For thousands of years, food and a person’s diet were key to medical treatment. Around 2600 BCE in China, the Nei Ching, an early medical text, prescribed combinations of yin and yang foods for healing. By the mid-fifth century BCE, Hippocrates and his followers prescribed diet, exercise and moderation in eating, drinking, sleeping and sexual indulgence. Even the Hippocratic oath, a vow still taken by all physicians, encourages physicians to consider a nutritional regime as part of the cure. “I will apply dietetic measures for the benefit of the sick according to my ability and judgment.”

The connection between food and health which was so critical to medical treatment through the Middle Ages has been somewhat neglected in today’s high-tech medical environment. Antibiotics, analgesics, and a myriad of new drugs and surgical techniques have lessened the emphasis on healthy eating habits. Nevertheless, poor diet remains a significant factor in our society’s overall health. The USDA Economic Research Service estimates that healthier diets could prevent $71 billion each year in medical costs, lost productivity, and the value of premature deaths associated with coronary heart disease, cancer, stroke, diabetes, hypertension, obesity, and osteoporosis.

Although medical professionals continue to focus technology to treat society’s health problems, researchers have made great strides in unraveling the mysteries of what early medical practitioners knew inherently: the importance of dietary nutrients.

Fundamentally, food nutrients are chemical compounds that break down in the digestion process to strengthen bones and muscles, grow hair, improve eyesight and even alter individuals’ emotional well being, to name a few of their effects.
One of the most interesting results of food is its varying interaction with the structures of the brain. This interaction occurs at the most basic levels of the human nervous system and is dependent on a group of chemicals, called neurotransmitters.

In order to understand food and many of its effects on humans, physiological and emotional, we will examine briefly the structure of the brain, where the chemical reactions occur.

Valentines are nice, but, for Americans, chocolate may well be their true love. According to studies, chocolate is the single most craved food in America. In fact, in 1998, Americans consumed 3.3 billion pounds of chocolate—more than 12 pounds per person—at a retail cost of $13 billion! Unfortunately, this love affair is more likely to be “love at first bite” rather than real chemistry.1,2

Although chocoholics hope to find some physiological basis for their cravings, no real proof exists. One theory postulates that women who are low in magnesium crave chocolate during their premenstrual period. However, women rarely crave leafy green vegetables or cheddar cheese—foods that are higher in magnesium than chocolate.1,2

Another theory suggests that chocolate lovers may be addicted to caffeine. Even though chocolate contains caffeine and theobromine (a caffeine-like substance), the low amounts of these chemicals are unlikely to create an addiction. One ounce of milk chocolate contains only 5 mg of caffeine; a cup of coffee may contain 100-150 mg.1

Rumors of a marijuana-like compound in chocolate attempted to explain chocoholics’ cravings. Although chocolate does contain a cannabinoid-like substance, it would be nearly impossible to consume enough chocolate at one sitting to have such an effect.1

Because carbohydrates raise the levels of serotonin and fat affects endorphin production in the body, some theories postulate that chocoholics seek a mood high or crave chocolate when their levels drop. Unfortunately, research does not yet confirm this pharmacological cause of chocolate cravings.1,2

On the bright side, chocolate consumption, in moderation, has some very beneficial effects. Americans get much of the trace mineral copper from chocolate and candy. Copper is essential to bone metabolism, and, when combined with calcium in milk chocolate, can help strengthen bones.1

Chocolate also contains the antioxidants epicatechin and catechin, flavonoid polyphenols that are found in red wine too. These chemicals may lower cholesterol by helping slow the body’s LDL oxidation process and neutralizing free radicals.1

For those who truly love chocolate, chemistry doesn’t change our affection. Its sweet taste and smooth texture are enough to keep us coming back for more.

receptors in the skin, muscles, joints, viscera, and sense organs. Efferent neurons transmit motor impulses away from the brain and spinal cord to muscles or glands.\textsuperscript{3,4,5}

The neuron is composed of three organs: the cell body, dendrites, and axon. (Figures 1 and 2) The cell body of the neuron is composed of the same organelles that make up other cells of the body, such as the mitochondria, lysosomes, Golgi apparatus, nucleus, and microtubules. But structures specific to the nerve cell include fine thread-like structures that help provide support for the cell processes, called neurofibrils, and Nissl bodies, membranous sacs with ribosomes that manufacture protein molecules for proper cell functioning, similar to the endoplasmic reticulum of other types of cells.

The dendrites of the neuron are numerous and highly branched. These receptive fibers, along with the cell membrane of the neuron, provide the network for communication with other neurons.

The single, cylindrical axon of the neuron conducts electrical impulses away from the cell body. The axon typically arises from the body as a single fiber, but may eventually give off collateral branches that conclude in fine processes called axon terminals. These processes balloon into bulb-shaped synaptic end-bulbs that contain membranous sacs called synaptic vesicles. The vesicles release chemicals, called neurotransmitters, into the small space known as the synapse to influence the action of other neurons, muscle fibers, or gland cells.\textsuperscript{3,4,5,6}
The cytoplasm of the axon is surrounded by a plasma membrane called the axolemma. Within the cytoplasm of an axon are contained mitochondria and neurofibrils, but no Nissl bodies; therefore, there is no protein synthesis within the axon.

Larger axons of the peripheral nerves (the nerves outside of the brain and spinal cord) are enclosed by neuroglial cells called Schwann cells. The membranes of these cells are composed of a lipoprotein with a lipid content that is higher than any other type of cells’ surface membrane. This lipoprotein is called myelin and forms a myelin sheath around the axon that allows for faster speed of an electrical signal. The portion of the Schwann cell that lies outside of the myelin sheath and contains the cell’s cytoplasm and nuclei is referred to as the neurilemma.

Myelinated axons appear white, and are responsible for the appearance of white matter in the brain and spinal cord. The
brain and spinal cord’s myelinated axons are not produced by Schwann cells; however, they are generated by another type of neuroglial cell called an oligodendrocyte.3,4,5

The Synapse
Neurons communicate across a very small space between the axon and dendrite, or the axon and cell body, of a presynaptic and postsynaptic neuron. This gap between the two neurons is called a synaptic cleft. The synaptic end-bulbs of the presynaptic neuron’s axon contain membranous sacs called synaptic vesicles.

When an electrical impulse reaches the end-bulb, the vesicles are signaled to fuse with the end-bulb’s cell membrane, open, and release neurotransmitters into the synaptic cleft. These neurotransmitters diffuse across the synaptic cleft and are taken up (uptake) by their respective receptors within the membrane of the postsynaptic neuron.3,4,5

The kind of receptors housed by each neuron depends on its connections. Not all neurons have receptors for every kind of neurotransmitter. For example, neurons in the nucleus basalis branch into the cerebral cortex and the limbic system. These neurons contain receptors for acetylcholine.6 In this instance, this release of acetylcholine in the nucleus basalis will influence only the neurons with a corresponding receptor, sending a signal through an intertwining network of neurons to affect memory function.

Communication between neurons occurs across the membrane, called the resting membrane potential, and a variety of pores within the membrane.

Communication between neurons depends upon an electrical voltage across the membrane (called the resting membrane potential) and a variety of pores within the membrane called ion channels. Some neurotransmitters may cause ion channels to open and others to close. Some may have an inhibitory action on the synaptic potential; others are excitatory. For example, if neurotransmitters are taken up by their respective receptors on the postsynaptic membrane and cause sodium channels to open, then sodium ions infuse, causing depolarization of the membrane. This triggers an action potential, or impulse.

If, on the other hand, a neurotransmitter is taken up by its respective receptor on the postsynaptic membrane and causes potassium channels to open, then potassium ions will diffuse. This results in inhibition of the action potential.3,4,5

### Table 1: Types of Neurotransmitters

<table>
<thead>
<tr>
<th>Neurotransmitter</th>
<th>Examples</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acetylcholine (ACH)</td>
<td>• Angiotensin, • Antiadrenergic hormone (vasopressin), • Atrial natriuretic peptide (ANP), • Bombesin, • Cholecystokinin (CCK), • Corticotropin, • Delta sleep factor, • Dynorphins, • Enkephalin, • Endorphin, • Galanin, • Gastrin, • Glucagon, • Hypothalamic-regulating hormones</td>
</tr>
<tr>
<td>Amino Acids</td>
<td>• Inhibin, • Melatonin, • Motilin, • Neuropeptide Y (NPY), • Neurotensin, • Oxytocin, • Secretin, • Vasoactive intestinal peptide (VIP), • Substance P, • Somatostatin, • Thyrotropin-releasing factor, • Corticotropin-releasing factor, • Gonadotropin-releasing factor</td>
</tr>
<tr>
<td>Aspartate</td>
<td>• Adrenocorticotrophin (ACTH), • Melanocyte-stimulating hormone</td>
</tr>
<tr>
<td>Gamma aminobutyric acid (GABA)</td>
<td>• Angiotensin, • Antiadrenergic hormone (vasopressin), • Atrial natriuretic peptide (ANP), • Bombesin, • Cholecystokinin (CCK), • Corticotropin, • Delta sleep factor, • Dynorphins, • Enkephalin, • Endorphin, • Galanin, • Gastrin, • Glucagon, • Hypothalamic-regulating hormones</td>
</tr>
<tr>
<td>Glutamate</td>
<td>• Inhibin, • Melatonin, • Motilin, • Neuropeptide Y (NPY), • Neurotensin, • Oxytocin, • Secretin, • Vasoactive intestinal peptide (VIP), • Substance P, • Somatostatin, • Thyrotropin-releasing factor, • Corticotropin-releasing factor, • Gonadotropin-releasing factor</td>
</tr>
<tr>
<td>Glycine</td>
<td>• Adrenocorticotrophin (ACTH), • Melanocyte-stimulating hormone</td>
</tr>
<tr>
<td>Biogenic amines</td>
<td>• Angiotensin, • Antiadrenergic hormone (vasopressin), • Atrial natriuretic peptide (ANP), • Bombesin, • Cholecystokinin (CCK), • Corticotropin, • Delta sleep factor, • Dynorphins, • Enkephalin, • Endorphin, • Galanin, • Gastrin, • Glucagon, • Hypothalamic-regulating hormones</td>
</tr>
<tr>
<td>Dopamine (DA)</td>
<td>• Inhibin, • Melatonin, • Motilin, • Neuropeptide Y (NPY), • Neurotensin, • Oxytocin, • Secretin, • Vasoactive intestinal peptide (VIP), • Substance P, • Somatostatin, • Thyrotropin-releasing factor, • Corticotropin-releasing factor, • Gonadotropin-releasing factor</td>
</tr>
<tr>
<td>Norepinephrine (NE)</td>
<td>• Adrenocorticotrophin (ACTH), • Melanocyte-stimulating hormone</td>
</tr>
<tr>
<td>Epinephrine</td>
<td>• Angiotensin, • Antiadrenergic hormone (vasopressin), • Atrial natriuretic peptide (ANP), • Bombesin, • Cholecystokinin (CCK), • Corticotropin, • Delta sleep factor, • Dynorphins, • Enkephalin, • Endorphin, • Galanin, • Gastrin, • Glucagon, • Hypothalamic-regulating hormones</td>
</tr>
<tr>
<td>Histamine</td>
<td>• Inhibin, • Melatonin, • Motilin, • Neuropeptide Y (NPY), • Neurotensin, • Oxytocin, • Secretin, • Vasoactive intestinal peptide (VIP), • Substance P, • Somatostatin, • Thyrotropin-releasing factor, • Corticotropin-releasing factor, • Gonadotropin-releasing factor</td>
</tr>
<tr>
<td>Serotonin (5-HT)</td>
<td>• Adrenocorticotrophin (ACTH), • Melanocyte-stimulating hormone</td>
</tr>
<tr>
<td>Tyramine</td>
<td>• Angiotensin, • Antiadrenergic hormone (vasopressin), • Atrial natriuretic peptide (ANP), • Bombesin, • Cholecystokinin (CCK), • Corticotropin, • Delta sleep factor, • Dynorphins, • Enkephalin, • Endorphin, • Galanin, • Gastrin, • Glucagon, • Hypothalamic-regulating hormones</td>
</tr>
<tr>
<td>Octopamine</td>
<td>• Inhibin, • Melatonin, • Motilin, • Neuropeptide Y (NPY), • Neurotensin, • Oxytocin, • Secretin, • Vasoactive intestinal peptide (VIP), • Substance P, • Somatostatin, • Thyrotropin-releasing factor, • Corticotropin-releasing factor, • Gonadotropin-releasing factor</td>
</tr>
<tr>
<td>Tetrahydroisocyaninelines</td>
<td>• Adrenocorticotrophin (ACTH), • Melanocyte-stimulating hormone</td>
</tr>
<tr>
<td>Neuropeptides</td>
<td>• Angiotensin, • Antiadrenergic hormone (vasopressin), • Atrial natriuretic peptide (ANP), • Bombesin, • Cholecystokinin (CCK), • Corticotropin, • Delta sleep factor, • Dynorphins, • Enkephalin, • Endorphin, • Galanin, • Gastrin, • Glucagon, • Hypothalamic-regulating hormones</td>
</tr>
</tbody>
</table>

**SOURCES:**
Neurotransmitters

The body may use as many as 60 chemical substances, stored in the synaptic vesicles of neurons, as neurotransmitters. (Some are listed in Table 1) They may act immediately to open channels in the cell membrane, or have a delayed action to influence the reactions of enzymes inside the cell. Some transmitters intensify or diminish a cell's response to other transmitters, allowing neurons in one area to influence neurons in neighboring or distant areas. Categories of neurotransmitters include acetylcholine, amino acids, biogenic amines and neuropeptides.3

Acetylcholine (ACh) affects memory in the brain and may be excitatory or inhibitory.3

Glutamine, an excitatory nonessential amino acid that is concentrated in the hypothalamus and thalamus, is connected with memory function and reduces the craving for alcohol.6,7 Alcoholics may have problems during the conversion of glutamine, which prevents the entry of glutamic acid (a metabolic byproduct) into the brain.7 Gamma-aminobutyric acid (GABA) is also an amino acid and the most common inhibitory neurotransmitter in the brain and central nervous system. Antianxiety drugs enhance GABA function.3,6,7

Dopamine, norepinephrine, epinephrine, and serotonin are biogenic amines.3,6 Dopamine is a hormone that acts as a neurotransmitter. It works in the cerebral cortex to influence emotional responses and in the basal ganglia to regulate movement.3 Researchers believe dopamine is connected with the feeling of pleasure. When synthesized, dopamine becomes norepinephrine.6 Norepinephrine (NE) is concentrated in the

GUT FEELINGS?

The gut has a brain of its own, “the enteric nervous system.” Like its larger counterpart in the head, the latest research demonstrates that the enteric nervous system also sends and receives impulses, records experiences and responds to emotions. The same neurotransmitters, including serotonin, dopamine, glutamate, norepinephrine and nitric oxide, can exert similar effects in both parts of the human body. The gut can upset the brain and the brain can upset the gut.

The enteric nervous system appears in the sheaths of tissue lining the esophagus, stomach, small intestine and colon. Viewed as a single unit, it is composed of over 100 million neurons, neurotransmitters and two dozen small brain proteins (neuropeptides) that communicate messages between neurons and support cells in the brain. In addition, major cells of the immune system, enkephalins (members of the endorphin family) and benzodiazepines (family of psychoactive chemicals that includes valium and xanax) are also present in the gut.

The gut’s circuitry permits it to act independently, learn, remember, and actually produce gut feelings. The gut’s brain is reportedly influential in human emotions, such as happiness and misery. Researchers have found that many gastrointestinal conditions such as colitis and irritable bowel syndrome arise from problems within the gut’s brain.

Scientists are now understanding the reasons why the body has two brains. David Wingate, MD, professor of gastrointestinal science at the University of London and consultant at Royal London Hospital said, “The first nervous systems were in tubular animals that stuck to rocks and waited for food to pass by.” However, as life evolved animals required a more sophisticated brain to find food and reproduce. Consequently, the central nervous system developed. However, according to developmental biologists, the gut’s functions of eating and digestion were too important, so the body kept the enteric nervous system as an independent structure in higher animals. Only loosely connected to the central nervous system, the gut can function almost independently without directions from the brain.

Early in embryogenesis, tissue, named the neural crest, forms and divides. One section develops as the central nervous system, and another piece moves down to become the enteric nervous system. At a later stage in development, the vagus nerve links these two systems, stated Michael Gershon, MD, professor of anatomy and cell biology at Columbia-Presbyterian Medical Center in New York.

The brain alerts the gut by communicating with a small number of “command neurons” which control the level of activity in the gut. In turn, these neurons signal the gut’s interneurons which carry messages up to the brain and back down to the gut. Command neurons and interneurons are located on two layers of gut tissue, the “myenteric plexus” and the submucosal plexus.” The volume of activity is regulated by changing the rate of firing by the vagus nerve.

Residing in the plexus are glial cells that nourish neurons, mast cells related to immune responses and a “blood brain barrier” that protects major neurons against harmful substances. The plexuses also contain sensors to detect sugar, protein, acidity and other chemical factors that might regulate digestion and determine how the gut mixes and moves it contents.

Gradually, researchers are beginning to evaluate the relationship between the two brains and understand why people experience particular emotions and pursue certain actions. For example, when the central brain confronts a frightening circumstance, stress hormones are released to prepare the body to fight or flee. These stress hormones stimulate many sensory nerves in the stomach, and individuals have described this feeling as “butterflies.” During the stress of battle, the higher brain com-
neurons that contact the hypothalamus, cerebellum, cerebral cortex and spinal cord. NE regulates mood, dreaming and waking (alertness), and also serves as a hormone. A third neurotransmitter that serves as a hormone is epinephrine. Epinephrine’s function is related to fear and anxiety. Norepinephrine and epinephrine are both released by the adrenal medulla. The catecholamine structure of these three amines allows for reuptake.

Neurons that contain receptors for serotonin (5-hydroxytryptophan or 5-HT) are located in the intestinal wall, blood vessels, and the central nervous system, where it projects into the hypothalamus, thalamus, and spinal cord. Serotonin is a neurotransmitter that is involved in sleep, appetite, sensory perception, temperature, cardiovascular function, muscle contraction, endocrine regulation, libido, and mood control. Researchers believe that serotonin reduces pain, decreases appetite, produces a sense of calm, and induces sleep. The amount of serotonin synthesized by the brain depends on the availability of tryptophan. Antidepressant drugs are often selective inhibitors of serotonin reuptake.

Transmitters in the nervous system are released in response to nerve impulses, working in milliseconds to affect muscle and gland cells, as well as other neurons. In the endocrine system, blood delivers transmitters in the form of hormones to all types of body cells. Unlike neurotransmitters, the action of hormones could require hours or days to take effect.

Neuropeptides, chains of two or more amino acids, are formed in neurons and act as neurotransmitters and as hormones.

Dangers the gut to shut down because an animal fleeing danger will not stop to defecate, reports Gershon. Fear also stimulates the vagus to increase activity and “turn up the volume” on the serotonin agents in the gut. When the gut gears up, the consequence is diarrhea. In other instances, when nerves in the esophagus are stimulated, people “choke” with emotion.

The famous “Maalox moment” results from the interaction of the two brains, according to Jackie D Wood, chair of the department of physiology at Ohio State University in Columbus, Ohio. When the brain sends out stress signals, changes occur in the nerve function between the stomach and esophagus. The result is heartburn.

During instances of high stress, the brain appears to protect the gut and signals the mast cells in the plexus. These mast cells generate histamine, prostaglandin, and other agents, resulting in inflammation. The negative side effects of these chemicals are diarrhea and cramping.

Drugs that are used for psych effects on the central brain also exert significant, often unanticipated, influence on the lower brain. Scientists have observed that nearly 25 percent of the patients who take Prozac or similar antidepressants also suffer from gastrointestinal problems, such as nausea, diarrhea and constipation. These drugs act on serotonin and stop its uptake by target cells so more remains in the central nervous system. When pressure receptors in the gut are stimulated, serotonin is released and starts the reflexive motion of peristalsis. In small doses, scientists have found that Prozac has successfully been used to relieve chronic constipation, whereas in larger amounts Prozac can produce constipation.

Antibiotics, including erythromycin, act on the gut receptors and generates oscillations that result in cramps and nausea. Heroin and morphine attach to the gut’s receptors and cause constipation. Interestingly, both brains can be addicted to opiates. More evidence of the dual relationship between the central brain and the gut is demonstrated by victims of Alzheimer’s and Parkinson’s diseases who suffer from constipation. Simply, the nerve cells in the gut are as ill as the nerve cells in the brain. One affects the other.

One question currently under investigation is why the gut has receptors for benzodiazepine, a drug that is used to alleviate anxiety. Current theories are pointing to the body’s own natural manufacture of the drug. A recent discovery was made with a patient who suffered liver failure and fell into a deep coma. By administering a drug that block benzodiazepine, the coma was reversed in minutes. Observers noted that when the liver fails, substances that usually are broken down by the liver can reach the brain. Some of these chemicals are identical to benzodiazepine. “We don’t know if they come from the gut itself, from bacteria in the gut or from food, but when the liver fails, the gut’s benzodiazepine goes straight to the brain, knocking the patient unconscious,” reported Anthony Basile, a neurochemist in the Neuroscience Laboratory at the National Institutes of Health in Bethesda, Maryland.

To finally understand the relationship between the gut and central brain would have far-reaching benefits for individuals who experience allergies, autoimmune diseases, such as Krohn’s disease or ulcerative colitis. One larger question remains—can the gut’s brain learn? Some anecdotal evidence points to the possibility, but more substantive research is needed. In the future, people may have a more concrete understanding of why they get flip-flops in a spring romance or become nauseated about an overdue project.

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Accessed 1/7/2000
The body’s ability to metabolize food depends on the time of day, the type and composition of food consumed, the amount consumed, person’s age and dietary history.

mones to regulate physiological responses to other parts of the body, especially digestion. The digestive system assimilates protein into amino acids, the most basic elements of proteins.6 Two types of neuropeptides, endorphins and dynorphins, are called opioid peptides, because they have strong pain-relieving effects. Endorphins and dynorphins are present in the gastrointestinal tract and endocrine glands and in the brain in the thalamus, hypothalamus, and limbic system. The function of these neurotransmitters is linked to memory, learning, feelings of pleasure, and regulation of sexual hormones, and mental illnesses (depression and schizophrenia). Acupuncture may increase the release of opioid peptides.3,6

EFFECTS OF FOOD
Certain nutrients in food, such as amino acids, are precursors to the development of neurotransmitters. (Table 2) The amount of the precursor nutrient in the diet determines the amount of neurotransmitter produced. Because foods are made up of many nutrients, their interactions become important to the production and release of neurotransmitters.

The body contains more than 40 amino acids and 1600 types of proteins, but only eight are considered essential amino acids: isoleucine, leucine, lysine, methionine, phenylalanine, threonine, tryptophan and valine. Animal-derived foods, such as meat and eggs, contain high amounts of these essential amino acids.7

When broken down, some essential amino acids become biogenic amines and act as neurotransmitters. For example, phenylalanine is metabolized into norepinephrine. Phenylalanine stimulates the cerebral cortex to release a hormone called cholecystokinin, which acts on the hypothalamus to suppress appetite.7 If a person doesn’t get enough phenylalanine, he or she may tend to overeat—never getting quite enough cholecystokinin to suppress their appetites. In the conversion to norepinephrine, phenylalanine also creates a nonessential amino acid called tyrosine, which is the precursor to epinephrine and dopamine. Some forms of depression may be caused by a body’s inability to convert phenylalanine to tyrosine.7

Turkey, milk, and other animal and fish proteins contain the amino acid tryptophan, which is essential to the production of serotonin. When consumed with carbohydrates, tryptophan moves quickly to the brain to synthesize serotonin, explaining why a big meal of turkey and stuffing causes drowsiness. However, when consumed with other proteins, the amino acids in those proteins compete with tryptophan to cross the blood brain barrier, preventing some of its benefits.6,7

The body’s ability to metabolize food into the micro and macronutrients it needs depends on a number of factors.

<table>
<thead>
<tr>
<th>TABLE 2 NEUROTRANSMITTER-RICH FOODS</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>FOODS RICH IN SEROTONIN AND TRYPTOPHAN:</strong></td>
</tr>
<tr>
<td>• Bananas</td>
</tr>
<tr>
<td>• Clams</td>
</tr>
<tr>
<td>• Escargots</td>
</tr>
<tr>
<td>• Milk</td>
</tr>
<tr>
<td>• Nuts</td>
</tr>
<tr>
<td>• Oysters</td>
</tr>
<tr>
<td>• Octopus</td>
</tr>
<tr>
<td>• Pineapple</td>
</tr>
<tr>
<td>• Plum</td>
</tr>
<tr>
<td>• Squid</td>
</tr>
<tr>
<td>• Turkey</td>
</tr>
<tr>
<td><strong>CHOLECYSTOKININ (CCK)</strong></td>
</tr>
<tr>
<td>Although CCK isn’t found naturally in food, studies show that consuming milk, soy protein (contains phenylalanine) may help increase the level of CCK circulating in the blood.</td>
</tr>
<tr>
<td><strong>CYCLO (HIS-PRO)</strong></td>
</tr>
<tr>
<td>This chemical affects satiety, hunger and behavior and is found in milk, yogurt, butter milk, shrimp, tuna, and nutritional supplements derived from milk and soy proteins.</td>
</tr>
<tr>
<td><strong>EXORPHINS</strong></td>
</tr>
<tr>
<td>These peptides are opposites of endorphins and act on brain opiate receptors to affect mood and suppress appetite. Foods that contain casein (milk protein), gluten (a wheat product), or zein (corn protein) all act as exorphins.</td>
</tr>
</tbody>
</table>

**SOURCE:** Brazilian Journal of Medical and Biological Research [online]
Researchers have made significant progress in understanding how nutrients encourage good health and prevent disease. They are also beginning to discover how drugs replace or mimic chemicals inherent in or derived from the foods we eat. Neuropharmacological research is being undertaken for hundreds of uses and the results show much promise for creating the drugs of the future.

One area of research focuses on serotonin’s receptor cites. Serotonin (5-HT) affects as many as seven different receptor cites. Scientists hope to design specific agonists and antagonists for each receptor based on its function. 5-HT1 receptors seem to be involved in muscle relaxation, specifically in the body’s cardiac and vascular systems. It prevents the release of certain neurotransmitters, and it creates effects in the central nervous system.

Four types of 5-HT1 appear to affect humans. Receptors for 5-HT1A, located in the central nervous system, are connected with sexual behavior, hypotension, food intake, and, possibly depression. This receptor may also produce hypothermia and be connected with reducing anxiety. Antagonists of 5-HT1B receptors inhibit aggressive behavior and food intake. 5-HT1C (renamed 5-HT2C) may regulate fluid in the brain and spinal chord, as well as affect the regulation of pain, sleep and cardiovascular function. 5-HT1D is being studied in connection with migraine headaches.

Receptors for 5-HT2 are being examined in connection with hypertension and migraine. Located in smooth vascular muscle, platelets, lungs, the central nervous system and the gastrointestinal tract, they appear to affect gastrointestinal and vascular muscle contraction, platelet aggression and depolarization of neurons. Antagonists of these sites may be useful as antipsychotic agents.

5-HT3 receptors, located mostly in peripheral and central neurons, seem to be involved in pain and the emesis reflex. These receptor sites are also being studied for use in treating migraines, anxiety, and cognitive and psychotic disorders.

5-HT4 receptors are involved in neurotransmitter release in the central nervous system, heart and gastrointestinal tract. In addition to studying the actual receptor sites, researchers are investigating micronutrients and how they affect those sites. These chemicals may enhance production, inhibit production, inhibit uptake or inhibit reuptake of neurotransmitters—all affecting their amount and action in our systems.

Research on selective 5-HT reuptake inhibitors (SSRIs) has been making news recently. By limiting chemicals that prevent serotonin reuptake, these drugs raise the levels of serotonin in the body. One use for SSRIs is for the treatment of depression. Scientists believe that depression may be caused by an abnormal function of norepinephrine and/or serotonin in the body, leading to a deficiency of these transmitters. Drugs such as Prozac, Zoloft and Paxil affect the reuptake of serotonin, but don’t affect other receptor systems, such as those for dopamine or histamines. SSRIs are being used to treat obsessive-compulsive disorder, and are being researched for Alzheimer’s disease, obesity, PMS, chronic pain, diabetic neuropathy, and to limit aggressive behavior and reduce alcohol intake.

CONCLUSION
With dozens of neurotransmitters and an exponential number of chemicals and receptor sites to investigate, the possibilities for new drugs—some of which could even prevent surgery—are endless. Among the conditions that scientists hope to affect through neuropharmacological research are anxiety and other mood disorders, such as schizophrenia and seasonal affective disorder, Alzheimer’s disease, ADHD, nicotine withdrawal, substance abuse, gastroesophageal reflux disease, stroke, hypertension, vascular disorders, heart disease, migraine, obesity, anorexia nervosa, bulimia, PMS, chronic pain, and nausea.

Continued research in neuropharmacology promises more precise, and possibly more natural, treatments to disease and dysfunction.
BRAIN POWER: THE PSYCHOLOGY OF FOOD

Obviously, physiological changes have an effect on our mood and desire for food, but our minds play an even more powerful role. Our emotions, dieting patterns, environmental cues, conditioned responses, and our desire for pleasure are psychological factors that trigger eating.

As humans, we seek pleasure and avoid or attempt to improve unpleasant experiences. When the human body receives a stimulus, it is conditioned to respond. Although people are aware of stimuli, they may choose not to respond outwardly, but their bodies must still respond to disperse the anger. Often, conditioned patterns in the brain's limbic system are formed as a result. Instead of getting angry at our managers or coworkers, we choose to busy ourselves with work. Our bodies are still trying to work out our anger and overcome its unpleasantness. When we choose to stop by the vending machine to grab a snack, the physiological response to food is recorded in connection with the anger. The limbic system remembers "when I'm angry, potato chips make me feel better." The next time we get angry and choose not to deal with the anger, our brains create a physiological craving for potato chips to respond to the anger.

We form many of these patterns as children. The limbic system remembers those patterns, long after we've forgotten those early experiences. For instance, 4-year-old Sara is tired and unhappy while shopping for groceries with her mom. Her mom buys her a candy bar, which she eats and feels better. The physiological reaction of her body to the chocolate and sugar and the corresponding pleasurable response is stored in her limbic system. If Sara often receives something sweet when she's unhappy, her limbic system becomes conditioned to crave sweets. As an adult, when Sara faces unpleasant circumstances, she may not even remember her childhood trips to the store, but she will still crave sweets to make her feel better.

Conditioned responses aren't the only psychological factors that lead us toward food. People also respond to emotions—anger, anxiety, boredom, depression, loneliness, even joy—by eating. Sometimes, a fear of an anticipated emotion or the feeling of being out of control urges food consumption. People use food to regulate their physiological feelings and ignore their psychological ones. Eventually, they find it difficult to tell the difference.

A TV commercial for some tantalizing treat, the aroma of fast food, easy access (sight) to a candy bar in the checkout lane, and eating out with friends can be situational eating triggers. Negative self-talk—self expectations, self criticism, excuses and rationalizations—can also lead to poor eating habits. Dieting, eating habits and types of medications can cause physiological changes, such as decreased blood sugar, which affect our moods. A change in body chemistry can trigger cravings for certain, often forbidden, foods. Too much caffeine or not enough food, for example, can cause low blood sugar levels and a desire for sweets. When people succumb to cravings, they often mentally punish themselves. Cycles of binging and dieting break down our self-esteem and leave people with feelings such as depression, embarrassment, guilt, and loss of control.

With so many psychological factors to influence our eating habits, it's no wonder that we've lost touch with our body's need for certain types of food. Often we use the physiological changes caused by certain foods to manipulate or deal with our psychological feelings.

REFERENCES