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Motivation concepts in behavioral neuroscience

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Abstract

Concepts of motivation are vital to progress in behavioral neuroscience. Motivational concepts help us to understand what limbic brain systems are chiefly evolved to do, i.e., to mediate psychological processes that guide real behavior. This article evaluates some major motivation concepts that have historic importance or have influenced the interpretation of behavioral neuroscience research. These concepts include homeostasis, setpoints and settling points, intervening variables, hydraulic drives, drive reduction, appetitive and consummatory behavior, opponent processes, hedonic reactions, incentive motivation, drive centers, dedicated drive neurons (and drive neuropeptides and receptors), neural hierarchies, and new concepts from affective neuroscience such as allostasis, cognitive incentives, and reward '*liking*' versus '*wanting*'.

Keywords: Motivation; Behavioral neuroscience; Limbic brain systems; Drive; Hunger; Thirst; Sex; Aggression; Homeostasis; Pleasure; Reward; Incentive; Addiction; Hypothalamus; Nucleus accumbens

Contents

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1. Introduction

Motivation has resurged as a topic for behavioral neuroscience. Motivational concepts are becoming widely recognized as needed to help neuroscience models explain more than mere fragments of behavior. Yet, if our motivational concepts are seriously wrong, our quest for closer approximation to brain-behavior truths will be obstructed as much as if we had no concepts at all. We need motivational concepts, and we need the right ones, to properly understand how real brains generate real behavior.

The time seems right to review and evaluate some major concepts of motivation of traditional or contemporary importance in behavioral neuroscience. Eating and drinking motivation will be highlighted here because this collection of journal articles is targeted principally to hunger, thirst, and related ingestive motivation, but the concepts are relevant to a wide range of other motivated behaviors too. This review is not complete and omits many motivational concepts that also deserve consideration. But my hope is to provide an initial assortment that may be useful to students and colleagues in behavioral neuroscience as they continue to evaluate brain systems of motivation in light of new discoveries.

For over 100 years, motivation concepts have been considered necessary, chiefly to understand two features of behavior. First is the variability of an individual's behavior over time in the face of constant stimuli. That is, why do individuals choose to do different things at different times? Internal brain and physiological processes of motivation are especially useful in explaining behavioral variability when the external environment stays constant. Second is the short-term stability and directedness of behavior as an individual seeks to obtain a goal or avoid a threat. That is, why do individuals seek out specific things at particular times? And why do they react as they do to affectively important stimuli encountered on the way? Motivation concepts are aimed at helping us understand these questions. When we combine these concepts with behavioral neuroscience research, we gain a better understanding of both brain and behavior.

2. Homeostasis and drives

Chief among the concepts of motivation in behavioral neuroscience is homeostasis and drive. Among the oldest in the motivation armamentarium, homeostatic drive concepts continue to underlie the thinking of many behavioral neuroscientists today. Hence, it is fitting that we start with homeostasis and drive. In practice, these concepts have usually been combined into one: homeostatic drive.

Homeostasis means maintaining a stable internal state. The word was coined in 1925 by the physiologist, Walter Cannon, and developed in a book several years later [30]. Cannon acknowledged that his ideas drew heavily on earlier ideas of Claude Bernard and other physiologists, but his homeostasis term was an original and useful focus for thinking about physiological regulation and stability.

In behavioral neuroscience of the past 50 years, homeostasis usually means a specific type of regulatory system that uses a setpoint, or built-in goal value, to maintain a stable physiological state (Fig. 1). The setpoint is compared constantly to the real physiological state of the moment, and that comparison detects whenever error or mismatch occurs between the physiological state and its setpoint goal value. The regulated value might be a physiological parameter that is crucial to normal function or, else, some other parameter that is merely a correlated marker of the more crucial one. In either case, an error will occur whenever the monitored parameter strays away from its goal value. When an error is detected, a homeostatic mechanism triggers appropriate correction responses. Thus, the modern homeostasis concept requires several mechanisms in the brain: a setpoint, an error detector to measure the actual physiological situation and decide if a deficit exists, and an error correction mechanism such as a motivated drive to activate appropriate responses (e.g., eating). Those responses provide negative feedback that corrects the deficit and brings physiological reality back to the setpoint (Fig. 1).

Homeostatic motivation has often been compared with the operation of a thermostat that regulates a room's temperature. A thermostat is designed to be a homeostatic mechanism. The thermometer in the thermostat continuously measures the actual temperature of the room and compares it with the previously chosen setpoint, or thermostat setting. If the measured temperature deviates too far from the setpoint, an error detector in the thermostat activates the furnace or air conditioning system to bring the temperature back to the setpoint range. In practice, the setpoint can be conceived as either a single optimum level or, more likely, a narrow range of acceptable levels. A range allows mechanisms to rest for

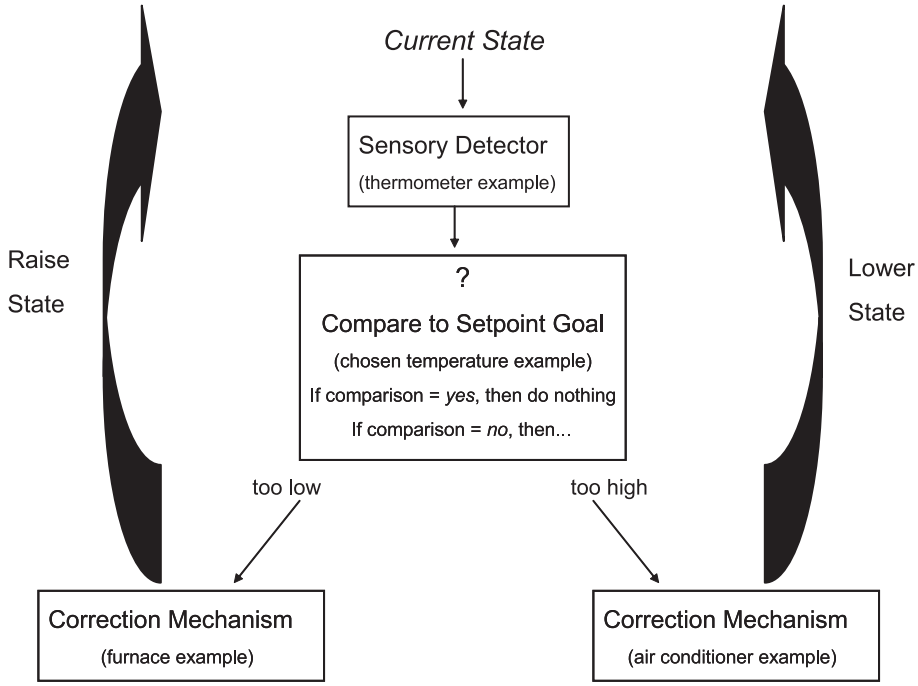

Fig. 1. Homeostatic mechanism. The mechanism uses negative feedback to correct errors in current state. The current state of the moment is compared with the setpoint or desired goal value. If the current state is too high or too low, an error-correcting mechanism is activated until the current state returns toward the setpoint. A room thermostat is an example of a homeostatic mechanism based on setpoint, goal comparison and error detection, and negative feedback correction. In the thermostat example, the setpoint is the thermostat level you set, the current state is the room temperature detected by the thermostat's thermometer, and errors of room temperature are corrected by thermostat activation of heating or cooling systems.

longer periods, without requiring nearly continual adjustments to chase after one elusive single value.

The important task for a homeostatic mechanism is to avoid error states outside of the narrow setpoint range, and this is important because a deviation out of the regulated range might be dangerous. Walter Cannon's phrase "the wisdom of the body" highlighted the usefulness of this regulation for survival [30]. Homeostatic error detection mechanisms embedded in neural, hormonal, and other physiological systems are presumed to become activated whenever the monitored parameter becomes too low (or too high in some cases). The goal parameter might be body water level, neuronal glucose level, nutrient storage level, or some other physiological factor. Error detection initiates correction responses to bring the level back into the regulated range. For example, too low levels of body fluid and blood pressure should trigger angiotensin hormonal responses that activate thirst and drinking behavior [46,52,79,140].

Similarly, if the brain really uses homeostatic mechanisms for hunger, then, it should have a setpoint for some specified range of body weight, blood glucose, nutrient storage, neuronal metabolism, or some other physiological variable relevant to hunger and satiety that could be monitored for deficit [18,55,58,63,123,137,138,157,192]. What is important to recognize is that behavioral neuroscience concepts of homeostasis require the existence of both setpoints and error detectors to monitor physiological deficits. This is easy to imagine for ingestion-related motivation like hunger, thirst, salt appetite, calcium appetite, or another specific nutrient appetite. Homeostasis, with a little stretching, has also been suggested for other motivations such as sex or aggression. In those cases, homeostatic models sometimes postulated that motivated behavior was triggered when the crucial regulated factor, such as levels of sexual hormones or related steroids in blood, reached too high a level after a long period of sexual deprivation or after long repression of aggression. For example, the hydraulic model of motivation of Lorenz [94] discussed below triggers aggressive or other motivated behavior almost homeostatically when the motivational signal overflows. Behavioral expression of sex or aggression in such homeostatic models is then presumed to lower hormonal or other factors back to below-threshold range. Other motivations such as drug addiction also have been argued to involve homeostatic properties, although they do not maintain a physiological parameter in the same sense as hunger or thirst does [87,152]. For example, Solomon's opponent process theory of motivation discussed below suggests that homeostasis logic applies to brain hedonic systems that mediate many different types of motivation [87,152].

Homeostasis has dominated behavioral neuroscience thinking about motivation. It is not too much of an exaggeration to say that the behavioral neuroscience of hunger, thirst, salt appetite, and other ingestive behavior in the past 50 years has been primarily a search for physiological setpoints and deficit signals. The concept has remained

the same although the particular cast of deficit signals focused on has varied with the interests of the neuroscientist: neuronal glucose uptake or glucose metabolism in brainstem or hypothalamus neurons, hepatic vagal signals about the nutrients in the liver, neurochemical actions of angiotensin, neuropeptide Y, leptin, and related neuropeptide receptors on neurons, etc.

An implication of this homeostatic emphasis is that motivation has often been taken to be nearly understood once the homeostatic-deficit trigger and its receptors are found. Naturally, that approach has led to exciting progress on the identification of deficit receptors and signals. Still, the emphasis on homeostatic deficit detection may also have had a cost in decades past, by diverting attention from other equally pressing questions about motivation in the brain, such as how brain systems mediate motivational functions beyond deficit detection. Fortunately, these questions are now being actively pursued too.

2.1. Homeostasis-like outcomes without homeostatic mechanisms

Although homeostasis has remained the dominating concept, splits and schisms have recurred over the years about whether brain mechanisms of motivation are truly homeostatic in operation or whether they simply look that way at first sight. Remember that contemporary behavioral neuroscience considers a truly homeostatic mechanism to operate by a setpoint and an error detector. But what if a mechanism maintains stable regulation without those components? Is it homeostatic? The answer is no if we are talking about the *mechanisms*, which behavioral neuroscientists usually are. By the definition that has reigned for decades, a homeostatic mechanism must use a setpoint and detect errors—like a thermostat. A mechanism that does not use those is not homeostatic. Yet the answer could be, "well, yes, sort of, more or less, at least, for most practical purposes" if we were only concerned with the *constant outcome*. Homeostatic outcomes without homeostatic mechanisms can occur if stability is maintained either by anticipatory mechanisms (that initiate motivated behavior before a deficit ever occurs) or by stability resulting from "settling points" (a stable balance among opposing forces) instead of setpoints.

2.1.1. Anticipatory motivation

Anticipatory drinking, eating, or other motivated behavior can be elicited as a classically conditioned response or by another preemptive mechanism before a physiological depletion ever occurs [53,130,139,182]. For example, thirst may be activated around mealtime, before the eaten food has transferred any water via secretion from the blood plasma into the intestines (producing hypovolemia), or added any ingested salts via absorption to produce hyperosmotic blood or stimulate osmotic brain detectors [53]. Similarly, many meals in ordinary life may be initiated before there is any detectable drop in blood glucose or in other physiological nutrient signals [192]. From the outside, anticipatory motivated behavior may look homeostatic because it helps maintain a stable physiological state over the long term. Anticipatory drinking or eating provides water, food, etc. that will eventually be needed, just before it is actually needed. But the mechanism is not homeostatic in such cases because there has been no physiological deficit and, hence, no error detection. When current state is compared with the setpoint goal, the two remain essentially the same (Fig. 1). If anything, a temporary surplus exists when you ingest in advance of need. No violation of physiological setpoint is involved in the behavior because no actual water deficit or nutrient deficits ever occur in these cases. Therefore, the homeostatic comparison and error detector mechanism depicted in Fig. 1 cannot be the mechanism that triggers anticipatory prevention response. An anticipatory mechanism is different.

Yet, this is not to say that brain systems of homeostatic thirst are not activated during anticipatory thirst when, say, you sit down for a meal or that you do not really feel thirsty—really and truly thirsty. Nor does it mean that the fluid you drink because of your anticipatory thirst is not soon needed in a physiological sense. Although no fluid deficit yet exists in your plasma nor any hyperosmality in your brain at the moment you sit down, the thirst motivation is real, and the water you drink is soon useful. And certain brain circuits may be activated that also would be activated in real homeostatic thirst. Whether triggered by predictive cues or by an actual physiological depletion, the final brain state and psychological motivation of thirst may well be identical, equally real, and equally thirsty. Anticipatory thirst may preemptively contribute to eventual stability as effectively as homeostatic thirst does and may use some of the same brain mechanisms. For example, anticipatory thirst triggered by intestinal hyperosmality or other early cues that predict future blood hypovolemia or brain hyperosmality can be blocked by some of the same neurochemical receptor antagonists that block depletion-triggered thirst (e.g., Angiotensin II antagonists) [130]. In real life, most instances of eating and drinking may occur in the absence of classical homeostatic deficits, acting either to coopt or preopt the depletion-cue detectors that trigger thirst or hunger in emergency cases of real deficit [46,52,79,130,139,192].

2.1.2. Settling points and illusory homeostasis

Another way of maintaining an outer illusion of homeostasis without true homeostatic mechanisms inside is through settling-point regulation [187]. A settling point is a stable state caused by a balance of opposing forces, but without any setpoint or error detection. There are many examples of nonhomeostatic settling point mechanisms. In nature, for example, sea level is maintained as a settling point without a homeostatic setpoint. Sea level has been so stably constant over centuries that the term 'sea level' has a definite meaning for altitude. It is the level 8850 m below the peak of Mt. Everest, which is used to define the height

of that mountaintop and nearly everything else. Yet, no one thinks for a moment that sea level has a setpoint or goal value, or that the level has been maintained so constantly by a homeostatic mechanism. There is no error detector or correction mechanism. No one measures the level and compares with to a preset value, no one fills up the ocean when it gets too low, or lets water out when too high. The stable sea level has settled as a balance between factors such as evaporation or polar freezing, which reduce water level, and opposing factors such as rainfall and polar melting, which increase it. The balance has settled around the point we call sea level and has stayed there throughout history because the forces are in equilibrium. Settling points are sometimes that stable. Still, settling points can change, and will change if opposing forces alter their balance. For example, sea level might conceivably rise if global warming causes the polar icecap to melt and release its water into oceans. The balance would settle at a new higher level, and some currently landlocked zones would become oceanfront properties without ever moving. Settling points are only settled at best, never regulated via error detection to match a true setpoint.

Just so, hunger, body weight, and other apparently homeostatic motivations might actually reflect physiological settling points rather than true setpoints, arising as a balance among opposing neural–hormonal–behavioral systems [22,119,187]. For example, in a provocative 1980 article, the biopsychologist Robert Bolles [22] argued that hunger and eating behavior have no homeostatic mechanism. He argued that there is no body weight setpoint, and, thus, hunger can never be triggered by an error deviation from a setpoint. Instead, body weight simply settles around a point that is only moderately stable. The settling point is determined not only by internal appetite and satiety mechanisms, but also by the external availability and palatability of foods, as well as other factors related to eating behavior. If obesity rates have risen in recent decades, Bolles would have argued that no brain setpoints have been changed. Rather, external conditions, foods, and norms have changed. What endures is the trait of people and many other animals to eat tasty treats whenever they can and to overeat treats if they are plentiful, tempting, and continuously within reach (especially if eating is not restricted to meals by cultural norms or other external factors). People persist in overeating in such situations despite no homeostatic deficits and even in the face of a body-weight surplus.

Bolles suggested that our homeostatic concept of body-weight setpoint is simply a fiction kept because it has a priori plausibility and seems a comfortable explanation. But instead of a homeostatic setpoint, he argued that body weight is kept relatively stable by opposing neuroendocrine and psychological reflex mechanisms that simply happen to be in balance at your current weight settling point—as is sea level. Palatability and appetitive signals in brain mesolimbic dopamine systems, or in NPY activation of hypothalamic neurons, act as positive signals to stimulate eating. Leptin or other satiety cues act as negative signals to stop eating. Many of these inputs to the settling point are tonic, being constant over weeks or months, whereas some others are phasic, occurring only around meals. But all act as opposing groups of mere linear signals, similar with reflexes, which cause food intake and body weight to settle at some point. They are all at equilibrium wherever your body weight is now.

Bolles argued that body weight itself was no more regulated than other signals that influence eating, including our perception of food palatability, because all these signals modulate the brain impact of others. For example, body weight can change palatability, not just vice versa. If we are truly starving, even ordinary food becomes especially delicious. Nearly inedible foods that now are unappealing to you may then seem worth eating. Similar malleability may apply to all other internal signals that influence eating, each of which is modulated by feedback from the others. The result of this interactive feedback is that the system tends to arrive at a settling point and to stay there as long as prevailing conditions remain unchanged. There is no homeostatic setpoint above and beyond this settling point, Bolles concluded. Similar considerations have recently been revived by Pinel et al. [119] to argue against homeostatic setpoints in controlling human hunger.

The rather startling implication of settling point ideas is that the brain's goal-oriented mechanisms of hunger, thirst, and other motivations might not be truly homeostatic in mechanism after all [18,102,139,187]. By this view, obesity caused by a lesion of ventromedial hypothalamus, a leptin deficit, or another neural cause is not due to a raised setpoint, but rather to an alteration in the balance of opposing neural factors that favors a higher settling point. Similarly, homeostatic setpoints have neither prevented nor caused the recent trend toward human obesity in American and some other societies, or in pet animals that have similar access to abundant palatable foods. Many people might be happier if a setpoint existed that could prevent them from gaining unwanted weight, but such human setpoints seem missing just when we need them. Instead, by this view, expanding waistlines reflect a higher settling point among many factors, only some of which have changed. Those changed factors are mostly external and include abundance of tasty calorie-rich foods, cultural patterns of extensive snacking outside of meals as well as large meal portions and low exercise rates [132,180]. Conversely, aphagia and stable, low body weights caused by lateral hypothalamic lesions or diet drugs are due to an opposite shift in opposing factors, which balance at a lower settling point, and are not due to the suppression of a body-weight setpoint. Thus, perhaps, brain mechanisms of regulation need to be divorced from the setpoint concept and instead incorporate mechanisms that operate in a settling point fashion.

In retrospect, setpoint and homeostasis concepts never needed to be as tightly conjoined as they have been in the past half-century. Walter Cannon, the inventor of homeo-

stasis, actually never wrote about setpoints, setpoint comparisons, or error detections. Instead, Cannon thought about homeostasis exclusively in terms of the concept of opposing reflexes, with which he was already familiar, such as spinal reflexes of leg extension versus leg retraction [30]. For Cannon, a high stimulus triggered an internal reflex that reduced the stimulus and restored homeostasis—essentially as a settling point. Later ideas of setpoint, error detection, and negative feedback arose only 15 years later. The setpoint concepts arose from cybernetic theory in engineering and computer science, where control theory concepts of setpoint, goal/reality comparison, and negative feedback correction were well established [183]. Cybernetic setpoint concepts were stunningly elegant as explanations and, hence, were irresistibly seductive to brain scientists. After all, if machines were operated by setpoints, then, it seemed plausible that brains did too. However, in science, as in life, seductive appearance is not necessarily a guarantee of lasting value. The trend in thinking now seems to be that setpoints are misleading for understanding how brain systems of hunger or thirst really work. Clearly, the question of setpoint versus settling point is important for behavioral neuroscientists who want to identify the true mechanisms of motivations such as hunger and thirst.

2.1.3. Allostasis

A last point worth mentioning on homeostasis is its relation to the alternative concept of allostasis [88,97,136,141,159]. Allostasis is usually offered in contrast to homeostasis and refers to physiological regulation of *changed* states.

In its strongest sense, allostasis can involve *positive feedback responses* such as snowballing neuroendocrine responses to stress [97,141]. Positive feedback happens when initial responses to a change contribute themselves to larger later responses to subsequent changes. For example, when a microphone gets too close to a loudspeaker, positive feedback can cause a sound system to spiral into harsh reverberating noise. This is opposite to the negative feedback of homeostasis, where a response opposes an original change and restores the original balance. In behavioral neuroscience, positive feedback may characterize some physiological reactions to prolonged stress, when the hypothalamic–pituitary axis responds stronger and stronger to a series of repeated stressors. In the most extreme cases, rising and cumulative stress reactions may eventually cause damage to the brain structures such as the hippocampus or amygdala [97,141].

Sometimes, allostasis has also been used to describe cases where regulation and levels change over time, but which otherwise behave homeostatically, and use negative feedback responses. Examples may include addicts who take drugs to escape or avoid drug withdrawal [88]. Originally, those people felt relatively normal without drugs, but once withdrawal symptoms are caused by their heavy drug use, they might conceivably take more drugs just to make those withdrawal symptoms go away. In such cases, there may be no positive feedback responses. Instead, the descent into withdrawal is caused by homeostatic negative feedback changes such as the down-regulation of neurotransmitter receptors or other settling point factors [86]. Rather than allostasis, the shift might as easily be called fluctuating homeostasis—the state simply chases a moving settling point (i.e., moving from normal drug-free state to a down-regulated withdrawal state after drugs). Indeed, some behavioral neuroscientists have used both homeostasis and allostasis terms to describe essentially the same withdrawal events [87,88].

Allostasis is a relatively new term, thus, it is difficult to specify now which meaning will become most accepted. If we wish allostasis and homeostasis to be distinctly different concepts for brain mechanisms, then, it might be best to restrict allostasis to cases that involve positive feedback responses and use homeostasis concepts for all negative feedback responses. But if we merely use allostasis to indicate that a homeostatic settling point has shifted, then, the two concepts will overlap in cases where an equilibrium changes over time (e.g., culturally induced obesity, drug withdrawal, global warming). Future usage by behavioral neuroscientists may prove the best guide for deciding which sense of allostasis will endure.

2.2. Intervening variable definitions of drive

We began with homeostatic drives. Perhaps, this is a good point to step back, separate drive from homeostasis, and see what remains. You may wonder—if drive is not defined as homeostasis, then how can drive be defined at all? Do not despair. Drive concepts flourished throughout the 20th century, often in homeostatic forms, yes, but homeostasis was not their reason for being. The reason for drive's being was the explanation and prediction of behavior. Drives were accepted because they not only provided homeostatic explanation for motivated behaviors (hunger, thirst, etc.), but also were useful to make the most efficient causal descriptions and predictions. Drives were useful even if the explainer was an atheoretical behaviorist, who eschewed motivation and other psychological concepts as a matter of principle. For the purpose of seeing the logical usefulness of drives, when stripped down to their bare minimum, let us try for a moment to view them from the behaviorist perspective.

To understand why, imagine ourselves to be rigid behaviorists restricted to explanations based only on observable stimuli and responses (S–R relationships). In principle, no motivational explanation at all might be needed as long as the behaviorist considers only one type of stimulus (for example, amount of water deprivation) and one type of motivated behavior (e.g., amount of water drunk later). Then, the behaviorist could just do the behaviorist thing, which is to measure the objective relationship between deprivation and drinking (stimulus and response). But the situation begins to change as soon

as additional stimuli and/or additional responses are looked at. That is when a drive concept becomes valuable to everyone, at least defined in its most minimal form as an *intervening variable*.

There are two reasons why we might wish to know about the intervening variable view of drive. First, by understanding the logic that once predominated thinking about drives, we can better understand much of what is meant when drives are talked about, even today. Second, there still remain today many contemporary counterparts to the behaviorist of the last century, in the form of neuroscience reductionists. Some of these reductionists still adhere to a basically logical positivist or extreme materialist conviction that all behavior must be explained without recourse to psychological levels of concepts and, instead, solely in terms of directly observed physical events: neurons, neurotransmitters, etc. Such extreme reductionism is a vestige of 20th century positivist tendencies toward explanatory concreteness at all costs and, though now relatively rare, it has not yet disappeared. For modern reductionists, just as for traditional behaviorists, motivation is a difficult explanatory concept because it cannot be directly observed as a physical event. For some reductionists, as for behaviorists, the only acceptable definition of drive or motivation is the intervening variable one. Thus, it is worth spending a page or so on the logic of drive as intervening variable.

Neal Miller, an important behavioral neuroscientist in the 1940s–1980s, made perhaps the clearest argument for the intervening variable view of drive as an explanatory concept, using thirst as an example [99]. To understand his argument, first, think of how a behaviorist/reductionist might explain what causes a rat or person to drink water (drinking and other behavioral responses are called dependent variables in this parlance, and stimuli or events that cause them are called independent variables; thirst or other motivational states stand in between cause and response as intervening variable). Deprivation of water is one cause of subsequent drinking, of course, but thirst and drinking also can be caused by other independent variables. You might get thirsty after becoming overheated, or after eating dry or salty food, or even after being injected with a hypertonic solution of sodium chloride, which, as it is saltier than normal blood, triggers drinking via hyperosmotic brain detectors [46,52,130]. Conversely, rats and people may drink more water when thirsty, but they also do other things, such as being more willing to work to get just a sip of water or being willing to drink even a bitter-tasting fluid (such as one that contains quinine).

A pure behaviorist/reductionist restricted to S–R (stimulus–response) descriptions would have to posit three different causal relationships between the independent variable of water deprivation and the three dependent variables of water consumption, lever presses for a sip, and quinine toleration (Fig. 2). Plus, if we add a new independent variable such as being too hot, the S–R explanation needs three more causal S–R relationships between it and the three dependent variables, plus three more S–R relationships if we add eating salty food, and three more for hypertonic injections, and so on for every new independent variable (Fig. 2). If we keep on adding independent variables or begin to add any new dependent variable responses, the number of S–R relationships soon explodes. We could end up with hundreds of causal S–R links and would need to find a brain mechanism for every one of them. Small wonder that an efficient scientist looks for some way to get explanatory parsimony and reduce the number of S–R relations that need to be explained.

Better causal parsimony can be achieved, Miller noted, simply by positing thirst as an intervening variable (Fig. 2). The intervening variable "thirst drive" connects all the causes to all the behavioral expressions of thirst. By connecting the dependent variables and independent variables through one common route, an intervening variable dramatically reduces the number of causal relationships and mechanisms. Adding a new cause adds only one new relationship—not three or more new relationships. Although the intervening variable is invisible and not physical, it is a purely objective relationship. For even the most positivist criterion of what it takes to be real, quantitative relationships have reality status in almost the same sense that mathematical entities such as π are real (π is the ratio between a circle's circumference and diameter). By the behaviorist/reductionist criterion of what is real (which we adopt for the moment, although it is arguably far too restrictive for understanding brain function), the reality of an intervening variable is manifest in its control of the dependent variable outputs. Therefore drive, as an intervening variable, has objective quantity that can be measured via its correlated effects on behavioral dependent variables.

2.2.1. Escaping circularity

One potential worry about motivational explanations is that they can become circular if used carelessly. A circular explanation is one that attempts to explain an observation in terms of itself. It just reasserts what has been observed and does not really add any new explanation. For example, if we explain why you are reading this article by supposing you to have a "drive to read about behavioral neuroscience concepts", we merely have restated what we've already seen you do. Everyone agrees that a circular explanation is no real explanation at all.

But motivational explanations need not be circular. The key to escaping circularity lies in using drive (or any other motivational concept) to make new predictions, not just restate what has already been seen. For example, if we infer that hypertonic injections cause thirst drive because we see it makes a rat drink, we can then predict it should also increase behaviors that we have not yet measured, such as lever pressing for water or quinine toleration. Similarly, Miller predicted that a thirsty rat would be more willing to get water even if it received a slight shock when crossing an

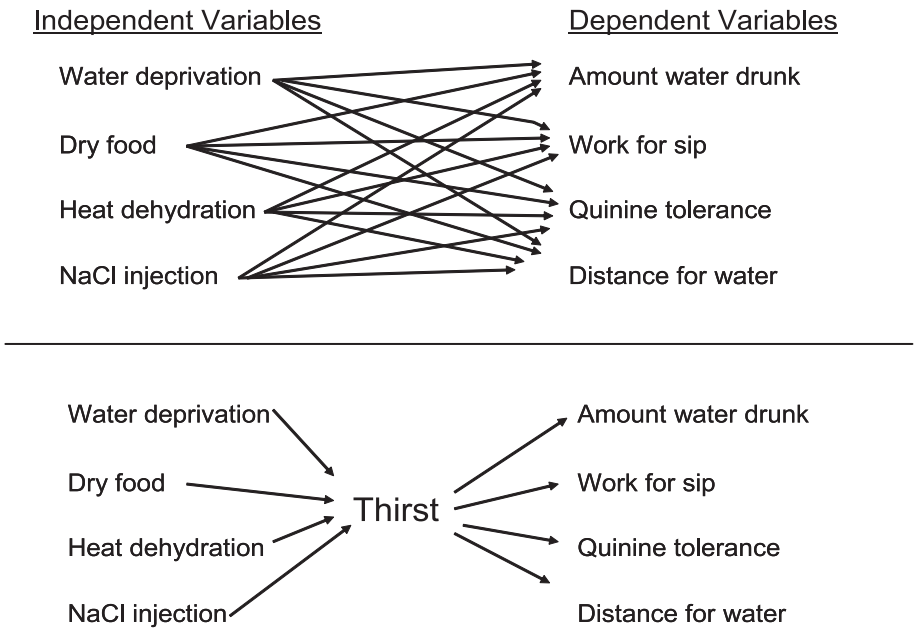

Fig. 2. Intervening variable concept of drive simplifies S–R relations. Top panel shows the 16 causal arrows needed to explain drinking (given four manipulations and four behaviors) without an intervening variable. Bottom panel shows the number of causal relations required, which is cut in half to eight when thirst is posited as an intervening variable. Modified from Miller [99].

electrified floor in front of the water spout compared with a rat that was less thirsty. If such predictions are correct, then, our drive concept has been validated and is no longer circular. Such predictions generally are correct [99]. Because usefulness is defined in science by being able to make true predictions before the experiment, thirst drive is clearly scientifically useful.

Still, what if a drive increases behaviors, but differently for different responses of the same motivation? This often happens in real life and is a point that worried Miller. For example, what if the amount drunk, lever presses, and quinine tolerance go up at different rates when thirst increases (Fig. 3)? We do not know which behavior most accurately reflects thirst, hence, we do not know exactly how much thirst has changed. Does that mean that we cannot measure thirst, or that it is not a distinct drive after all? The answer is no, not necessarily. Even one independent variable will produce multiple different effects on several responses if they have different mechanisms that execute them. One reason is that different dependent responses produce different consequences that feed back to constrain later performance. The amount drunk is constrained by how big your stomach is, but stomach size does not affect how much you can work for a sip, or how much bitter quinine or painful shock you might be willing to tolerate as the price of a drink. Likewise, muscle fatigue constrains how much work can be done for a sip by a lever-pressing rat, but not amount drunk or quinine toleration. In other words, feedback from specific behavioral expressions of motivation may alter the way they express a motivation. This arises from the effector feedback or feedback from behavioral consequences. A more central or motivational explanation of why different behavioral outputs sometimes diverge, even for one drive, is provided by the Lorenz hydraulic model of motivation discussed further below.

3. Raising the bar for motivation: flexible goals, affective displays

We have seen that drives, triggered by internal depletion cues and directly activating behavioral responses, are one

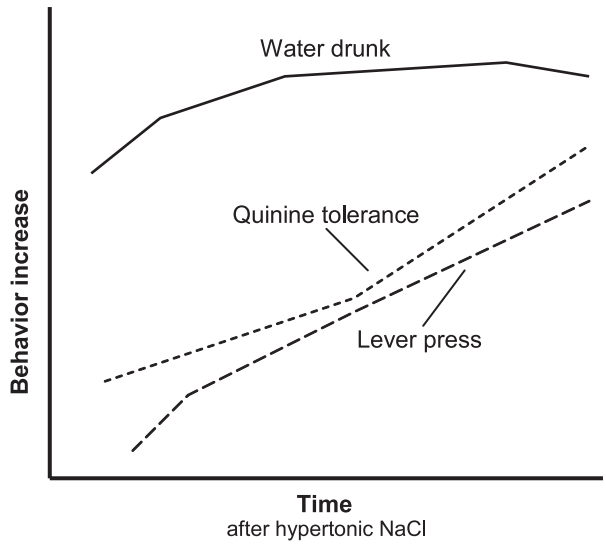

Fig. 3. Correlation of thirst behaviors after hypertonic NaCl. All behavioral measures increase together, but the rates of increase are different. Modified from Miller [99].

way of conceiving of internal motivational states. But the intervening variable is only the most minimalist concept of motivation. It is relatively impoverished and sterile, leaving out lots of what makes motivation interesting in the psychological sense and lots about how motivation actually works in brain systems. Even a mere hungry fly has motivation in the intervening variable sense of drive [43]. The phrase hungry fly was coined by Dethier [43] to describe his influential 1960s studies of the neurophysiological reflexes in a housefly, reflexes which control how much sugar it will eat [43]. Basically, a fly has two eating reflexes. An excitatory one makes it eat whenever it lands on food, and an inhibitory one stops eating when the fly stomach becomes full. If the inhibitory reflex was removed by cutting a sensory nerve from the fly gut, Dethier [43] found that the fly would continue to eat until it burst its tiny stomach. This pair of opposing reflexes can be viewed as controlling the fly's eating drive, but although that intervening variable may be causally elegant, it is almost appallingly simple both neurally and psychologically. If that is all that hunger is, then, the brains of mammals, like us, seem to contain a lot of unnecessary neurons and limbic circuits. But two ingestive reflexes are hardly satisfying as a complete model of motivation—at least not to anyone unwilling to accept a fly as adequate model for motivation as we know it. Flies may get hungry in Dethier's sense, but hunger in a fly lacks several aspects of hunger in you. How can we identify the difference?

To avoid oversimplification and to identify what makes motivation interesting, several behavioral neuroscientists have suggested that we set minimum criteria or bottom limits for defining real motivation [44,46,61,163,164]. A mere drive or intervening variable that activates responses will never qualify for these more complex and interesting senses of motivation.

For example, Teitelbaum [163,164] suggested, around 1970, that real motivation must be able to motivate flexible instrumental behavior. In practice, an animal or person must learn a new operant response to gain a goal to prove they were motivated for that goal (e.g., learning to press a bar for reinforcement). An operant response can be selected arbitrarily at the whim of the experimenter, to ensure it was not activated by simpler reflexive or instinctive mechanisms. Learning of an operant demonstrates for Teitelbaum that the creature was motivated in a crucial sense of the word—in the sense of being willing to do most anything to gain the goal.

Teitelbaum's operant criterion drew conceptually on an earlier descriptive classification of motivated behavior, which was proposed a century ago by the early American ethologist, Wallace Craig (building on even earlier formulations by Sherrington and others; [34,147]). Craig proposed, based on careful study of animal behavior, that all motivated behavior could be divided into two sequential phases, an *appetitive phase* followed by a *consummatory phase*. Craig's appetitive phase of motivated behavior is the flexible approach behavior that an animal or person emits before the motivational goal is found. Flexible appetitive behavior helps find the goal. Instrumental behavior or operant responses performed to gain access to a goal are a type of appetitive behavior, easily produced, and measured in standard behavioral neuroscience laboratories. The consummatory phase follows only once the goal object is actually obtained. Consummatory behavior is elicited by the goal stimulus, and thus consummates the appetitive phase. Consummatory behavior often is a stereotyped and species-typical pattern of movements: chewing and swallowing food, licking and drinking water, etc. But the real root meaning of the word consummatory is not consumption but rather consummation. It is almost accidental that ingestive consummatory behavior involves consumption of food and water; it does not for sex, aggression, or other non-ingestive-motivated behaviors. Consummatory behaviors for those motivations are sexual copulatory patterns or aggressive biting. Consummatory behavior terminates an appetitive phase of behavior and gives actual transaction with the sought-after goal, consummating the flexible seeking that went on before.

Teitelbaum's definition of motivation as the operant pinpoints the appetitive phase as essential. In other words, it was not enough to qualify as motivation to have consummatory behavior or even to modulate consummatory behavior via homeostatic drive in an intervening variable sense. One needed also appetitive behavior, flexible enough to interact with instrumental associative learning to shape new operant responses.

However, even flexible operants may leave out some important aspects of motivation. Computer programs that are extremely simple can learn operant responses (e.g., "if response X is followed by reinforcement, then repeat X with increased frequency"). The reinforcement in this simple computer program has no motivational properties other than that it increases the emission probability of future responses. By itself, mere response reinforcement does not capture the essence of what many people mean by motivation, including many behavioral neuroscientists.

Epstein [46] suggested around 1980 that three additional criteria are needed to distinguish truly motivated behavior. These criteria are (1) flexible goal directedness or means–end readiness, (2) goal expectation, and (3) affect. Goal directedness essentially builds upon Teitelbaum's operant learning idea. This criterion means behavioral demonstration that the target was a true goal, shown by flexible learning and coordinated appetitive behavior aimed at obtaining the goal, both changing appropriately when the alteration of circumstances necessitate new strategies to obtain the goal. It means to rule out both simple forms of learning and simple drive activation of behavior. Instrumental learning is one form of demonstration of goal directedness, but is not the only one, nor the most complex or most convincing. For example, cognitive inference of a new spatial route to the goal would qualify even better as evidence for goal directedness because it could not be

solved by a simple algorithm such as "increase response frequency after reinforcement".

The second criterion of expectation for the goal carries this further. When you walk to your refrigerator, you do it because you expect to find something nice inside. You may imagine what that something will look and taste like, even before you open the door. These are declarative, cognitive forms of goal expectation, and very much a part of the motivation that causes behavior in human everyday life. That is why Epstein wished to include expectation as a defining feature of motivation, even for animals. In a lesser sense, goal expectation might mean forms of associative learning that anticipate the goal, without necessarily involving a cognitive type of expectation, such as classical conditioning of an anticipatory conditioned response. Epstein was not always clear about what form or level of goal expectation he meant and tended to combine them in his discussion. In any case, he was willing to accept a rat experiment effect called incentive contrast (also known as the Crespi effect after its 1940s discoverer; [35,54]). For example, if a rat has learned to run down a path for a particular food reward, its running speed gradually gains a value proportionate to that reward. Now, if the reward is suddenly increased, the rat is likely to run rather dramatically faster on its very next trial after the new and improved reward experience—even though it has never yet been reinforced for the new faster speed. This effect is usually explained (as Crespi suggested) by supposing that the rat had learned to expect its original food reward, and that it was pleasantly surprised by its larger reward when it occurred. The surprise could not have occurred if the rat had no expectation—no representation of what the reward should have been. Hence, the occurrence of an incentive contrast effect could be taken as evidence that a goal expectation did exist, even in the rat [54]. Modern psychologists' criteria for expectation in animals tend to be more demanding [3,32,44,120]. For example, one might need to show that the animal possesses a declarative and continually updated representation of an outcome's current value (gained from past experiences) or demonstrate that the animal understands the causal relation between a particular outcome and the particular action that produces it [3,32,44,120]. Still, expectation in some sense was an important criterion for Epstein's concept of motivation.

Third, Epstein suggested that real motivation is always accompanied by affective reactions to the goal itself. By affective reaction, Epstein meant behavioral, autonomic, or similar physiological responses that indicated the presence of some hedonic or emotional state. As Epstein put it, "What I mean by affect is discernable patterns of somatic and autonomic-glandular (both exocrine and endocrine) responding that are expressed as integral aspects of appetitive-consummatory sequences of behavior" (Ref. [46, p.44]). His point was that motivation is directed toward hedonically laden goals, and if a goal is hedonic, it should elicit an affective reaction. Thus, the presence of hedonic reactions confirms that the behavior was truly motivated.

The criteria for motivation proposed by Epstein and Teitelbaum were intended to extend the reach of behavioral neuroscience and ensure that it explained more than hungry flies.

3.1. Opponent process drive concept

The psychologist Richard Solomon suggested a useful general concept for thinking about many drives involving affectively valenced stimuli that are pleasant or unpleasant (Fig. 4). Opponent process theory posits that all hedonic

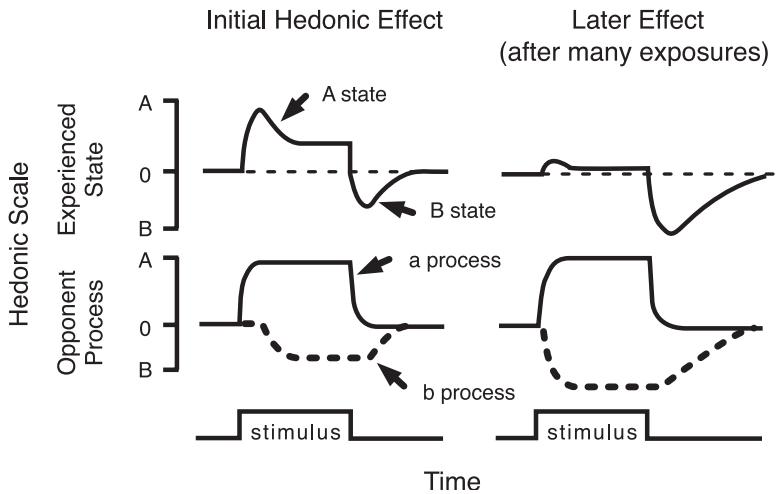

Fig. 4. Solomon's opponent process model of hedonic motivation. The first hedonic experience of a stimulus is modeled at left. The experienced state is depicted at the top panel, and the next line shows the separate processes that add together to cause the experienced state. The occurrence of the stimulus is shown at the bottom. After many exposures to the same stimulus, its later experience of the stimulus is shown at right. The a-process has not changed, but the b-process has strengthened. A stronger b-process causes the experience to be dominated by a B-state instead of an A-state. Modified from Solomon and Corbit [152].

stimuli, if strong and prolonged, activate not only their own direct hedonic reaction in the brain, but also an opponent process of opposite hedonic valence [152]. The oppositely valenced opponent is actively generated by the brain, in response to the first hedonic reaction, which was generated by the stimulus. If the hedonic stimulus is pleasant, then, the opponent is unpleasant. If the stimulus is unpleasant, then, the opponent is pleasant. This opponent concept is strongly related to homeostasis and extends to all affective reactions the homeostatic notion that physiological systems are designed to maintain a neutral balance. An opponent process is always actively generated by the brain to counteract the effect of the original hedonic stimulus. The hedonic opponent helps bring the brain back toward a neutral affective balance. This hedonic opponent model was inspired by a sensory opponent process known to be involved in color processing in brain visual systems [76].

Let us take a hedonic example such as a pleasant addictive drug: heroin. The opponent process theory posits that the pleasant heroin stimulus directly activates first an *a-process* in brain reward circuits, which produces the positive affective reaction (experienced as the *A-state*). But the a-process, in turn, indirectly triggers activation of a negative or opponent *b-process*. The b-process in this case would be unpleasant if experienced by itself (a *B-state*), but when combined with the strong heroin a-process, the b-process partially cancels it and reduces the original A-state. Tolerance is the result of the reduced A-state, manifest as diminishment of the heroin pleasure. Tolerance becomes more pronounced the more often heroin is repeated. An important part of Solomon's theory is that if the heroin stimulus is repeatedly taken again, only the b-process gets strengthened, and not the a-process, which stays proportional to the unchanged stimulus. The unpleasant b-process gets more intense and longer lasting with each heroin use. Finally, an unpleasant withdrawal experience (B-state) is caused after each drug use because at that point, the opponent b-process outweighs the heroin a-process.

For unpleasant stimuli, such as painful shocks, Solomon posited that the a-process was hedonically negative, and, thus, the opponent process works in reverse. For example, pain causes an aversive a-process, which then indirectly activates an analgesic b-process to oppose the pain (such as brain opioid systems). The b-process in this case may reduce pain during the noxious stimulus and, possibly, even cause a rebound into a pleasant B-state after the pain stimulus ends. This has been suggested to be a mechanism for "runner's high" and other positive affective states sometimes reported to be induced in accustomed practitioners by experiences that appear to outsiders to be rigorous ordeals.

Other behavioral neuroscientists have proposed similar opponent models, but some such as Shepard Siegel, Stephen Woods and their colleagues, suggest that classical conditioning gives learned associations the ability to activate opponent process without needing an a-process. That causes conditioned stimuli for pleasant drugs to elicit b-processes, such as conditioned tolerance and withdrawal, and causing pain predictors to elicit conditioned analgesia [121,143,148]. In addition, behavioral neuroscientists such as George Koob [87] have suggested specific neural mechanisms to mediate opponent b-processes, especially for drug addiction, such as drug-induced tolerance or down-regulation in the mesolimbic dopamine system or activation of stress responses in brain such as amygdala release of corticotropin-releasing factor (CRF).

The limits of the opponent process concept are that b-process effects do not always occur for every affective a-process event, and even when they do occur, the opponent b-process is not always the chief motivational factor involved in the behavior. For example, although drug withdrawal occurs as a b-process after heroin, it is surprisingly often not the reason why addicts keep taking drugs [126,145]. However, opponent process concepts often remain useful in thinking about interactions between motivational processes that have different valence.

3.2. Hydraulic drives

Drive models of motivation that extended beyond mere intervening variables had to have explanatory properties that could explain complex aspects of motivated behavior. Why does a stimulus sometimes elicit motivated behavior but sometimes not? Why are more motivated behaviors recruited as a motivation grows? It is worth noting the hydraulic drive model of motivation proposed by Konrad Lorenz, an ethologist and Nobel laureate [94].

Lorenz's hydraulic model is essentially a metaphor that suggests that motivational drive grows internally and operates a bit like pressure from a fluid reservoir that grows until it bursts through an outlet (Fig. 5). Internal causes of a motivational drive (e.g., physiological depletion cues or secreted hormones related to hunger, thirst, aggression, and sex) are like incoming streams that trickle into the reservoir, replenishing the hydraulic fluid for that motivation. Motivational stimuli in the external world (food, water, sexual and social stimuli, etc.) act to open an outflow valve, releasing drive to be expressed in behavior. Although brains do not contain reservoirs, it is easy to imagine internal physiological signals that might linearly increase to fulfill the metaphoric role of fluid accumulation, such as hormone secretion, neurotransmitter receptor activation, neuronal gene expression, or neural firing rates.

In Lorenz's model, internal drive strength interacts with external stimulus strength. If drive is low, then, a strong stimulus is needed to trigger motivated behavior. If the drive is high, then, a mild stimulus is sufficient. As the valve opened more, more drive fluid would flow out, covering more drain holes in the collecting pan below, and so, more behavioral responses would be recruited to express the motivation.

If the drive is extremely high, it may burst into outflow even with no external stimulus at all. Lorenz

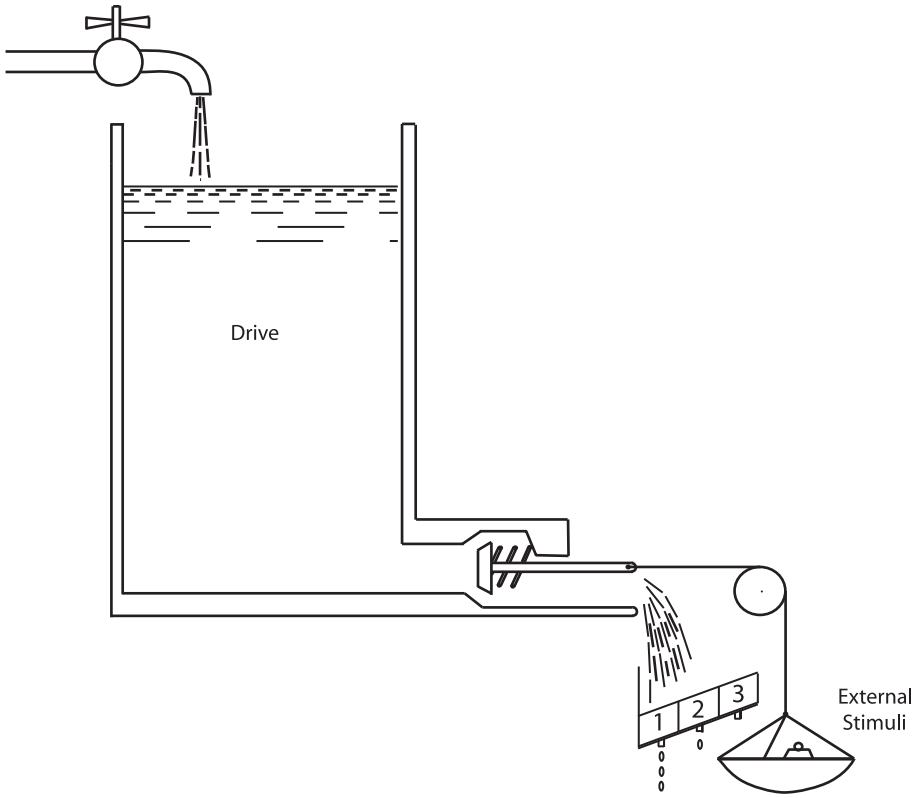

Fig. 5. Hydraulic model of motivational drive. A causal factor such as hormone accumulation or deprivation time pours drive energy into the reservoir. As drive accumulates, pressure increases on the output valve. The valve opens when the internal pressure and/or external stimulus weight becomes sufficient. Then, drive flows out into the behaviors (the numbered pan at the bottom). Behavior 1 is triggered first and most easily. If the drive flow is great enough, Behaviors 2 and 3 will be triggered too. Modified from Lorenz and Leyhausen [94].

called this spontaneous burst a *vacuum reaction*. One example is the nest-building drive observed in birds (canaries) kept in captivity during their normal nest-building season [69]. These birds lived in an aviary without access to twigs, straw, grasses, or other natural nesting material. Unable to find the proper stimuli to build nests, the birds became increasingly restless as the nesting season progressed. Eventually, some birds began to use their own feathers, still attached to their bodies in bizarre "vacuum reaction" attempts to build a nest. A bird would fly from its perch to other spots in the aviary, moving from place to place and looking about in a fruitless search for nest materials. Finally, the searching bird turned its head toward its own tail feathers, found a feather, and seized it in its beak. Still with its own tail in its beak, the bird flew awkwardly back to its perch. There, the bird began to try to build a nest out of its tail, a dance of head weaving movements while holding the feather. Normally, those movements would weave whatever material was in its beak into a nest under construction. Only after building its nonexistent nest would the bird release its tail feather, which flipped back into place, and then possibly fly off to repeat the whole pathetic sequence. The Lorenz hydraulic model explains vacuum reactions, such as this nest building, as an irrepressible expression of nest building drive that had been bottled-up past the capacity of its reservoir. Using similar logic, Lorenz also explained displacement behaviors, such as when a bird, alternating between attacking a rival in a territorial dispute and fleeing from the rival's attacks, suddenly broke off and began to engage intensely in an irrelevant third behavior such as eating or grooming [167]. In these cases, Lorenz postulated that excessive attack/flee drives had blocked each other, and together spilled over to activate the irrelevant eating or grooming behavior, displacing the original dominant behaviors.

There are some problems with this hydraulic model. Most motivated behaviors do not actually erupt in the inexorable way as Lorenz's model suggests, although, perhaps, some do. In addition, behavioral expressions of motivation often do not reduce the motivation but, instead, actually primes or enhances its subsequent intensity. You may have experienced this 'cocktail peanut' phenomenon: After taking one tidbit without desire and merely to be polite, you suddenly find you want to eat a few more. Priming is well known in animal studies of drug and brain stimulation reward. For these rewards, responding may often be low in the beginning of a session, unless a free reward is given or until the first reward is finally earned. Then, after its free or first reward, the animal sets hard to

work for more. Priming is explained better by incentive motivation concepts discussed below.

Lorenz's hydraulic model was never strongly adopted by behavioral neuroscience because it offered few details on neural mechanisms. Perhaps, drive displacement also sounded disagreeably similar with Freudian displacement to some neuroscientists (although Lorenz's displacement concepts are much simpler and more falsifiable by experiment). Still, Lorenz's drive concepts can sometimes be useful, at least for comparing against behavioral observations, regarding temporal build up in motivation, the effects of preventing behavioral expression, and the interaction between internal motivational factors and external stimuli in controlling motivated behavior.

3.2.1. Drive reduction and reward

Before leaving drive concepts altogether, a final feature to consider is the relation of drive reduction to reward or reinforcement. Many drive theories of motivation between 1930 and 1970 posited that drive reduction is the chief mechanism of reward [75,100,101,153]. If motivation is due to drive, then, the reduction of deficit signals should satisfy this drive and essentially could be the goal of the entire motivation. Thus, food could be a reward because it reduces hunger drive, water is a reward when thirsty because it reduces thirst drive, and so on. The drive reduction concept of reward is so intuitive that it was thought to be self-evident for decades. The power of this idea is so great that some behavioral neuroscientists today still talk and write as though they believe it. All the more pity, perhaps, that the idea turns out not to be true. Drive reduction is not really a chief mechanism of reward.

Evidence against drive reduction came from several sources in the 1960s. Even for food and hunger, reducing physiological drive via intravenous feeding turns out to be relatively ineffective at stopping eating. Although in favor of drive reduction were several reports between 1950 and 1975 that intravenous or intragastric nutrient feeding was reinforcing by itself, and could suppress normal eating behavior, the reinforcing effects were sometimes hard to replicate in other studies and suppression of eating was usually incomplete [47,100,106]. And other evidence indicated that something else was really more important for controlling motivated behavior. A vivid early counterexample against pure drive reduction is the anecdotal case of a man named Tom whose esophagus was permanently damaged in childhood when he accidentally drank scalding soup without knowing it was too hot [190]. The burn sealed his esophagus and, thereafter, blocked the passage of food to the stomach. Hence, a surgical opening or gastrostomy fistula was implanted in his stomach, and he was sustained afterwards by placing food and drink directly through the fistula into his stomach. There was no longer any apparent purpose in putting food in his mouth first because food in the mouth could not descend though the closed esophagus. Yet, Tom insisted on munching food at meals, when he would chew and then spit out the food before placing it in his stomach. Why? Because "introducing (food) directly into his stomach failed to satisfy his appetite" (Ref. [190, p.8]).

The idea that satisfying appetite is not merely a matter of physiological drive reduction was supported further by results of experimental studies with animals. For example, dogs intravenously fed the full amount of nutrients they would ordinarily eat but still consumed their normal meals by mouth when given a chance, in addition to receiving their intravenous calories. They quickly become overweight, but still continued to eat [173]. Homeostatic drive was not the reason they ate, and their motivation to eat was not satisfied by physiological drive reduction. Similarly, a classic experiment by Miller and Kessen [100] compared rats learning to go down an alley either for pure drive reduction by intragastric feeding of milk or, instead, for the taste and feel incentive stimuli of being able to drink the milk normally. Rats quickly learned to run to their tasty drink, but merely walked for the drive reduction of intragastric intubation. Similarly, when rats learn signals either for an oral sugar water reward or for intragastric delivery of the same reward, they later show motivation to approach only the signal that means oral delivery of the sweet taste [103]. All these instances suggest that motivation is more compatible with incentive concepts of taste reward discussed below than with earlier drive reduction concepts.

The most important evidence against drive reduction concepts came in the 1960s from studies of brain stimulation reward and related studies of motivated behavior elicited by "free" brain stimulation. A single electrode in the lateral hypothalamus could both elicit motivated behavior (if just turned on freely) and have reward or punishment effects (if given contingent on the animal's response). Many behavioral neuroscientists of the time believed in drive reduction theory. So, at first, they expected to find that the brain sites where stimulation would reduce eating (presumably by reducing drive) would also be the sites where stimulation was rewarding (again, presumably by reducing drive). Conversely, they believed the opposite would be true too. They expected to find that punishing electrodes would sometimes activate drives like hunger.

The best descriptions of these beliefs come from the experimenters' own words. As James Olds (the codiscoverer of brain stimulation reward; Ref. [108, p.89]) put it, he was originally guided by the drive reduction hypothesis that an "electrical simulation which caused the animal to respond as if it were very hungry might have been a drive-inducing stimulus and might therefore have been expected to have aversive properties". In other words, if the drive reduction theory were true, an "eating electrode" should also have been a "punishment electrode".

Conversely, a "satiety electrode" that stopped eating should have been a "reward electrode". As Miller (Ref. [98, pp.54–55]), a major investigator of brain stimulation effects, recounted later in describing how drive reduction

concepts guided his research, "If I could find an area of the brain where electrical stimulation had the other properties of normal hunger, would sudden termination of that stimulation function as a reward? If I could find such an area, perhaps recording from it would provide a way of measuring hunger which would allow me to see the effects of a small nibble of food that is large enough to serve as a reward but not large enough to produce complete satiation. Would such a nibble produce a prompt, appreciable reduction in hunger, as demanded by the drive-reduction hypothesis?". Thus, if the drive reduction theory were true, you might actually be able to watch hunger drive shrink by recording the shrinking activity of the drive neuron each time a nibble of food reduced that drive and caused reward.

Disappointingly for a generation of behavioral neuroscientists, the theory was not true, and nearly all of the predictions based on it turned out to be wrong. In many cases, the opposite results were found instead. The brain sites where the stimulation caused eating behavior were almost always the same sites where stimulation was rewarding [175,177]. Eating electrodes were not punishing electrodes. Instead, the eating electrodes were reward electrodes. Stimulation-induced reward and stimulation-induced hunger drive appeared identical or, at least, had identical causes in the activation of the same electrode. This meant that the reward could not be due to drive reduction. The reward electrode increased the motivation to eat, it did not reduce that drive. Instead reward must be understood as a motivational phenomenon of its own, involving its own active brain mechanisms. Today, the active brain mechanisms of reward are the topic of much research in affective neuroscience [5,10,31,39,44,62,84–86,107,109,129,133,135,142].

3.2.2. Early steps toward hedonic reward concepts

Central to most incentive motivation theories is the concept of hedonic reward. For behavioral neuroscience to study pleasure, hedonic reward must be mapped onto brain systems. In an classic 1960 paper titled *The Pleasures of Sensation*, Pfaffmann (Ref. [117, p.254]) issued an early call for better understanding of neural bases of sensory pleasure, focusing on "the relation between hedonic processes and afferent nerve discharges, preference behavior, and taste reinforcement". Pfaffmann [117] drew on the results of 1940s–1950s biopsychology experiments by Young [195] and Sheffield [146] to show that sensory pleasure was an important cause of behavior.

The experiments of Young [195] had cleverly demonstrated that tasty hedonic rewards caused sudden and real changes in rats' behavior, and that hedonic reward could overturn previously well-established habits. Young had kept hedonic concepts alive in experimental psychology during behaviorist decades when associative learning and drive were the only concepts used by others. He also developed experimental methods for separating hedonic reward from learned habits in laboratory rats. Similarly, experiments by Sheffield showed that pure sensory rewards were behaviorally reinforcing even when they did not reduce drives. For example, rats avidly drank saccharin solutions that had no nutrients, and male rats worked to gain brief access to a female for copulatory intromission, even if no time was allowed for the subsequent ejaculation that presumably might have reduced their sex drive [146]. As an aside, it is interesting to note that the interpretation of Sheffield [146] of his own experiments did not use hedonic concepts, although the results are now regarded as classic experimental examples of hedonic incentives. Instead, Sheffield [146] invented a rather bizarre drive induction theory to explain them, positing the taste or sex sensations to induce frustration or a related excitement that spurred behavior. Sheffield's drive induction theory directly contradicted most conventional drive reduction theories of the time, such as that of Mowrer [101]. Mowrer [101] posited the opposite of Sheffield, that the taste of saccharin, feel of copulation, and other reward-associated stimuli acted essentially as conditioned drive reducers (sensory signals that reinforced behavior by reducing drive, via their learned associations with the physiological drive reduction of food digestion, sexual orgasm, etc.). Both were wrong. Sheffield [146] eventually abandoned his frustration version of drive-induction theory, and the theory of Mowrer [101] soon evaporated with other drive reduction theories of the era. However, perhaps, the main point to be taken from this battle over sex and saccharin between drive induction versus drive reduction is that until 1960s, almost no one could conceive of any reinforcement explanation couched in concepts other than drive. Drive was always the explanation, even if some explainers posited drive to go up while others posited drive to go down for the same event (e.g., saccharin reinforcement).

The article on pleasures of Pfaffmann [117] was influential in part because he made a clean break with drives and interpreted all of the Young [195] and Sheffield [146] experiments as behavioral examples of sensory hedonic reward. Pfaffmann [117] connected those hedonic examples to behavioral neuroscience by pointing to the electrophysiological firing patterns of taste sensory pathways to the brain. Pfaffmann [117] argued that the neural encoding of sweet taste, sexual sensation, and other hedonically laden sensations must be rewarding and motivating all by itself, without any need of drive reduction.

Decades later, Eliot Stellar [156] further championed the need for behavioral neuroscience to study affective reactions in an article entitled *Brain Mechanisms in Hedonic Processes*. Stellar (Ref. [156, p.378]) urged that "it is time that we again address questions of sensation, feeling, and affect in humans, and animals as well, and ask about the biological basis of hedonic experience". Specifically, regarding the use of basic animal studies to reveal insights into brain mechanism of affective reaction, Stellar [156] asserted that "what we identify as hedonic experience in man emerges over phylogeny in a wide range of behavioral precursors" and "Some of the precursors of hedonic experience may occur in infrahumans, as judged primarily by approach and withdrawal behavior, affective expression, and the potent

effects of reinforcement. These can only be inferred, but should not be ignored'' (p. 404). Calls such as Pfaffmann's and Stellar's helped to direct behavioral neuroscience research toward hedonic reward, an important step for the development of incentive motivation concepts.

4. Incentive motivation concepts

Incentive motivation concepts rose as drive concepts fell beginning in the 1960s. Several new realizations about brain and motivation, including some already mentioned, led many psychologists and behavioral neuroscientists to reject simple drive and drive-reduction theories. Specific alternative theories were developed in the form of incentive motivation theories [11,19,21,109,117,156,169,177,195].

To give you a sense of how these concepts developed in 1970s and 1980s, it is instructive to briefly consider the origins of what I call the Bolles–Bindra–Toates theory of incentive motivation [19–21,169,170]. This has grown to be a useful motivational concept, and each of those three biopsychologists made major incremental contributions to its development.

First, Bolles (the same ''settling point'' Bolles) reviewed many experimental failures of drive motivations and drive reduction concepts of reward, such as those discussed above. He proposed instead that individuals were motivated by incentive expectancies, not by drives or drive reduction [21]. Incentive expectancies were essentially learned expectations of a hedonic reward, essentially indistinguishable from cognitive predictions. Bolles called these expectations S–S* associations. He meant that a predictive neutral stimulus (S), such as a light or a sound, became associated by repeated pairing with a hedonic reward that followed (S*), such as a tasty food. The S caused an expectancy of the S*. The S was what Pavlovian psychologists would call a conditioned stimulus (CS or CS+), and the S* they would call an unconditioned stimulus (UCS). Bolles' point in calling them S and S* was to emphasize that the chief motivational difference between these stimuli was that the S* already carried a motivational value even before learning while the S did not. He also wanted to stress that learning resulted in a predictive expectancy of reward. But it was not clear why an S–S* expectancy would cause motivation. Why not just passively wait and enjoy the predicted reward that you expect?

The psychologist Dalbir Bindra [19,20] therefore rejected the idea that expectation per se was the most important factor for basic incentive motivation for rewards (although expectations might well be important to cognitive strategies to obtain the reward), but otherwise adopted the incentive prediction framework of Bolles. Bindra suggested that a CS for a reward actually evokes the same incentive motivational state normally caused by the reward itself, as a consequence of classical conditioning. The learned association does not simply cause expectation of the reward. It also causes the individual to perceive the CS as a hedonic reward, and lets the CS elicit incentive motivation just as would the original hedonic reward (the unconditioned reward, or UCS; or, in Bolles' terminology, the S*). The CS takes on specific motivational properties that normally belong to the S* itself. These motivational properties are specifically incentive properties (at least for reward S*, for painful S* motivation would be based on fear or punishment properties). The establishing S* is typically a pleasant taste, nasty foot shock, or other sensory stimulus with hedonic valence. But sometimes, the S* also involves more subtle physiological processes too. For example, Booth, Sclafani, and others showed that pairing a new CS flavor with physiological calories delivered in the flavored food or intravenously causes an increase in the taste's incentive value—an internal S* that causes people and rats to later prefer its S flavor over another flavor [23,104,144,172]. In such cases, conditioned incentives attract approach, elicit goal-directed behavior, and, sometimes, even consumption. Conditioned incentives may also carry hedonic or affective properties: The CS often becomes a '*liked*' reward in its own right.

Critics of Bindra noted that if conditioned stimuli simply became permanent incentives because of learning, then one should always respond to them as incentives, whether hungry or thirsty [57]. Yet, clearly, physiological drive state is important to motivation, even if drive is not equivalent to motivation. You do not seek out food when you are thirsty. Physiological deficits such as hunger or thirst depletion signals do modulate motivation for rewards such as food.

To incorporate physiological drive/deficit states into incentive motivation, Frederick Toates [169] modified the Bolles–Bindra concepts. He suggested that physiological depletion states could enhance the incentive value of their goal stimuli. This was essentially a multiplicative interaction between physiological deficit and external stimulus, which determined the stimulus' incentive value. Physiological deficit signals did not have to drive motivated behavior directly. They could magnify the hedonic impact and incentive value of the actual reward (S*). Physiological drive signals could also magnify the hedonic/incentive value of predictive stimuli for the reward (CSs). This is a three-way interaction between physiological deficit, the CS/S stimulus, and its learned association with the UCS/S*. Tasty food, refreshing drinks, sexual partners, addictive drugs, and social and other rewards were all hedonic incentives that might be modulated by internal states. The sights, smells, and other predictive CSs for these rewards could also be modulated in incentive value. Finally, Toates [169] posited that both cognitive expectancy (as suggested by Bolles and familiar to cognitive psychologists) and these more basic incentive motivation processes might occur simultaneously within the same individual's brain, and both be recruited in different ways to control goal-directed behavior (an idea developed further by Dickinson and Balleine and their colleagues in the 1990s, discussed below).

4.1. Alliesthesia: changing hedonic value

The key to understanding how physiological drive states modulated basic incentive motivation for Toates [169] lies in a concept called "alliesthesia", a word coined by Michel Cabanac [28], which means essentially a change in sensation (though to be fully accurate, the phenomenon is a change only in the pleasure of the sensation). Toates began with an idea that the pleasure of hedonic incentives could be modulated by relevant physiological drive states. Cabanac [27,28] showed, for example, that people gave higher subjective ratings of pleasure to the taste of sugar when they were hungry than when they had recently eaten. The pleasure of the sensation changed with their physiological state, although the sensory quality of the sweetness was the same. In human adults, alliesthesia is evident in our subjective ratings of stimulus pleasantness. In human infants and animals, alliesthesia also has been detected using measures of affective facial expressions to taste pleasantness and measures of brain neurochemical responses [1,29,37]. Thus, alliesthesia is a very basic biopsychological phenomenon. Cabanac [28] argued alliesthesia applies to most hedonic sensations. A hot bath feels delightful if we are cold, but may seem positively unpleasant on a hot day—when a cold plunge into a cool pool seems much more pleasant. Similarly, the saltiness of seawater is unpleasantly intense to most individuals, but even the saltiest tastes become pleasant in "salt appetite" that follows dramatic loss in body sodium [6,140].

What if conditioned stimuli, which predict rewards, also have their incentive/hedonic impact modulated via alliesthesia? Toates [169] suggested that they do. He argued that physiological drive states play a role in motivation primarily by modulating the incentive/hedonic value of their relevant food and drink rewards, and of their predictive cues or conditioned stimuli. In this way, conditioned motivation could follow Bindra-type incentive rules, and yet, be modulated flexibly by internal depletion states, just as natural taste alliesthesia is modulated. Tests of this hypothesis have tended to support Bindra–Toates. For example, during a physiological sodium depletion state, a sour/bitter taste that was previously associated with saltiness becomes greatly enhanced in its hedonic palatability—just as the pleasure of saltiness itself is increased by the sodium depletion [14].

4.2. Splitting incentives: 'liking' versus 'wanting'

The Bindra–Toates incentive concept suggests that learned Pavlovian incentive stimuli become both '*liked*' and '*wanted*' as a consequence of reward learning. Conditioned incentive value is equivalent to conditioned incentive/hedonic value according to the original Bindra–Toates model [169]. Individuals can literally move along a gradient of conditioned hedonic stimuli to find their goal, according to this concept, following stimuli that are more and more '*wanted*' and '*liked*'. '*Liking*' and '*wanting*' are almost synonyms for the same incentive value in the original model. But my colleagues and I have suggested that a split may sometimes occur between the incentive processes of '*liking*' and '*wanting*' because these two components of reward have different brain mechanisms. The result is what we call an incentive salience model or a modified Bindra–Toates model of incentive motivation [11,13,127].

Incentive salience ('*wanting*') follows the Bindra–Toates rules for incentive conditioning but identifies separable brain substrates for '*liking*' a reward versus '*wanting*' the same reward. The incentive salience model was proposed around 1990 as a way to reconcile why brain dopamine sometimes seemed to mediate sensory pleasure, when it actually does not [15,16]. The incentive salience concept drew on Bindra–Toates concepts of psychological incentive motivation [19–21,169,170], combined with aspects of earlier hedonia, appetitive behavior, and reward expectancy models of brain dopamine function [50,59,111,189], to clarify brain mechanisms of reward '*wanting*' and '*liking*'.

'*Liking*' is essentially hedonic impact—the brain reaction underlying sensory pleasure-triggered by immediate receipt of reward such as a sweet taste (unconditioned '*liking*'). '*Liking*' can also sometimes be triggered by a CS, as Bindra had suggested (conditioned '*liking*'; [14,25,42,72,104]). '*Wanting*', or incentive salience, is the motivational incentive value of the same reward [13,126]. But incentive '*wanting*' is not a sensory pleasure. '*Wanting*' is purely the incentive motivational value of a stimulus, not its hedonic impact.

Why did brains evolve separate '*wanting*' and '*liking*' mechanisms for the same reward? Originally, '*wanting*' might have evolved separately as an elementary form of goal directedness to pursue particular innate incentives even in advance of experience of their hedonic effects. Later incentive salience became harnessed by evolution to serve learned '*wanting*' for predictors of '*liking*', following Bindra–Toates incentive motivation rules, and guided by Pavlovian or classical associations [44]. Or, '*wanting*' may have evolved as distinct from '*liking*' to provide a common neural currency of incentive salience shared by all rewards, which could compare and decide competing choices for food, sex, or other rewards that might each involve partly distinct neural '*liking*' circuits. The important point is that '*liking*' and '*wanting*' normally go together, but they can be split apart under certain circumstances, especially by certain brain manipulations.

'*Liking*' without '*wanting*' can be produced, and so can '*wanting*' without '*liking*'. '*Liking*' without '*wanting*' happens after brain manipulations that cause mesolimbic dopamine neurotransmission to be suppressed. For example, disruption of mesolimbic dopamine systems, via neurochemical lesions of the dopamine pathway that projects to nucleus accumbens or by receptor-blocking drugs, dramatically reduces incentive salience or '*wanting*' to eat a tasty reward, but does not reduce affective facial expressions of '*liking*' for the same reward [13,114]. Dopamine suppression leaves individuals nearly without motivation for any pleasant incentive at all: food, sex, drugs, etc. [24,50,60,95,150,160,174]. Yet, '*liking*', or the

hedonic impact of the same incentives, remains intact, at least in many studies where it can be specifically assessed by either facial affective expressions or subjective ratings [13,24,114]. '*Liking*' in animal affective neuroscience studies has usually been measured based on affective facial expressions elicited by the hedonic impact of sweet tastes, which is discussed more below under 'affect and hedonic reactions'. Similarly, in humans, drugs that block dopamine receptors may completely fail to reduce the subjective pleasure ratings that people give to a reward stimulus such as amphetamine [24,179].

Conversely, '*wanting*' without '*liking*' can be produced by several brain manipulations in rats (and perhaps by real life brain sensitization in human drug addicts [126]). For example, electrical stimulation of the lateral hypothalamus in rats, as mentioned before, triggers a number of motivated behaviors such as eating. In normal hunger, increased appetite is accompanied by increased hedonic appreciation of food, as Cabanac [28] showed regarding alliesthesia. But eating caused by electrical stimulation of the lateral hypothalamus is not accompanied by enhanced hedonic reactions to the taste of food. For example, Elliot Valenstein and I found that during lateral hypothalamic stimulation, rats facial expressions to a sweet taste actually became more aversive, if anything, as though the taste became bitter, although the same electrode made them eat [15]. The hypothalamic stimulation did not make them '*want*' to eat by making them '*like*' the taste of food more. Instead, it made them '*want*' to eat more despite making them '*dislike*' the taste. Mutant mice whose brain receptors receive more dopamine than normal because of their genetic mutation also show excessive '*wanting*' of sweet reward, while nonetheless '*liking*' sweetness less than normal mice do [115]. Recent experiments in our laboratory have further traced the neural causation of incentive salience to GABAergic spiny neurons in regions of the nucleus accumbens—the neurons that receive mesolimbic dopamine. The activation of dopamine signals onto those neurons by amphetamine microinjection or GABAergic feedback from the same neurons onto themselves produces '*wanting*', despite '*disliking*' that is similar to hypothalamic stimulation [115,122,194]. All of these brain manipulations make rats '*want*' to eat food that they fail to make the rats '*like*' (and sometimes even make the rats actually '*dislike*').

What is '*wanting*' if it is not '*liking*'? According to the incentive salience concept, '*wanting*' is a mesolimbic-generated process that can tag certain stimulus representations in the brain. When incentive salience is attributed to a reward stimulus representation, it makes that stimulus attractive, attention grabbing, and a target for many Bindra–Toates-style goal-directed strategies [13,126,194]. When attributed to a specific stimulus, incentive salience may make an autoshaped cue light appear food-like to the autoshaped pigeon or rat that perceives it, causing it to try to eat the cue (in autoshaping, animals sometimes direct behavioral pursuit and consummatory responses towards the (Pavlovian CS+cue; [78,171,184]). When attributed to the smell emanating from a bakery, incentive salience can rivet a person's attention and trigger sudden thoughts of lunch.

But '*wanting*' is not '*liking*', and both together are necessary for normal reward. '*Wanting*' without '*liking*' is merely a sham or partial reward, without sensory pleasure in any sense. However, '*wanting*' is still an important component of normal reward, especially when combined with '*liking*'. Reward in the full sense cannot happen without incentive salience, even if hedonic '*liking*' is present. Hedonic '*liking*' by itself is simply a triggered affective state—there is no object of desire or incentive target, and no motivation for reward. It is the process of incentive salience attribution that makes a specific associated stimulus or action the object of desire, and that tags a specific behavior as the rewarded response. '*Liking*' and '*wanting*' are needed together for full reward. Fortunately, both usually happen together in human life.

4.2.1. Addiction and incentive sensitization

For some human addicts, however, drugs such as heroin or cocaine may cause real-life '*wanting*' without '*liking*' because of long-lasting sensitization changes in brain mesolimbic systems. Addicts sometimes take drugs compulsively even when they do not derive much pleasure from them. For example, drugs such as nicotine generally fail to produce great sensory pleasure in most people, but still are infamously addictive.

In early 1990s, Terry Robinson and I proposed the *incentive-sensitization theory of addiction*, which combines neural sensitization and incentive salience concepts [126,127]. The theory does not deny that drug pleasure, withdrawal, or habits are all reasons people sometimes take drugs [77,88,124], but suggests that something else, sensitized '*wanting*', may better explain compulsive long-lasting addiction and relapse. Many addictive drugs cause neural sensitization in the brain mesocorticolimbic systems (e.g., cocaine, heroin, amphetamine, alcohol, nicotine; [77,125,127]). Sensitization means that the brain system can be triggered into abnormally high levels of activation by drugs or related stimuli. Sensitization is nearly the opposite of drug tolerance. Different processes within the same brain systems can simultaneously instantiate both sensitization (e.g., via increase in dopamine release) and tolerance (e.g., via decrease in dopamine receptors) [77,87,125,127,128]. However, tolerance mechanisms usually recover within days to weeks once drugs are given up, whereas neural sensitization can last for years [112]. If the incentive-sensitization theory is true, it helps explain why addicts may sometimes even '*want*' to take drugs that they do not particularly '*like*'. It may also help explain why recovered addicts, who have been drug-free and out of withdrawal for months or years, are still sometimes liable to relapse back into addiction, especially on occasions when they reencounter drug-associated cues such as drug paraphernalia or places and social settings where they used to take drugs [126,127].

The incentive-sensitization theory is an explanation of a human clinical problem that sprang entirely from basic behavioral neuroscience research and concepts. The theory applies to human addicts, but originally was developed as a deductive concept wholly from results of animal laboratory experiments on neural sensitization and on incentive salience functions of brain dopamine systems [126,127]. The concept was never mentioned in scientific articles on human addiction prior to its development in the behavioral neuroscience laboratory. If it turns out to be true, it is an example of how basic behavioral neuroscience research can produce new concepts that are clinically useful. If eventually proved false, it will join the list of outdated concepts (such as drive reduction), whose primary use now is to delineate how brains might have motivated behavior (but do not) from yet-to-be-proposed concepts that more closely approximate the actual truth. In this regard, the future of incentive sensitization may be interesting to watch.

4.2.2. Cognitive goals and ordinary wanting

Before leaving this topic, it is useful to note how the incentive salience meaning of the word '*wanting*' differs from what most people mean by the ordinary sense of the word wanting. A subjective feeling of desire meant by the ordinary word wanting implies something both cognitive (involving an explicit goal) and conscious (involving a subjective feeling; [3,45,56,168]). When you say you want something, you usually have in mind a cognitive expectation or idea of the something-you-want: a declarative representation of your goal. Your representation is based usually on your experience with that thing in the past. Or, if you have never before experienced that thing, then, the representation is based on your imagination of what it would be like to experience. In other words, in these cases, you know or imagine cognitively what it is you want, you expect to like it, and you may even have some idea of how to get it. This is a very cognitive form of wanting, involving declarative memories of the valued goal, explicit predictions for the potential future based on those memories, and cognitive understanding of causal relationships that exist between your potential actions and future attainment of your goal.

By contrast, none of this cognition need be part of incentive salience '*wants*' discussed above. Incentive salience attributions do not need to be conscious and are mediated by relatively simple brain mechanisms [12,17,126]. Incentive salience '*wants*' are triggered by relatively basic stimuli and perceptions (not requiring more elaborate cognitive expectations). Cue-triggered '*wanting*' does not require understanding of causal relations about the hedonic outcome [11,12,44]. Sometimes, as described for addiction, incentive salience may lead to irrational '*wants*' for outcomes that are neither liked nor even expected to be liked [126].

Studies by Dickinson and Balleine [3,44] have led the way in demonstrating the differences between cognitive wanting and incentive salience—'*wanting*'—in behavioral neuroscience experiments with animals, showing these forms of wanting may depend on different brain structures. For example, incentive salience '*wanting*' depends highly on subcortical mesolimbic dopamine neurotransmission, whereas cognitive forms of wanting depend instead on cortical brain regions such as orbitofrontal cortex and insular cortex [4,44]. The implication for behavioral neuroscience concepts of motivation is that there may be multiple kinds of wanting, with different neural substrates [12].

4.3. Affect: hedonic 'liking', 'disliking', fear and other affective reactions

Pleasure, pain, fear, and other affective reactions are becoming of more and more interest to behavioral neuroscientists (and cognitive neuroscientists), resulting in a new field now called affective neuroscience [10,41,62,90,109,110,129,150]. The goal stimuli of virtually all biologically based motivations elicit affective reactions. It would be surprising that evolutionary selection built affective reactions so strongly into brain organization if hedonic processes had no purpose or consequences [26,27]. Hedonic or affective reactions to food, water, sex, and other rewards may play a vital causal role in future motivated behavior. For example, as the discussion of incentive motivation above makes clear, '*liking*' of rewards is a determinant of future incentive salience '*wanting*', even though they have separable neural mechanisms [13]. Hedonic experience and memories of reward are also the chief input to cognitive incentive valuation mechanisms [44].

4.3.1. Subjective affect and objective affect

The subjectivity of affect in everyday experience has led some to reject it from scientific understanding, but that rejection has been mistaken and unnecessary. Rejection is unnecessary even for reductionists/behaviorists who believe that subjective phenomena are not amenable to scientific study because behavioral neuroscientists can study affective reactions in ways that are objective, not necessarily subjective. Fortunately, for behavioral neuroscience's prospects of understanding affect in the brain, affective reactions to hedonic stimuli have objective aspects just as vision and memory do. Indeed, several neuroscientists have been forceful advocates for the objective scientific study of affective reactions in brain and behavior [12,38,89]. For example, Damasio [38] argues that emotional processes, in general, are purely objective, even though the conscious feeling of them is subjective. He writes, ''the term 'feeling' should be reserved for the private, mental experience of emotion''. But ''The term 'emotion' should be used to designate all the responses whose perception we call feeling''. Similarly, LeDoux [89] has argued that negative affective reactions such as fear can be studied by behavioral neuroscience in a purely objective fashion. He writes ''When we use the term 'fear', we are naturally inclined to think of the feeling of being afraid. (But) As important as

subjective feelings like fear are to our lives, it seems likely that these were not the functions that were selected for in the evolution of the fear systems or other emotion systems". "And we can study the fear system in animals, even if we cannot prove that they experience feelings of fear" (p. 131).

Even ordinary people, under some conditions, can have affective reactions that are purely unconscious, and therefore merely objective. For example, Winkielman and colleagues [17,185] found that a subliminally brief view of happy facial expressions may produce no elevation in subjective feeling or mood ratings in thirsty people at the moment it occurs, but can still cause them to consequently consume more of a fruit drink presented moments later and to give higher subjective value ratings to the drink's pleasantness, attractiveness, and monetary value. Conversely, exposure to subliminally brief angry facial expression produces no reduction in subjective mood or emotion ratings, yet, can cause these people to consume less of the drink later and give it lower ratings of pleasantness, attractiveness, and price. Because the people did not feel more positive after viewing subliminal happy expressions, nor more negative after viewing subliminal angry expressions, no subjective feelings could have changed their subsequent affective response to the drink. Instead, subliminal happy expressions appeared to act by causing an unconscious affective reaction, inaccessible to intervening conscious introspection at the moment it arises [17,185]. Such unconscious affective reactions are purely objective by definition because they do not influence subjective ratings when people monitor and report their feelings. Similarly, human drug addicts, in some circumstances, will work for low doses of stimulants or morphine, doses so low they produce no subjective effects and even no autonomic responses, without being aware that they are doing so [51]. In both of these cases, the human affective reaction lacks any subjective feeling at all detectable, even by the person who has it. The affective reaction then exists solely as an objective behavioral or physiological reaction, with no subjective component. Such demonstrations have led us to suggest the term '*liking*' for objective core affective reactions that are observed in behavior or physiology, whether or not objective '*liking*' is accompanied by conscious feelings of subjective liking [12,17,126,127]. Unconscious '*liking*' is different from conscious liking, just as incentive salience '*wanting*' is different from ordinary conscious wanting. The important point here is that if humans can have affective reactions that are purely objective, then, behavioral neuroscientists need not be deterred by worries about subjectivity from studies of objective affective reactions in either animals or humans.

4.3.2. More on specific pleasures: a limbic circuit for taste 'liking'

Affective neuroscience aims to understand how '*liking*' or sensory pleasure, fear, and other affective reactions are generated in the brain. To succeed at this task, behavioral neuroscientists must study particular affective reactions—pleasure of this sweet taste, fear of that shock, etc. We need real affective reactions for scientific study to find neural bases, and real reactions are always to some particular stimulus.

To probe the brain circuit of a specific pleasure, such as sweetness, we need an objective measure able to specifically detect affective '*liking*' reactions (Figs. 5 and 6). After all, unless we can identify when '*liking*' occurs, we will never be able to identify the brain mechanism that generates it. Equally important, we must be able to identify brain events that cause '*liking*' to change up or down. That is because we want to be able to distinguish affective '*liking*' and its brain causes from the many other psychological processes that also might be activated simultaneously with '*liking*' when a reward stimulus is encountered. '*Wanting*', associative learning and reward prediction, cognitive representations, actions, and behavioral response generation all might be triggered by an affective stimulus at the same time. How can one know which brain activation mediates a '*liking*' process, rather than those others? Only by showing that manipulation of particular brain systems causes changes in '*liking*'. This is just where the need arises for specific '*liking*' reactions to reveal when brain activation causes specific change in '*liking*'.

Examples of objective hedonic '*liking*' reactions include affective facial expressions elicited by the hedonic impact of tastes in newborn human infants (Figs. 6 and 7; [154,155]). Sweet tastes elicit positive facial '*liking*' expressions (tongue protrusions, etc.), whereas bitter tastes instead elicit facial '*disliking*' expressions (gapes, etc.). Many animals also display similar '*liking*/*disliking*' reactions elicited by sweet/bitter tastes [65,118,155]. These affective expressions seem to have developed from the same evolutionary source in humans, orangutans, chimpanzees, monkeys, and even rats and mice [9,155]. For example, the evolutionary relationships of these species can be traced in a taxonomy based on the details of their affective facial expressions (Fig. 6B). The behavior-based taxonomy looks essentially the same as traditional phylogenetic taxonomies that are based on details of skull structure or molecular gene sequences [9,155]. Another indicator that humans and animals share the same taste '*liking*' expressions is that they share identical microstructural features produced by neural-generating circuits. For example, all observe the same brain-generated timing equation for determining how long each rhythmic tongue protrusion lasts [9,155]:

$$\text{Duration in msec} = 0.26(\text{species}' \text{ adult weight in kg})^{0.32}$$

That means that a human or gorilla tongue protrusion or gape may appear languidly slow, whereas a rat or mouse reaction seems blinkingly fast, yet, all have identical timing deep structure scaled to their evolved size (and it turns out

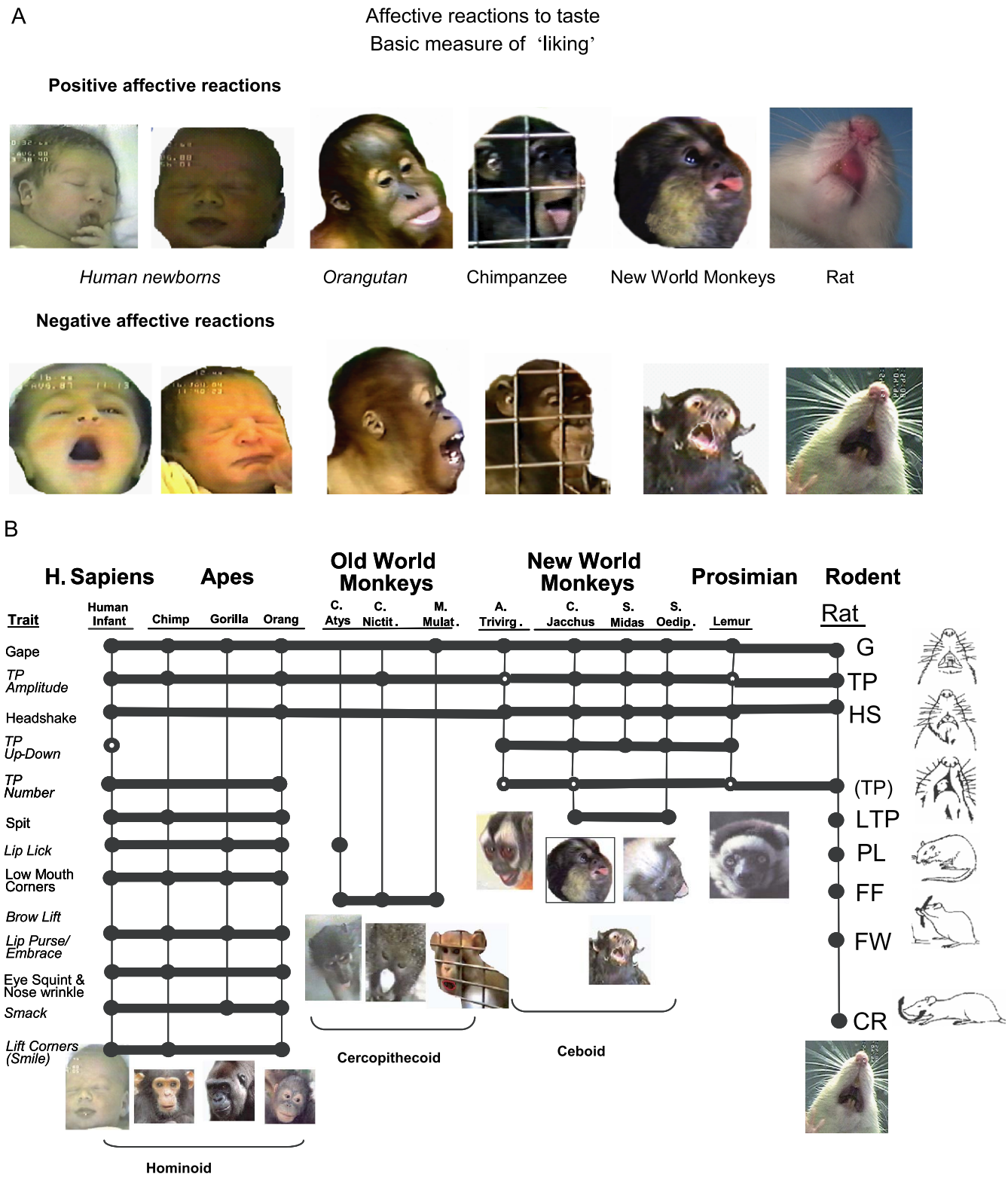

Fig. 6. (A) Examples of affective facial expressions to taste by human infants, apes, monkeys, and rats. Modified from Steiner et al. [155] and Berridge [9]. (B) Evolutionary relations revealed in facial expression: Taxonomic tree based on shared details of affective facial expressions to taste. Behavioral expression taxonomy mirrors phylogenetic relationships among humans, 11 other primate species, and rats. Species that are closely related share the most components (indicated by connecting horizontal lines). All species share some universal components, such as gapes to bitter. Modified from Steiner et al. [155] and Berridge [9].

that this timing is programmed by their brains, not a passive result of physical constraints, e.g., even small infants have timing already scaled to their future sizes; [9,155]). The implication is that modern brain mechanisms of affective taste '*liking*' are likely to be highly similar in humans and other animals today.

In a 1970s affective neuroscience study, Steiner [154] showed that the elemental neural circuit for generating '*liking*' facial expressions is contained in the human brainstem. The crucial demonstration was that basic positive or negative facial expressions are still found in anencephalic infants. Anencephalic infants have a midbrain and hindbrain, but no cortex, amygdala, or classic limbic system, due to a congenital defect that prevents prenatal development of their forebrain. Yet, sweet tastes elicit normal positive affective facial expressions from them, and bitter or sour tastes elicit negative expressions. At about the same time, Grill and Norgren [66] showed that a decerebrate rat's brainstem also remained able to generated normal taste reactivity expressions after it was surgically transected.

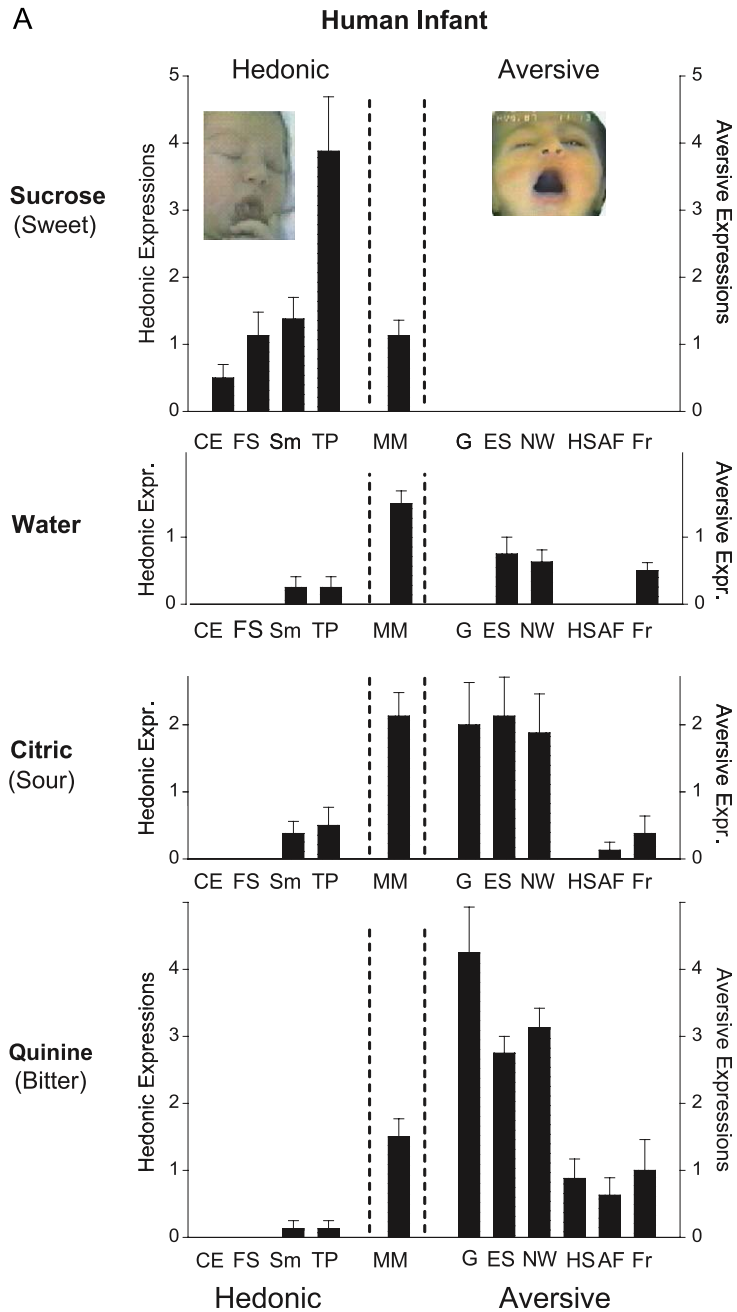

Fig. 7. Measuring hedonic impact in human infants and rats. Intensity of '*liking*' and '*disliking*' for tastes is revealed in affective facial expressions of human infants (A) and in adult rats (B). Human infant data are from Steiner et al. [155] and rat data from Berridge [9].

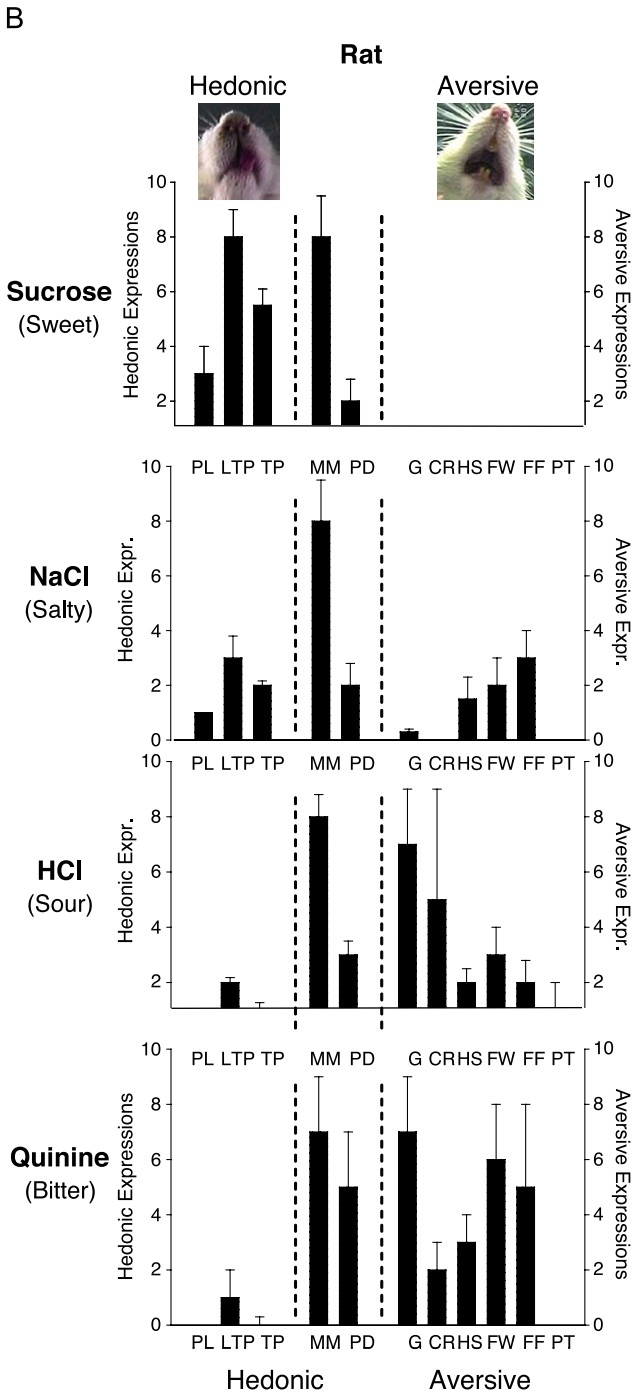

Fig. 7 (*continued*).

Demonstration that brainstem anencephalics or decerebrates show basic affective expressions to sweet or bitter tastes means that the brainstem participates importantly in these reactions, but does not mean that '*liking*' lives only in the brainstem. Instead, brain circuits of '*liking*' are organized hierarchically, involving both the forebrain and the brainstem. Normally, '*liking*' is determined by limbic structures in the forebrain too, arranged in a distributed neural network. Forebrain mechanisms can overrule the brainstem to control affective expressions to tastes [10]. One hedonic forebrain mechanism able to cause '*liking*' is opioid neurotransmission onto GABAergic spiny neurons in the nucleus accumbens [10,33,83,91,113]. For example, microinjection of drugs that stimulate opioid receptors in the shell of the nucleus accumbens cause increased facial '*liking*' reactions to sweetness [113]. Similarly, GABA receptor feedback

onto the same spiny neurons in the nucleus accumbens causes either increased or decreased sensory '*liking*', depending on the precise location of the microinjection in the shell of nucleus accumbens [92,122]. Other hedonic brain substrates causing '*liking*' include nucleus accumbens outputs to the lateral hypothalamus and ventral pallidum (where lesions cause sweet tastes to become '*disliked*', so rats react to them as though they were unpleasantly bitter) and connected structures elsewhere in the brain [10,36,82,84,109,134,158,196].

Affective neuroscience studies of hedonic facial expressions to taste have also identified a number of false hedonic brain substrates that do *not* mediate '*liking*' for sensory pleasures—even though they were once thought to do so. For example, as mentioned above, false hedonic substrates include mesolimbic dopamine projections to the nucleus accumbens. Dopamine suppression or lesion does not suppress taste '*liking*' facial expressions [13,81,114]. Instead, the hedonic impact of sweetness remains robust even in a nearly dopamine-free forebrain (also, still robust is the ability to learn some new reward values for a sweet taste, which indicates that '*liking*' expressions remain faithful readouts of forebrain '*liking*' systems after dopamine loss) [13]. Supporting evidence also comes from PET neuroimaging studies of humans, which report that dopamine release triggered when people encounter a food or drug reward may better correlate to their subjective ratings of wanting the reward than to their pleasure ratings of liking the same reward [93,178]. Thus, popular concepts of dopamine as a pleasure neurotransmitter now appear to be less tenable than they once were (though dopamine seems important to '*wanting*' rewards, even if not to '*liking*' rewards; [12,13,44,96,133]). Separating true '*liking*' substrates from false ones is a useful step in identifying the real affective neural circuits for hedonic processes in the brain [10,49,80,84].

5. Brain concepts of drive and motivation

Finally, we consider the concepts of functional brain wiring. In behavioral neuroscience, drive and motivation have often been conceived as arising from neural activation of a dedicated brain center or dedicated brain circuit, made up of dedicated brain neurons. A neural substrate is *dedicated* to its motivation if the neuronal activation of that substrate always produces that particular motivation. Early brain models of motivation typically viewed motivation to be mediated by a particular brain region or center localized in one place. A classic example was the hypothalamic center model of hunger drive versus satiety of Stellar ([157]; Fig. 8). Stellar [157] proposed that the circuit for hunger drive was contained in the lateral hypothalamus, and its stimulating effect on eating behavior was opposed by a satiety center contained in the ventromedial hypothalamus. Each hypothalamic region received internal signals about energy depletion and stores and also interacted with outside stimuli, behavior, and feedback from the world. The hypothalamic outputs led to hunger and eating and to homeostatic regulation.

5.1. Drive-dedicated neurons

A hunger brain center should contain specialized hunger neurons. If so, dedicated hunger neurons, when activated, could cause motivation to eat. By the logic of dedicated neurons, hunger neurons would receive homeostatic deficit signals and other hunger-relevant cues and be necessary and sufficient causes of psychological hunger and eating behavior. Similarly, a thirst drive needs a thirst center or circuit containing dedicated thirst neurons. By this concept, there could be dedicated neurons for sex, others for predation, aggression, parental attachment, etc. Every specific motivation could have its own dedicated neurons.

5.1.1. Evidence against drive centers and dedicated neurons

Problems became apparent for the concepts of motivational brain centers and dedicated drive neurons by the late 1960s. One problem was that no center actually seemed to contain an entire motivation, and brain lesions rarely eliminated a motivation completely. For example, after lateral hypothalamic lesions purportedly destroyed hunger centers, at least, some aspects of eating behavior and hunger gradually recovered. Rats first begin to eat palatable foods and, later, ordinary foods too, and then even respond to at least a few physiological hunger signals [165,186]. The reemergence of motivated behavior during recovery after a brain lesion meant that motivational control must be partly distributed elsewhere in the brain, not entirely contained in the destroyed center [18,63,116,149,181]. Even a mere decerebrated brainstem contains circuits able to mediate some aspects of hunger, such as the ability to change the amount swallowed when food is put in the mouth, in response to hunger hypoglycemia signals or to satiety hormones such as leptin or melanocortin [64]. Hence, a motivation must be mediated by a distributed brain circuit, rather than localized in a center.

Even more fundamental, problems appeared also for the idea of a dedicated drive neuron itself, no matter where in the brain it might be imagined to be. The most dramatic evidence against dedicated drive neurons came from studies of motivation elicited by electrical brain stimulation, which discovered that, sometimes, the activation of one single brain site could elicit many different motivated behaviors, depending on environmental situations, individual predisposition, and experience [70,73,176,177]. For example, if one stimulated the lateral hypothalamus of different rats, many rats might show eating behavior. But a few rats might show drinking behavior, a few show sexual behavior, or others show predatory aggressive behavior, depending on the availability of stimuli and on the disposition of the individual rat being stimulated.

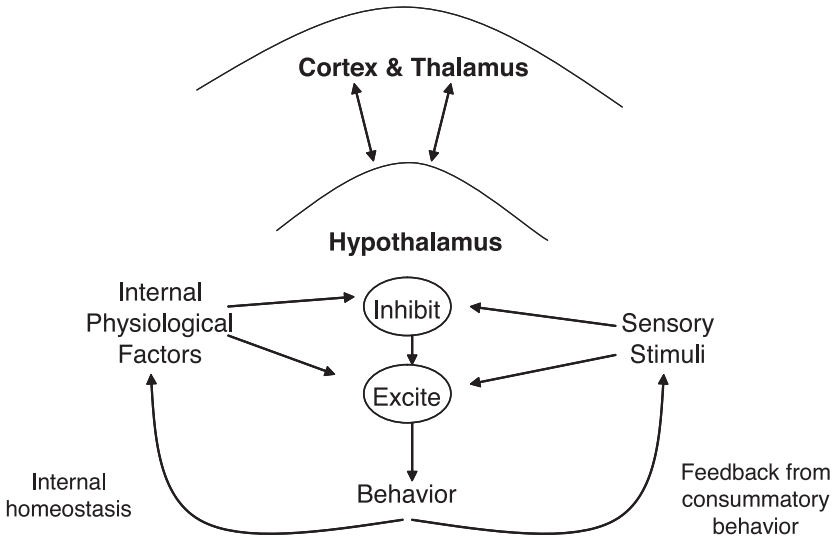

Fig. 8. Hypothalamic center model of hunger motivation of Stellar [157]. Modified from Ref. [157].

Individual disposition was particularly important and particularly problematic for the dedicated drive neuron hypothesis. This was demonstrated around 1970 by an important series of brain stimulation studies by Elliot Valenstein et al. [177]. For example, once a particular rat had showed one motivated behavior after its lateral hypothalamus was stimulated (e.g., drinking from a water spout), you could predict that other electrodes implanted in the lateral hypothalamus would also be likely to elicit drinking in that same rat. Conversely, if one electrode caused a rat to eat, other electrodes in the same rat also mostly caused eating. Every rat seemed to have its own "*prepotent* behavior", the single type of motivated behavior that was elicited by nearly all its LH electrodes [177]. A rat's particular prepotent behavior tended to remain stable even if one physically moved the stimulating electrode across the hypothalamus [188] and even if one destroyed the hypothalamic neurons closest to an electrode (by creating an electrolytic lesion) and then stimulated a second time with enough intensity to activate neurons remaining farther away [2]. If anyone had believed that thirst or hunger was caused by dedicated neurons in the hypothalamus, it was as though one rat had a hypothalamus full of thirst neurons, but another rat had only hunger neurons in its hypothalamus. That is clearly no way for evolution to build rats, and so, evolution likely never did. Instead, this discovery suggested that, perhaps, the brain did not assign hypothalamic neurons to be dedicated to particular motivations after all.

Further problems for the dedicated drive neuron concept arose from Valenstein's demonstrations that stimulation-evoked motivated behavior was flexible or plastic. *Plasticity* meant that the motivated behavior evoked by a hypothalamic electrode could be changed gradually by manipulating an individual's experiences under the brain stimulation [177]. For example, at first, a given lateral hypothalamic electrode might elicit only eating behavior from a rat. Naturally, one might infer from that first observation that the electrode must stimulate hunger neurons. However, the stimulation-evoked behavior changed if one restricted the rats' opportunities for a while. If food was taken away during the periods while the rat received its daily brain stimulation, after several days, the typical rat began to drink from a nearby waterspout. One might think that the rat was merely falling back on a second-best substitute. Perhaps, the rat would still prefer to eat if again given a choice? But this was not so. Eventually, even if the food were put back, those rats ignored it and did not eat while their hypothalamus was activated. Instead, they continued to drink during stimulation, sticking with the experience-induced alternative behavior and eschewing their original motivation. Originally, it seemed that the electrode activated the hunger neurons, but later, it activated thirst neurons. Yet, this electrode had never moved, only the motivated behavior had changed.

These demonstrations of motivational plasticity, when combined with the demonstrations of hypothalamic prepotency described above, effectively put an end to the plausibility of the dedicated drive neurons in lateral hypothalamus [177]. The concept simply seemed wrong that hunger is caused by dedicated hunger neurons, thirst by thirst neurons, sex by sex neurons, and other motivations by their own dedicated drive neurons. Instead, lateral hypothalamic electrodes activated more complex motivational brain systems, which tended to express whatever prepotent motivation was dominant for an individual at that moment, but which could be shifted by a variety of factors, such as individual experience, that were irrelevant to its biological regulation. Thus ended a "dedicated drive neuron" chapter in the behavioral neuroscience of motivation.

5.2. Dedicated neuropeptides

Or did it? In the past 10 years, the concept that dedicated neurons cause hunger/satiety, for example, has been partly

resurrected in the form of dedicated neurochemical coding by leptin, neuropeptide Y, cholecystokinin, ghrelin and related peptide signals for appetite or satiety [18,71,193]. Contemporary hypotheses that hunger is caused by hypothalamic neuropeptide Y or agouti-related protein, or that satiety is caused by hypothalamic leptin or alpha-melanocyte-stimulating hormone (MSH), may sometimes seem to imply that neuropeptide receptors are dedicated to particular motivational drives, just as hypothalamic neurons were once thought to be. The old hypothesis that the hypothalamus contains dedicated hunger neurons has metamorphosed into the new hypothesis that the hypothalamus contains dedicated hunger peptides and dedicated hunger receptors. Similar stories of dedicated neurochemical coding could be framed for other motivations too. For example, thirst may be said to be caused by brain receptors for angiotensin II, and social motivations, such as attachment, have been suggested to be caused by activating brain peptide receptors for oxytocin and vasopressin [105,162].

Do dedicated neuropeptides trigger distinct motivated behaviors, as neurochemical keys slipping into their own motivational locks to unlock hunger or thirst or social attachment? Such concepts are highly attractive to some behavioral neuroscientists because they appear to give a simple and concrete basis for how hunger differs from thirst, sex, aggression, or affiliation. Neuropeptide coding of motivation is appealing for exactly the same reasons as motivational brain centers or dedicated motivational neurons. All these concepts suggest that there is a specific physical substrate that we can point to as the cause for a specific motivation. But several neuroscientists have sounded calls for caution [18,131,191]. For example, although neuropeptide Y is sometimes called the hunger neuropeptide, it also causes effects that are quite different and, in some ways, even opposite to normal hunger, such as promoting a conditioned aversion to the taste of the food it made a rat eat [191]. In other words, neuropeptide receptors may turn out to be similar to electrical stimulation of the hypothalamus in their motivational effects, in that neither produces true natural categories of motivation, such as pure hunger, and both produce broader motivational effects. If so, the dedicated neurochemical coding concept of motivation may turn out to be as misleading as the simplistic neuroanatomical concepts that came before it. This debate will probably continue for some time. Perhaps, new versions of old ideas may prove more enduring than originals will. Or, a better balance may be struck this time for behavioral neuroscience between attractively simple concepts and persistently complex realities. History will be the judge, based on future data and concepts.

5.3. Neural hierarchies of motivation

Final mention should be given to neural hierarchy, a brain concept as enduring as dedicated centers and neurons. Hierarchical concepts of brain organization have long been important in thinking about motivation [7,58,74,118,147,166,181]. For instance, simple motivated consummatory behaviors, like chewing and swallowing food, are known to be generated by the brainstem, at least in their most basic forms [8,66,147]. Yet, somehow, these brainstem circuits are under the continual governance of forebrain circuits in normal brains—as when you decide its time to eat.

Concepts of brain hierarchy were shaped a century ago by the insightful clinical deductions of John Hughlings-Jackson, a British neurologist. Hughlings-Jackson studied the symptoms of human patients who had lesions of the brain from strokes or accidents. He concluded that brain hierarchies controlled most psychological functions involved in movement, motivation, emotion, and cognition. Hierarchy was often dramatically revealed in his human patients after forebrain injury by their inability to call upon lower functions for voluntary purposes, even though their lower brain still retained basic capacity to generate the function [74]. For example, a patient with damage to one side of their motor neocortex was no longer able to smile voluntarily on one half of the face. One might conclude that the person had a unilateral paralysis of facial muscles on seeing the voluntary attempt made without success. Indeed, other patients with brainstem damage, not cortical damage, have true facial paralysis that prevents them from smiling. Yet, when told a funny joke, the patient with cortex damage could suddenly smile quite normally on both sides. The emotion involved in humor still was able to cause a normal smile, even though the smile was lost to the patient's repertoire of purposeful acts to do by voluntary will. Smiling itself, as a facial expression, and the emotion that ordinarily spurs it when we hear a joke were both intact beneath the damaged cortex. All that was lost after the cortical lesion, Hughlings-Jackson proposed, was the voluntary, top level of the hierarchy in the neocortex. By this hierarchical concept, the same smile is represented in several levels of the brain: the brainstem (as a motor pattern), the lower forebrain (as an emotional response), and the cortex (as a polite smile under voluntary control).

Multiple neural levels of function representation, with higher levels acting as the boss of lower levels, is the essence of a brain hierarchy [58]. In the above example, the patient lacked a neocortical command element that ordinarily enabled a voluntary decision to smile and bossed the lower levels into producing it. But the lower levels by themselves were sufficient for more basic emotional generation of smiles. For motivation, the simplest and traditional hierarchical view is that the brainstem mediates mere reflexes, whereas the forebrain mediates controlling motivational functions. However, that view ignores the evidence that in a whole healthy brain even, the brainstem elements contribute to causing true affect and motivational functions [38,151]. A more integrative

hierarchical concept is that the brainstem mediates core motivational functions (including aspects of core affective reaction as well as consummatory behaviors), whereas the forebrain mediates higher functions that interact with lower ones to elaborate their functions, add new features, and exert hierarchical descending controls over brainstem functions [58].

Modern concepts of brain motivation hierarchies have been strongly influenced by ideas of the behavioral neuroscientist Charles R. Gallistel [58]. As Gallistel puts it, each level in a brain hierarchy is semiautonomous. Even lower levels are autonomous, in the sense that they exercise final control over the function they perform. No other level can perform the autonomous function of a lower level—not even higher ones. If the low level is damaged, the function is truly lost. For example, after a paralyzing the brainstem lesion, a person cannot smile ever at all. But the levels are only semiautonomous, not fully autonomous, because they are subject to being bossed by higher brain levels. Hierarchically superior levels decide when and if to activate a function. Higher levels can boss lower levels by sending down hierarchical signals of excitation or inhibition [58].

Today, hierarchical concepts continue to guide behavioral neuroscientists' thinking about motivation [7,63,67,149,161,168]. Hierarchy concepts work extremely well for describing large-scale relations between the forebrain and the lower brain. For example, Smith, Grill, and others suggest that the forebrain can usefully be posited to initiate appetitive motivated behavior by a hierarchical mechanism, whereas the brainstem executes consummatory aspects of motivated behavior [63,64,147,149].

5.3.1. Limitations to hierarchy

Yet, even brain hierarchy concepts of motivation go only so far. They may possibly not go much farther than beyond the top of the midbrain, at least in one sense. Although hierarchy may nicely capture relations between the forebrain and the brainstem, it does not always seem to do so well in capturing the relations between, say, the cortex and the hypothalamus [181]. Yes, the neocortex might plausibly be proposed to hierarchically boss the hypothalamus, perhaps, during cognitive or voluntary regulation of emotion [40]. But often, emotional reactions overpower voluntary regulation. Which is the boss when you have an irrepressible emotion, the cortex or subcortical brain? And what of the relations between each of the limbic structures contained in the forebrain? Does amygdala boss the mesolimbic accumbens system or vice versa? Does the nucleus accumbens boss the hypothalamus or the reverse, or how about the hypothalamus and ventral tegmental area? And does the cortex always boss these other subcortical structures? Or is it ever bossed by them?

Behavioral neuroscience evidence about forebrain hierarchies is contradictory. One reason may be the lack of clear anatomical hierarchy in forebrain limbic wiring (Fig. 9). There are not just straightforward top-down connections from top forebrain structures to slightly lower ones. Instead, complex reentrant loops connect together the nucleus accumbens, ventral pallidum, hypothalamus, amygdala, septum, hippocampus, ventral tegmental area, cingulate, and prefrontal cortex, and other forebrain limbic structures [161,181]. Looking at a forebrain limbic wiring diagram, one is struck by the looping swirls of complexity, not just a neat linear top-down hierarchy [48,67,68,161,181,196].

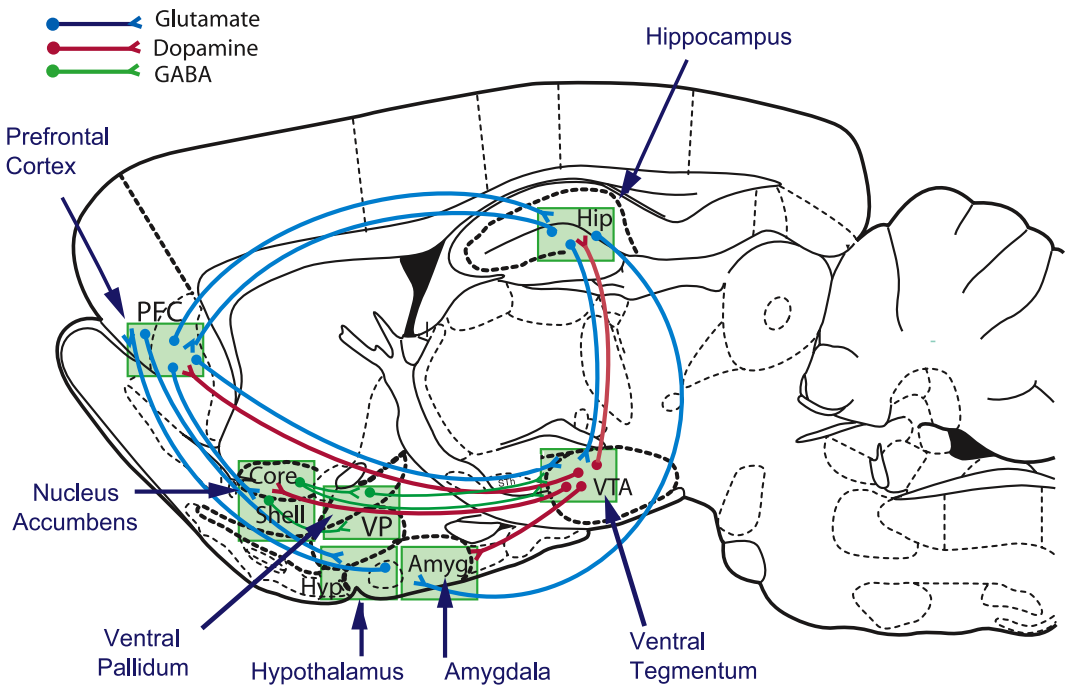

Fig. 9. Where is the hierarchy in forebrain? Some neuroanatomical connections among forebrain limbic structures. Modified from Kelley and Berridge [84].

Hence, perhaps, we need new brain organization concepts in addition to hierarchy, especially for forebrain circuits. The term heterarchy can be applied to a nonhierarchical system of equals, in which multiple structures play relatively equal roles. Heterarchy may be a step toward the truth for limbic circuits. But of course, heterarchy by itself means little more than not hierarchical. It tells us little about the positive organizational features of the limbic circuit. More specific concepts are slowly being evolved for what heterarchy means in the context of limbic forebrain circuits [31,48,67,68,82,84,161,181,196]. The development of better neuroanatomical and functional brain concepts for limbic circuit organization in the future will certainly prove useful to progress in behavioral neuroscience of motivation.

6. Conclusion

Motivational concepts are needed to understand the brain, just as brain concepts are needed to understand motivation. Motivation concepts can aid behavioral neuroscience to live up to its potential of providing brain-based explanations of motivated behavior in real life. Without them, neuroscience models would remain oversimplified fragments, removed from the behavioral reality they purport to explain.

Trying to explain how the brain controls motivated behavior without motivational concepts is like trying to understand what your computer does without concepts of software. Erich von Holst, an important early behavioral neuroscientist, emphasized that we need what he called level-adequate concepts to understand how brains control behavior [73]. Level adequate means an explanation that adequately matches the level of complexity of the thing we are trying to explain. Concepts of hedonic reaction, incentive motivation, homeostatic reflex, hierarchy, heterarchy, etc., are all level adequate in the sense that they are the simplest possible concepts to adequately capture the crucial corresponding aspects of what brains actually do.

Still, motivation concepts must be chosen carefully and continually evaluated against new data. Fundamentally wrong concepts of motivation are as bad as none at all. As we have seen, inadequate concepts must sometimes be tossed out, and useful concepts made better, based on experimental evidence.

Additional goals for the future will include mapping new neuroscience developments onto new motivation concepts. How do brain levels of neuroanatomical structures and systems, neurochemical signals and modulation, intracellular biochemistry, and molecular gene signalling levels all interact to instantiate psychological processes and control behavior? Behavioral neuroscience can help answer such important questions. Answering them is a shared goal of neuroscientists, psychologists, and others who conduct behavioral neuroscience research. Good concepts of motivation are vital to reach that goal.

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