Biopsychological Aspects of Motivation

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1. A primer on biopsychology and its methods

As a discipline, biopsychology aims to explain experience and behavior based on how the brain and the rest of the central nervous system work. Biopsychological approaches to motivation, then, try to explain motivational phenomena based on an understanding of specific functions of the brain. Work in this area is primarily done using mammalian animal models, such as rats, mice, and sometimes primates, too, with the assumption that the way motivational processes and functions are carried out by the brain is highly similar across related species and that findings obtained in other mammals will therefore also hold for humans.

When studying motivational processes, biopsychologists often use lesioning (i.e., selective damaging) techniques to explore the contributions of specific brain areas or endocrine glands to motivational behavior, reasoning that if destroying a specific brain area or gland alters a motivational function, then the lesioned substrate must be involved in that function. Other techniques often utilized in this type of research include direct recordings from neuron assemblies in the behaving animal to determine, for instance, which brain cells fire in response to a reward, and brain dialysis, which allows the researcher to examine how much of a neurotransmitter is being released in a behaving animal in response to motivationally relevant stimuli. Finally, biopsychologists also frequently use pharmacological techniques to, for instance, increase synaptic activity associated with a specific neurotransmitter by administering a transmitter agonist (which mimics the action of the neurotransmitter) or decrease synaptic activity by administering a transmitter antagonist (which blocks neurotransmitter activity). This is often done locally in the brain to determine the contribution of specific neurotransmitter systems to a function subserved by a circumscribed brain area. Frequently these methods are combined with each other, and they are almost always combined with behavioral or learning paradigms designed to reveal the contribution of a brain area,

neurotransmitter, or hormone to specific aspects of motivation (e.g., instrumental learning, responding to reward).

One major advantage of the biopsychological approach to motivation is that it can go beyond circular explanations of motivation, which often arise if only behavioral measures are used to infer causal effects of motivation. For instance, one could explain the observation of aggressive behavior (the explanandum) by the presumed existence of an underlying aggression drive (the explanans), which is in turn inferred from the observation of aggressive behavior. As long as there is no independent means of assessing the presumed aggression drive, the explanation for aggressive behavior will remain circular (e.g., "Why is he shouting at Mary?" "Because he has a strong aggressive disposition." "How do you know that's the case?" "Because he's shouting at Mary"). In contrast to purely behavioral accounts of motivation, biopsychologists would argue that activity in certain brain regions or the release of certain transmitters and hormones, in interaction with environmental cues, precede or cause aggressive behavior, thus separating the explanandum from the explanans. For instance, one very successful account of aggressive behavior, Wingfield's challenge hypothesis (Wingfield et al., 1990), holds that increased levels of testosterone predispose animals to assert their dominance, but only if their dominance is challenged by competitors and in certain situational contexts, such as breeding seasons. Clearly, the explanans (testosterone) is not only more specific and concrete here than the postulation of a putative "aggression drive", it is also clearly separated from the explanandum (aggressive or dominant behavior), and its causal relationship to the explanandum can be studied empirically by, for instance, removing the animal's gonads, administering testosterone, or a combination thereof.

What animal models of motivated behavior can not reveal, however, is the relationship between the brain and subjective states that accompany and characterize some aspects of motivation. Animal research is therefore increasingly complemented with studies on humans that allow

researchers to relate measures of brain activity or physiological changes to both behavior and subjective states. With the advent of sophisticated brain imaging methods, such as functional magnetic resonance imaging (fMRI), that provide relatively high temporal and spatial resolution in assessments of the active human brain, biopsychological research on motivational and emotional processes has undergone both an unprecedented growth spurt and a transformation, giving rise to the new and burgeoning field of affective neuroscience (Panksepp, 1998).

In the present chapter, we will review the current status of biopsychological research, focusing on the key brain systems and processes that have been found to mediate motivational phenomena in studies on animals and humans. Our aim is to provide the reader with an overview of the key substrates of motivation and emotion and highlight some important current findings and developments in the field. For more comprehensive and detailed treatments of the biopsychology of motivation, we refer the reader to the excellent books by LeDoux (2002), Panksepp (1998), Rolls (1999), and Toates (1986).

2. Hallmarks of motivation

To make sense of biopsychology's contributions to the understanding of motivation, we feel it is important to first provide an overview of the core phenomena and processes of motivation on which biopsychologists tend to focus. This will equip us with the proper conceptual framework to understand the biopsychological contributions to the science of motivation. We will therefore cover in the following what biopsychologists consider to be hallmarks of motivation, before we move on to describe key brain structures and processes involved in motivation.

2.1 *Motivated behavior comes in two basic flavors: approach and avoidance motivation*

The first key characteristic of motivated behavior is that it can be aimed at attaining a pleasurable incentive (= reward) or avoiding an aversive disincentive (= punishment). This hallmark of motivation has taken a central role in the conceptual frameworks of motivation proposed by major

theorists in the field (e.g., Atkinson, 1957; Carver & Scheier, 1998; Craig, 1918; Gray, 1971; Mowrer, 1960; Schneirla, 1959) and today is an important and active area of research in biopsychology and the affective neurosciences. While an organism in the approach motivation mode works to decrease the distance from a desired goal object (e.g., prey, a food pellet, or a good exam grade) until it is reached, an organism in the avoidance motivation mode needs to increase the distance from an aversive goal object or state (e.g., predator, starvation, bad exam grade). Avoidance of a disincentive can happen in two fundamentally different ways: active avoidance and passive avoidance.

Active avoidance characterizes the behavioral strategy of actively executing behavior that is instrumental in bringing the individual away from the disincentive. This can be as simple as fleeing from the dangerous object and as complex as spending a great deal of time learning for a biochemistry exam in order to avoid a bad grade. Some theorists have posited that avoidance motivation is a particular inefficient form of motivation, because it is never clear to the individual how far is far enough (Carver & Scheier, 1998). Approach motivation terminates upon contact with the goal object or state; but when does avoidance motivation stop? When a predator is 100 yards away? When it is out of sight? But when it is out of sight, how does the organism know it is far enough removed? In other words, it could be argued that avoidance motivation is problematic because, on the one hand, it requires the presence of the disincentive as a reference point to enable the organism to gauge its spatial or psychological distance to it and, on the other hand, does not provide a clear-cut criterion when the distance has become large enough to terminate behavior aimed at avoiding the feared goal object or state.

Based on earlier work by Mowrer (1960), Gray (1971) has proposed that one way out of the active avoidance dilemma is to conceive of objects or places that during past learning episodes have been associated with non-punishment as safety signals that have actual reward value. In other words,

instead of running away from a feared object, the organism reframes the situation, and in a sense switches from avoidance to approach motivation by reorienting its behavior with reference to a safe and thus rewarding object or place. This also solves the problem of how far away the individual needs to be from the aversive object in order to feel safe: as soon as the safety object or place is reached, the motivational episode can be terminated. A classic study by Solomon and Wynne (1953) illustrates this switch from avoidance of danger to approach to safety: Solomon and Wynne trained dogs to jump from one compartment to another as soon as a stimulus signaling impending foot shock appeared. Remarkably, most dogs not only learned to avoid shock by jumping to the safe compartment within very few trials, but also were amazingly resistant to extinction: some continued to traverse over to the safe compartment upon presentation of the warning signal for more than 600 trials! Equally remarkably, they quickly ceased to show any sign of fear after they had learned how to cope with the threat of shock.

The other mode of avoidance motivation is *passive avoidance*. The following are all examples of this behavioral manifestation of motivation: an animal ceasing all foraging behavior and keeping very still when it spots a predator; a rat that learns to stop bar-pressing in the presence of specific discriminatory stimuli, because bar-pressing then reliably produces foot shock; and a student refraining from participating in a class discussion in order not to be ridiculed for saying something stupid. The fundamental difference between passive avoidance on the one hand and active avoidance and approach on the other is that in the case of the former, behavior is *inhibited* in order to avoid a certain goal state or object, whereas in the case of the latter two, behavior is *executed* in order to avoid or attain something. Thus, active and passive avoidance represent behaviorally very different solutions for dealing with the same problem, namely, avoiding a punishment.

2.2 *Motivation consists of two distinct phases*

Motivation consists of relatively distinct segments or phases that serve different functions,

and, as we will see, biopsychological studies strongly support this view. Most theorists agree that the motivational process needs to feature at least two consecutive elements: a *motivation phase* during which the organism works to attain an reward or avoid a punishment and a *consummation phase* in which the outcome is evaluated, that is, in which the organism consummates and determines the "goodness" of the reward or assesses whether a danger or punishment has been successfully avoided (e.g., Berridge, 1996; Craig, 1918). Thus, an animal may become motivated to eat either because it sees a tasty morsel, because of a state of nutrient depletion indicated by hunger, or a combination of both, and it starts working towards the goal of obtaining food. The motivation phase can be as simple as taking a few steps towards a food trough and starting to eat and as complex as hunting down an elusive prey in the jungle. Note also that the motivation phase is characterized by observable behaviors (instrumental activity to attain a reward or avoid a punishment) and an affective-motivational state, which in humans can be characterized subjectively with such terms as craving, longing, or being attracted to (or repelled by) the goal object, but in animals can only be inferred from behavior. Berridge (1996) has labeled this phase of the motivational sequence *wanting*, and differentiates it from *liking*, that is, the evaluation of the hedonic qualities of the reward (or punishment) accompanying the consummation of an incentive.

While most people intuitively assume that you want what you like and vice versa, research indicates that the two phases of motivation are actually dissociable. For instance, drug addicts feel compelled to take "their" drug even though there is no longer any pleasure in taking it (wanting without liking; cf. Robinson & Berridge, 2000). Conversely, people subjectively and objectively respond with signs of liking to tasty food, regardless of whether they are hungry or have just eaten a meal – thus, liking can stay the same despite strong differences in wanting (Epstein et al., 2003). As we will see later, the two phases of motivation are also associated with separable brain systems.

2.3 *Many qualitatively different types of rewards can give rise to motivation*

Many different types of reward (or punishments) can give rise to motivated behavior, and what motivates behavior can vary across individuals and also within an individual across time. Rewards are often conceived of by learning psychologists as unconditioned stimuli towards which all Pavlovian and instrumental learning is ultimately directed. Types of reward and the motivational systems associated with them that have been studied for a long time in biopsychology include food in the case of feeding and hunger motivation, water in the case of thirst, orgasm in the case of sexual motivation, social closeness in the case of affiliation motivation, and being on top of the social hierarchy in the case of dominance motivation. Social and personality psychologists, who study humans instead of animals, would add other candidates to this list, such as achievement motivation, in which mastery experiences are rewarding; intimacy, in which deepening one's relationship to a specific other is rewarding; and power motivation, in which having impact on others is experienced as rewarding (this is similar to, albeit more subtle than, dominance motivation as studied in animals). One additional fundamental motivational system, curiosity or exploration, does not even seem to have a specific reward associated with it, except maybe the discovery of *any* kind of new and pleasurable unconditioned stimulus. Some of these rewards can even be differentiated into several kinds of specific rewards. For instance, research on hunger and feeding reveals that the amount of protein, fat or carbohydrates contained in food all represent separable kinds of reward that organisms are differentially sensitive to, depending on what kind of nutrient they most urgently need.

While these are all very different kinds of rewards, fulfilling a variety of functions related to the organism's individual and genetic survival, they are also similar in the sense that animals (including humans) want them, feel compelled to attain them repeatedly, and will show invigorated responding in situations in which their behavior could lead to the attainment of a reward. Whether an individual feels more or less wanting for a given reward depends, of course, on the individual's need state (e.g., how long has it been since s/he last ate?), as well as on the individual's liking of that

reward or, in the parlance of human motivational psychology, whether the individual has a *motive* for attaining a given reward (McClelland, 1987). The more he or she responds with pleasure to obtaining the reward, the stronger the motive to seek it out in the future.

2. 4 *Motivation is dynamic*

Another key feature of motivation emerges from the interplay of wanting and liking, namely, that motivation is a dynamic process. Even the most dedicated glutton, for instance, won't spend all available time eating, but switch to the pursuit of a different kind of reward after he or she has eaten to satiety. However, because the glutton enjoys food so much (= high liking for the reward), he or she will eventually be quicker to become motivated to eat again and thus eat with greater frequency or intensity than a person who takes little pleasure in the reward of tasty food. Moreover, one and the same reward can change in the degree to which it is liked simply as a function of how much of it an individual has already consumed. One piece of chocolate, for instance, can be quite tasty and rewarding. But even a chocolate connoisseur will probably only experience disgust if he or she is forced to eat 2 pounds of chocolate at once. Cabanac (1971) has termed this phenomenon *alliesthesia*, that is, changing subjective evaluation of the same reward over time. Alliesthesia is assumed to track the usefulness of a given reward as a function of the changing needs of the organism. Clearly, food is highly useful, and thus very pleasant, for a semi-starved individual, but becomes less useful, and thus less pleasant, for someone who has already eaten to satiety.

Thus, motivation for a particular type of reward waxes and wanes, depending on the recency of reward consummation, the degree to which the reward is experienced as pleasurable, but also other factors, such as the presence or absence of cues in the environment that predict the availability of a particular reward as well as the strength of competing motivational tendencies. The dynamic nature of motivation, which can even be mathematically modeled (cf. Atkinson & Birch, 1970), is clear to anyone who studies motivation through observation in humans and other animals, but has frequently

been overlooked by personality trait researchers who emphasize the consistency of behavior over time (for a discussion of this issue, see Atkinson, 1981).

2.5 *Motivation can be need-driven, incentive-driven, or both*

Obviously, motivation is often triggered by the physiological needs of the organism. Falling nutrient levels induce hunger; increasing blood saltiness induces thirst. As a consequence, we seek for food or drink to quench the need. But somewhat less obviously, motivation can also be triggered solely by cues in the environment. Such motivation-arousing cues are called *incentives*, and a good illustration of incentive motivation is the salted-peanut phenomenon. Imagine yourself sitting in front of the TV after a good, filling dinner. Next to you, you find a bowl of salted peanuts. You are actually full, but why not try one? After you have eaten one and found it quite tasty, your hand goes back to the bowl to fish for more, and half an hour later, you have eaten the contents of the entire bowl, *although you were not even hungry to start with!* In this case, it was some rewarding aspect about the peanuts themselves that made you eat them, not an unsatisfied physiological need for nutrients. Thus, how pleasurable a reward is depends not only on our need state but also on the nature or quality of the reward itself, which can sometimes motivate us even though we are not experiencing any need at all.

This principle is illustrated by an experiment in which the independent effects of incentive and need factors on food intake behavior were studied (Panksepp, 1998; cf. Fig. 1). Animals' need state was manipulated by allowing them to eat regular chow whenever they wanted (ad lib group; low need state) or by starving them for 24 hours (high need state). When tested on their food intake, half of all animals were offered regular chow (low incentive value) or hamburger (high incentive value). Among animals that were offered chow, there was a clear effect of need state: hungry, food-deprived rats ate more than ad lib-fed rats that had had access to chow all the time. But the results also document a clear incentive effect on motivation to eat: regardless of need state, *all* animals

gorged themselves on the hamburger treat. These findings illustrate that motivation can sometimes reflect differences in need state (in the chow condition) and sometimes differences in the incentive value of a goal object (in the hamburger condition).

Of course, need- and incentive-driven motivation can also go hand in hand. Incentives can be more attractive, rewarding or pleasurable when a person is in a high need state and less so when the person is in a low need state. For instance, a hungry person may perceive and experience a bland piece of bread as deliciously tasty, whereas that same piece of bread may be considerable less attractive when the person is in a state of satiety.

2. 6 *Motivation is characterized by flexibility of cue-reward and means-end relationships*

Motivation drives, and in turn is influenced by, Pavlovian and instrumental learning processes. Hungry rats are quicker than satiated rats to learn that a certain sound (the conditioned stimulus, or CS) reliably predicts the presentation of a food pellet (the unconditioned stimulus, or US), and anxious people (i.e., individuals who are particularly motivated to avoid punishments) are quicker to learn that a particular face (CS) presented on the computer screen predicts an aversive noise (US) presented on their headphones (Pavlovian conditioning; e.g., Morris, Öhman, & Dolan, 1998). Similarly, hungry rats show better learning of bar-pressing behavior if the bar pressing produces a food pellet. Anxious people are better at learning to respond to a complex stimulus sequence presented on the computer screen if speedy responding to the stimuli prevents the loss of points or money (instrumental learning; e.g., Corr, Pickering, & Gray, 1997). And power-motivated individuals show enhanced implicit learning of a visuomotor sequence if its execution leads to the presentation of a face with a low-dominance expression and to impaired learning if the sequence is followed by a face with a high-dominance expression (Schultheiss, Pang, Torges, Wirth & Treynor, 2005).

Learned cues can, in turn, trigger motivation. This phenomenon is powerfully demonstrated

in the case of post-traumatic stress disorder (PTSD; Brewin, Dalgleish, & Joseph, 1996). People who suffer from PTSD have typically acquired this disorder during a traumatic episode in their lives. One key characteristic of PTSD is that a stressful reliving of the traumatic event can be triggered by any kind of stimulus that happened to be present in the original, PTSD-inducing situation. For instance, a sudden loud noise can lead to a powerful panic response in a person who has been in combat and has learned to associate this noise with the imminent danger of enemy fire, whereas the same noise will only lead to a slight startle response in a person without PTSD. Thus, for the PTSD patient, sudden loud noises are conditioned danger signals that give rise to a powerful fear response. On the brighter side, mice and rats that have learned to associate a particular place in their environment with access to a sexual partner will show hormonal changes characteristic of sexual motivation whenever they revisit this place (Graham & Desjardins, 1980). Here, the place is the conditioned cue that elicits the motivational state.

In a sense, Pavlovian and instrumental learning processes make motivation possible in the first place, because they free the individual from fixed, instinctual responses to built-in trigger stimuli, allowing the individual to become motivationally aroused by wide variety of stimuli that predict the availability of a reward and develop an adaptive repertoire of behaviors that are useful for obtaining a reward. Although these learning processes are not entirely unconstrained in many species and domains of behavior (e.g., Seligman, 1970), they nevertheless make goal-directed behavior enormously flexible and adaptive.

2.7 *Motivation has conscious and nonconscious aspects*

Traditionally, biopsychology has not dealt with the issue of consciousness in the study of motivation, because most research in this field was carried out in animals lacking the capacity for symbolic language and introspection. Almost by default, then, the majority of biopsychological accounts of motivation assume that consciousness is not necessary for goal-directed, reward-seeking

behavior. At the intersection of biopsychology, neuropsychology, psychopharmacology, and social psychology, however, researchers did examine the issue more closely, but still came to essentially the same conclusion. For instance, Berridge (1996) reviewed evidence suggesting that even for as fundamental a motivational system as feeding, humans rarely have accurate insight into what drives their appetites, or what makes them start or stop eating -- self-reports of motivation often contradict behavioral data. Similarly, Rolls (1999) has suggested that most of the brain's considerable power for stimulus analysis, cognitive processing, and motor output are primarily in the service of implicit (i.e., nonconscious) motivational processes, representing the organism's various needs for physical and genetic survival, whereas conscious, explicit motivation is the exception from the rule in the brain, depends on language and serves primarily to override implicit processes.

Berridge and Robinson (2003) have recently pointed out that implicit/explicit dissociations exist not only in the domain of motivation, but can also be documented for emotion and learning. For instance, learning and memory can be divided into declarative (conscious, explicit) and nondeclarative (nonconscious, implicit) processes, with the former including memory for events and facts and the latter including Pavlovian conditioning and instrumental learning (Squire & Zola, 1996). In this context, it is worth noting that the human brain has evolved for a long time in the absence of symbolic language and hence the ability to report on mental states. So perhaps it is not surprising that language-based functions are the newcomers in an otherwise already highly developed and adaptive brain and that many motivational, emotional, and cognitive functions, which ensured our prelinguistic ancestors' survival, do not depend on or require conscious introspection.

On the other hand, humans clearly are able to explicitly set goals and pursue them in their daily lives. If we were governed exclusively by phylogenetically shaped motivation needs, it would be, for instance, almost inconceivable that any human would ever return to the dentist's office after having experienced the pain of a root canal procedure. Of course, conscious regulation of

motivational processes is not restricted to overriding raw motivational impulses and needs, but also extends to the formulation of short- and long-term goals and the elaboration of plans to attain them. Traditionally, contributions of the brain to these uniquely human faculties have been studied by neuropsychologists and neurologists, who examined the role of frontal lobe lesions in higher-order brain functions in humans. Presently it remains unclear to what extent brain structures subserving conscious self-regulation and goal pursuit are integrated with, dissociate from, or interact with brain structures subserving implicit motivational processes and systems. The elucidation of this issue will be an important task for affective neuroscience in the coming years.

2.8 Summary

Biopsychological research focuses on a set of intersecting properties of motivation. Motivation can be directed towards a positive incentive (approach motivation) or away from a negative incentive, either through behavioral approach towards a safe place (active avoidance) or suppression of behavior until the danger is over (passive avoidance). The motivational process consists of two phases, one that involves decreasing (or increasing) one's distance from an incentive (wanting) and one in which, once the incentive has been reached, the hedonic qualities of the incentive are evaluated (liking). Different types of incentives (e.g., novelty, food, water, sex, affiliation, dominance) can give rise to motivated behavior. Motivated behavior changes its goals dynamically, depending of how recently a given need has been satisfied and what kinds of incentives are available in a given situation. Motivation can reflect the presence of a strong need state (e.g., energy depletion); it can be triggered solely by strong incentives, even in the absence of a profound need (pure incentive motivation); or it can be the product of the confluence of a need state and the presence of suitable incentives. Motivation is characterized by flexibility of cue-incentive and means-end relationships and drives, and in turn is influenced by, Pavlovian and instrumental learning processes. Finally, biopsychological approaches to motivation do not assume that motivation requires

conscious awareness, but acknowledge that in humans, specialized brain systems support the conscious setting and execution of goals.

3. Brain structures generally involved in motivation

While different motivational needs engage different networks of brain areas and transmitter systems, some systems fulfill such general, fundamental motivational functions that they are recruited by almost all motivational needs. This is particularly true of the amygdala, the mesolimbic dopamine (DA) system, and the orbitofrontal cortex (OFC) (cf. Cardinal et al., 2002). We will also examine the lateral prefrontal cortex (LPFC), one of several brain structures that are involved in the regulation of motivational impulses. Figure 2 provides an overview of the location of these structures in the human brain.

3.1. Amygdala: recognizing rewards and punishments at a distance

The amygdala is an almond-shaped structure located in the temporal lobes of the brain. Its critical role in motivational processes was first documented by Klüver and Bucy (1937, 1939) who observed a phenomenon in monkeys whose temporal lobes had been lesioned that they termed "psychic blindness". Klüver and Bucy described what they observed in one monkey: "The […] monkey shows a strong tendency to approach animate and inanimate objects without hesitation. This tendency appears even in the presence of objects which previously called forth avoidance reactions, extreme excitement and other forms of emotional response." (1939; p. 984). Thus, loss of the amygdala leads to an inability to assess the motivational value of an object from afar ("psychic blindness") and the monkey needs to get in direct contact with the object to figure out its significance. Also notable is the loss of fear accompanying amygdala lesion.

Research over the last 60 years has led to a much more nuanced understanding of the "psychic blindness" phenomenon observed by Klüver and Bucy. The amygdala turned out to be a key brain structure for Pavlovian conditioning. It helps to establish associations between stimuli that

do not initially carry any motivational meaning with unconditioned rewards or punishers, if the former reliably predict the latter (LeDoux, 1996). Thus, an intact amygdala enables an individual to learn that, for instance, the sight of a banana (conditioned visual cue) predicts a pleasant taste if the banana is eaten (food reward), whereas the sight of a rubber ball does not predict a rewarding taste if the ball is taken into the mouth. Similarly, the amygdala is necessary for rats or humans to learn that a visual stimulus like a blue light predicts a shock, and thus to express fear to the blue light. With an intact amygdala, CS-US associations can be learned within a few trials, and sometimes even based on a single trial; with a lesioned amygdala, humans and animals need hundreds of trials to learn such associations and may even fail to acquire them altogether.

The amygdala consists of several, highly interconnected nuclei (i.e., groups of neuronal cell bodies that serve similar purposes), and two of them are particularly important in emotional and motivated responses to CS and US (cf. Fig. 3; LeDoux, 1996, 2002). Through its *central nucleus*, the amygdala influences primarily *emotional reactions* mediated by hypothalamic and brainstem structures. For instance, the central nucleus triggers the release of stress hormones (e.g., cortisol) through its effect on the endocrine command centers in the hypothalamus; increases arousal, vigilance, and activation through its projections to major neurotransmitter systems (e.g., dopamine); and activates various autonomic nervous system responses (e.g., galvanic skin response, pupil dilation, blood pressure). Through the *basolateral nucleus*, the amygdala influences *motivated action* through its projections to the nucleus accumbens, a key structure of the brain's incentive motivation system (see below). If the central nucleus is lesioned, animals are still able to show motivated responses (e.g., bar-pressing for food) in response to a CS, but preparatory emotional responses are impaired (e.g., salivation is lacking). Conversely, if the basolateral amygdala is lesioned, animals will still show an emotional response to a CS, but fail to learn instrumental responses to elicit (or avoid) the presentation of a CS (Killcross, Robbins, & Everitt, 1997).

Another important feature of the amygdala is that it receives input from virtually all stages of sensory processing of a stimulus (LeDoux, 1996). This starts at the earliest stages of stimulus analysis at the level of the thalamus, which can give rise to a "knee-jerk" amygdala response to crude stimulus representations (e.g., something that roughly looks like a snake), and ranges all the way to highly elaborated multi-modal representations from cortical areas that can trigger or further amplify amygdala responses ("It really *is* a venomous Cobra slithering towards me!") or dampen down amygdala responses ("Oh, it was just an old bicycle tire lying on the ground…"). The amygdala in turn sends information back to stimulus processing areas like the visual areas at the occipital lobe to influence stimulus processing, which may give rise to various forms of motivated cognition, such as an enhanced focus on emotionally arousing features of the environment (Vuilleumier et al, 2004). The amygdala also influences memory for emotional events (Cahill, 2000).

The involvement of the amygdala in emotion and motivation has frequently been studied using procedures that involve punishments, such as foot shock, because many noxious stimuli are universally aversive and it is therefore relatively easy to obtain fear-related amygdala activation and learning with such procedures (LeDoux, 1996). While the amygdala undoubtedly is involved in states of fear and other negative emotions, it should not be overlooked that this brain structure also plays a critical role in approach motivation and reward (Baxter & Murray, 2002). For instance, Pavlov's famous dogs would have had a hard time learning to salivate in response to the bell sound (CS) predicting food (US) if their amygdalae had been damaged. Other research shows that an intact amygdala is crucial for animals to show second-order reinforcement learning (i.e., learning to bar press in order to make a light appear that has previously been paired with the presentation of food or a sexual partner; e.g., Everitt, 1990), and humans depend on it to generate affective "hunches" that guide their decision-making and behavior (Bechara et al., 1997).

In summary, the amygdala can be characterized as a motivational "homing-in" device whose

activity is influenced by sensory information at all stages of cognitive processing and that allows individuals to adjust their physiological states and overt behavior in response to cues that predict the occurrence of unconditioned rewards and punishers. In the case of rewards, an intact amygdala allows the individual to learn about cues that signal proximity to a desired event or object and to navigate the environment in order to approach the reward, moving from more distal to more proximal reward-predictive cues until the reward itself can be obtained. In the case of punishers, the amygdala enables individuals to respond to punishment-predictive "warning signals" either by freezing and an increase in vigilant attention or by active avoidance behavior that removes the individual from a potentially harmful situation.

3.2 The mesolimbic dopamine system: scaling the "magnetic" pull of incentives

The mesolimbic dopamine (DA) system is a key brain system for the invigoration of motivated behavior. This system has its root in DA-producing neuronal cell bodies located in the ventral tegmental area (VTA) of the upper brain stem. The axons of these neurons terminate in the nucleus accumbens, a small cluster of neurons in the ventral striatum, as well as in the prefrontal cortex. The nucleus accumbens receives input from the amygdala and the OFC and has been characterized as a gateway through which sensory information can influence motor response preparation in the basal ganglia (Mogenson, Jones, & Yim, 1980).

Conditioned and unconditioned reward stimuli will make tegmental DA cells release bursts of DA in the accumbens and prefrontal cortex, thereby exerting a broad, general influence on synaptic transmission in these structures (Schultz, 1998). Notably, however, these DA bursts accompany the actual occurrence of reward only initially. After learning of reward-predictive stimuli has taken place, DA no longer increases in response to the reward itself, but in response to the reward-predictive CS. If that stimulus is itself reliably predicted by another (second-order) predictive CS, the DA puff will ratchet back further and occur in response to the second-order CS, but no longer

in response to the first-order CS or the reward, and so on (cf. Fig. 4; Schultz, Dayan, & Montague, 1997).

So what is the function of DA being released into the accumbens? To answer this question, researchers have conducted studies in which the mesolimbic DA system was lesioned or DA agonists or antagonists were used to alter the effects of DA release in the accumbens. Results of a typical study of this type are presented in Figure 5 (Ikemoto & Panksepp, 1999). Rats were trained to run down a runway to a goal box filled with a tasty sucrose reward. On each trial of this task, they either received varying amounts of a DA antagonist dissolved in a fluid (vehicle) and injected into the nucleus accumbens or just the vehicle as a control condition. The DA antagonist was intended to block the effects of natural DA release on synaptic transmission in the accumbens, whereas treatment with the vehicle was not expected to interfere with the effects of DA release. After the first trial, rats who had received the highest dose of DA antagonist differed from all other groups in that they traversed the runway to the goal box much more slowly than any other group (left panel of Fig. 5). This difference also persisted on subsequent trials. Notably, these rats' consumption of the sweet sucrose solution was just as high as all the other rats once they had reached the goal box (right panel of Fig. 5).

A recent study by Pecina et al. (2003) complements these illustrative results. Pecina et al compared hyperdopaminergic rats (i.e., rats that had been genetically engineered to have higher-than-normal DA levels in the brain) with normal rats in their learning of the runway task and their consumption of sucrose solution available in the goal box at the end of the runway. Compared to control rats, hyperdopaminergic rats needed fewer trials to learn that running to the goal box was rewarded with sucrose, showed faster running once they had acquired this knowledge, and were less distractible on their way to the goal box. However, just like the DA-impaired rats in the previously described study, hyperdopaminergic rats' affective liking responses to sucrose in the goal box did not

differ from those of control-group animals.

These findings illustrate that DA transmission in the accumbens is required for the invigoration of goal-directed behavior (i.e., running toward the goal box), but does not play a role in the hedonic response to the incentive itself (i.e., consumption of the sweet solution). In other words, the mesolimbic DA system is a key structure for *wanting* a reward, but does not mediate *liking* of the reward (Berridge & Robinson, 1998). In a sense, then, the mesolimbic DA system functions like a magnet that pulls the organism closer to a desired goal or object, and the findings of Pecina et al suggest that greater availability of DA in the accumbens can be equated with a stronger magnetic pull of incentives.

Brain imaging studies have shown that synaptic activity in the accumbens is also related to incentive seeking in humans. In these studies, accumbens (and sometimes also VTA) activation has been observed in response to such varied incentives as beautiful opposite-gender faces, listening to chills-inducing music, or playing a computer game (Aharon et al, 2001; Blood & Zatorre, 2001; Koepp et al, 1998). It is notable in this context that the human trait of extraversion seems to be related to the sensitivity of the mesolimbic DA system (cf. Box 1).

However, just like the amygdala is often described too exclusively as the substrate of negative emotions, the mesolimbic DA system is often exclusively portrayed as the biological substrate of approach motivation. This view is incorrect. A large body of research shows that the mesolimbic DA system is also involved in active avoidance, that is, in tasks or situations in which the individual needs to do something in order to avoid a punishment (Ikemoto & Panksepp, 1999; Salamone, 1994; but see Ungless, 2004). Animals with a functionally impaired mesolimbic DA system (e.g., through lesions or DA antagonist administration) have a harder time learning to avoid an aversive stimulus, just as they have difficulties learning how to get to a reward. In contrast to active avoidance, the mesolimbic DA system is not required for passive avoidance; lesions of this

system do not lead to impaired performance on tasks that require the inhibition of ongoing behavior in order to dodge a punishment. These findings point to a larger conclusion about the function of the mesolimbic dopamine system, namely, that it is involved in the *facilitation* of behavior that is guided by incentives such as the attainment of reward or relief from punishment (cf. Box 2).

3.3 The orbitofrontal cortex: evaluating rewards and punishments

The OFC derives its name from the fact that it is the part of the cortex that lies directly above the eye orbits and thus on the ventral (i.e., downward-facing) side of the frontal cortex. The OFC receives highly processed olfactory, visual, auditory, and somatosensory information. Notably, it is interconnected with both the amygdala and the mesolimbic DA system, making it one of three major players in the brain's incentive motivation network. The OFC plays a key role in scaling the valence of a broad array of primary and conditioned reinforcers, including perceived facial expressions, various nutritional components of food, monetary gains and losses, and pleasant touch (Rolls, 2000).

Two notable features characterize the OFC. First, different types of reinforcers are represented by anatomically distinct areas of the OFC. And second, each area's activity changes with the motivational value of a given reinforcer. Evidence for the existence of anatomically distinct reward areas comes from many studies conducted by Rolls and colleagues (reviewed in Rolls, 2000; 2004). These studies revealed, for instance, that different subregions of the OFC respond to the degree to which a given food contains glucose, fat, salt, or protein (e.g., de Araujo et al, 2003). Similarly, brain imaging studies conducted with human subjects show that specific OFC regions are activated in response to monetary gains and losses (O'Doherty et al, 2001). Monetary punishment was associated with activation of the lateral (i.e, towards the side) OFC, whereas monetary reward was associated with activation of the medial (i.e, towards the body's midline) OFC.

The OFC's response to a specific reward is not fixed, however, but changes dynamically with the exposure to or consummation of a given reward and with changes in reward contingencies. Data

from responses of single neurons recorded through hair-thin electrodes in primates provide powerful illustration for the dynamic representation of reward value in the OFC (Rolls, 2000; 2004). If a monkey is given a single drop of glucose syrup -- a highly rewarding energy-rich food substance -- glucose-specific cells in the OFC show a strong burst of activity. If the monkey is fed more and more glucose over time, however, the firing rate in these neurons decreases in a fashion that is closely correlated with the monkey's acceptance of further glucose administrations, all the way up to the point when the OFC neurons stop firing and the animal completely rejects the glucose syrup (cf. Fig. 6). If the animal is given sufficient time after it has gorged itself on glucose syrup, however, it will eventually again accept more syrup, and its glucose-specific OFC neurons will resume their vigorous firing in response to the sweet taste. Findings such as these suggest that OFC neurons encode the individual's hedonic response to reinforcers, and that as the individual becomes "satiated" on a given reinforcer, neural responding dies down – a neurobiological manifestation of the alliesthesia effect.

Findings from brain-stimulation reward studies are consistent with this interpretation of OFC functioning (Rolls, 1999). In this type of research, an electrode is implanted in the brain, and the animal can activate the flow of current at the electrode tip by pressing a lever. Depending on where in the brain the electrode is located, the animal is sometimes observed to press the lever frantically, as if the stimulation leads to a pleasurable sensation, and this increase in lever pressing is taken as an indication that a brain reward site has been located. Brain-stimulation reward effects have been documented for many OFC sites, suggesting that pleasurable emotions are indeed experienced if these sites are activated. Notably, for food-related OFC reward sites, it has been observed that lever-pressing varies with the need state of the organism: if the animal was hungry, it displayed vigorous lever pressing at this site, but when the animal had eaten, lever-pressing ceased (Rolls, 1999). This suggests that OFC reward sites are sensitive to the degree of satiation an organism has reached with regard to a specific reinforcer and must therefore integrate information about the rewards incentive

value with the organismic need states.

OFC reward areas can also become activated by conditioned incentives (e.g., sounds or sights that predict food; Rolls, 2000; 2004). For instance, an area that responds strongly to the taste of food can, through learning, also become activated by the sight of that type of food. Together with the findings on the pleasurable properties of OFC activation, this observation suggests that conditioned incentives can feel just as pleasurable as the "real thing", that is, the actual reward. This idea is at the core of many modern theories of incentive motivation (e.g., Bindra, 1978). Interestingly, the OFC is also able to break or even reverse learned CS-reward associations very rapidly (Rolls, 2000; 2004). For instance, through learning, OFC neurons will respond to a triangle shape that reliably precedes food reward, but not to a square shape that is not associated with food. As soon as the relationship is reversed and the triangle no longer predicts the food but the square does, the same OFC neurons will cease responding to the triangle and start responding to the square. Thus, the OFC encodes not only the reinforcement value of rewards, but also of stimuli that are associated with them, and it can rapidly change its evaluations as soon as the reward value of a conditioned incentive changes. Not surprisingly, lesions to the OFC abolish the individual's ability to represent changing CS-reward contingencies, and emotional responses may become "unhinged" and persevere for long periods (Damasio, 1994; Rolls, 1999).

The OFC is not the only site of the "incentive motivation network" that codes for the pleasantness of a reward. For instance, some research suggests that portions of the nucleus accumbens and of the ventral pallidum (both parts of the basal ganglia, a subcortical brain structure involved in motor control and instrumental conditioning) code for the pleasantness of food reward (Berridge, 2003). Conversely, the OFC is involved not only in reward evaluation, but also plays a role in response inhibition and emotion regulation (Bechara, Damasio, & Damasio, 2000).

3.4 The lateral prefrontal cortex: Motivational regulation and override

The lateral prefrontal cortex (LPFC) is the portion of the frontal cortex just behind the forehead, extending to the temples. Along with the OFC and the medial PFC, it is one of the latest parts of the cortex to appear phylogenetically and is also the latest to come to maturation, taking as long as to early adulthood to reach its full functional capacity (Fuster, 2001). The LPFC supports a host of important mental functions, including speech (Broca's area in left LPFC), working memory, memory encoding and retrieval, and motor control. Most important from a motivational perspective are two specific functions of the LPFC. First, the LPFC is the place in the brain where goals and complex plans to enact them are represented. Second, and related to the first function, the LPFC can regulate the activation of core motivational structures of the brain, such as the amygdala.

Evidence for a key role of the LPFC in goal-directed action comes from neurological case studies (Luria, 1973; Luria & Homskaya, 1964). Perhaps it is not surprising that individuals with LPFC lesions that destroy language capability and working memory have a hard time initiating and executing voluntary behavior, particularly if it is complex. They lack the ability to instruct themselves, pacing themselves verbally through complex action sequences (language center lesion) or may not be able to retain all the elements of a complex plan in memory for long enough to execute it in its entirety (working memory lesion). Even more subtle forms of volitional deficits appear in LPFC lesions that spare both working memory and speech centers. The Russian neuropsychologist Alexander Luria (1973; Luria & Homskaya, 1964) has described people with this type of lesion who were perfectly able to understand and remember a verbal action command such as "Please take the pencil and put it on the table" and could repeat it to the experimenter, but were unable to use it to guide their behavior. Thus, an intact LPFC is critical for the execution of complex plans that rely on working memory and language for representation and updating of their elements and an intact connection of these structures to motor output. Note that the key role of language in the pursuit of complex goals and plans also makes the LPFC one critical point of entry for the social regulation of

behavior. Specifically, although people with LPFC lesions may be relatively unimpaired in their ability to respond motivationally to innate or learned nonverbal social cues (such as facial expressions, the prosody of spoken language, or gestures), they lose their ability to coordinate their behavior with that of others flexibly through the pursuit of verbally shared goals or to adapt their behavior to changing demands and expectations of their sociocultural environment.

The LPFC's capacity to represent and enact complex, verbally "programmed" goals also entails that it needs to be able to regulate and override already ongoing motivational needs and impulses and thus to resolve conflict between competing behavioral tendencies. Anyone who ever had to learn for an exam on a beautiful sunny day knows that it takes some effort and self-control, often mediated through verbal commands directed at oneself, to keep the focus on the books instead of jumping up and running outside. The LPFC seems to achieve this feat through its inhibiting effects on activity in structures related to incentive motivation such as the amygdala. Studies show that nonverbal stimuli with strong incentive properties, such as facial expressions of emotion or pictures with negative affective content (such as depictions of mutilated bodies; Adolphs & Tranel, 2000) cause activation of the amygdala in humans. However, these findings are usually obtained under conditions of passive viewing that do not require LPFC participation in the task. As soon as participants are asked to verbally label the expression of a face or to reappraise a negative scene such that it becomes subjectively less aversive, LPFC becomes activated and amygdala activation decreases (Ochsner et al., 2002). This disrupting effect of LPFC activation on amygdala activity may enable people, for example, to refrain from impulsive aversive responses or to remain seated at their desk when having to learn for an exam instead of following their impulse to engage in motivationally more exciting activities. These findings suggest that engagement of the LPFC's verbal-symbolic functions in dealing with an emotionally arousing stimulus dampen down activity in emotion generators such as the amygdala (cf. Lieberman, 2003).

In summary, LPFC supports the planning and implementation of complex behavior through its ability to adopt or formulate explicit (i.e., verbally represented) goals and keep them activated in working memory and by controlling activation in the brain's incentive motivation network and thereby inhibiting impulsive responding to motivational cues.

3.5 Summary

Many motivational processes make use of what we have termed the brain's incentive motivation network, consisting of the amygdala, the mesolimbic dopamine system, and the orbitofrontal cortex. The amygdala is involved in learning which environmental cues predict the occurrence of a reward or punishment and thereby guiding the organism towards pleasant and away from noxious outcomes. The mesolimbic dopamine system regulates how vigorously the individual engages in reward seeking, but also in active avoidance of punishments, and it does so by receiving information about conditioned cues from the amygdala. The orbitofrontal cortex evaluates the "goodness" of primary and secondary rewards based on the individual's current need state and learning experiences. Motivational processes rely on these three structures to act in concert so that cues that predict (amygdala) stimuli that have been experienced as pleasant (orbitofrontal cortex) elicit behavioral invigoration (mesolimbic dopamine system) directed at reward attainment. Behavioral impulses generated by this incentive motivation system are influenced by other functional structures, such as the lateral prefrontal cortex. The lateral prefrontal cortex guides behavior in part through the formulation of complex, verbally represented goals and plans for their implementation and through regulation of output of the brain's incentive motivation network, can shield explicit goals from interference of incentive-driven motivational impulses.

We should emphasize at this point that in the preceding sections we have selectively discussed only some of the most important brain areas involved in motivation and its regulation and left out other key structures such as the hippocampus (involved in context-dependent modulation of

emotional and motivational states) and the medial prefrontal cortex including the anterior cingulate cortex (involved in regulation of attention, response conflict resolution, and movement initiation). Instead, we want to dedicate the remainder of the chapter to the discussion of specific motivational systems, which are rooted in hypothalamic structures (cf. Fig. 2 for the location of the hypothalamus in the human brain), but also harness the brain's incentive motivation network to guide behavior.

4. Specific motivational systems

Certain tasks and goals in an organism's life are recurrent. All animals need to find food and eat regularly to get energy; they need to drink so as not to dehydrate; they are driven to find a mate to pass their genes on to their offspring; in order to do that they need to compete with and dominate other same-sex members of their species, and so on. These tasks have been keeping not only currently living beings busy, but also their ancestors, reaching back millions of years in evolutionary history. Hence, it should not be too surprising to find that evolution has dealt with such recurring needs and goals by equipping brains (and bodies) with special systems that ensure that the needs of the individual's day-to-day survival and the need of the individual's genomic generation-to-generation survival are met adaptively and efficiently. Such specialized systems that coordinate and support the attainment of specific classes of incentives have been identified and described in considerable detail for drinking, feeding, affiliation, dominance, and sex. In the following, we will illustrate the ways in which evolution has shaped motivational systems by taking a closer look at four of them.

4.1 Feeding

The primary reason to eat is to provide energy for functioning of the body. Hunger reflects the need to replenish nutrients. However, in the modern, developed world, in which food is overabundant, there are many other factors that motivate us to eat. These include routine (i.e., "It's noon- it's lunch-time!"), stress, pleasure, and social factors (i.e., when other people are eating). The

physiological mechanisms that control the regulation of eating involve an interplay between the brain (especially the hypothalamus, a key brain area for regulating basic physiological needs) and other organs, such as the liver, stomach, and fat stores. This section will cover some of the neurobiological signals that turn on and turn off the drive to ingest food: the need for energy as well as the desire for the pleasures of taste.

4.1.1 Energy needs

All organisms need nutrients for energy to sustain the chemical processes of life. Our cells use glucose as their primary energy source. Glucose can be stored as *glycogen* in the liver, and fat serves as a longer-term storage of energy. However, the body has multiple ways of sensing when more energy might be needed, such as when glucose levels drop, fat stores decline or intestinal motility changes. These conditions trigger activity in brain circuitry that generates a feeling of hunger, or motivation to eat.

Many of the body's systems for sensing energy needs begin in the digestive tract. The stomach contains stretch receptors that send signals of fullness to the brain. The gut also produces many neurohormones that act on the brain to let it know how recently and how much food has been consumed. One such neurohormone is *cholecystokinin* (CCK.) The more food enters the gut, the more CCK is released. CCK acts on the vagus nerve, which sends a satiety (i.e., fullness) signal to the brain. Thus, CCK helps to turn off motivation to eat. High levels of CCK actually cause nausea – a "warning sign" to stop consumption (Greenough et al., 1998).

Another satiety signal comes from fat. Fat cells produce a hormone called *leptin*, which travels through the blood and acts at the hypothalamus to inhibit feeding. The more fat on the body, the more leptin is produced. When leptin levels are low, one feels hungry and eats more; when they are high, one eats less. Leptin thus serves as a signal to the brain of the amount of fat stored in the body, and helps to regulate body weight in the long term. Leptin also acts as a short-term signal:

leptin levels in the blood increase at the end of a meal, promoting satiety, and decrease some hours post-meal, promoting hunger (Friedman and Halaas, 1998).

The brain also contains specialized neurons that monitor levels of glucose in the blood. These "glucostat*"* neurons, located in the hypothalamus, react when glucose levels drop. They send a signal to other regions of the hypothalamus to trigger feeding (see, for example, Stricker and Verbalis, 2002).

What are the brain systems to which CCK, leptin and glucostat neurons communicate? They are numerous, but include neurons in the hypothalamus producing *neuropeptide Y* (NPY). NPY is a potent hunger-inducing molecule: miniscule amounts of NPY injected into the brains of laboratory animals cause them to eat voraciously. One of the ways leptin acts is by inhibiting the neurons that produce NPY- therefore, by turning hunger off. Similarly, CCK turns off NPY production in the hypothalamus (Levine and Billington, 1997; Billington and Levine, 1992).

In the hypothalamus, there are also neurons producing and responding to a class of neuropeptides called *melanocortins*. Peptides that activate melanocortin receptors, such as alpha-melanocyte-stimulating-hormone (α-MSH), lead to satiety, whereas peptides that block these receptors, like Agouti-related protein (Agrp), stimulate hunger (Irani and Haskell-Luevano, 2005; Stutz et al., 2005). Just as leptin and CCK "turn off" the hunger signal of NPY, they also cause α-MSH neurons to increase their firing rate, releasing more α-MSH and promoting satiety.

Gonadal steroids, whose primary role is to regulate fertility and sexual motivation (see section 4.4.3), also have an impact on food intake. In female animals, estrogen has a significant restraining effect on food intake. After ovariectomy, which stops the production of estrogen in the ovaries, female rats increase their food intake and gain about 25% of body weight. Progesterone counteracts the effects of estrogen. High levels of progesterone lead to increased food intake and body mass, an effect that is consistent with progesterone's role as a hormone that promotes and

safeguards pregnancy, which is characterized by steeply increasing energy needs.

4.1.2. Reward

Need for energy is obviously not the only reason we eat. Eating is pleasurable, and, like other pleasurable activities (sex, addictive drugs, etc.), eating causes release of dopamine (DA) in the nucleus accumbens, part of the brain's reward learning system (see section 3.2, "The mesolimbic dopamine system"). In particular, sweet and fatty foods are naturally rewarding to humans, rats, and other omnivores. In rats, it has been shown that diets containing extra fat and sugar lead to greater activity in brain structures involved in pleasure and reward (Levine et al., 2003).

The body's natural opioids participate in the pleasurable experience of eating. Opioids are released in the brain during food intake, especially of sweet and fatty foods. Injecting laboratory rats with opioids causes them to eat somewhat more of a plain chow, but a great deal more of a palatable sweet or high-fat chow. Unlike NPY, opioids do not seem to participate in hunger driven by energy needs: an injection of NPY into the brain increases animals' intake of bland yet energy-rich chow, but not of tasty but energy-dilute sugar-sweetened water. On the other hand, an injection of opioids causes a large increase in sugar-water intake, without having much effect on chow intake (Levine and Billington, 2004).

Sweet and fatty foods are not the only ones we seek out. A flavor called *umami*, present in meats, sea-foods and soy, is very rewarding to humans and laboratory animals, possibly because it serves as a good indication that the food is rich in protein (Yamaguchi and Ninomiya, 2000). The flavor additive monosodium glutamate (MSG) powerfully activates umami taste receptors on the tongue, which is why foods containing MSG taste so good to us.

Finally, we are naturally motivated to seek out a variety of foods. Humans and laboratory animals exposed repeatedly to a single flavor, even one that is highly rewarding at the start, will rapidly tire of it and consume less of it. However, if they are then exposed to a different flavor, the

rewarding nature of the first one will be renewed (Swithers and Martinson, 1998). Because of this phenomenon (alliesthesia), the best way to make a lab rat gain weight is to put it on a "cafeteria diet": a choice of multiple foods (e.g., Gianotti et al., 1988). That rat will gain considerably more weight than rats given only one highly tasty food to eat.

Recently, researchers have found that different flavors activate different parts of the orbito-frontal cortex (OFC) in humans. The OFC is a region involved in tracking reward. Thus, different tasty flavors seem to be registered by distinct parts of this brain structure as separate kinds of reward. This finding gives a hint as to the neurobiological basis of the phenomenon that we crave a variety of flavors, rather than just one (Rolls, 2005).

4.1.3 Summary

Hormonal signals from the organs, such as leptin (from fat) and cholecystokinin (from the digestive tract) enter the brain and act on neurons in the hypothalamus to affect hunger and satiety. In the hypothalamus, neuropepride Y and Agouti-related protein stimulate hunger, whereas alpha-melanocyte-stimulating-hormone reduces hunger. Opioids play a role in the pleasurable aspects of eating.

4.2 Affiliation and attachment

While almost all organisms have social interactions with others of their species, attachments formed between parents and young and between mates are only common in mammals and birds. Parent-offspring attachments, which can be thought of as motivations to be near the parent or the offspring, probably evolved in mammals and birds because these animals require extended parental care, including warmth and nourishment, during immaturity. Mating pair bonds, which give rise to a long-term motivation to be near the mate, exist in species that engage in cooperation between mates in rearing of offspring. Interestingly, the majority of bird species form pair bonds between mates, but very few mammalian species do - humans being one notable exception.

In this section, we will cover the basic biopsychology of the parent-offspring bond and the mating pair bond. We will also briefly discuss neurobiological aspects of other kinds of attachments, such as friendships.

4.2.1. Parent-offspring attachments

Maternal-offspring attachments have been extensively studied in the rat and the sheep. In these species, there is little or no paternal involvement in care for young - this generally occurs only in those mammals that also form mating pair bonds.

Rat pups cannot regulate their body temperature in infancy, so the dam (mother) must spend time huddling over them to provide them with warmth. She also nurses the young and retrieves pups that get separated from the rest of the litter. Male rats and nulliparous females (females that have not had offspring) do not display these behaviors upon initial contact with pups. In fact, nulliparous females find the odor of rat pups aversive, and avoid them.

So how do females develop the motivation to care for their young? Estrogen and progesterone levels are very high during pregnancy, and these hormones set the stage for maternal behavior. As levels of these hormones drop at the end of pregnancy, levels of *prolactin* and *oxytocin* rise - two hormones released by the pituitary gland which are necessary for lactation. The oxytocin surge at the end of pregnancy is also necessary for uterine contractions of labor. All of these hormones are necessary for full expression of maternal behavior (Mann and Bridges, 2001). Nulliparous female rats or castrated male rats treated with progesterone and estrogen followed by prolactin and a jolt of oxytocin - mimicking the hormonal status of the end of pregnancy - engage in maternal behaviors towards pups just as frequently as a dam that has just given birth. A major site of action for these hormones is the medial preoptic area (MPOA), a brain region in the hypothalamus that is also important for sexual behavior (Young & Insel, 2002; see section 4.4.2 for more on the MPOA and sexual behavior). The hormones also influence the brain's olfactory system (which

handles perception of odor) so that the dams don't mind the odor of pups. There is evidence that hormones also affect the olfactory system in humans at the end of pregnancy: new mothers rate smells associated with human babies as less unpleasant than nulliparous women or men do (Fleming et al, 1993).

The same hormones are also necessary for maternal behavior in sheep. In sheep, oxytocin performs an important function for recognition of one's own young. Sheep live in large herds, and a lactating ewe must allow her own lambs to nurse while keeping other lambs away. However, without a sufficient oxytocin surge at the end of pregnancy, ewes will reject their own lambs. It turns out that oxytocin is necessary for the ewe to learn to recognize the smell, sight and sound of her lambs as distinct from other lambs. Once this learning is complete, oxytocin is no longer necessary for recognition (Keverne and Kendrick, 1994; Kendrick, 2004).

In species where fathers help take care of the young, such as Siberian hamsters, Tamarin monkeys, and humans, male animals undergo hormonal changes towards the end of their mate's pregnancy, and these hormones are necessary for paternal behavior. Prolactin appears to be important for paternal behavior in many species. In many cases, including humans, prolactin levels go up in fathers along with mothers at the end of pregnancy. In male wolves, prolactin rises seasonally, corresponding with the season in which pups are born. Other hormonal changes also tend to echo those of females in pregnancy. As in mothers, testosterone increases in fathers in species that need to defend the pups against hostile intruders (Wynne-Edwards, 2001).

Hormones may set parental behavior into motion- however, the hormones of pregnancy quickly subside, and the behavior, once learned, continues. Hormones like oxytocin may cause long-term changes in the nervous system that support the attachment to one's young and the motivation to care for them. Rats that have already had litters in the past provide better, faster maternal care than new mothers. In primates, learning may be even more important. Monkeys that have not had a

normal social environment growing up are severely deficient in maternal behavior as adults (Harlow & Harlow, 1966). A famed female chimpanzee raised in captivity had to be trained by humans in proper nursing and care of her infant (Matsuzawa, 2003). Clearly in this species, and most likely in humans, hormones alone are not sufficient to produce maternal behavior or a bond to one's offspring.

What about the bond of the infant to its parent(s)? When rat pups are separated from their dams, they show signs of distress, including making ultrasonic vocalizations which alert the dam that the pup has become separated from the litter. Applying warmth to the pups calms them and makes them cease vocalizing. Injections of opioid peptides- brain chemicals involved in pleasure and suppression of pain- achieve the same effect. Similar effects have been seen in young dogs, chickens, and primates: opioid drugs reduce separation distress, even at doses too low to cause sedation or other effects (Nelson and Panksepp, 1998). More evidence for opioid involvement in affiliation and attachment will be addressed below in "4.2.3: Other attachments."

In many species that have been studied, opioids and warmth are not the whole picture. Rat pups prefer to huddle close to a warm object with the smell of their particular dam, indicating that they can recognize their dam by smell (e.g., Sullivan et al., 1990). In other species, as well, the young seem to form an attachment to their own primary caregiver in particular. For example, young dogs preferred their mother to other dogs, even in adulthood after not having had contact with her for two years (Hepper, 1994). In primates, including humans, infants quickly learn to recognize and prefer to be with their primary caregiver(s) (e.g., Porter, 1998). Again, it is thought that hormones like oxytocin may play a role in the formation of these bonds by facilitating long-term changes in the nervous system, which persist (along with the bond) after the hormones have subsided.

4.2.2 Mating pair-bonds

The best-studied neurobiological animal model of pair bonding is in the prairie vole. Once these small rodents mate for the first time, the pair forms an attachment that lasts until one of the

animals dies. They live in a nest together, both contribute to rearing of their young, and continue to mate with each other and produce young in subsequent seasons. When separated, the voles exhibit considerable distress, similar to the distress infants of many mammalian species experience during separation from their mother.

Oxytocin and a closely-related hormone, *vasopressin*, turn out to be crucial for the formation of this pair bond. Oxytocin and vasopressin levels surge during mating. As in mother sheep learning to recognize their young, these hormones establish the attachment to the mate, which then continues- represented in long-term changes in the brain- long after hormone levels have returned to normal. We can experimentally block oxytocin/vasopressin effects in the brains of the voles before their first mating, and prevent the formation of a pair bond. Similarly, pair bonds can be formed without mating by injecting these hormones into the brains of a pair of animals. Oxytocin seems to be the key hormone in females, and vasopressin in males (Insel 1997; Insel et al. 1998), although newer research implicates oxytocin in pair bonding in both sexes.

While prairie voles form pair-bonds, a closely-related species, montane voles, do not. Like many other mammals, montane voles mate with multiple partners and only females care for the young. The difference between these two species lies in the pattern of oxytocin and vasopressin receptors in the brain. Pair-bonding prairie voles have lots of oxytocin and vasopressin receptors in the nucleus accumbens and ventral pallidum, brain areas involved in reward. The oxytocin and vasopressin released during the first mating between two animals act at these brain sites to permanently change the dopamine (reward-learning) system, such that being with the mate becomes rewarding. In a sense, after mating, the brain forms an "addiction" to the mate (Keverne and Curley, 2004).

Does oxytocin underlie pair bonding in other species, such as humans? Although this has been speculated to be the case (e.g., Taylor et al., 2000), conclusive evidence is still lacking.

Certainly humans form attachments differently than meadow voles: a single sex act does not lead to a life-long commitment in our species! Nonetheless, oxytocin may play a role in formation of bonds or attachments in humans. As in other mammals, oxytocin levels rise during sex (in particular at orgasm) and during massage or other soothing tactile contact (Uvnas-Moberg, 1998). This oxytocin increase may facilitate bonding. Also, brain imaging studies in which people viewed photos of their significant other or child compared to photos of acquaintances or other children revealed comparatively greater activity in the ventral striatum - a region encompassing reward-related circuitry, such as the nucleus accumbens – when people viewed their loved ones (Bartels and Zeki, 2000, 2004). Thus, the same reward circuitry that is crucial for vole pair-bonding seems to play a role in human attachment.

4.2.3 Other attachments

Mating bonds and parent-offspring bonds are not the only attachments that animals form. Individuals of many species show signs of stress and pathology if isolated. Rodents, canines and primates, among other creatures, tend to live in close-knit groups, and have strong motivations for contact and interaction with others in their group. In primates, in particular, attachments can form between unrelated, non-kin individuals. These are often supported by mutual grooming, which serves to strengthen ties and soothe a distressed ape. Motivation to be groomed seems to involve *beta-endorphin*, a naturally occurring opioid: levels of this opioid in the nervous system rise when groomed, and when they are low, individuals seek out grooming (Keverne et al., 1989; see also Taira & Rolls, 1996).

Some studies suggest that opioids are involved in human affiliation, as well. After viewing an affiliation-related movie, people high in a “social closeness” trait felt more affiliative and also had higher tolerance to heat-induced pain (opioids help to reduce pain.) Both of these effects were blocked by naltrexone, an opioid antagonist (Depue and Morrone-Strupinsky, 2005). This evidence

suggests that the affiliation-related movie caused an increase in opioid release in this group of people.

Oxytocin has social functions beyond parent-infant and pair bonds, including an important role in social memory. Mice lacking the gene for oxytocin behave in a new encounter with a familiar mouse as they would with a stranger. When the missing oxytocin is replaced in their brains, they can learn who's who just like normal mice (Winslow and Insel, 2002).

Recent intriguing studies even suggest that, in humans, oxytocin plays a role in trust shown towards a stranger. Participants in one experiment played an economic game in which Player 1 was given a sum of money, part of which they could entrust to Player 2, in whose hands the money would triple. Player 2 could then return whatever amount s/he chose (including zero) to the Player 1. It was found that Player 2's who received higher sums of money from their Player 1's had higher blood levels of oxytocin, and oxytocin levels were also related to how much money Player 2 returned to the Player 1 (Zak et al., 2005). In a follow-up study, one group was given a dose of oxytocin intra-nasally and another group received placebo. (From the nose, some small molecules like oxytocin may enter parts of the brain such as the hypothalamus.) In the oxytocin group, Player 1's entrusted more money to the Player 2's (Kosfeld et al., 2005). In both studies, when people played the game with a computer that randomly allocated money, oxytocin had no relationship to money received or given. This suggests that oxytocin actually increases our ability to trust another person.

4.2.4 Summary

The hormones estrogen, progesterone, prolactin and oxytocin are involved in the initiation of maternal behavior. Similar hormones are also involved in paternal behavior. In mothers, oxytocin facilitates learning to recognize and bond with her offspring. Oxytocin and vasopressin are also necessary for the formation of pair bonds. After an attachment is formed, these hormones are no longer necessary to sustain the bond. Opioids play a role in the attachment of an infant to its parent,

as well as in affiliation in primates.

4.3 Dominance

Most animals do not only have to evade predators, find sustenance, and a mate to survive as individuals and as sets of genes, they also have to compete with members of their own species to secure resources necessary for survival. Behavior directed at defeating others in resource competitions are called dominance behaviors and they often give rise to relatively stable dominance hierarchies within the group.

4.3.1 Mechanisms and benefits of dominance

Dominance issues are most obviously at stake when the males of a species compete with each other for a mate. The competition can be carried out intrasexually, against other males, to defeat them and keep them away from females, and/or intersexually, to attract the attention of a female and advertise one's genetic fitness. In Darwin's (1871) own words, this is the difference between "the power to conquer other males in battle" and "the power to charm females". The two often go hand in hand, such as when a male's large body size makes him not only more likely to win fights with other males, but also more attractive to females (Wilson, 1980).

But dominance extends beyond assertiveness and success in the mating game and often involves privileged access to other resources, such as food or protected nest sites. In some species, such as many birds, dominance is a relevant attribute only during mating and has to be re-negotiated in every new mating season; in others, particularly in animals living in social groups, dominance rank is a more stable individual attribute, determined and changed in occasional violent fights and reinforced frequently through non-violent signals of dominance (e.g., a warning stare, bared teeth) and submission (e.g., exposure of the throat area in dogs and wolves).

The establishment of stable dominance hierarchies within a social group benefits not only the "top dog", the alpha animal at the tip of the hierarchy, but also the lower-ranking animals (Wilson,

1980). A stable dominance hierarchy means that all group members can save energy by sticking to a pecking order at the food trough, because who gets first pick and who does not does not have to be fought about on each feeding occasion. In many species, the dominant animal will also actively enforce peace among subordinate group members by breaking up fights. And although dominant animals usually are more successful at procreating, subordinate members get to promote their genes, too, either by "sneak copulations" or by helping dominant animals with which they share genetic ties to raise their offspring.

In humans, of course, things become more difficult, because it is much harder to pinpoint one specific dominance hierarchy that is binding for all. A student in a course may be subordinate to the high-expertise professor. But the professor may rank rather low among his or her colleagues in the department, whereas the student may be an undefeated ace on the tennis court and excel in the student debate club. Thus, humans' dominance ranks are much more fluid than other animals', reflecting the fact that each one of us is a member of many different groups, not just one.

4.3.2 Brain correlates of dominance

The biopsychological roots and correlates of dominance are well-described for the rat, biopsychology's favorite animal model (Albert, Jonik & Walsh, 1992). A male rat tries to establish or maintain dominance by launching an attack during which he pushes an intruder with his hind legs or flank and then chases him away. He also shows piloerection, that is, the hair on his body rises and makes him look bigger and more intimidating. This pattern of lateral attack and piloerection can also be observed in rat mothers trying to protect their pups. A hypothalamic network centered on the anterior nucleus (AN) of the hypothalamus plays a critical role in lateral attack and piloerection and thereby in rats' dominance behavior (Albert et al., 1992; see also Delville, DeVries, & Ferris, 2000). If the AN is lesioned, lateral attack is no longer displayed against intruders; if it is stimulated, lateral attack can be elicited much more quickly and it is displayed more intensively. This effect is

particularly strong in the presence of high levels of testosterone in males or testosterone and estradiol in females. In the regulation of dominant behavior, the hypothalamus interacts with other brain areas involved in incentive motivation and reward learning. For instance, lesions of the nucleus accumbens decrease rats' inclination to attack an intruder (Albert et al., 1989). On the other hand, by binding to steroid receptors and thereby increasing transmission at dopaminergic synapses in the accumbens (Packard, Cornell, & Alexander, 1997), elevated levels of gonadal steroids like testosterone and estradiol facilitate motivation for attacking an intruder in non-lesioned rats.

4.3.3 Dominance and aggression

At this point, a word of caution is in order about the relationship between aggression and dominance. First, aggression is only one way to attain and secure dominance in many species, a fact that a narrow focus on the rat as an animal model of dominance may obscure. For instance, aggressive and violent behavior as a means of attaining dominance often backfires in primate groups and is almost universally outlawed in humans. Work in primates even suggests that high levels of the neurotransmitter serotonin, through its restraining effect on impulsive aggression, promote the attainment of high social rank (Westergaard et al., 1999). Thus, considerable social finesse is required to become dominant, and in humans more than most other species non-aggressive means of achieving dominance have become critical for social success.

And second, not all forms of aggression are related to dominance (Panksepp, 1998). Besides the type of offensive aggression associated with dominance in many species, there is also defensive aggression elicited by threat and predatory attack directed against prey. However, the latter two are mediated by brain systems that are different from the one we have described for offensive aggression, they serve very different functions, and they are not influenced by hormone levels. Thus, equating dominance with aggression would be a mistake, because there are many forms of dominant behavior that are not overtly violent or aggressive (particularly in higher mammals) and there are

forms of aggression that have nothing to do with dominance.

4.3.4 Hormonal factors in dominance behavior

As suggested by the facilitating effect of gonadal steroids on AN-mediated offensive aggression, hormones play a key role in dominance. In many species, including humans, high levels of testosterone facilitate aggressive and non-aggressive dominance behaviors (Nelson, 2005). For instance, seasonal variations in testosterone levels are strongly associated with seasonal changes in aggression and territorial behavior in many species: when testosterone is high, aggression is, too. As testosterone production increases in male mammals and birds around puberty, so does aggression; castration abolishes increases in both. Among humans, it has been observed that male and female prisoners who are high in testosterone are also more prone to aggressive behavior and rule infractions (Dabbs et al., 1987; Dabbs & Hargrove, 1997). And in most species, those who are high in testosterone are also more likely to engage in fights for dominance.

Success or defeat in such dominance contests in turn leads to increased or decreased levels of testosterone. Elevated levels of testosterone have been observed, for instance, in winners of sports competitions, chess matches, and even in simple games of chance, whereas losers' testosterone typically decreases (Mazur & Booth, 1998). These differences in testosterone responses to contest situations even extend to observed dominance. Research has shown that after a soccer match, fans of the winning team have increased testosterone, whereas fans of the losing team have decreases testosterone (Bernhardt et al., 1998). Thus, the relationship between testosterone levels and dominance outcomes is a two-way street in which one's testosterone level influences dominance seeking and the results of this behavior in turn affect one's testosterone levels (Mazur, 1985; Oyegbile & Marler, 2005).

Although basal levels of gonadal steroids like testosterone are usually under hypothalamic control (the hypothalamus regulates release of hormones from the pituitary, which in turn regulate

the release of hormones such as testosterone from glands in the body), its control is relatively sluggish and changes can take an hour or more. But the testosterone increases and decreases typically observed in winners or losers of dominance contests happen within 10 to 20 min and thus much faster than hypothalamic control would permit. So what drives these quick testosterone changes?

Robert Sapolsky (1987) solved this riddle in a series of elegant field experiments with wild-living baboons in Kenya. He stressed both high-ranking and low-ranking male baboons by darting and immobilizing them (baboons, like many other mammals, experience immobilization as stressful). Sapolsky observed that within minutes, low-ranking animals showed a drop in testosterone, whereas high-ranking animals' testosterone surged. To find out what explained these differences in testosterone response to a stressor, he next applied a variety of hormone agonists and antagonists and studied their effect on testosterone release. Sapolsky found that the stress hormone cortisol increased more in low-ranking than in high-ranking baboons; also, administration of dexamethasone (a cortisol-like substance) suppressed testosterone release in all animals by making the testosterone-producing cells in the testicles less sensitive to signals from the pituitary. In contrast, administration of a substance that inhibited the release of the sympathetic catecholamines epinephrine and norepinephrine (also called adrenaline and noradrenaline) abolished the post-stress testosterone increase in high-ranking baboons, which suggests that these hormones normally have a stimulating effect on testicular testosterone release. Sapolsky concluded from these findings that the balance between cortisol, which is more likely to be released in response to overwhelming stressors, and sympathetic catecholamines, which are released very quickly in response to stressors that are perceived as manageable, has a fast and direct effect on testosterone. If the cortisol response to a stressor outweighs the catecholamine response, testosterone levels dip quickly – an outcome that is more likely in low-ranking, powerless animals. If the catecholamine response to a stressor outweighs the cortisol response, testosterone increases – a typical outcome for dominant animals who are used

to calling the shots.

These findings from a relatively unusual darting-and-immobilization procedure mirror exactly what Sapolsky and others have observed in many mammalian species. Often, dominant and non-dominant animals do not differ strongly in their basal testosterone levels (Sapolsky, 1987; Wingfield et al., 1990). But when they are challenged, dominant animals respond with a quick testosterone rise, which increases muscle energy and aggressiveness and thus makes them more likely to win the challenge, whereas non-dominant animals respond with a testosterone decrease, lowering their pugnacity and thus their likelihood to get hurt in a fight. In humans, high levels of implicit power motivation may be the equivalent to a dominant status in animals (Schultheiss, in press). Power-motivated people respond with increased sympathetic catecholamines and decreased cortisol to dominance challenges in which they can keep the upper hand (McClelland, 1982; Wirth, Welsh, & Schultheiss, 2006). The net result is a testosterone increase within 15 min after the challenge. In contrast, low-power individuals respond with increased cortisol levels and low catecholamine levels to such a dominance challenge, which suggests even when they can keep the upper hand, they are stressed out and uncomfortable with the situation. A drop in testosterone is the consequence (Schultheiss, Wirth et al., 2005).

4.3.5 Summary

Dominance behaviors are aimed at gaining privileged access to resources that ensure the individual's personal and genetic survival. Established dominance hierarchies bestow benefits on dominant and subordinate members of a group by lowering the incidence of costly fights for resources. Dominance is not synonymous with aggression; while offensive, hormone-dependent forms of aggression clearly play a role in the establishment of dominant status, dominance also extends to non-aggressive behavior, and predatory and defensive aggression typically are unrelated to dominance. Dominance motivation is supported by the anterior nucleus of the hypothalamus and its

interconnections to brain substrates of incentive motivation, and by high levels of gonadal steroids such as testosterone and estradiol which facilitate signal transmission in brain structures related to dominance motivation. In a multitude of species, high testosterone facilitates dominance and aggression, and the outcome of dominance fights in turn leads to rapid changes in testosterone, particularly in males, with winners registering an increase and losers a decrease. These testosterone changes are triggered by the effects of stress hormones on the gonads. Elevated cortisol levels inhibit, and elevated sympathetic catecholamine levels stimulate the release of testosterone. In humans, high levels of implicit power motivation predispose individuals to respond with low cortisol, elevated sympathetic catecholamines and increased testosterone to a dominance challenge, whereas low-power individuals respond with increased cortisol, low sympathetic catecholamines and decreased testosterone.

4.4 Sex

The need for sex is at once one of the most potent and in a sense most peculiar of all motivational systems. One doesn't have to be a Freudian to recognize that a lot in our and other living beings' lives revolves around sexual reproduction. At the same time, not having sex does not threaten our survival as individuals in the same way as not having food, water, or social protection would. But if one considers that passing on our genes to our offspring is the ultimate and perhaps in a sense most magnificent goal of all sexually reproducing animals, because it extends a billion-year old uninterrupted chain of life by another generation, then it makes sense that evolution made sure that no living thing would forget about procreating by making the sexual urge an extremely powerful one. In the following, we will review how sexual motivation is shaped through the interaction of biological factors and experience.

4.4.1 Developmental origins of sex and gender

Although for birds and mammals, biological sex initially resides in the genes, the gonads take

over pretty early in fetal development. For the rest of our lives, the gonads govern sexual behavior to a large extent, partly through their permanent (= organizational) effects on the developing brain, and partly through their temporary (= activational) effects on the adult brain (Nelson, 2005). If at conception a gene on the Y chromosome, which is present only in males, is expressed, testes develop and start producing testosterone and other androgenic hormones, leading to male body morphology (e.g., development of male genitals) and brain organization. If at conception the gene is not activated -- as is the case in females, who are lacking the Y chromosome -- ovaries develop. And because ovaries release almost no hormones during development, brain and body develop in the female mode.It should be noted that sexual development is not all-or-none, either-male-or-female. Rather, different parts of the body and of the brain are influenced by the interplay of hormones, hormone-metabolizing enzymes, and the expression of hormone receptors at different times during development, which can lead to variations in the fit between "brain sex" (sexual identity; sexual preferences) and body sex. Thus, although in many cases male body sex goes along with male sexual identity and a preference for female partners and female body sex goes along with female sexual identity and a preference for male sexual partners, this is by no means a certain outcome and variations (e.g., transsexuality, homosexuality) do occur (LeVay & Hamer, 1994; Panksepp, 1998).

4.4.2 Hypothalamic command centers of sexual behavior

The differential "marinating" of the brain in gonadal hormones during fetal development leads to differences in the organization of hypothalamic control of sexual behavior. These differences, and their effect on sexual motivation and behavior, have been most thoroughly studied in rats (Nelson, 2005; Panksepp, 1998). In female rats, the key command center of sexual behavior is the ventromedial nucleus (VMN) of the hypothalamus. If this nucleus is lesioned, female rats won't show any interest in mating with a male, as reflected in the absence of proceptivity, that is, the active solicitation of male sexual interest, and receptivity, that is, the readiness to allow males to mate with

them. In rats, receptivity is easily observable as a behavior called lordosis, which consists of the female arching her back and deflecting her tail to allow the male to copulate with her. Electrical stimulation of the VMN, on the other hand, can trigger both proceptivity and receptivity, but only in the presence of the gonadal steroids estrogen and progesterone, which bind to steroid receptors in the VMN and are released during the fertile phase (estrus) of the rat's estrous cycle. Of course, the central coordinating function of the VMN is integrated with the operation of brain structures supporting incentive motivation generally. For instance, female rats in estrus show increased DA release in the nucleus accumbens at the sight of a male rat, and this increased DA release reflects increased motivation to approach the male (Pfaus et al., 1995).

The key command center of male sexual behavior is the medial preoptic area (MPOA) of the hypothalamus, which, as a result of organizational effects of gonadal steroids, is larger in males than in females. MPOA lesions in males lead to an inability to copulate. Electrical stimulation of the MPOA, on the other hand, makes male rats ejaculate earlier than normal; also, testosterone treatment in castrated male rats restores normal levels of neuronal firing in the MPOA. Like in females, the hypothalamic control of sexual behavior is integrated with general-purpose motivational brain systems and hormonal factors in males. In a series of elegant studies, Everitt (1990) showed that MPOA lesions lead to a loss of copulatory ability, while sexual motivation remained intact (e.g., animals continued to bar-press for access to females). Conversely, if the basolateral amygdala was lesioned and the MPOA was spared, animals were no longer motivated to gain access to a female in estrus, but were able to copulate with her once placed on top of her. Likewise, a reduction of DA transmission in the mesolimbic DA system led to a decrease in sexual motivation, but did not affect copulatory ability. Notably, castration, which leads to an almost complete loss of testosterone, impaired both sexual motivation and copulatory ability.

4.4.3 Hormonal factors in sexual motivation

This last finding suggests that hormones, which gave rise to differential organization of the hypothalamus in males and females in the first place, later play a key role in sexual motivation. Even with a fully functional brain, sexual behavior in mammals and other species is strongly dependent on sufficient levels of gonadal steroids (i.e., testosterone, estrogen, and progesterone; Nelson, 2005). In females of many species, including our own, initiation of sexual activity coincides with the high-estrogen phase of the reproductive cycle (Wallen, 2001; note, however, that in most other species, females not in estrus show no sexual interest at all). Removal of the ovaries leads to a loss of sexual appetite, which can be restored through the administration of estrogen (Zehr et al., 1998). Similarly, male sexual motivation in humans and other species depends on sufficiently high levels of testosterone (Nelson, 2005). Notably, in many parts of the brain testosterone needs to be converted to estrogen first before it can have an effect on behavior, and studies have shown that male sexual motivation requires the presence of both testosterone and testosterone-converted-to-estrogen in the brain (Baum, 1992).

The release of gonadal steroids does not just fuel sexual motivation, but can itself be the outcome of a motivational process. For instance, research on rats has shown that conditioned sexual cues can trigger the release of testosterone in males (Graham & Desjardins, 1980). And a study with human subjects revealed that heterosexual men experience a transient testosterone rush when meeting an attractive woman (Roney, Mahler, & Maestripieri, 2003).

4.4.4 Learned sexuality

Findings about the roles of hypothalamus and hormone levels in sexual motivation may be taken to suggest that sexual motivation is a purely biological phenomenon, without any influence of environmental factors. Contrary to this idea, biopsychologists have collected ample evidence that sexual behavior is strongly dependent on social learning processes, even so much so that some talk about “learned sexuality” (Woodson, 2002). The just described conditioned hormone release effect is

one example of learned sexuality. Moreover, rats reared in social isolation show clear deficits in sexual motivation and copulatory performance later in adulthood, and even animals that were reared socially need to learn, through Pavlovian and instrumental conditioning processes, how to tell male from female, what types of signals are sent by a potentially willing partner, and how to copulate appropriately. Even something as "biological" as male sperm production is amenable to learning: male Japanese quail release more spermatozoa and an overall greater volume of semen during copulation if they have been exposed to a Pavlovian-conditioned sexual cue which stimulated sperm production in the gonads in a preparatory fashion before copulation (Domjan, Blesbois, & Williams, 1998). This dependence of sexual behavior on learning may also explain why in species whose behavior is particularly open to learning, such as humans, sexual motivation and performance can remain intact for a long time even after sudden loss of gonadal function and why the females of our and some other primates (e.g., the bonobo chimpanzee) show sexual motivation and behavior even during low-estrogen, non-fertile phases of the reproductive cycle.

4.4.5 Summary

During development hormonal factors play a critical role in the organization of gendered body morphology and brain structures. After maturation, sexual motivation and performance depend on activational effects of gonadal steroids. The ventromedial nucleus and the medial preoptic area are the hypothalamic control centers for sexual behavior (particularly copulation) in females and males, respectively, and are functionally integrated with the brain's incentive motivation network (i.e., amygdala, mesolimbic dopamine system). Adaptive sexual behavior also depends on learning processes that allow organisms to learn about and discriminate sexual cues and to acquire behaviors that are instrumental for successful mating.

5. Conclusion

In this chapter, we have tried to provide an overview of the biopsychology of motivation, an

incredibly vast, multifaceted, fascinating and lively field of study often overlooked by social-cognitive motivation psychologists who rely predominantly on self-report and experimental studies with humans. As a consequence, with relatively few exceptions, the biospychological and social-cognitive approach to the study of motivation have gone their separate ways for a long time, the former exploring the brain correlates of basal needs such as hunger, sex, or affiliation, and the latter examining people's goals, self-views, attributions, and information-processing biases. However, the fact that we were able to weave into this chapter many studies conducted with human subjects (and we are certain that only a couple of years from now, we could include many, many more) suggests that the divide between the two fields of motivation research is about to break down. It is our hope that as biopsychologists become more interested in the way that fundamental motivational needs play out in the human brain, human motivation researchers will become more curious about how motivational processes and constructs that are uniquely human are "embrained" and embodied.

Guiding questions

Question	Answer
1. Please describe three frequently used research strategies in the biopsychology of motivation. What are these strategies almost always combined with?	Biopsychological research on motivation often uses (1) lesioning techniques to study the contributions of specific brain areas to a behavior; (2) recording techniques (e.g., single-cell recording; in-vivo dialysis) to study the behavior of specific neurons; and (3) pharmacological manipulations of synaptic signal transmission to study the role of specific transmitter systems. These strategies are almost always combined with behavioral methods (e.g., Pavlovian or instrumental learning procedures) to illuminate the contributions of specific brain areas or transmitter systems to specific cognitive or behavioral functions.
2. What are the hallmarks of motivation from the perspective of biopsychology?	Motivated behavior can be directed towards the attainment of rewards (approach motivation) or away from punishers (avoidance motivation). Motivation consists of two distinct phases: a motivational phase proper, during which the individual engages in the pursuit of a reward (or avoidance of a punisher) and an evaluation phase, during which the individual consummates

	the reward and evaluates its "goodness". Although there are many different classes of reward (e.g., food, sex, dominance), they can all engage similar motivational processes (e.g., response invigoration, learning). Motivated behavior changes its goals dynamically, depending of how recently a given need has been satisfied and what kinds of incentives are available in a given situation. Motivation can be induced through a physiological need, the presence of incentive stimuli, or both. Motivation makes use of, and shapes, learning of stimulus-stimulus (Pavlovian conditioning) and means-ends (instrumental conditioning) relationships. Biopsychological approaches to motivation do not assume that motivation requires conscious awareness, but acknowledge that in humans, specialized brain systems support the conscious setting and execution of goals.
3. What is a key function of the amygdala in motivation?	The amygdala forges associations between affectively neutral stimuli (CS) that reliably predict affectively charged events or stimuli (US). In the process, the predictive stimuli take on affective meaning themselves and can induce

	motivational states. The amygdala thus acts as a motivational "homing-in" device that allows individuals to adjust their physiological states and overt behavior to cues that predict the occurrence of unconditioned rewards and punishers and bring them closer to the former or away from the latter.
4. What is the key function of the mesolimbic dopamine system in motivation?	The mesolimbic dopamine system invigorates active behavior directed towards the attainment of reward or safety.
5. What is a key function of the orbitofrontal cortex (OFC) in motivation?	The OFC evaluates the "goodness" of primary and secondary (i.e., learned) rewards based on the individual's current need state, learning experiences, and previous exposure to the reward.
6. What is a key function of the lateral prefrontal cortex (LPFC) in motivation?	The LPFC guides behavior through the formulation of complex, verbally represented goals and plans for their implementation. It also influences behavior by regulating the output of the brain's incentive motivation network and can shield explicit goals from interference by incentive-driven motivational impulses.
7. What is the difference between active and passive avoidance? Which structure of the	The difference between passive avoidance and active avoidance is that in the case of the former,

motivational brain plays a critical role in the former, but not in the latter?	behavior is *inhibited* in order to avoid a punisher, whereas in the case of the latter, behavior is *executed* in order to attain safety. Active avoidance, but not passive avoidance, depends on functions of the mesolimbic dopamine system.
8. What is alliesthesia? Please provide an example.	Alliesthesia is the changing subjective evaluation of a reward over repeated exposures or across changing stimulus contexts. For instance, most people experience a little piece of chocolate as quite tasty and pleasant, but would respond with nausea and aversion after eating one pound of it.
9. Imagine that you have just finished a large meal. Describe the signals sent to your hypothalamus to indicate that you are full, and how neuropeptide systems in the hypothalamus would respond.	Leptin levels rise in the bloodstream; levels of CCK from the gut also rise. CCK sends signal to the vagus nerve. Leptin and CCK/CCK signal from vagus act on the hypothalamus to increase activity of α-MSH neurons and decrease activity in NPY neurons.
10. How do opioids and NPY differ in their control of food intake / motivation to eat?	NPY is involved in hunger due to energy needs. NPY drives animals to prefer the most calorically dense food available, even at the expense of taste. Opioids are involved in motivation to eat for pleasure. Opioids drive animals to choose the tastier option, at the expense of calories/energy.

11. Describe one role of opioids in affiliation or attachment.	Any of the following: A) opioids reduce distress in infant mammals separated from their mothers, implicating opioid systems in infant-to-parent attachment. B) In primates, opioids are involved in motivation to groom each other. C) In humans, opioid systems may be involved in feelings of affiliation, as evidenced by higher pain tolerance in high-trait affiliation people who watched an affiliation-related film, an effect that was blocked by an opioid antagonist.
12. Describe the role of oxytocin in parent-offspring and pair-bond attachments. Is oxytocin necessary for the initiation of attachment? For the maintenance of the attachment? Is it sufficient?	High oxytocin levels in the bloodstream are necessary for the formation of parent-offsping attachments and pair bonds. However, oxytocin is not sufficient- other hormones and learning factors also are necessary. Oxytocin is not necessary for the maintenance of the attachment, once it is formed.
13. What is the difference between intrasexual and intersexual competition?	Intrasexual competition occurs when members of one gender fight or compete with each other to establish who will be allowed access to members of the other gender, whereas intersexual competition occurs when members of one gender vie for attention and acceptance as a mate of members of the other gender.

14. What is the relationship between dominance and aggression?	Aggression is one form of dominance behavior. But not all forms of aggression serve dominance (e.g., predatory or defensive aggression are not aimed at dominance) and dominance also encompasses non-aggressive behaviors, which are particularly critical for success in primate species.
15. Which hypothalamic structure plays a critical role in dominance and how can this be demonstrated?	The anterior nucleus (AN) of the hypothalamus plays a critical role in dominance, as assessed by piloerection and lateral attack. If the AN is lesioned, dominance behavior ceases; if the AN is stimulated, dominance behavior is facilitated.
16. What is the relationship between dominance and gonadal steroid hormones?	High levels of gonadal steroids (primarily testosterone, but also estradiol) facilitate dominant and aggressive behavior, and dominance success can in turn increase gonadal steroid levels. Thus, the relationship between dominance and gonadal steroids is reciprocal.
17. Which mechanism drives the rapid testosterone changes observed in the context of male dominance challenges?	In males, rapid changes of testosterone release is governed by the stimulatory effects of sympathetic catecholamines (norepinephrine and epinephrine) and the inhibitory effects of cortisol on the testes. In dominant individuals, the effect of sympathetic catecholamines outweighs that of

	cortisol, which in turn leads to a net testosterone increase. In non-dominant individuals, the effect of cortisol outweighs that of the sympathetic catecholamines, which in turn leads to a net testosterone decrease.
18. Which hypothalamic centers regulate male and female sexual behavior, and which specific aspects of sexual behavior are particularly dependent on these centers?	The ventromedial nucleus (VMN) and the medial preoptic area (MPOA) are the hypothalamic control centers for sexual behavior in females and males, respectively. In females, both proceptivity (active solicitation of male sexual interest) and receptivity (readiness to allow males to mate with them) depend on an intact VMN and sufficiently high levels of estradiol and progesterone. In males, copulatory ability depends on an intact MPOA and sufficiently high levels of testosterone, whereas sexual motivation does not depend on the MPOA.
19. What kind of evidence suggests that in sexual motivation, hypothalamic control centers of sexual behavior are functionally integrated with other structures of the brain's incentive motivation network?	Female rats in estrous show increased dopamine (DA) release in the nucleus accumbens at the sight of a male rat, and this increased DA release reflects increased motivation to approach the male. In males, a reduction of DA transmission in the mesolimbic DA system leads to a decrease in sexual motivation, but does not affect

	copulatory ability. Moreover, MPOA lesions lead to a loss of copulatory ability in males, while sexual motivation remained intact. Conversely, if the amygdala is lesioned and the MPOA is spared, male rats are no longer motivated to gain access to an estrous female, but are able to copulate with her once placed on top of her. These findings suggest that sexual motivation depends not just on the hypothalamus for copulatory ability, but also on the amygdala and the mesolimbic DA system for guiding and invigorating an animal's behavior to gain access to a mate.

Box 1: Extraversion: An incentive-motivation trait?

Extraversion is perhaps the most salient personality trait, and individual differences on the continuum from introversion (= low extraversion) to high extraversion have been linked to a biological basis as early as in the second century AD by the Greek physician Galen. The first modern biopsychological account of extraversion was formulated by Hans Eysenck (1967), who mapped individual differences in extraversion onto differences in brainstem arousal systems. He argued that extraverts suffered from low levels of arousal and therefore engaged in vigorous social and physical activities to bring their brains up to a comfortable arousal level at which they can function properly. Introverts, in contrast, have high arousal levels to start with and appear withdrawn because they avoid vigorous activity which would push their arousal level "over the edge" and thus impair their overall functioning.

Although there is evidence supporting the validity of Eysenck's arousal theory of extraversion, it does not seem to capture the complete story. For one thing, as Gray (1981) pointed out, high levels of extraversion resemble a disposition to impulsively seek rewards, whereas high levels of introversion are linked to the avoidance of punishments. Gray's reinterpretation of the extraversion-introversion continuum, which is supported by considerable evidence from animal and human studies, suggests that this trait is less about differences in *arousal* than about differences in *motivation* (cf. Matthews & Gilliland, 1999). A second criticism that can be leveled against Eysenck's theory is that the construct of arousal itself is too undifferentiated. Eysenck developed his theory based on pioneering studies on the role of the brainstem in cortical arousal that were conducted in the 1940's. Later research indicated, however, that the brain houses several arousal mechanisms that serve a variety of different functions, some supporting sensory processes, others supporting attention and memory, and yet others being involved in motor arousal or activation (e.g., Tucker & Williamson, 1984).

Both criticisms were taken into account in a new theory of the biological basis of extraversion formulated by Richard Depue and Paul Collins (1999). According to these authors, individual differences in people's extraversion levels are based on variations in the degree to which the mesolimbic dopamine (DA) system, which can be characterized as a motor arousal system, responds to signals of reward with an increase in DA-modulated synaptic transmission. People high in extraversion, like Pecina et al's (2003) hyperdopaminergic rats, respond with greater activation of the mesolimbic DA system, and thus stronger *wanting,* to incentives than people low in extraversion. As a consequence, their behavioral surface appears more activated, lively, and invigorated than that of introverts. To test his theory, Depue and colleagues (1994) administered DA agonists or placebo (i.e., a substance lacking any neurochemically active compounds) to extraverts and introverts and measured hormonal and behavioral indicators of increased DA-dependent synaptic signal transmission, such as the suppression of the lactation hormone prolactin and increased eye blink rate. As expected, after DA agonist administration, but not after placebo, extraverts showed more prolactin suppression (Fig. 7) and a greater increase in eye blink rate than introverts. These findings suggest that extraverts have a greater capacity for mesolimbic DA system activation, both naturally stimulated by incentive signals and artificially induced through DA agonists, than introverts.

Depue et al's (1994) findings also suggest, for one thing, that people do seem to have some insight into the functioning of their motivational brain. Individuals who endorse many extraversion items on personality questionnaires (i.e., extraverts) may have an accurate perception that they are behaviorally turned on by a lot more things than people who do not endorse such items (i.e., introverts). Yet this does not mean that they can introspectively access the operating characteristics of their mesolimbic DA system; rather, they may perceive in themselves and their behavior the same things that people who know them well perceive, too: namely, that they are outgoing, active, and full of energy a lot of the time. Notably, at the same time, they seem to be largely unaware of what

exactly engages their incentive motivation system in the first place. As Schultheiss and Brunstein (2001; see also Pang & Schultheiss, 2005) have shown, peoples' implicit motives, which reflect which incentives they *like* and will work for, do not correlate with measures of extraversion. In other words, although people do not have introspective access to what is particularly rewarding for them (determined by their implicit motives), they do seem to have a relatively accurate perception of how strongly they respond to reward-predictive cues once they encounter them (represented by their self-reported extraversion level).

Box 2: Two biopsychological accounts of approach and avoidance motivation

In the introduction to this chapter, we have already pointed out that the distinction between approach to rewards and avoidance of punishments is fundamental for the psychology of motivation. Perhaps it is not surprising, then, that a variety of biopsychological accounts for the approach-avoidance distinction have been proposed over the years, and not all of them show strong agreement on which basic structures and systems in the brain are involved in these motivational states. Two of the most influential models have been proposed by the late Jeffrey Gray and by Richard Davidson.

Gray (1971; Gray & McNaughton, 2000) differentiates between three motivational systems: a behavioral activation system (BAS), a flight-fight-freeze system (FFFS), and a behavioral inhibition system (BIS; cf. Fig. 8). The BAS, which Gray associates with the mesolimbic dopamine (DA) system, responds to unconditioned and conditioned reward stimuli, unconditioned and conditioned non-punishment (safety) stimuli, and novel stimuli, which may potentially be rewarding. It is involved in states of approach motivation, but also in active avoidance, that is, if an individual has to generate certain behaviors in order to reach a safe place in the environment. The FFFS is housed in a system consisting of the periaqueductal grey (a sheath of grey tissue enclosing the channel from the 3rd to the 4th brain ventricle), the medial hypothalamus, and the amygdala. It is activated by unconditioned and conditioned non-reward (frustration) stimuli, unconditioned and conditioned punishment stimuli, and novel stimuli, which may be dangerous. The FFFS mediates behaviors that remove the aversive stimulation, such as panicked flight (to escape from a predator or harmful situation), freezing (to not draw attention to oneself or to appear dead), or defensive attack (as a last resort, if one is already cornered by an enemy or predator). Finally, the BIS is identified with the septo-hippocampal system consisting of the septum, the hippocampus, and the connections between these structures. It is activated whenever an approach-avoidance conflict occurs and needs to be resolved; that is, when both the BAS and the FFFS are activated equally strongly by stimuli that

predict reward or safety and stimuli that predict punishment or frustration (e.g., if nutritious food is available in an area that is also the prowling ground of predators). In this case, both approach and avoidance responses need to be inhibited for the individual to be able to assess the situation carefully in order to come to a better understanding of the risks and rewards involved in any further course of action. At the same time, the individual must be on high alert, able to respond to changes in the situation within a split second. This is exactly what BIS activation helps achieve: inhibition of prepotent responses from BAS and FFFS and increased arousal, attention, and analysis of the situation. The BIS is also activated by approach-avoidance conflicts of the kind in which a previously rewarded behavior is no longer rewarded or a previously safe behavior is suddenly punished. In both cases, the BIS is a key mediator for decreased frequency of the behavior, enabling the individual to show extinction of behavior in the former case and passive avoidance in the latter. Without an intact septo-hippocampal system, neither would occur.

Thus, Gray's theory distinguishes not only between approach and avoidance, but within the latter also between active and passive avoidance and escape, and maps these motivational states onto different systems. Approach and active avoidance (as approach to safety) are mediated by BAS activation, escape by FFFS activation and passive avoidance (and, more generally, approach-avoidance conflict resolution) by BIS activation.

The starting point of Davidson's (2000, 2001) theory is evidence that people who suffer a stroke or lesion in the left frontal cortex are much more likely to subsequently experience severe depression than individuals who suffer a stroke in the right frontal cortex or other cortex areas. Could the frontal cortices also be involved in affective states and traits in people with an intact, healthy brain? To examine this question, Davidson and his colleagues conducted studies on the effects of stimuli (e.g., movies or pictures) that induced strong positive or negative moods on asymmetries in frontal cortex activation, as assessed with electroencephalograms (EEG), positron emission

tomography (PET) and other techniques. They found that presentation of affectively positive stimuli led to greater left-frontal activation than negative stimuli, which elicited stronger activation in the right frontal lobe. These findings held not only for adults, but were already observable in 10-month-old infants (Fox & Davidson, 1988). Further research indicated that frontal asymmetries could not only be obtained with transiently induced mood states, but also when individual differences in stable traits were measured: individuals who indicated on personality questionnaires that they were prone to approach rewards showed stronger resting left-frontal activation, whereas individuals who characterized themselves as typically moody and prone to motivational withdrawal showed stronger resting right-frontal activation (Sutton & Davidson, 1997; see Fig. 9). These findings are not restricted to humans: resting asymmetries in frontal activation predict hormonal and behavioral indicators of positive and negative affect in monkeys, too (e.g., Davidson, Kalin, & Shelton, 1993). Davidson (2000) concluded from these findings that, both as a state and as a trait, approach motivation is associated with left-frontal activation and avoidance motivation is associated with right-frontal activation.

But what exactly is the role of the frontal lobes in approach and avoidance motivation? Do they *generate* these states or *regulate* them? Davidson (2000) argues that the latter is the case. He and his colleagues have gathered evidence using PET and fMRI suggesting that increased left-frontal activation leads to decreased activation of the amygdala, a structure that has been implicated in the generation of negative affect. In other words, the left frontal cortex (or specific parts of it) normally keeps the activity of the negative-affect-generating amygdala at bay, and this function can be temporarily weakened by the induction of negative mood states (the amygdala momentarily escapes from left-frontal-cortex control), chronically weakened through a strong avoidance-motivation trait (the left frontal cortex has a generally weak inhibitory effect on the amygdala) and completely abolished in victims of a left-frontal stroke or lesion (the amygdala runs unchecked).

Thus, Gray and Davidson present very different accounts of how and where in the brain approach and avoidance motivation become manifest, with Gray's theory being more concerned with the generators of core motivational states and how they contribute to learning and behavior and Davidson's theory placing more emphasis on the role of the frontal lobes in regulating the experience of positive and negative motivational states. But because both accounts are based on solid and extensive empirical evidence and are not inherently contradictory, it seems possible that both may one day become integrated into a more comprehensive theory of how approach and avoidance motivation are generated and regulated in the brain.

Box 3: Genes and obesity

Researchers discovered leptin via a mutant mouse strain which overeats and becomes very obese. This strain has a defective gene, which scientists termed the *ob* gene (for obesity.) Later, it was found that in normal mice, the *ob* gene codes for the hormone now known as leptin. Without a functioning *ob* gene, the mutant mice cannot produce leptin. Their brains respond as if their bodies contained no fat: the animal acts as if it is starving, and eats voraciously. Injections of leptin return the body weigh and food intake of the mice to normal (Friedman and Halaas, 1998).

Melanocortins were known to affect skin pigmentation in rodents, but their role in food intake was similarly discovered via a mutant mouse strain. This strain also overeats despite extreme obesity, and it has yellow fur- hence its name, the *Agouti mouse*. Researchers found that this mouse strain has a defective gene for a particular melanocortin receptor. The lack of this receptor means that melanocortins like α-MSH cannot have an effect in the brain or in the skin, resulting in obesity and different pigmentation (Carroll et al., 2004).

Do genetic mutations cause obesity in humans? For most obese people, the answer is no. A melanocortin precursor defect has been discovered in humans that leads to obesity, a pale complexion, and red hair, but this mutation is very rare. Genes may influence the propensity to gain weight, but diet and exercise are the most important factors in human obesity (Martinez, 2000).

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Table 1: Neuropeptides that affect hunger and feeding

Neuropeptide	Source	Effect on feeding	Effects on other neuropeptides
Leptin	Fat cells	Decrease	Increases α-MSH; decreases NPY
CCK	Intestine (and brain)	Decrease	Increases α-MSH; decreases NPY
NPY	Brain (hypothalamus)	Increase	
α-MSH	Brain (hypothalamus)	Decrease	
Agrp	Brain (hypothalamus)	Increase	

Figure captions

Figure 1. Effects of incentive (hamburger vs. chow) and need factors (food deprivation vs. ad lib feeding) on food intake. Adapted with permission from Panksepp, J. (1998). *Affective neuroscience: The foundations of human and animal emotions*. New York, NY: Oxford University Press.

Figure 2. Sagittal cut of the brain at the midline, with approximate locations of key structures of the motivational brain. Closed circles represent structures fully or partly visible in a sagittal cut; dashed circles represent structures that are hidden from view in a sagittal cut. The amygdala is hidden inside the frontal pole of the temporal lobe; the lateral prefrontal cortex is located on the outer side of the prefrontal cortex; the nucleus accumbens is a part of the striatum and lies at the front of the subcortical forebrain. The ventral tegmental area modulates activity in the nucleus accumbens via dopaminergic axons (arrow). Both structures are part of the mesolimbic dopamine system.

Figure 3. A schematic overview of the amygdala and some of its nuclei (LA: lateral nucleus; BLA: basolateral nucleus; CE: central nucleus) and the emotional-motivational functions they mediate. After LeDoux (2002).

Figure 4. Recordings from a striatal dopamine (DA) cell of a monkey who received rewarding drops of fruit juice (R) that he learned to associate with a predictive visual or auditory cue (CS). A histogram on top of each panel shows when the cell fired most frequently; single lines with dots below the histogram represent repeated recordings of the time before, during, and after a reward or cue was administered. Each dot indicates when the neuron was firing. Adapted with permission from Schultz, W., Dayan, P., & Montague, P. R. (1997). A neural substrate of prediction and reward. *Science, 275*, 1593-1599.

Figure 5. An illustration of the dissociation between wanting (running speed to goal box; left panel) and liking (intake of sweet solution; right panel). Adapted with permission from Ikemoto, S.,

& Panksepp, J. (1999). The role of nucleus accumbens dopamine in motivated behavior: a unifying interpretation with special reference to reward-seeking. *Brain Research Reviews, 31*(1), 6-41.)

Figure 6. An illustration of need-dependent reward evaluation in the OFC. On both panels, the x axis displays amount of glucose solution fed (in mL). Upper panel: The Y axis displays firing rate of sweet-responsive neurons responsive to glucose, relative to responses to drops of saline (SA) or blackcurrant juice (BJ). Lower panel: Behavioral acceptance of glucose solution. Adapted with permission from Rolls, E. T. (2005). Taste, olfactory, and food texture processing in the brain, and the control of food intake. *Physiology and Behavior, 85*(1), 45-56.

Figure 7. Relationship between responses to a DA agonist as assessed by the amount of prolactin suppression relative to placebo (higher levels =greater suppression) and scale scores on Positive Emotionality, a measure of extraversion. Greater DA activation is associated with higher levels of positive emotionality. Adapted with permission from Depue, R. A., Luciana, M., Arbisi, P., Collins, P., & Leon, A. (1994). Dopamine and the structure of personality: relation of agonist-induced dopamine activity to positive emotionality. *Journal of Personality and Social Psychology, 67*(3), 485-498.

Figure 8. Gray's conceptual-nervous-system model of motivation. Normal arrows represent activating effects, blocked arrows represent inhibiting effects. Modified with permission from Gray, J. A., & McNaughton, N. (2000). *The neuropsychology of anxiety* (2 ed.). Oxford (GB): Oxford University Press.

Figure 9. Frontal brain asymmetry and approach and avoidance motivation. Higher mid-frontal EEG asymmetry scores represent greater relative left-frontal activation, and higher scores on the BIS-BAS scales of represent greater relative approach motivation Adapted with permission from Sutton, S. K., & Davidson, R. J. (1997). Prefrontal brain asymmetry: A biological substrate of the behavioral approach and inhibition systems. *Psychological Science, 8*, 204-210).

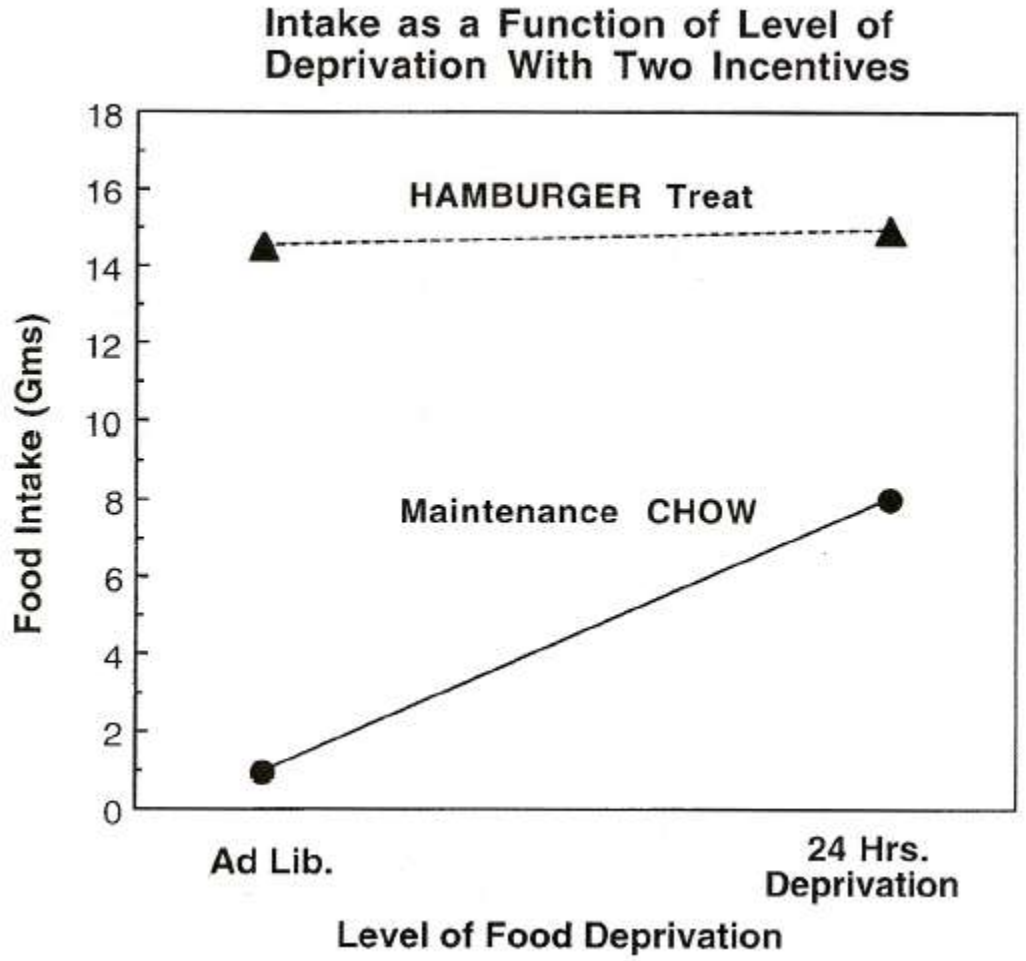
Intake as a Function of Level of Deprivation With Two Incentives
18
16
14
12
10
8
6
4
2
0
HAMBURGER Treat
Maintenance CHOW
Food Intake (Gms)
Ad Lib.
24 Hrs. Deprivation
Level of Food Deprivation

Figure 1

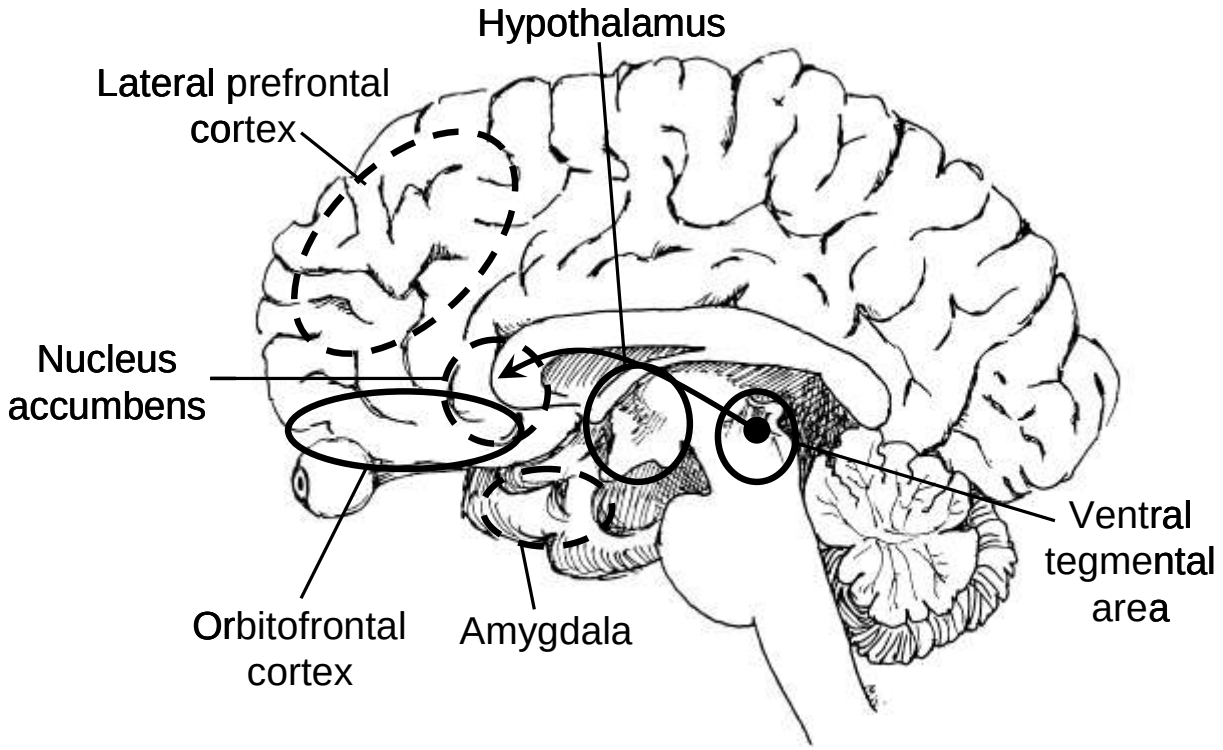
Hypothalamus
Lateral prefrontal cortex
Nucleus accumbens
Ventral tegmental area
Orbitofrontal cortex
Amygdala

Figure 2

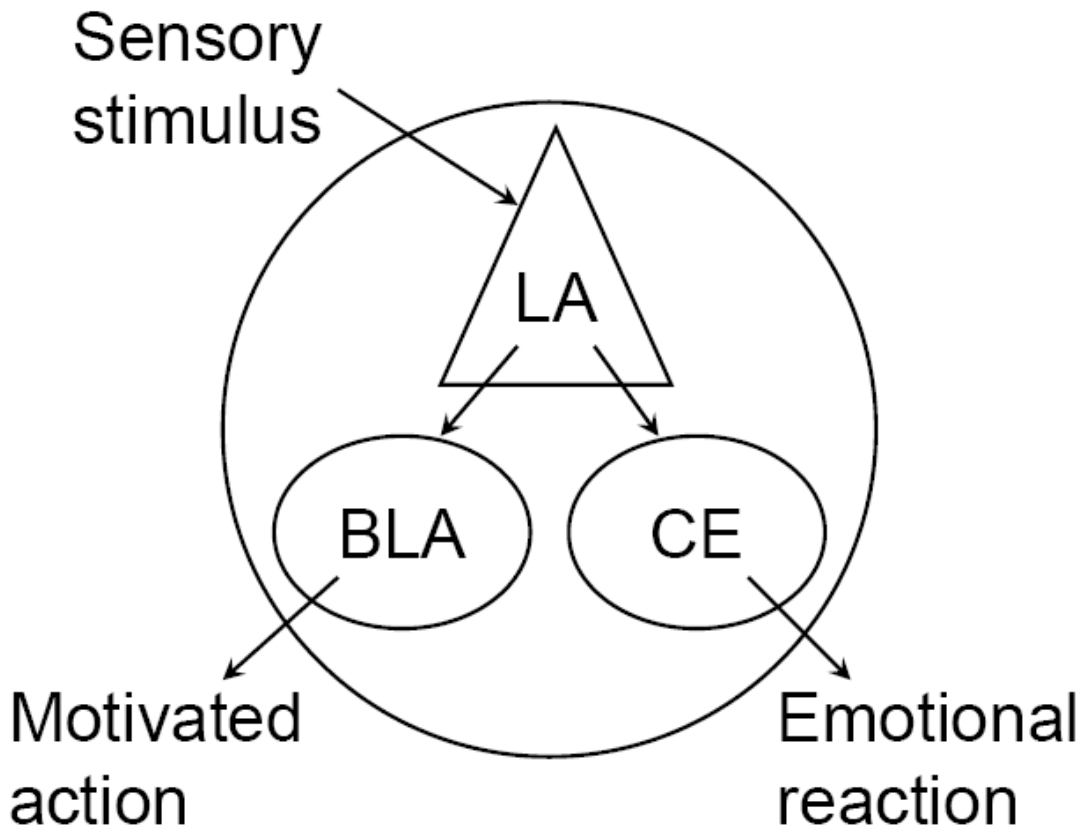
Sensory
stimulus
LA
BLA
CE
Motivated
action
Emotional
reaction

Figure 3

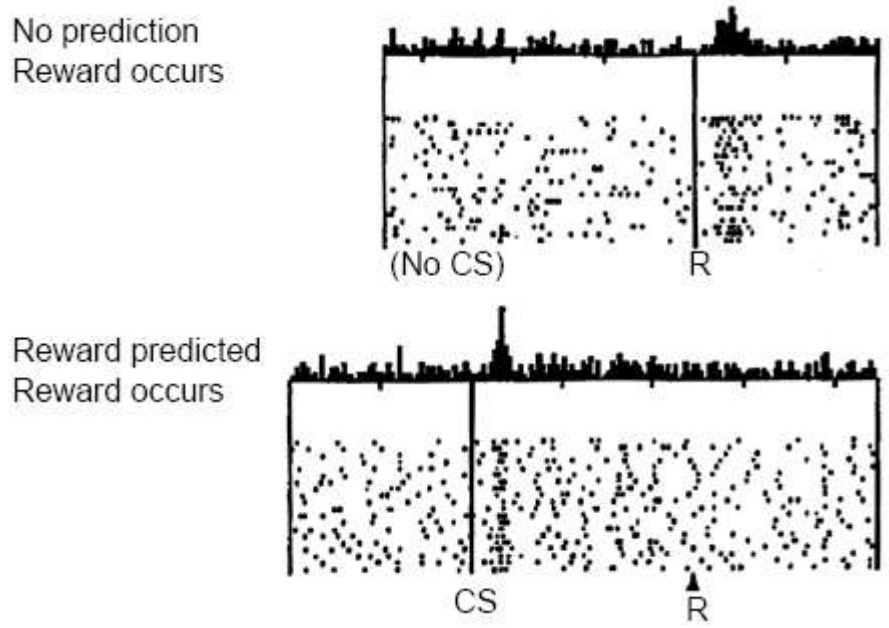
No prediction
Reward occurs
(No CS)
R
Reward predicted
Reward occurs
CS
R

Figure 4

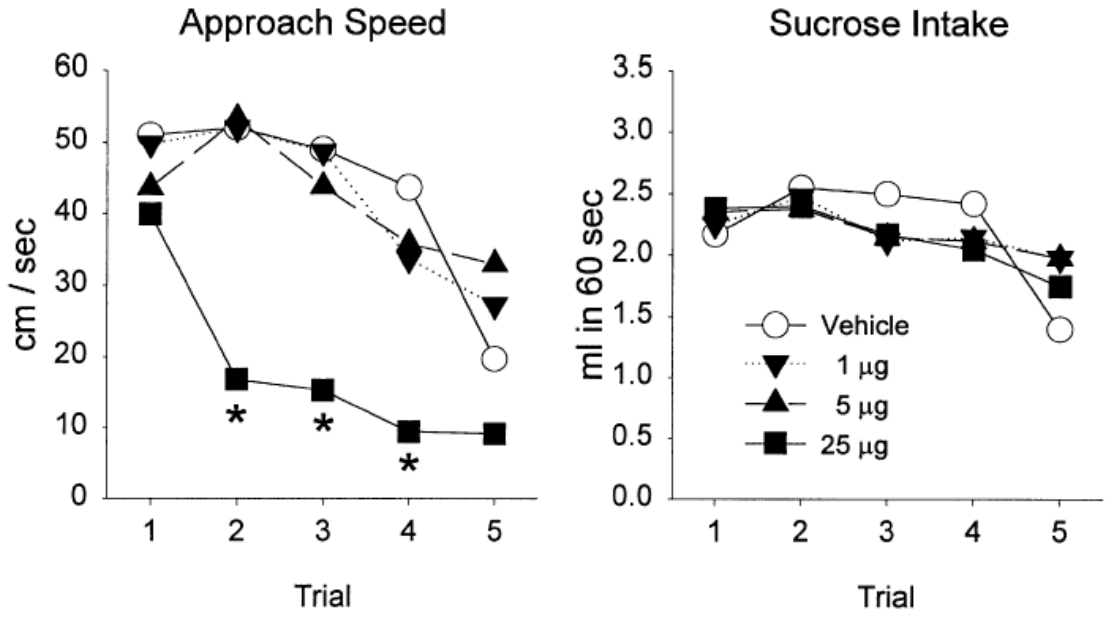
Approach Speed
cm / sec
60
50
40
30
20
10
0
1
2
3
4
5
Trial
*
*
*
Sucrose Intake
ml in 60 sec
3.5
3.0
2.5
2.0
1.5
1.0
0.5
0.0
Vehicle
1 µg
5 µg
25 µg
Trial

Figure 5

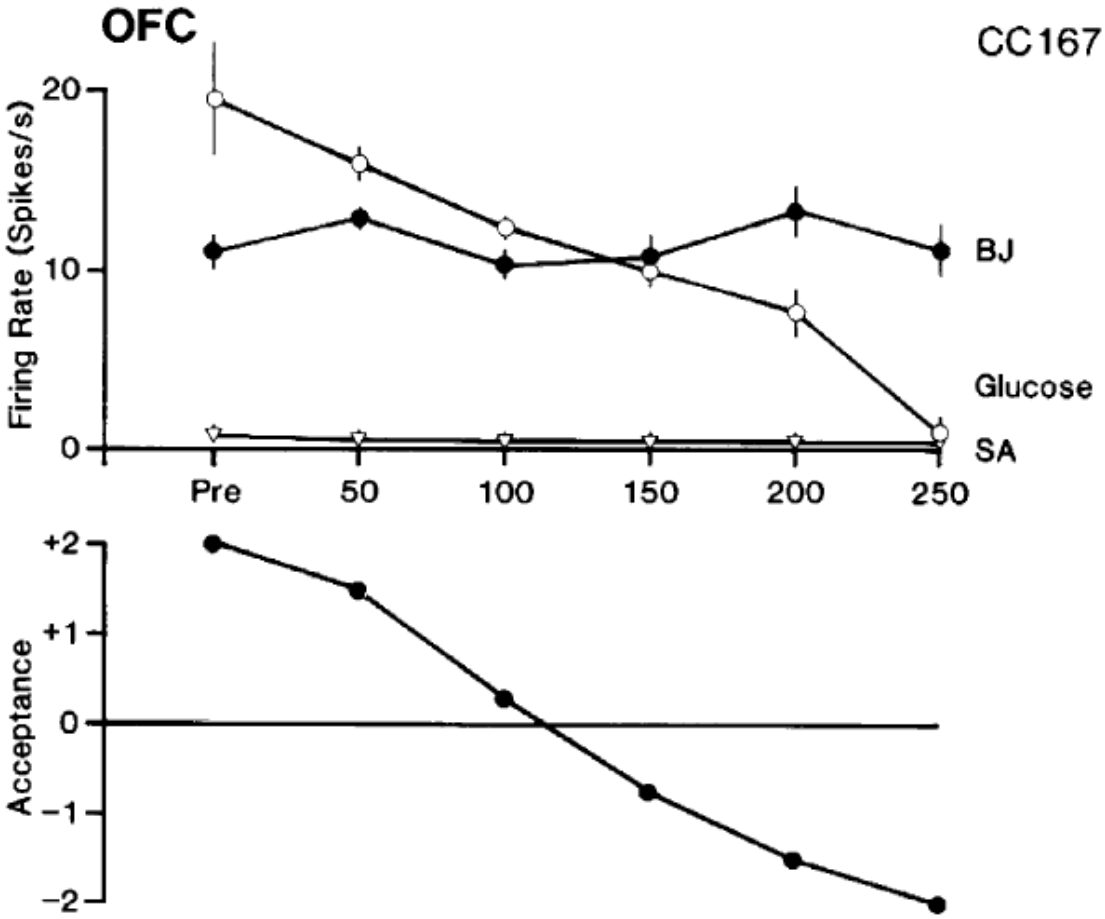
OFC
CC167
Firing Rate (Spikes/s)
20
10
0
BJ
Glucose
SA
Pre
50
100
150
200
250
Acceptance
+2
+1
0
−1
−2

Figure 6

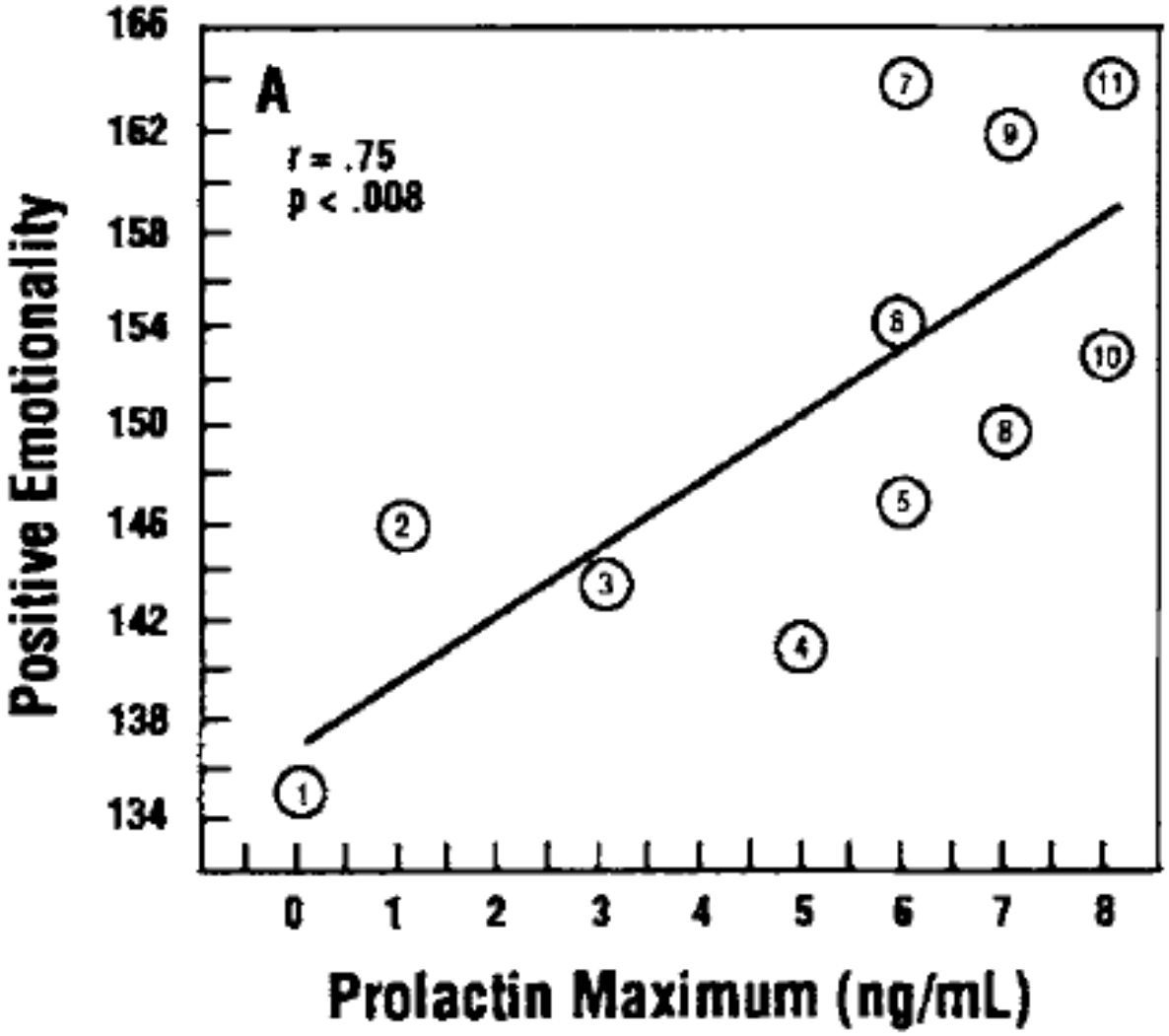
A
r = .75
p < .008
Positive Emotionality
166
162
158
154
150
146
142
138
134
0
1
2
3
4
5
6
7
8
Prolactin Maximum (ng/mL)

Figure 7

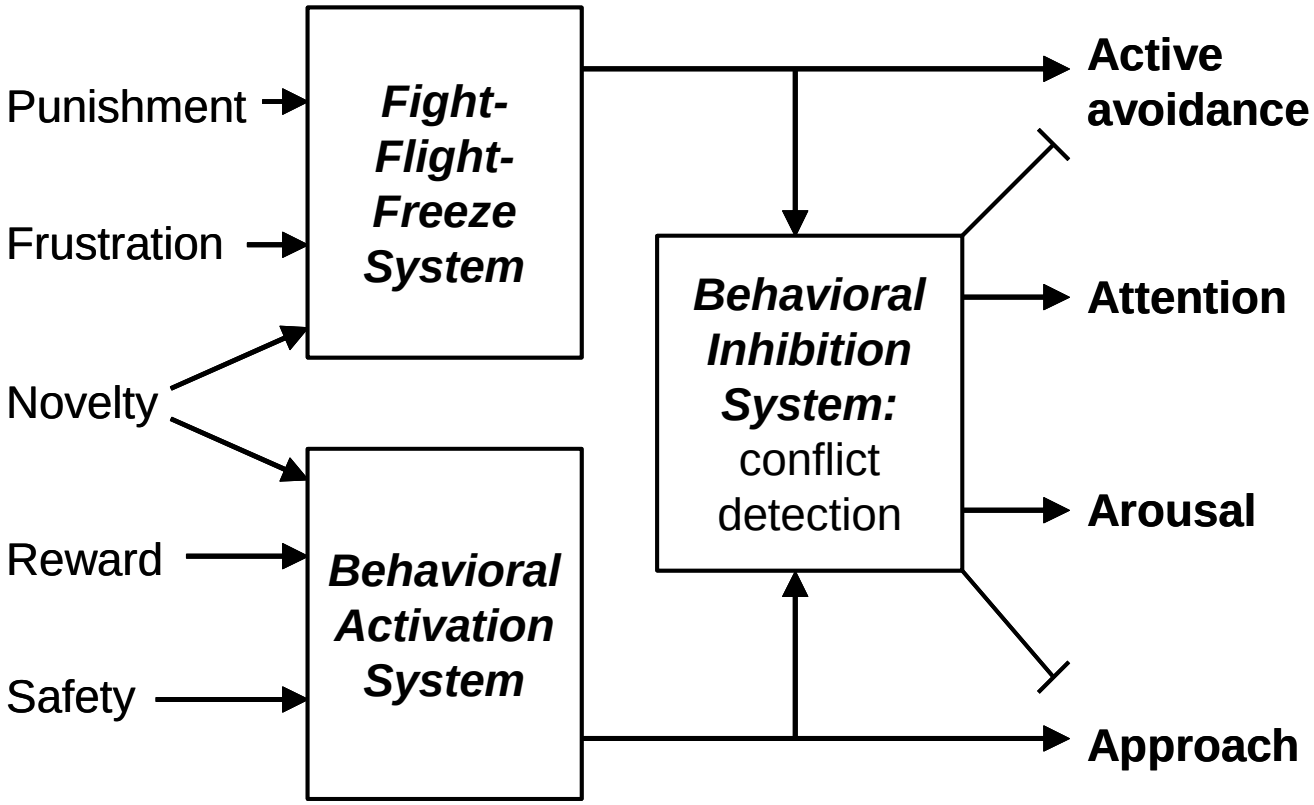
Punishment
Frustration
Novelty
Reward
Safety
Fight-Flight-Freeze System
Behavioral Activation System
Behavioral Inhibition System: conflict detection
Active avoidance
Attention
Arousal
Approach

Figure 8

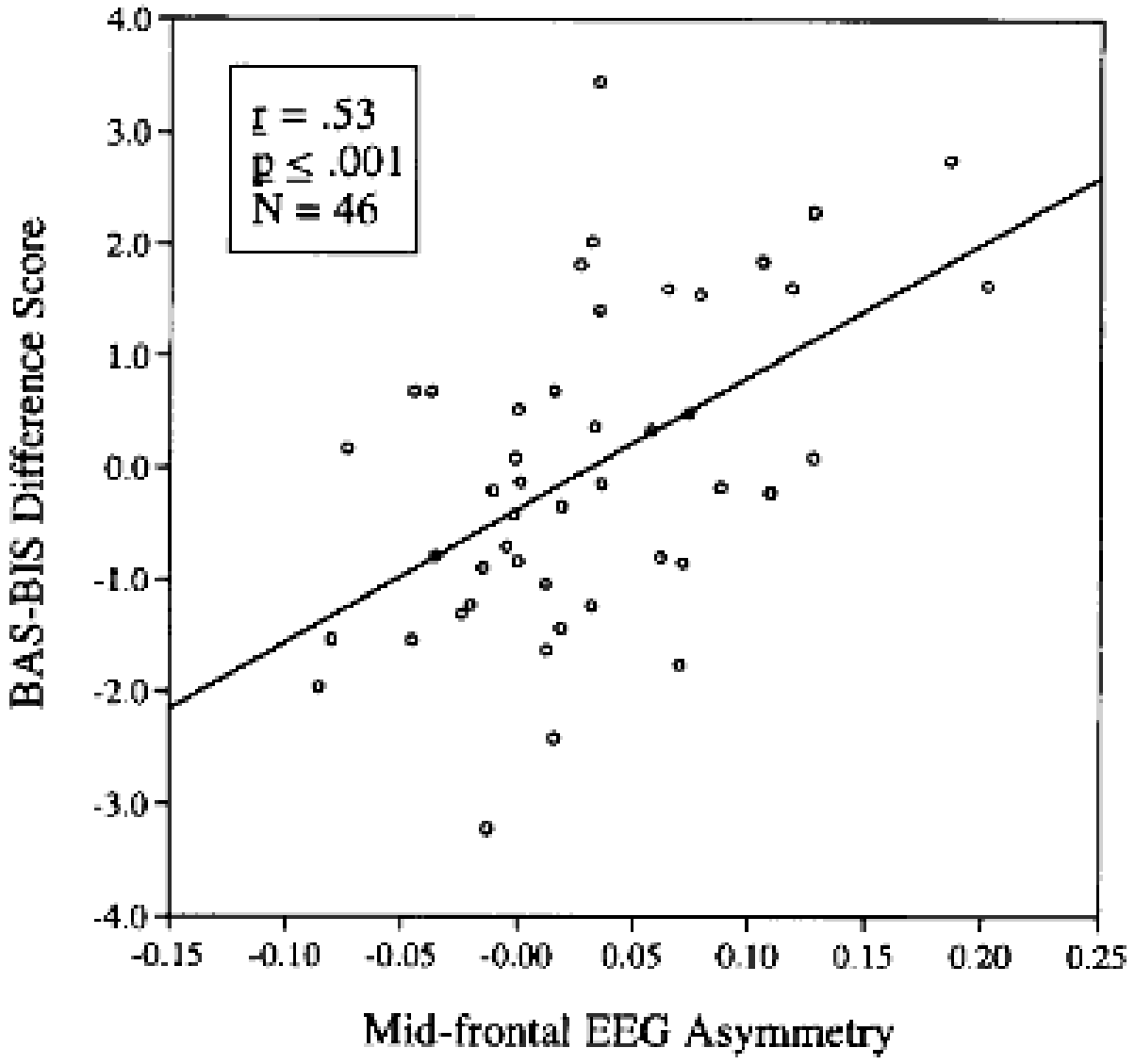
r = .53
p ≤ .001
N = 46
BAS-BIS Difference Score
4.0
3.0
2.0
1.0
0.0
-1.0
-2.0
-3.0
-4.0
-0.15
-0.10
-0.05
-0.00
0.05
0.10
0.15
0.20
0.25
Mid-frontal EEG Asymmetry

Figure 9