soul (i.e. the mind) occupied the heart. Only after several hundred years did Greek writers such as Alcmaeon finally prevail in their arguments that the faculties of the mind lay in the watery ventricles of the brain. The emphasis then shifted to the number of mental faculties and by ad 400 the Church Fathers, including St Augustine, proposed the cell doctrine of the mind, the cells being the ventricles and the faculties of sensation, imagination, reasoning, movement, and memory residing in separate cells, with some sharing since there were only three cells. Although the number of faculties eventually rose to seven or eight, little then changed for another thousand years — making the cell doctrine easily the most enduring theory of the physical basis of mind. But 18th-century anatomists like Sylvius were busy undermining it, convincing their contemporaries that the convolutions of the cerebral cortex were far too complex to be mere cooling pipes for the blood. However, the idea of cortical localization of function had to wait for Franz Joseph Gall in the early 19th century.

From first observing that the mental characteristics of his school friends appeared to be related to the shape of their heads, Gall believed that traits like cautiousness and mirthfulness - he announced 27 in all - were localized and that their magnitude was reflected in the size of a particular region, which in turn was indicated by the size of the overlying skull. Gall's phrenology enjoyed a brief ascendancy until about 1820 when Flourens, noting that damage to different parts of the brain often had similar and diffuse effects on behaviour, concluded that the brain acts as a whole. It was much later that localization of function acquired scientific respectability when Broca showed in 1861 that speech impairment followed damage to a restricted part of the left frontal lobe, and Gustav Fritsch and J. L. Hitzig reported their observations on the effects of electrically stimulating different parts of the exposed brain, first in soldiers with head injuries and then in animals. They found that stimulating discrete parts of what is now known as the motor cortex produced movements of different regions of the body. Interestingly, the doyen of Scottish phrenology George Coombe had observed similar patients in the 1830s and noted that the exposed brain swelled and reddened when the patient became excited.
Coombe had stumbled on changes in cerebral blood flow in relation to particular mental activity, which formed the basis of late 20th-century functional brain imaging.

The acceptance of the idea of localization of function had one unfortunate effect. It came to be taken for granted that the senses of touch and vision and hearing were mapped on the surface of the brain and that there was a similarly orderly representation of the muscles, as shown in Fig. 1. But why there is a map at all was not recognized as a question of fundamental importance, despite the fact that nature went to enormous trouble to evolve genetic instructions which ensured that the retina of the eye and the surface of the body are represented on the surface of the brain in an orderly map and not higgledy-piggledy. Furthermore, a computer programmed to recognize patterns does not need within its components anything like a geographical map of the original scene. So why does the brain have one?

It became increasingly difficult to sidestep this question with the demonstration from 1970 onwards of multiple maps of the eye in the brain. A map is demonstrated by recording the electrical activity of clusters of nerve cells, determining where a visual stimulus must lie on the retina for it to excite these particular cells, and then moving the recording electrode to another group of cells. Using this procedure in anaesthetized animals, it was shown that the retina is mapped not once but many times in the cortex. The macaque monkey has at least ten mapped representations of the retina, and about twenty others where it is the nature of the stimulus rather than its position that is computed. At least a third of the cerebral cortex in the owl monkey is concerned with the multiple mapped representations of visual space.

What is the purpose of such an arrangement, which is not confined to vision, for there are now known to be several topological representations of the surface of the body and the musculature in monkeys? A plausible explanation concerns a well-known physiological phenomenon called lateral inhibition. In the eye itself, adjacent differences in the brightness of colour of the image are given prominence in the nerve signals that leave the eye. This is accomplished by a system of lateral inhibitory connections in the retina which ensure that nerve cells tend to inhibit their immediate neighbours. In an area of uniform illumination or colour, all cells are equally excited by the light and equally inhibited by their neighbours. But where there is a sharp difference in illumination, as at the image of a contour, the highly illuminated cells exert a powerful inhibition on their neighbours in the shade, and the difference in signals sent by the two groups of cells is enhanced. Lateral inhibition cannot create something out of nothing, but it can enhance one feature of the visual image at the expense of another. Lateral inhibition of this kind ensures that edges and contours are prominently coded in the signals from the eye.

There is now incontrovertible evidence from physiology and anatomy that lateral inhibition works in the brain as well as in the eye, and this provides the major reason for the existence of a map of the eye on the cortex of the brain. If the differences in illumination of adjacent parts of the eye are to be accentuated in the cortex, the sensory connections between the nerve cells concerned with the two adjacent parts of the image should be close together. In a map they are as close together as possible, and lateral interactions will be maximally efficient. If there were no map, so that nerve cells concerned with adjacent parts of the image were often far apart in the relevant cortical area, the problem of interconnecting the cells becomes formidable and the average length of a connection would be much greater, about 20 to 30 times greater in visual area 1 of man and monkey. In a map of the sensory surface the lateral interconnections between cells can all be local, and anatomy has shown this to be so.

But why are there many maps for each of the senses rather than just one? The answer is really the same. Inhibitory connections between neighbouring nerve cells of the cortex are now believed to be involved in coding many attributes of the visual image, such as colour, movement, disparity, orientation, size, and spatial periodicity. If all of these were to be attempted within one map, the local
interconnections would again have to be longer and the problem of interconnecting the right cells would increase. By having many maps, each small and containing nerve cells concerned only with one or a few of the stimulus attributes just mentioned, the lateral interconnections can be kept as short as possible and the problem of interconnecting the right type of cell is minimized.

This simple idea has much to support it. First, although there are long fibre connections from one part of the brain to another, microscopy has shown that the connections within a particular map are short and predominantly inhibitory. Second, physiology has shown that nerve cells within a particular cortical representation of the eye tend to be concerned with a restricted range of stimulus qualities, such as orientation, distance, size, colour, or movement. Different maps deal with different stimulus qualities. Third, the human corpus callosum contains about 600 million nerve fibres connecting the two sides of the brain. They are grouped from front to back according to destination and function; if they were not, their average length would have to be longer. Fourth, there are many examples of very selective effects on visual perception of localized brain damage. Although they are rare, some patients suffer a highly selective disturbance of the perception of colour or position or depth or motion, as would be expected when the damage is occasionally restricted to one of the visual maps. Functional maps keep connections short and, therefore, keep the brain (and skull) small enough to be born through a narrow birth canal.

Although the different sensory qualities of the visual scene are initially coded in separate visual areas, our visual perception is unitary not fragmentated, which means that the timing of the activity of cells in different visual areas must be precisely coordinated. If we look at a moving, spinning, coloured object and the nervous signals in one visual area are out of phase with all the others, some distortion should occur in what is seen. Indeed, fever, toxicosis, and brain damage can all lead to temporary visual perceptual dislocations. For example, in one part of the visual field objects may appear too large or too small, smooth movement may look jerky, contours may be multiplied, position and orientation be greatly misperceived.

Multiple brain maps of sensory and motor systems are now established. They permit the maximum efficiency and economy in the myriad interconnections between nerve cells responsible for analysing sensory signals. Their existence also throws light on what is now seen as an unwarranted controversy about localization of function. The cortical representations of the sensory attributes of stimuli, such as colour, may be confined to a few areas. The cortical events underlying certain complex and cognitive actions are probably so widely dispersed that no brain damage, however great, can either destroy them entirely or leave them wholly unimpaired. See also neuropsychology.

Language and the brain

Many people assume the physical basis of language lies in the lips, the tongue, or the ear. But deaf and mute people can also possess language fully. People who have no capacity to use their vocal cords may still be able to comprehend language and use its written forms. And human sign language, which is based on visible gesture rather than the creation of sound waves, is an infinitely creative system just like spoken forms of language. But the basis of sign language is not in the hand, just as spoken language is not based in the lips or tongue. There are many examples of aphasics who lose both the ability to write as well as to express themselves using sign-language, yet they never lose manual dexterity in other tasks, such as sipping with a straw or tying their shoes.

Language is brain stuff--not tongue, lip, ear, or hand stuff. The language organ is the mind. More specifically, the language faculty seems to be located in certain areas of the left hemispheric cortex in most healthy adults. A special branch of linguistics, called neurolinguistics, studies the physical structure of the brain as it relates to language production and comprehension.
**Structure of the human brain.** The human brain displays a number of physiological and structural characteristics that must be understood before beginning a discussion of the brain as a language organ. First, the cerebrum, consisting of a cortex (the outer layer) and a subcortex, is also divided into two hemispheres joined by a membrane called the corpus callosum. There are a few points which must be made about the functioning of these two cerebral hemispheres.

1) In all humans, the right hemisphere controls the left side of the body; the left hemisphere controls the right side of the body. This arrangement—called contralateral neural control—is not limited to humans but is also present in all vertebrates--fish, frogs, lizards, birds and mammals. On the other hand, in invertebrates such as worms, the right hemisphere controls the right side, the left hemisphere controls the left side. The contralateral arrangement of neural control thus might be due to an ancient evolutionary change which occurred in the earliest vertebrates over half a billion years ago. The earliest vertebrate must have undergone a 180° turn of the brain stem on the spinal chord so that the pathways from brain to body side became crossed. The probability that such a primordial twist did occur is also born out by the fact that invertebrates have their main nerve pathways on their bellies and their circulatory organs on their backs, while all vertebrates have their heart in front and their spinal chord in back--just as one would expect if the 180° twist of the brain stem vis-a-vis the body did take place.

2.) Another crucial feature of brain physiology is that each hemisphere has somewhat unique functions (unlike other paired organs such as the lungs, kidneys, breasts or testicles which have identical functions). In other words, hemisphere function is asymmetrical. This is most strikingly the case in humans, where the right hemisphere—in addition to controlling the left side of the body—also controls spatial acuity, while the left hemisphere—in addition to controlling the right side of the body—controls abstract reasoning and physical tasks which require a step-by-step progression. It is important to note that in adults, the left hemisphere also controls language; even in most left-handed patients, lateralization of language skills in the left hemisphere is completed by the age of puberty.

Now, why should specialized human skills such as language and abstract reasoning have developed in the left hemisphere instead of the right? Why didn't these skills develop equally in both hemispheres. The answer seems to combine the principle of functional economy with increased specialization. In nature, specialization for particular tasks often leads to physical asymmetry of the body—witness the lobster's claws—where limbs or other of the body differentiate to perform a larger variety of tasks with greater sophistication (the same might be said to have happened in human society with the rise of different trades and the division of labor).

Because of this specialization, one hemisphere—in most individuals for some reason it is the right hemisphere—came to control matters relating to 3D spatial acuity—the awareness of position in space in all directions simultaneously. Thus, in modern humans, artistic ability tends to be centered in various areas of the right hemisphere.

The left hemisphere, on the other hand, came to control patterns that progress step-by-step in a single dimension, such as our sense of time progression, or the logical steps required in performing feats of manual dexterity such as the process of fashioning a stone axe. This connects with right-handedness. Most humans are born with a lopsided preference for performing skills of manual dexterity with the right hand—the hand controlled by the left hemisphere. The left hand holds an object in space while the right hand manipulates that object to perform tasks which require a step-by-step progression. Obviously, this is a better arrangement than if both hands were equally clumsy at performing complex, multi-step tasks, or if both sides of the brain were equally mediocre at thinking abstractly or at processing information about one's three-dimensional surroundings. So human hemispheric asymmetry seems to have developed to serve very practical purposes.
(By the way, left-handedness seems to be the result of inheritance of two copies of a gene which does not impart strong right-hand preference. The right-handed gene is dominant—in 25% of the population has no copy of this gene, presumably 12.5% percent of these non-handed individuals develop a righthandedness anyway, and 12.5% develop a tendency toward left handedness. At any rate, being left-handed doesn't seem to have any special effect on language acquisition or learning or on anything else innate to humans.)

This general pattern of cognitive asymmetry was probably well established in our hominid ancestors before the language faculty developed. So why did humans evolve in such a way that the language faculty normally localized in the left hemisphere? Why not in the right? Clearly, the reason is that language, like fashioning a stone axe, is also a linear process: sounds and words are uttered one after another in a definite progression, not in multiple directions simultaneously. In the modern human, the feature of **monolineal progression** seems naturally to ally language with other left brain skills such as the ability to perform complex work tasks, or abstract step-by-step feats of logic, mathematics, or reasoning. Even among natural left-handers (in about 12.5 % of any human population, language skills are localized in the cortex of the left hemisphere in all but about 2.5% of the cases. Some of these are individuals who received damage to the left hemisphere in childhood which, presumably, prevented language from localizing there; however, we don't know why language localizes in the right hemisphere of the brain in about one in fifty healthy adults. Like right or left handedness, it seems to correlate with nothing else in particular.

How do we know that the left hemisphere controls language in most adults. There is a great deal of physical evidence for the left hemisphere as the language center in the majority of healthy adults.

1) Tests have demonstrated increased neural activity in parts of the left hemisphere when subjects are using language. (PET scans--Positron Emission Tomography, where patient injects mildly radioactive substance, which is absorbed more quickly by the more active areas of the brain). The same type of tests have demonstrated that artistic endeavor draws normally more heavily on the neurons of the right hemispheric cortex.

2) In instances when the corpus callosum is severed by deliberate surgery to ease epileptic seizures, the subject cannot verbalize about object visible only in the left field of vision or held in the left hand.) Remember that in some individuals there seems to be language only in the right brain; in a few individuals, there seems to be a separate language center in each hemisphere.)

3.) Another clue has to do with the evidence from studies of brain damage. A person with a stroke in the right hemisphere loses control over parts of the left side of the body, sometimes also suffers a diminution of artistic abilities. But language skills are not impaired even if the left side of the mouth is crippled, the brain can handle language as before. A person with a stroke in the left hemisphere loses control of the right side of the body; also, 70% of adult patients with damage to the left hemisphere will experience at least some language loss which is not due only to the lack of control of the muscles on the right side of the mouth--communication of any sort is disrupted in a variety of ways that are not connected with the voluntary muscles of the vocal apparatus. The cognitive loss of language is called **aphasia**, and we will discuss various types of aphasia in great detail tomorrow; only 1% of adults with damage to the right hemisphere experience any permanent language loss.

Aphasics can blow out candles and suck on straws, even sing and whistle, but they cannot produce normal, creative speech in either written, spoken, or gestural form. Sign language users also store their linguistic ability in the left hemisphere. If this hemisphere is damaged, they cannot sign properly, even though they may continue to be able to use their hands for such things as playing the
drums, giving someone a massage, or other non-linguistic hand movements. Injury to the right hemisphere of deaf persons produces the opposite effect.

*Experiments on healthy individuals with both hemispheres intact.*

1) In 1949 it was discovered that if sodium amytal is injected into the left carotid artery, which services blood to the left hemisphere, language skills are temporarily disrupted. If the entire left hemisphere is put to sleep, a person can think but cannot talk.

2) If an electrical charge is sent to certain areas of the left hemisphere (exactly which areas we will discuss tomorrow), the patient has difficulty talking or involuntarily utters a vowel-like cry (although the production of specific speech sounds has never been induced by electrical charge). An electrical charges to the right hemisphere produces no such effect.

3) Musical notes and tones are best perceived through the left ear (which is connected to the spacial-acuity-controlling right hemisphere. In contrast, the right ear better perceives and processes the sounds of language, even linguistic tones (any form with meaning); the right ear takes sound directly to the left hemisphere language center.

4) When repeating after someone, most individuals have a harder time tapping with the fingers of the right hand than with the left hand. /Perform this experiment in class./

5) The language centers in the left hemisphere of humans actually make the left hemisphere bulge out slightly in comparison to the same areas of the right hemisphere. This is easily seen without the aid of the microscope. For this reason, some neurolinguists have called humans the **lopsided ape**. Some paleontologists claim to have found evidence for this left-hemispheric bulging in *Homo neanderthalus* and *Homo erectus* skulls.

Other primates also possess a left perisylvian area of the brain, but it doesn't seem to be involved in their communication. Animal communication seems in fact to be controlled by the subcortical areas of the animal brain, much like human vocalizations other than language--laughter, sobbing, crying, as well as involuntary, word-like exclamations which do form part of language--are controlled in humans in the subcortex, a phylogenetically older portion of the brain that is involved with emotions and reflex responses.

**Tourette's syndrome**, which produces random and involuntary emotive reflex responses, including vocalizations This type of disorder, which often affects language use, is caused by a disfunction in the sub cortex. There is no filter which prevents the slightest stimulus from producing a vocal response, sometimes of an inappropriate manner using abusive language or expletives. These words are involuntary and often the affected individual is not even aware of uttering them (like "um" in many individuals) and only realizes it when video is played back.

This syndrome is not so much a language disorder per se as a disorder of the filters on the adult emotional reflex system--a kind of expletive hiccup. True language is housed in the cortex of the left hemisphere, not in the subcortical area that controls involuntary responses.

**What can language disorders tell us about the brain's language areas?**

Certain types of brain damage can affect language production without actually eliminating language from the brain. A stroke that damages the muscles of the vocal apparatus may leave the abstract cognitive structure of language intact--as witnessed by the fact that right hemisphere stroke victims often understand language perfectly well and write it perfectly with their right hand--although their speech may be slurred due to lack of muscle control. We have also seen that certain disorders involving the subcortex--the seat of involuntary emotional response--may have linguistic side effects, such as in some cases of Tourette's syndrome.
But what happens when the areas of the brain which control language are affected directly, and
the individual's abstract command of language is affected? We will see that language disorders can
shed a great deal of light on the enigma of the human language instinct.

SLI. One rare language disorder seems to be inborn rather than the result of damage to a previously
normal brain. I have said that children are born with a natural instinct to acquire language, the so-called
LAD; however, a tiny minority of babies are born with an apparent defect in this LAD.

Certain families appear to have a hereditary language acquisition disorder, labeled specific
language impairment, or SLI. Children born with this disorder usually have normal intelligence,
perhaps even high intelligence, but as children they are never able to acquire language naturally and
effortlessly. They are born with their window of opportunity already closed to natural language
acquisition. These children grow up without succeeding in acquiring any consistent grammatical
patterns. Thus, they never command any language well—even their native language. As children and
then as adults, their speech in their native language is a catalog of random grammatical errors, such as:
It's a flying birds, they are. These boy eat two cookie. John is work in the factory. These errors are
random, not the set patterns of an alternate dialect: the next conversation the same SLI-afflicted
individual might say This boys eats two cookies. These sentences, in fact, were uttered by a British
tenager who is at the top of his class in mathematics; he is highly intelligent, just grammar
blind. SLI sufferers are incapable of perfecting their skills through being taught, just as some people
are incapable of being taught how to draw well or how to see certain colors. This is the best proof we
have that the language instinct most children are born with is a skill quite distinct from general
intelligence.

Because SLI occurs in families and seems to have no environmental cause whatsoever, it is
assumed to be caused by some hereditary factor—probably a mutant, recessive gene that interferes with
or impairs the LAD. The precise gene which causes SLI has yet to be located.

SUMMARY
Let's sum up three important facts about language and brain.

First, humans are born with the innate capacity to acquire the extremely complex, creative
system of communication that we call language. We are born with a language instinct, which
Chomsky calls the LAD (language acquisition device). This language aptitude is completely different
from inborn reflex responses to stimuli as laughter, sneezing, or crying. The language instinct seems to
be a uniquely human genetic endowment: nearly all children exposed to language naturally acquire
language almost as if by magic. Only in rare cases are children born without this magical ability to
absorb abstract syntactic patterns from their environment. These children are said to suffer from
Specific Language Impairment, or SLI. It is thought that SLI is caused by a mutant gene which
disrupts the LAD.

The LAD itself, of course, is probably the result of the complex interaction of many genes—not
just one—and the malfunction of some single key gene simply short-circuits the system. For example, a
faulty carburetor wire may prevent an engine from running, but the engine is more than a single
 carburetor wire. Many thousands of genes contribute to the makeup of the human brain—more than to
any other single aspect of the human body. To isolate the specific set of genes that act as the blueprint
for the language organ is something no one has even begun to do.

Second, the natural ability for acquiring language normally diminished rapidly somewhere around
the age of puberty. There is a critical age for acquiring fluent native language. This phenomenon seems
to be connected with the lateralization of language in the left hemisphere of most individuals—the
hemisphere associated with monolinear cognition (such as abstract reasoning and step-by-step physical
tasks) and not the right hemisphere, which is associated with 3D spatial acuity, artistic and musical
ability. Unlike adults, children seem to be able to employ both hemispheres to acquire language. In other words, one might say that children acquire language three-dimensionally while adults must learn it two dimensionally.

Third and finally, in most adults the language organ is the perisylvian area of the left hemispheric cortex. Yesterday we discussed the extensive catalog of evidence that shows language is usually housed in this specific area of the brain. Only the human species uses this area for communication. The signals of animal systems of communication seem to be controlled by the subcortex, the area which in humans controls similar inborn response signals such as laughter, crying, fear, desire, etc.

**Aphasia**

We know which specific areas of the left hemisphere are involved in the production and processing of particular aspects of language. And we know this primarily from the study of patients who have had damage to certain parts of the left hemispheric cortex. Damage to this area produces a condition called aphasia, or speech impairment (also called dysphasia in Britain). The study of language loss in a once normal brain is called aphasiology.

Aphasia is caused by damage to the language centers of the left hemisphere in the region of the sylvian fissure. Nearly 98% of aphasia cases can be traced to damage in the perisylvian area of the left hemisphere of the cerebral cortex. Remember, however, that in the occasional individual language is localized elsewhere; and in children language is not yet fully localized.

Strokes cause 85% of all aphasia cases; other causes include cerebral tumors and lesions. One in 200 people experiences aphasia, with males more at risk. Gradual recovery is possible in 40% of adult cases; pre-pubescent children are much more likely to recover from aphasia, with the language faculty localizing in another, unaffected area of the brain, usually the perisylvian cortex of the right hemisphere. Generally, the more extensive the injury, the greater the likelihood of permanent damage.

But we have seen that language is a complex of interacting components--consonants and vowels, nouns and verbs, content words and function words, syntax and semantics. Could it be that these components are housed in particular sub-areas of the left hemispericperisylvian cortex? We haven't pinpointed whether nouns are stored separately from verbs, or where the fricative sounds are stored. There is no conclusive proof for that type of specialization of brain tissue. But there is compelling evidence to believe that two special aspects of language structure are processed by different sub-areas of the language center. We know this because damage to specific areas of the peresylvian area produces two basic types of aphasia.

Each of these two types of language loss is associated with damage to a particular sub-region of the perisylvian area of the left hemispheric cortex.

(1861) Paul Broca discovered Broca's area (located in the frontal portion of the left perisylvian area) which seems to be involved in grammatical processing. (While parsing sentences such as *fat people eat accumulates*, there is a measurable burst of neural activity in Broca's area when the last word is spoken.) Broca's area seems to process the grammatical structure rather than select the specific units of meaning. It seems to be involved in the function aspect rather than the content areas of language.

**Broca's aphasia** involves difficulty in speaking. For this reason it is also known as emissive aphasia. Broca's aphasics can comprehend but have great difficulty replying in any grammatically coherent way. They tend to utter only isolated content words on their own. Grammatical and syntactic connectedness is lost. Speech is a labored, irregular series of content words with no grammatical morphemes or sentence structure. *(Read example)* Grammar rules as well as function morphemes are lost. Broca's aphasia is also known as agrammatic aphasia. Grammar is destroyed; the lexicon more or less preserved intact.
(1875) Karl Wernicke: **Wernicke's area** (in the lower posterior part of the perisylvian region) controls comprehension, as well as the selection of content words. When this area is specifically damaged, a very different type of aphasia usually results, one in which the grammar and function words are preserved, but the content is mostly destroyed.

Since **Wernicke's aphasia** involves difficulty in comprehension, in extracting meaning from a context, it is also known as **receptive aphasia**. Wernicke's aphasics easily initiate long-winded, fluent nonsense, but don't seem able to respond specifically to their interlocutor (unlike Broca's aphasics, who can understand but the have difficulty replying). Wernicke's aphasics often talk incessantly and tend to utter whole volumes of grammatically correct nonsense with relatively few content words or with jibberish words like "thingamajig" or "whatchamacallit" instead of true content words. *(Read example.)* Because Wernicke's aphasia patients can utter whole monologs of such contentless grammatical babble, hardly letting their interlocutor get a word in edgewise, their affliction is also known as **jargon aphasia**.

The normal human mind uses both areas in unison when speaking. Apparently, normal adults use the neurons of Wernicke's area to select sounds or listemes. We use the neurons of Broca's area to combine these units according to the abstract rules of phonology and syntax--the elements in language which have function but no specific meaning--to produce utterances.

**Review:**

**Broca's aphasia**--**emissive aphasia**--**agrammatic aphasia**: difficulty in encoding, in building up a context, difficulty in using the grammatical matrix of phrase structure, difficulty in using the elements and patterns of language without concrete meaning. Broca's area apparently houses the elements of language that have function but no specific meaning--the syntactic rules and phonological patterns, as well as the function words--that is, the grammatical glue which holds the context together.

**Wernicke's aphasia**--**receptive aphasia**--**jargon aphasia**: difficulty in decoding, in breaking down a context into smaller units, as well as in selecting and using the elements of language with concrete meaning. Wernicke's area apparently houses the elements of language that have specific meaning--the content words, the lexemes--that is, the storehouse of prefabricated, meaningful elements which a speaker selects when filling in a context.

Let's review what these two areas--Broca's and Wernicke's seem to be telling us about the way language is stored in the brain. Language obviously consists of these two aspects working together in unison:

1) A very large but finite number of elements with specific form and meaning (morphemes, words, phrases--the lexicon, or set of **listemes**, on the other hand--). **These ready-made elements seems to be stored in Wernicke's area.**

2) A fairly small number of patterns with virtually no limit on the specific meaning they can express (the phonology and syntax--the grammar of language, the abstract blueprint by which the prefabricated units of Wernicke's area are combined). **These abstract patterns seem to be stored in Broca's area.**

**Roman Jakobson**, a Russian born linguist who made extensive studies of aphasia in the 1950's, noted that both types of the aphasic lose language in the exact reverse order that language is acquired by a child--*s* of *plays*, the genitive *'s*, then finally plural *s*. This is true of the sound pattern, as well. In instances of gradual, progressive degeneration of the language centers of the left hemisphere, the aphasic's loss of phonology is the mirror image of the acquisition of elements in childhood.

These two areas have been implicated even more broadly with the human abilities to deal with signs. Roman Jakobson also noted that normal language function involves an interaction of two
different associative properties of meaning: association by **contiguity** and association by **similarity**. (Perform a word test with the word *knife.* ) Jakobson conducted aphasia studies in the 50's and 60's which revealed that each of the two basic types of linguistic aphasia--Broca's emissive, or agrammatic, aphasia and Wernicke's receptive, or jargon, aphasia-- also affects a specific one of these two aspects of linguistic association in a predictable way.

**Broca's aphasia (emissive, agrammatic) also involves contiguity disorder.** We have seen how Broca's aphasics have difficulty in building up a context. Jakobson showed that Broca's aphasics also lose their general ability to communicate in terms of spatial and temporal **contiguity:**

1.) The Broca's aphasic can name synonyms and antonyms but not contiguous concepts: *champagne, wine,* but not *cork, tipsy, hangover.* *knife*--->*dagger, sword,* but not *fork, spoon, table, to eat with.*

2.) Broca's aphasics also evince an inability to comprehend metonymy, synecdoche, tropes based on contiguity.

3.) All understanding of word building, connecting morphemes to build words, is lost. The Broca's aphasic can say *jewel* but cannot build such derivates as *jeweler, jewelry;* or he can say *employ* but not *employer, employee.* He shows an inability to combine or break down linguistic units. Compound words such as *Thanksgiving* are perceived as indivisible wholes. Broca's aphasics cannot pronounce new or unfamiliar words: *big, give,* but not *gib.* Cannot form the plural of *wug* or any other plural. If the word exists only as a ready-made unit, it cannot be built up out of smaller units. **Linguistic expression is limited to selection of ready-made units; all contiguity-based relations are impaired--content is retained but context is lost.**

**Wernicke's aphasia** (receptive, jargon aphasia), on the other hand, involves **similarity disorder.** We have seen that for Wernicke aphasics, conversation is easily initiated but lacks content. Connective words such as conjunctions, pronouns, prepositions remain, but selection of content words is impaired; content words tend to be absent or replaced by general terms such as *thing, stuff, whatchamacalit.*

Wernicke's aphasics also lose their ability to perform language skills based on association by similarity. They cannot form or comprehend metaphors and similes and compensate by using associations based on contiguity.

1.) Wernicke's aphasics cannot produce synonyms or antonyms: Instead, the patient will name things contextually associated with an object. When asked to define the word *knife,* a Wernicke's aphasis might say *to eat with* or *knife,* or even *knife and fork;* he would not say *dagger, sword,* or anything similar. When asked to repeat the word *glass* he might say *window,* or something contiguous with glass.

2.) Wernicke's aphasics evince an inability to use or comprehend metaphor, simile--tropes based on association by similarity.

3) **Linguistic expression is limited to contiguity-based relations--context is retained while content is lost; all skills based on the recognitions of similarity or dissimilarity are impaired and replaced by expressions of contiguity.**

Jakobson was the first to note that Broca's and Wernicke's area seem to control these different and complementary associative properties of meaning. In the conversation of a normal individual, both regions of the brain work in unison (healthy people even have a hard time separating out what associations are based on similarity and which are based on contiguity). But in aphasic patients, either context and contiguity (Broca's) or the content and similarity (Wernicke's) tend to be impaired (though each individual aphasic has a different combination of these impairments). If Broca's and Wernicke's regions are both severely damaged--in other words, if the entire linguistically relevant perisylvian area...
of the brain is damaged—the patient loses all language ability; he experiences **aphasia universalis**, or the total loss of language.

Recent studies have shown that Broca's and Wernicke's areas are actually contiguous portions of the brain—part of a single area—rather than separate areas (the connection is hidden by the convolutions of the brain). Some recent neurolinguists have called the band of linguistically relevant neural tissue which contains Broca's and Wernicke's areas the **perisylvian area**.

This perisylvian area, apparently, is the language organ in humans. Other animals lack this area, although monkeys and other primates show a small development of the area of their brain that is analogous to Broca's area, this area does not seem to play a role in their communicative skills. In humans, the perisylvian area seems to be the seat of the language skills in most adults. It is here that language skills are normally localized as the brain matures.

It is not possible to say precisely that Broca's and Wernicke's areas have the same language functions in all adults; sometimes language skills seem to be localized in slightly different areas of the adult brain. Broca's area does not always control grammar in the same way that the liver always produces bile and the pancreas always produces pancreatic juice. Unlike the liver, pancreas, and other organs, the developing brain seems to have a property called **plasticity**, which allows functions to be localized in a variety of possible places as the brain matures. This is why damage to Broca's area does not always cause the typical agrammatic aphasia; and damage to Wernicke's area does not always cause the typical jargon and babbling symptoms of Wernicke's aphasia.

There is also some evidence that sub-areas of Broca's area or sub-areas of Wernicke's area may store aspects of language as specific as the comprehension of nouns and verbs or the ability to break a sentence down into words, on the one hand, and the word into individual morphemes or phonemes, on the other. And yet in every individual the ability to communicate seems to involve an interaction of one part of the cortex which controls selection and another part which controls the combination of selected units. These areas, in turn, are connected by a dense set of neurons and so are really extensions of one another. The complex interaction of these neurons gives us our complete language faculty.

**The semiotic organization of the brain**

Jakobson's aphasia studies has implications for the study of the structure of human sign systems in general (**semiotics**). Language is only one of the human manifestations of semiotic (sign-sensitive) behavior. The dual aspects of similarity/selection and contiguity/combination, seen so clearly in the functioning and impairment of language, actually appear as primal forces in all forms of human expression, not just language

**James Frazer** (*The Golden Bough*)—describes two types of magic rites: charms based on similarity--sympathetic or imitative magic **vs.** contagious magic.

Different genres of literature rely to varying degrees on the two types of associations. Most poetry relies more on similarity and less on connected context; most types of prose, on the other hand, relies more heavily on contiguity, on a connected context.

Similarity and contiguity often alternate as dominant forces of expression in art and literature. romanticism **vs.** realism; impressionism **vs.** cubism. In other words, all of our meaning-based systems, not only language, seem to involve a constant interplay of Wernicke-based similarity relations, on the one hand, and Broca-based contiguity relations, on the other.

**Conclusion**

And so, our course began with a discussion of language and mind and it ends with a discussion of language and the brain. It would seem that the perisylvian area of the left hemisphere is indeed not
only the primary organ of language; it also seems to underlie a broader range of cognitive powers that make humans unique. Speech may consist of sound vibrations or visual symbols superficially not unlike the signs of animal communication, but language--the abstract system that underlies the production of speech--is a property of the uniquely human aspect of the mind. Language is brain stuff. And it seems that the human brain--among that of all other species--is uniquely constructed to manipulate complex sign systems such as language, art, and other representational behavior. We are born with the capacity to acquire language in childhood because of the genetically planned structure of our brains. This property of the brain has been called the language instinct. Bees seek nectar, birds build nests, spiders spin webs. We humans create language.

This language instinct is undoubtedly why we humans have become--along with such enormously successful creatures as earthworms and algae--one of the most influential species ever to inhabit the earth.

FUNCTIONS OF CORPUS CALLOSUM

The brain is divided into the right and left hemisphere, and the two halves are connected by the corpus callosum. This bundle of nerve tissue contains over 200 million axons (nerve fibers that carry electrical impulses from neurons’ cell bodies) by rough estimate. This neural tissue facilitates communication between the two sides of the brain. The corpus callosum is the largest collection of white matter within the brain, and it has a high myelin content. Myelin is a fatty, protective coating around nerves that facilitates quicker transmission of information. White matter should not be confused with gray matter. The brain uses gray matter for computation, thinking, memory storage, and more. White matter, like the corpus callosum, allows different parts of the brain to communicate with each other.

Some congenital (birth) defects include a complete lack of this neural tissue. In modern neurosurgery, some surgeons have surgically cut the corpus callosum as a means for treating epileptic seizures. By disrupting contact between the two brain hemispheres, a seizure can be isolated and kept from spreading.
The posterior portion of the corpus callosum is called the splenium; the anterior is called the genu (or "knee"); between the two is the truncus, or "body", of the corpus callosum. The rostrum is the portion of the corpus callosum that projects posteriorly following from the anteriormost genu.
Thinner axons in the genu interconnect prefrontal cortex areas between the two sides of the brain. Those in the posterior body of the corpus callosum interconnect parietal lobe areas. Thicker axons in the midbody of the corpus callosum and in the splenium interconnect areas of the motor, somatosensory, and visual cortex.

Using magnetic resonance diffusion tensor imaging, the studies of Hofer and Frahm suggest that the anterior sixth of the corpus callosum interconnect the prefrontal parts of the brain; the next third, the premotor and supplementary motor regions; the following sixth, the motor areas; then the next twelfth deals with the sensory areas; and the final quarter, the parietal, temporal, and occipital lobes.

**Evolution**

In primates, axon diameter, and hence its conduction velocity, has increased in the corpus callosum with increased brain size and so maintained the speed of communication between the two cerebral hemispheres particularly between its primary motor and sensory areas. However, this scaling between increased brain size and increased myelination of corpus callosum axons has not occurred between chimpanzees and humans. This has resulted in humans having double the delay time of communication between the two sides of the brain compared to that of macaques.

**Sexual dimorphism**

There are disputed claims about the difference of the size of the human corpus callosum in men and women and the relationship of any such differences to gender differences in human behaviour and cognition.

R. B. Bean, a Philadelphia anatomist, suggested in 1906 that the "exceptional size of the corpus callosum may mean exceptional intellectual activity" and claimed differences in size between males and females and between races, although these were refuted by Franklin Mall, the director of his own laboratory.

Of much more substantial popular impact was a 1982 Science article claiming to be the first report of a reliable sex difference in human brain morphology, and arguing for relevance to cognitive gender differences. This paper appears to be the source of a large number of lay explanations of perceived male-female difference in behaviour: For example Time magazine was reported to state in 1992 that the corpus callosum is "Often wider in the brains of women than in those of men, it may allow for greater cross-talk between the hemispheres—possibly the basis for women’s intuition." There is scientific dispute not only about the implications of anatomical difference, but whether such a difference actually exists. A substantial review paper performed a meta-analysis of 49 studies and found, contrary to de Lacoste-Utamsing and Holloway that males have a larger corpus callosum, a relationship that is true whether or not account is taken of larger male brain size. Bishop and Wahlstein found that "the widespread belief that women have a larger splenium than men and consequently think differently is untenable." However, more recent studies using new analysis and imaging techniques (e.g. diffusion-tensor imaging) revealed morphological and microstructural sex differences in human corpus callosum. A 2006 Serbian study found variations in morphology correlated with sex, but in ways too complex for simple direct comparison. Whether, and to what extent, these morphological differences are associated with behavioural and cognitive differences between men and women remains unclear.

**Other correlations**

The front portion of the corpus callosum has been reported to be significantly larger in musicians than non-musicians, and to be 11% larger in left-handed and ambidextrous people than right-handed people.

**Epilepsy**
The symptoms of refractory epilepsy can be reduced by cutting the corpus callosum in an operation known as a corpuscallosotomy.

Function:
The corpus callosum is the largest fiber bundle in the brain, containing nearly 200 million axons. It is involved in several functions of the body including:
- Communication Between Brain Hemispheres
- Eye Movement
- Maintaining the Balance of Arousal and Attention
- Tactile Localization

**Pathology**
- Alien hand syndrome: A complete or partial absence of it in humans is called agenesis of the corpus callosum.
- Split-brain
- Septo-optic dysplasia(deMorsier syndrome)
- Alexia without agraphia(seen with damage to splenium of corpus callosum)

**References:**