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Motivation

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Motivation is the study of the processes that cause animals and humans to exhibit varying sets of behavior at different times. Some examples of such behavior sets are eating, fighting, socializing, achieving, and studying. Traditionally, one distinguishes between biopsychological and sociopsychological approaches to the processes that cause these behaviors (Reeve, 1997). The processes addressed by the first tradition are principally physiological and those by the second tradition mainly cognitive. The biopsychological perspective has been particularly successful in the analysis of so-called biological motives common to animals and humans, such as hunger, aggression, or sex. The sociopsychological perspective has been effective in the analysis of so-called cognitive motives largely restricted to humans, such as power or achievement needs. To the extent that modern psychology has come to accept that all psychological processes are due ultimately to physiological activity, the division is now somewhat arbitrary. Nevertheless, explanations of biological motives, even when concerning humans, are mainly offered in terms of largely factual physiological mechanisms (neuronal activation, hormone secretions, etc.), whereas cognitive motives are mainly explained in terms of psychological constructs (intending, planning, executing, etc.). These constructs, modern neuroimaging techniques notwithstanding, can not yet be easily related with physiological events. This validates the dual approach offered in this chapter. Still, in some cases we have offered an integration of biopsychological and sociopsychological approaches, and other examples of such integration are given in Chapter 4. We look forward to further integration of these two approaches in the years to come.

11.1 THE BIOPSYCHOLOGICAL PERSPECTIVE

All organisms, including humans, are the product of biological evolution, a peculiar game that certain organic molecules with not perfectly self-replicating properties started up about four billion years ago. This observation is basic to the biopsychological perspective on motivation. Organisms are biological machines which are dedicated to the survival and reproduction of their genes, the present day descendants of these molecules (Dawkins, 1989). All behavior is the final product of a phylogenetic (evolutionary), ontogenetic (developmental), and physiogenetic (physiological) cascade of causes and effects. The biopsychological study of motivation is principally concerned with the last stages of this chain of events but at times it must also attend to their evolution and development. Genetics and

learning for example play a role in determining why some people over-eat and others under-eat, why some are aggressive and others timid, and so on. The modern biopsychological perspective, which originated with Claude Bernard and Charles Darwin during the nineteenth century, assumes that most behaviors of humans and other advanced animals are the consequences of a varied and interacting number of causal factors and processes, which can not be easily subsumed under one global theory or even a limited number of different theories. This is best conveyed by describing some of the mechanisms underlying the most salient biological motivation systems.

Thirst

The life functions of all organisms are based on physico-chemical reactions that take place in an aqueous medium and work best when there is a certain salt/water mixture. The circulation of some animals furthermore depends on a certain volume of blood. More than 90% of the human body consists of water, about a third of it in a chemically bound form. Water losses are continuously incurred through respiration, perspiration, urination, and defecation. Drinking water or very watery solutions is by far the main mechanism by which water deficits are compensated. Water saving through excretion of more concentrated urine, avoidance of dry foods, suppression of sweating (to the possible detriment of thermoregulation!), and other mechanisms can help temporarily but will not prevent death if water is not drunk within a couple of days (De Caro, 1986). Water swallowed wets the mouth. This stimulates chemo- and mechanoreceptors in the mouth by diluting the concentrated (salty tasting) and lesser (parched sensation) saliva secreted during water deprivation and relieves the drinking drive for some time. The sensory receptors send neural signals that reach an area of the hypothalamus responsible for integrating the neural messages about the lack or presence of water in the mouth and elsewhere. Its activity correlates well with the subjective feeling of thirst. However, filling the mouth with water and spitting it out again does not inhibit thirst for long. Only when water filling the stomach is sensed by mechanoreceptors and chemoreceptors is the neural center more lastingly inhibited. However, if the passage of the water to the intestine is blocked (in animals) thirst recurs after a while. A longer lasting quenching of thirst occurs only when the water is allowed to pass into the intestinal tract and from there into the blood. Here it replenishes the loss in blood volume which goes along with water deficits and increases the venous blood pressure sensed by baroreceptors near the heart. Loss of blood pressure is known to be an important elicitor of thirst because of the verbal report and drinking behavior by humans after massive bleeding. An increase in venous pressure decreases the hypothalamic thirst drive, but still not definitively.

A more lasting inhibition of thirst occurs when water-diluted blood reaches the anterior hypothalamus and water finds its way by diffusion into osmoreceptive neurons, swells them and causes them to fire neural signals that massively inhibit the neighboring hypothalamic thirst center. They are called osmoreceptors because they respond to the salt/water concentration differences between their outside and inside giving rise to osmotic pressure. In fact the same optimal salt/water concentration is vital for all body cells. The osmoreceptors act as samplers of that variable. We know that these cells are crucially important for the regulation of thirst because water-satiated animals will begin to drink copiously again if a minute quantity of concentrated saline solution is experimentally injected into the anterior hypothalamus. They overdrink drastically and as soon as the injection effect wanes they begin to urinate profusely. Generally, an excess of water intake can be compensated by an increased diuretic activity of the kidneys. This activity is regulated by many of the same factors that control drinking, but of course in an opposing manner. When their supply with blood decreases, the kidneys secrete a substance into the blood that activates the hormone angiotensin. Among other things this active form of angiotensin is capable of inducing drinking through special brainstem chemoreceptors that activate the hypothalamic thirst center. The hypothalamic osmoreceptors on the other hand do not only induce thirst but also lead to the secretion of the hormone vasopressin by the pituitary gland (hypophysis). Vasopressin reaches the kidneys via the blood circulation and acts to reduce their water excretion. The hypothalamic thirst drive activates the essential and largely innate swallowing of water, but of course only if water is directly available. Most often the quenching of thirst requires learned responses such as walking to a well and drawing water from it. Some drinking may also be motivated less by the osmotic needs of the body than by the fact that a solution contains substances such as caffeine or alcohol for which there is no real bodily need but which are capable of directly stimulating neural mechanisms responsible for a sensation of well-being or pleasure (see below).

Hunger

The life functions of all animals require the intake of carbohydrates, fats, proteins, vitamins, salts, and trace elements to replenish the loss of solid matter which occurs through excretion and respiration after metabolic turnover (Leeg, 1994). Deprivation of food induces the motivating state of hunger, which is stronger the longer the deprivation lasts. When we eat a meal, chemoreceptors and mechanoreceptors in the mouth, pharynx, and stomach signal to an area of the hypothalamus which functions as a satiation center. This area in turn temporarily inhibits a neighboring hypothalamic area functioning as a hunger center. It appears that inhibition is based on the synaptic transmitter serotonin and that hunger activation involves the synaptic transmitter noradrenaline. Fenfluoramine, a pharmacological blocker of noradrenaline, is used to control excessive eating by very obese individuals. When the food mass enters the intestine, chemoreceptors induce the secretion of the hormone cholecystokinin, which enters the blood stream and among other things, stimulates the satiety area of the hypothalamus. When food reaches the upper intestine, digestion has progressively broken down carbohydrates into glucose, fats into fatty acids and glycerin, proteins into amino acids, and trace elements have been freed from organic molecules. Vitamins are not modified and salts are dissolved. All of these substances enter the bloodstream and are transported to all the cells of the body where they are further metabolized in the service of the life-supporting functions.

We have already mentioned the need for salts in connection with the maintenance of a precise water/salt balance in the body cells. The regulation of salt appetite is mediated by the same osmoreceptors which are also important for thirst regulation; but more immediately it is simply the pleasant taste that salt confers to food which normally ensures that we take up enough salt. A fall in salt concentration signaled by the hypothalamic osmoreceptors might even cause a relative upgrading of the hedonic value of the taste of salt. Any excess in salt intake that might occur through this rough and ready mechanism causes the kidney to excrete more salt than it normally does, at the cost of extra water loss. This increased salt excretion is elicited by aldosterone, a hormone secreted by the adrenal glands. Exaggerated ingestion of salt provokes increased thirst. The drinking that normally follows restores the osmotic state of the body's cells but also indirectly enables the disposal of salt through increased urine production.

Glucose (blood-sugar) is the main metabolic fuel for the cells and is thus required in appreciable quantities, not least so by the nervous system. Nevertheless, its concentration in the blood should not exceed a certain measure because too much glucose has a poisonous effect, as in diabetes. To prevent this, glucoreceptors in the hypothalamus activate the satiety center inhibiting any further eating. Also, under the influence of the hormone insulin, the lack of which causes diabetes, the liver regulates blood glucose levels by converting it into the starch glycogen, which it stores. This glycogen is converted back into glucose, under the influence of the hormone glucagon, when the glucose level falls again. Both insulin and glucagon are secreted by the pancreas gland under hypothalamic neural control.

Fatty acids can also supply metabolic energy except in neurons that are fully glucose-dependent. The latter, however, can benefit from the fact that the liver can convert glycerin into glucose. Glycerin and fatty acids, recombined to fat, can however also be stored in special adipose cells located under the skin and elsewhere in the body. While storing fat, these cells secrete leptin, a hormone which has a satiating effect when it reaches the brain (Kalat, 1997). Some people may in fact be obese because their brain is genetically under-sensitive to leptin or because their leptin is chemically aberrant (Rosenzweig, Leiman, & Breedlove, 1999). However, there certainly are other causes of obesity, such as individual differences in the basal metabolic turnover controlled by the hormone thyroxin. The mechanism that causes fat cells to release fatty acids and glycerin, which serve as energy sources during extended physical exercise or long-term fasting, is not yet clear. Although we have left out many of details, it should be obvious that the intake of carbohydrates and fats is normally regulated homeostatically, ensuring the maintenance of a fairly constant body weight. Increased levels of metabolites of these substances are sensed and these signals cause a satiation of hunger. However, these post-resorptive satiation signals arise too late to be relevant for the loss of appetite that normally limits the size of a meal. The latter arises from the much faster feedback originating from the food passage through mouth and stomach.

There do not seem to be any sensors comparable to the glucose or leptin receptors which could ensure the separate regulation of ingestion of proteins, vitamins, and trace minerals. Less definite regulatory mechanisms seem to ensure that we eat enough of them. One such mechanism is our preference for varied rather than monotonous food. The pleasurable connotation of eating is much reduced if we persistently eat

the same tasting food. This habituation causes us to seek some variety in foods. There are some hypotheses about how this may be implemented neurally. Chances are that the varied diet that comes about in this manner ensures a balanced intake. There are also learning processes that help to ensure a balanced diet. Vitamin deficits are known to induce a feeling of sickness. Ingestion of food which contains the missing vitamin quite rapidly brings relief. Animals, and probably also humans, can associate this relief with the taste and odor of the food which they ate shortly before, and will then seek it out later. Conversely, animals and humans also learn to associate sickness with the taste and odor of food which they ate earlier. This aversion to the taste or odor of the particular food is virtually beyond conscious control. This device, of course, ensures the avoidance of poisonous foods.

It is possible that as in the case of salt appetite, deficits and excesses of particular substances or elements may affect states of the brain in ways that subjectively feel like cravings for or aversions to foods containing these substances. These states may be equivalent to the special hungers that we at times are able to identify in ourselves: a definite appetite for sweets, for meat, for vegetables, or some other kind of food. Childhood experience also influence taste preferences and might result in diet customs such as the eating of hot Indian curries or of American 'junk-food'. Cultural fashions can also lead to a conscious regulation of food intake with the aim of maintaining sportive fitness or social attractiveness. Anorexia, a life-threatening under-eating which arises often in adolescent girls, is exacerbated by a learned beauty-of-slimness fad. Culture often connects the simple act of eating and drinking with complex ceremonies (formal dinners, tribal feasts) that serve social needs more than the need for nutrition. Or again, the hunger motive may underlie complex cognitive operations such as organized hunting or commercial agriculture. But even at this sociocognitive level genetics can affect eating habits. Certain human populations are genetically unable to digest lactose (milk-sugar) as adults and therefore some of these groups bleed rather than milk their cattle.

Sleep and Wakefulness

The necessity of keeping oneself fed and hydrated obviously requires physical and mental activity. The satisfaction of most other motives usually also involves activity. Even when all needs seem satisfied there is still an intrinsic motive for intermittent activity. It serves to keep the neuro-musculo-skeletal movement apparatus in a fit condition. There is also an intrinsic exploration or curiosity drive that makes us survey the environment, inspect novel items, and generally acquire knowledge that may be useful later (Schneider & Schmalt, 1994). All this requires a state of wakefulness associated with a heightened responsiveness to external stimuli and a general readiness for behavioral action. But activity is also connected with increased chemical turnover and physical stress due to the drain on metabolic resources and wear on body structures. These must be replenished and repaired in periods of rest. In humans as well as in animals, the cycles of activity and rest are organized on the basis of a daily rhythm (Pinel, 1997). Diurnally active animals, humans included, tend to sleep at night and be awake during the day. In nocturnally active animals this pattern is reversed. Even when humans or animals are experimentally kept in a constantly lit, even-temperatured, sound-insulated, clockless, artificial environment they persist with an activity/rest cycle that is close to 24 hours in duration, the so-called circadian rhythm. It is driven by a neuro-humoral oscillator (biological clock) with the nucleus suprachiasmaticus and the pineal gland (epiphysis) as interacting elements. The basic rhythm, however, is modulated by homeostatic processes. Temporary deprivation of sleep or activity is partly compensated by a relative lengthening of subsequent sleep or activity phases.

Wakefulness is largely determined by the activity of a dense network of neurons along the axis of the brainstem. It receives collateral signals from all sensory stimuli and is capable of activating most cortical circuits. This is reflected by high frequency, small amplitude oscillations seen in electroencephalograms (EEG). As a person becomes drowsy and falls asleep, EEG recordings are dominated by low frequency, large amplitude oscillations. This slow wave sleep is periodically interrupted by episodes of EEG activity very similar to that seen during waking, although the person remains asleep. Rapid eye movements (REM sleep) can be observed through the sleeping person's closed eyelids. If a person wakes up during this phase she/he mostly reports having been dreaming. Deprivation of REM sleep for a night increases the number and length of REM sleep phases during the next night. This indicates that the organism needs REM sleep. The function of this dream sleep is not totally clear but animal studies have suggested that its prevention interferes with the consolidation of memories. It may serve to reorganize cognitive information that accumulates during wakefulness. Both types of sleep and their alternation are controlled by nuclei of the midbrain and thalamus, where the

synaptic transmitter serotonin may be involved together with the epiphysial hormone melatonin in inducing the basic sleeping state. The addition of the transmitter acetylcholine induces REM episodes whereas wakefulness state may be maintained by the synaptic transmitter noradrenaline. Noradrenaline and serotonin have already been implicated in the regulation of hunger and satiety, and acetylcholine is among other things the transmitter that acts at the neuro-muscular endplates. Thus the same transmitters are acting in the regulation of different behaviors in different parts of the nervous system. Certain affective disorders are associated with sleep-irregularities and precisely timed doses of melatonin can bring relief. In any case, disturbance of the various mechanisms of sleep results in symptoms such as insomnia, sleep-walking, or narcolepsy.

Aggression and Fear

Survival and reproduction are dependent upon individual organisms securing environmental resources. When resources are freely available, as for example water in a humid country, there is no need to fight for them. However, food acquisition by predatory species entails hunting, and the prey may resist. Both predators and prey may engage in aggressive attack and defensive fleeing during such encounters. This agonistic behavior is often similar to that seen in intra-specific interactions, as individuals of the same species often have to compete for resources such as food or shelter. Intra-specific antagonism may also occur during competition for social resources, such as mating partners or allies. Such agonism often develops over prospective resources such as winter-barren land in expectancy of its summer fertility, or over social status in expectancy of prime access to all kinds of resources. Moreover, agonistic behavior may be supported by social groupings such as families, tribes, or nations. These conflicts take on the character of wars as the size of the groups involved increases and as weapons potentiate the group's power to injure and kill. Xenophobia, sectarianism, obedience, and fanaticism are some cognitive/cultural factors that can exacerbate aggression.

Fighting is connected with risk of injury and thus aggression is often balanced by fear. This frequently results in threat behavior. The individual development of agonistic motivation is driven by genetically determined behavioral dispositions which are modulated by experience. It is easy to breed genetically increased aggressiveness; fighting fish, cocks, and dogs being examples. Children probably become more aggressive if they discover that aggression is more often followed by reward than by punishment. Agonistic behavior is mainly controlled by non-homeostatic mechanisms, triggered by situational factors such as pain, offence, jealousy, or frustration (Renfrew, 1997). Nevertheless, deprivation from channeled, socially accepted aggressive outlets such as competitive sports or verbal disputes might lead to increased aggressive drive in at least some individuals. Frustrated children are more likely to behave aggressively to a bystander if they have previously experienced an aggressive scene than if they have not. Sensory information about situations provoking anger or fear is apparently transmitted by a special thalamo-amygdalar pathway. Stimulation through electrodes implanted into a neural circuit that extends from the amygdala through to the nucleus striae terminalis, the medial hypothalamus, and the medial midbrain elicits agonistic behavior in animals. In humans, responses such as slapping a medical attendant, accompanied by an angry facial expression, were observed when stimulation of the amygdala was necessary as a pre-surgical exploration. Male animals and humans generally are more prone to show aggression than females. In animals this is mediated by the hormone testosterone (Rosenzweig, Leiman, & Breedlove, 1999). Castrated male animals display less aggression than normal males and in both humans and animals testosterone injections heighten readiness for aggression. In female animals the pregnancy hormone progesterone appears to increase readiness for defensive aggression when offspring are threatened.

Sex and Parenthood

The central property of genes is replication. Sexual reproduction, a common complication of gene replication, is advantageous but also costly. Advantageous, because it enables the mixing of parental genes and thus guarantees offspring diversity. This increases the chance that some offspring will succeed in a permanently changing environment. Costly, because in many species it requires the synchronized coupling of ovum and sperm within the body of the female who then also bears the costs of pregnancy and in mammals, lactation. Sex is energetically cheap for males but they must compete for females. Females must be choosy about males, who are co-responsible for the genetic quality of their offspring. Females assess the quality of males and their willingness to continue investing in the common progeny after mating. The dominance of monogamy in human cultures is likely derived from the poor prospects single mothers

have of raising healthy progeny under rough conditions. In many species, a maintained pair bond is essential for successful parenting. Males' reproductive efforts could be wasted if they do not contribute to the welfare of their offspring. Love probably strengthens this pair-bonding. Among humans, cultural customs produce much variation in courtship and partnership habits. The role that off-reproductive mating plays in pair maintenance is probably why sex is connected with intense pleasure. The dissociation of sexual activity from reproduction by contraceptive technology has reduced sex to a largely hedonic motive.

The existence of women and men is determined by the sex chromosomes. An XX fertilized ovum is predetermined to develop into a female, an XY fertilized ovum to develop into a male. However, this only determines whether the embryos develop female or male reproductive organs. Occasionally occurring XO (only one X chromosome) individuals develop neither testes nor ovaries. The differences in female and male behavior, or rather in the details of brain structures that control these differences, come about through a more complicated mechanism. A few weeks before birth the testes of male (human) embryos secrete testosterone which biases brain development in a male direction. Female embryos have no such burst of testosterone, nor indeed of estrogen, as the ovaries exhibit no perinatal activity. This allows the relevant brain structures to go on developing undisturbed (Kalat, 1997). Quite early in life these brain differences have the consequence that girls tend to learn to speak earlier than boys and boys are more prone to show rough-and-tumble (fighting) play than are girls. At puberty (testes secreting again testosterone, ovaries secreting for the first time estrogen), the brain differences determine that most girls begin to develop an interest in boys and most boys are attracted to girls. Why some do not and become homosexual rather than heterosexual is not yet altogether clear. For a proportion of male homosexuals it seems likely that brain differentiation is partly blocked by a mutant gene located on their X chromosome.

Readiness for the sex act, or libido, is in part influenced by the presence of circulating testosterone in males and, to a lesser extent, circulating estrogen and progesterone in females. Erotic stimuli and stimulation bring about mating readiness in both sexes. In males, more than females, this arousal is helped by the novelty of erogenous stimuli. This so-called Coolidge effect is an easily demonstrable phenomenon in male animals. They appear to tire if the same female is repeatedly presented but show renewed sexual efforts if a new receptive female is presented. Sexual arousal results in penile and clitoral erections and lubricatory gland secretions. This entails the expansion of vein irrigated cavernous tissue and glandular contractions under the control of nervous signals from the brain. Mechanostimulation of penile and vaginal tissue results in ejaculation of sperm in the male and contractions of the vaginal walls in the female. Both events are associated with a sensation of pleasure (orgasm), due to neural signals transmitted to the brainstem reward areas. The contractions associated with female orgasm may be coupled with a particularly effective transport of sperm to the uterus. The release of mature ova by the ovary occurs at four-weekly intervals and menstruation is connected with the post-ovulatory sloughing off of tissue lining the uterus. This tissue develops within the cycle in preparation for the possible implantation of a fertilized ovum and is rejected if such implantation does not take place. A complicated interaction between hypophysial and ovarial hormone secretions regulates the ovulatory/menstrual cycle. In contrast to many animals, the cycle has little influence on the day-to-day sexual receptivity of the human female. Even the widespread intercourse bar connected with menstrual bleeding is probably culturally sanctioned and not physiologically determined (Abramson & Pinkerton, 1995).

When implantation occurs and gestation begins, female libido is at first not inhibited despite a much changed hormonal situation. This suggests that sexual receptivity in the human female, additionally to fertilization also supports pair-bonding. As birth approaches, the hypophysial hormone prolactin readies mammary gland tissue for milk production. Emotional attachment to the baby seems to be facilitated by mechanical stimulation of the uterus wall during child birth. When the baby nurses, an innate response activated by low blood glucose, this stimulates through a quite direct neural pathway the secretion of oxytocin, another hypophysial hormone that causes the release of milk.

Parental care is the most basic form of genetically driven altruism (Rosenblatt, 1996). Although genes are essentially selfish in their operations, the evolutionary game allows that carriers of identical genes (e.g., offspring, kin) can benefit from genetically instructed altruistic behavior. The gene sets of children are of course combinations of half-sets of maternal and paternal gene copies. They can rely on being the preferred recipients of altruistic parental attention. Parents are prepared to bestow advantages on their children at cost to themselves but mothers more so than fathers. Mothers can nearly always