*Chapter* **3**

# Biological Motives: Hunger and Thirst

*Water was the only thing on my mind. One of the men led me into a cabin where I downed four China mugs of water in quick succession. The skipper, who was barely my age, became alarmed. "Aren't you overdoing it?" he asked. I said yes, maybe too much water would be bad. So I had a couple of mugs of pineapple juice and a mug of hot beef broth, one after the other.*

—EDWARD V. RICKENBACKER*

Let us begin with two motives that are essential to life. The motivational systems that keep the body supplied with fuel and, in land-dwelling animals, with water, *must* be intact and functioning. Otherwise, we simply wouldn't be here. And if the demands of these motives were not met with some regularity, we would not stay around for very long.

In examining feeding and drinking, we will look for *principles*—what kinds of systems are we dealing with, and what do they do?—and we will see some investigations that illustrate these principles. These simple biological motives may seem a long way removed from attacks with guns, career choices, and other complex human actions. They are. But in tackling something as complex as motivation, it makes sense to start with simple cases. Then too, we will see that *simplicity* is a relative matter. We are a long way from understanding just what happens even when a laboratory rat is made hungry.

*Quoted by Wolf (1958). Captain Edward Rickenbacker had just spent twenty-one days on a life raft floating in the Pacific, with seven companions (six survived).

## DRIVES AND MOTIVATIONAL STATES

At the end of Chapter 1, we saw that behaviorism had become the dominant school of thought at least in American psychology. Its program, as John B. Watson declared it, was to determine the relations between objectively specifiable *stimuli* and objectively measurable *responses.*

Even to behaviorists, however, it became apparent that that program would not work. Something more was needed, for two reasons.

### Behavioral Variation in a Constant Environment

First, responses may vary even when the stimulus is constant. Thus Curt P. Richter,[1] one of the founders of modern motivational psychology, saw great differences from one day to another in the amount of activity displayed by rats in a *constant stimulus situation.* When he measured this activity (Figure 3-1), he found it to be affected by the internal state of the animal. Female rats showed peaks of activity every fourth or fifth day, and these peaks corresponded roughly to the times of sexual receptivity,

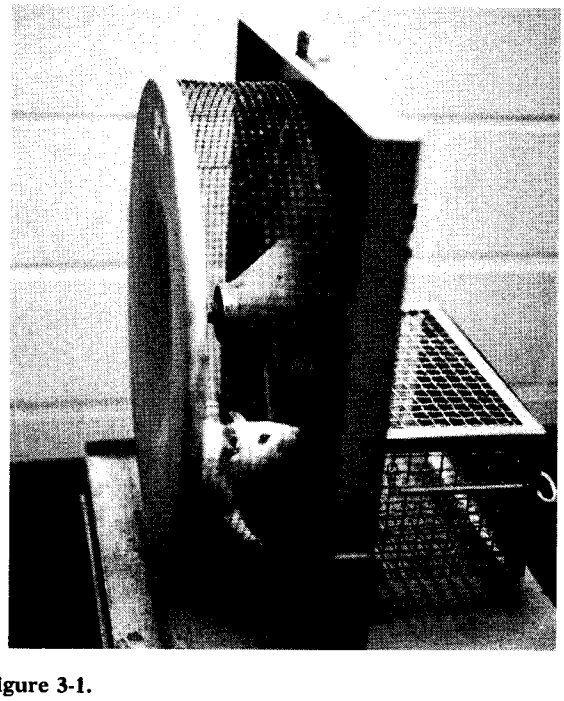

**Figure 3-1.**
Activity wheel for the rat. The rat turns the wheel as it runs, and each revolution of the wheel advances a counter, providing a record of the amount of running that occurs.

[1]Richter, 1922.

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or *estrus.* Male rats showed no such cycles of activity. But if food was withheld, both males and females became progressively more active as time without food elapsed—again, even in an unchanging external environment.

Clearly, in addition to the stimulus situation outside the animal, one has to consider its *internal state.* Behavior changes when the environment does not. If the causes of that change are not in the situation, then they must be in the rat.

## Multiple Outputs: Goal-directed Action

Second, an effect of internal state can be expressed by any of a number of specific responses. Offer solid food to a hungry rat, and it will chew; offer it liquid food, and it will lap. But it may do *none of these things* if it is not hungry.

### Arbitrary Responses and Purposive Behavior

Appropriate training can add more members to the class of actions. If food is available, a food-deprived rat will approach it directly. If food is not available, and if the rat has been trained to run a maze for food, it will run that maze. If trained to press a lever for food, it will press the lever.

This is an important fact. It is the reason we speak of *motivation,* and not just of *feeding or drinking behavior.* The behavior itself is not fixed; rather, the rat will make any of a variety of specific responses to get its food. More important, the connection between, say, lever-pressing and food is a totally arbitrary one, set up by the experimenter. There is nothing in the nature of food, or of rats, that connects lever-pressing with food.

To many writers, it is this *arbitrariness* of the response that is diagnostic of purposive, goal-directed behavior.[2] It is how we distinguish goal-directed behavior from reflexive or instinctive behavior that is not arbitrary, but a natural part of an animal's feeding repertoire. Some writers would say that it shows *desire* or *wanting.* If we see that the rat will make an arbitrary response—that it will *do whatever it has to do, make whatever response is available* to obtain food—then we may say that it *wants* food.

### Hierarchical Organization

All this suggests some mechanism, or state, that is different from any specific response. For convenience, we will call such a state a **motivational state.** Here, such a state is activated by food deprivation, and it can activate in turn any of a number of specific actions. We might as well call

[2]For example Tolman, 1932; Teitelbaum, 1966.

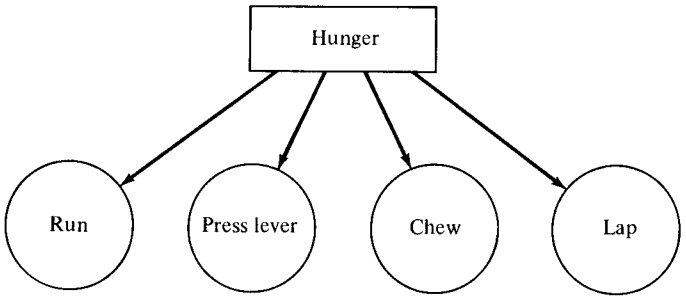


**Figure 3-2.**
Hunger as a motivational state that can be expressed by any of a number of responses.

it *hunger.* Figure 3-2 shows the idea and also introduces another important concept: the *hierarchical organization* of behavior.

A **hierarchy** is any system that is organized from top to bottom, so that its units are composed of subunits which in turn are composed of sub-subunits, and so on down. Think of a military chain of command, in which the general can order any or all of his field officers to get something done, the officers order their sergeants to get it done, and the sergeants order their foot soldiers to do it.

Well, feeding and food-getting behavior are organized in that way. A state of hunger, the general, can tell the officer in charge of running, "Run in that direction." (Or it could tell the lever-pressing officer, "Press the lever.") The running officer gives orders to the leg-moving sergeants, which control patterns of muscle contractions: "Advance the left foot, then the right foot . . ." The muscles themselves are the foot soldiers that perform the actual movements.

Further examples abound. Later we will see that a male stickleback fish in reproductive condition will fight a rival male, court a female, or build a nest if he has no nest. But he will do *none of these* unless his hormonal state puts him in reproductive condition.[3] The hormones must act at the level of *reproductive motive(s)* to activate the whole system, with all its components.

Before moving on, it is worth noting that such a motivational state is not observed directly. It is *inferred* from behavior. Or, better: It is a way of *talking about* the behavior. If we see that an actor may achieve the same end in a variety of ways, or that a number of specific movements are affected together by some condition, then *we say* that a motivational state exists.

[3]Tinbergen, 1951.

### A Look Backward: The Drive Concept

Now let us put these pieces together. A **drive**, we will say, is a hierarchically organized **motivational state**, evoked by some change or condition inside the animal. We need such a concept for two reasons: (1) Behavior can vary in an unchanging environment, and (2) any of a number of specific responses, including arbitrary learned ones, can be activated. That is why we distinguish motivational states from the specific behaviors that express them.

## MOTIVATION, NEGATIVE FEEDBACK, AND HOMEOSTASIS

The concept of drive was attractive to scientists who sought to understand the physiological causes of action. It provides a direct link between motivated behavior and physiological regulatory processes. These relationships introduce two concepts that are fundamental to motivational thinking: homeostasis and negative feedback.

### Homeostasis

The body has mechanisms for regulating a number of physiological variables within the narrow limits that the body requires. This regulation is called **homeostasis.*** It is achieved both by physiological means and by behavioral means.

For example, if temperature rises too high, our bodies can drive it down again by perspiring: if it falls too low, our bodies drive it up again by shivering. But behavior extends our range of options. If temperature rises too high, we can move into the shade; if it falls too low, we can build a fire or put on a sweater. In other words, behavior can be a part of the homeostatic system that maintains temperature at or near an optimum value.

Other basic biological drives play a similar role. Because thirst leads us to find and drink water, the body receives water when it needs it. Because hunger drives us to eat, the body's fuel is replenished as it begins to run low. Thus a relative constancy of body water, and of available fuel, is maintained by behavioral as well as physiological means.

### Negative Feedback

Homeostatic regulation depends upon **negative feedback**, another fundamental concept of motivation. A *negative-feedback loop* is any case in which the output of a system is "fed back" to reduce the input. An air conditioner controlled by a thermostat is such a system. The temperature

*The term was coined by the physiologist Walter B. Cannon (1871–1945). It has come to mean either the optimal range of values necessary for life, or the processes by which they are kept within that range.

in the room rises (input). That calls the air conditioner into play (output), and its action reduces room temperature. That is, it reduces the input; that is what makes it a negative-feedback system.

A separate loop could keep temperature from falling too low. If the temperature drops (input), the furnace is switched on (output), and the temperature rises again to take the input away (negative feedback). With these two back-to-back systems, one can *regulate* the temperature of the room as precisely as the sensitivity of the thermostat and the speed of action of the output mechanisms permit (Figure 3-3).

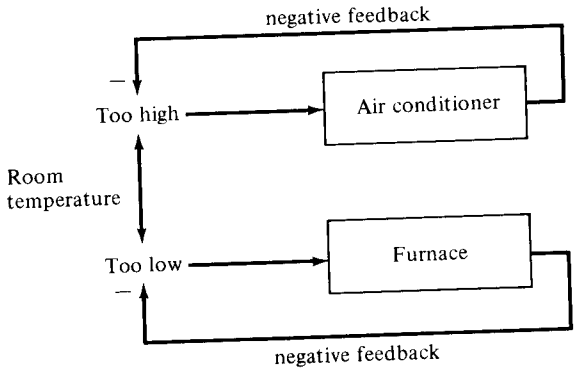


**Figure 3-3.**
Control of room temperature by back-to-back negative-feedback loops.

Finally, in either case, the output of the system could be behavior rather than the action of a machine. If external temperature uses too high, we may move to a cooler place or turn on the air conditioner manually. If it drops too low, we put more clothes on, light a fire, or turn on the furnace.

The same sort of thing happens in the body. Take blood sugar level, for example. Sugar is supplied by the blood to the cells of the body, which burn it as fuel. If the cells are to work properly or even go on living, a steady supply of fuel must be maintained. The level of sugar in the blood must be regulated within quite narrow limits. And it is—an instance of homeostasis.

How does such regulation take place? Briefly, the body has a kind of sugar bank, in which sugar is stored outside the bloodstream in the form of complex molecules known as *glycogen.* If the blood sugar level drops too low (input), there are reflex reactions that write a check on the account; glycogen is broken down into sugar molecules which are released into the blood (output), and blood sugar level rises again (negative feedback). Conversely, if blood sugar level rises too high (input), the body writes a deposit slip: Glucose molecules are withdrawn from the blood

and stacked into glycogen molecules (output), and blood sugar level decreases (negative feedback). The result is regulation of blood sugar level within a narrow range, because these back-to-back feedback loops—these *homeostatic* mechanisms—prevent it from drifting either too high or too low.

Where then does behavior come in? It can be seen as another output of the homeostatic mechanism. If the body's stores of glycogen are used up, it can draw on its fat or even its protein to keep blood sugar level from dropping. But at some point, the body's fuel reserves must be replenished. How? By finding and eating food.

In summary, hunger and thirst are negative feedback loops. When low on water (input), we find water and drink it (output), and are not low on water any more (negative feedback). When low on fuel (input), we find food and eat it (output), and are not low on fuel any more (negative feedback). The system's output—behavior—reduces the input that aroused it.

## THE LOCAL SIGN THEORIES

But how do these systems work? To present them as negative-feedback loops shows what they do; but how do they do it? In particular, what are the all-important *inputs* that call the systems into action? What tells an organism that it's time to eat or drink?

### Cannon's Experiments

The most influential early experiments were the work of the physiologist Walter B. Cannon. He regarded hunger as a sensation arising from the gastrointestinal tract, the stomach in particular.

In his most famous experiment, Cannon had a human subject swallow a balloon that in turn was coupled to a pneumatic system (one worked by air pressure) that permitted Cannon to record muscle contractions in the subject's stomach. When the empty stomach showed a series of rhythmic contractions, that was when the subject reported feeling hungry. The discovery was verified and extended by many others, who also discovered that the stomach contractions (already known as "hunger contractions") could be caused in turn by a decrease in blood sugar level. Since sugar carried by the blood is the fuel used by the cells, a drop in blood sugar level would indicate the need for food. That need is made conscious, so the argument went, by way of stomach activity, which the person senses.*

Thus Cannon concluded that hunger was a sensation evoked by a stimulus, or "sign," coming from a particular location in the body; hence the term *local sign. Thirst* received a similar analysis. Cannon concluded that the sensation of thirst was elicited by the local stimulus, dryness of the mouth.

*For a detailed presentation of this theory see Carlson (1916).

There was a subsidiary assumption in these theories—that the sensations of hunger and thirst are unpleasant. That accounts for the negative-feedback characteristics of these motives, and also for the fact that they can be expressed by multiple responses. When we experience these unpleasant sensations, we try to get rid of them (negative feedback), and we do it by whatever means is available (multiple outputs).

### The Shift to Behavior

More recent investigations have made Cannon's theories unpopular. In the first place, his data were correlational. A person may feel hungry when his stomach contracts, but that doesn't mean he feels hungry *because* his stomach contracts. Moreover, it is clear that hunger does not *require* the experience of stomach contractions. Human patients, for example, have undergone cutting of the *vagus nerve,* the nerve that carries sensations from the stomach up to the brain, and have still reported feelings of hunger. Similarly, numbing the interior of the mouth, which ought to block sensations of dryness, does not prevent thirst from arising.

This does not mean that local signs play no role in hunger and thirst. It does mean that if they do, they are not the *only* sources of these motives; there must be others as well. Therefore, scientists began looking at how the *internal state of the body* influences feeding and drinking, and food- and water-getting behaviors. Often these experiments used animals as subjects. Animals can't tell us whether they feel hunger or thirst sensations, but they can eat and drink, and we can ask how conditions inside the body affect their doing so. Let us turn to these investigations.

## THIRST

Land-dwelling animals lose water continuously, humans along with the rest. The loss occurs through many channels. You and I are losing water all the time through the lungs as we exhale moisture, and through the skin as we perspire. Still more water is lost in urine and feces.

We lose water in all these ways, but we have only one way of replenishing it. We must drink. But how do we, and other animals, know when it is time to drink?

### The Arousal of Thirst

As it became clear that the dry-mouth theory could not be the whole story of thirst, investigators began looking for the internal changes that trigger drinking.

To understand this work, we must consider that the water in our bodies can be divided into two parts, or compartments. Some of our body's water is inside the cells. That is the intracellular fluid. The rest of it is outside the cells—in the blood plasma and in the fluid that surrounds and bathes the cells (Figure 3-4). This is the extracellular fluid. The thirst system, it turns out, is responsive to both fluid compartments.

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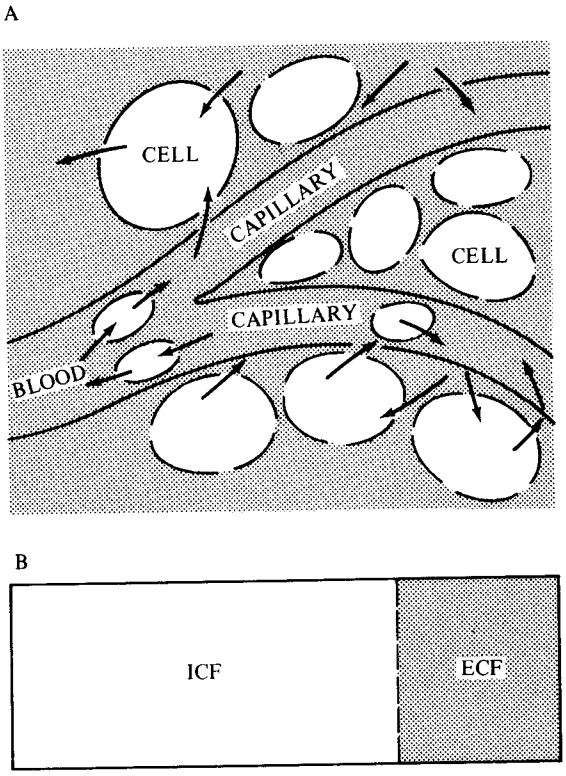
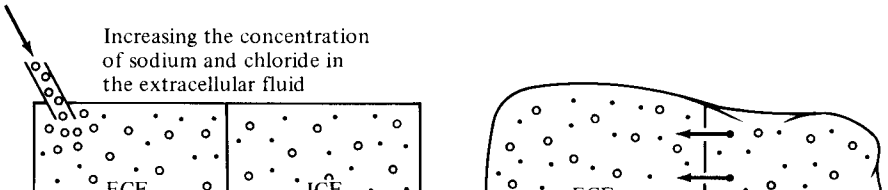



**Figure 3-4.**
(A) Distribution of body fluids outside the cells (shaded areas) and inside the cells (unshaded areas). Arrows show movement of water into and out of the bloodstream, and into and out of the cells. (B) For simplicity, we often speak of the body fluids as if they were divided into two compartments, one containing all the fluid inside all the cells (intracellular fluid, ICF), and the other containing all the fluid outside the cells (extracellular fluid, ECF).

### Intracellular Thirst

A classic experiment was performed by Alfred Gilman in 1937,[4] to show that *cellular dehydration* is a stimulus for thirst. Gilman injected a solution of sodium chloride into the bloodstreams of his subjects (dogs). Sodium and chloride, so injected, do not penetrate the cells of the body; they stay outside the cells. If the solution injected has a higher concentration of dissolved particles than do the body fluids (as Gilman's solution did), then the fluid outside the cells, the **extracellular fluid**, will have a higher concentration than the fluid inside the cells, the **intracellular fluid**. To dilute the concentrated fluid outside, water rushes out of the cells (this action is known as **osmosis**). That expands the extracellular

[4]Gilman, 1937.

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compartment shrunken.

**Figure 3-5.**
Gilman's experiment. Sodium and chloride particles in the body fluids are shown as open circles, water molecules as dots.

fluid but leaves the cells dehydrated and shrunken (Figure 3-5). Gilman injected control solutions of urea at other times; these do not have this effect, because urea does move into the cells, so that the concentration of dissolved particles remains the same inside and outside the cells.

Thus, injections of concentrated sodium chloride solutions draw water out of the cells and leave them dehydrated. Injections of concentrated urea solutions do not do that. Sure enough, Gilman's sodium chloride injections caused copious drinking, but urea injections did not.

This demonstration in the dog has since been repeated in various species, humans included. The results are the same: Strong sodium chloride solutions lead to copious drinking and, in humans, reports of intense thirst. Moreover, rats will run a maze, or press a lever, to obtain water after such injections. Therefore, cell dehydration arouses not just drinking per se, but a drive or motivational state with its multiple specific responses (see Figure 3-6 below).

### Extracellular Thirst

It later became clear that cell dehydration, like a dry mouth, could be only one stimulus for thirst, not *the* stimulus. For example, extensive bleeding from wounds can provoke very severe thirst. Yet in these cases, the fluid is lost not from the cells but from the extracellular fluid. This is the fluid outside the cells, which includes the blood plasma and the fluid that surrounds the cells themselves. Might a decrease in extracellular fluid volume also be a stimulus for thirst?

Apparently it is. James Fitzsimons[5] in England and Edward Stricker[6] in America showed that a reduction in extracellular fluid volume could

[5]Fitzsimons, 1961.
[6]Stricker, 1966.

provoke drinking. This happened even though direct measurement of the body fluids showed that no cell dehydration was present. Reduction of extracellular fluid also evoked a motivational state, not just drinking; a rat would press a lever for water in response to such reduction.

These findings and later ones led to the *double-depletion hypothesis,* which is now generally accepted. There is not one state of the body that evokes thirst, but at least two. Reduction of fluid inside the cells is one; reduction of fluid outside the cells is another; and either of these can say to a rat or a human, "Time to drink." The two sources of thirst seem to have additive effects. If one reduces the fluid both inside and outside the cells, the drinking that results is about equal to the sum of the drinking that each alone would produce.

Not one thirst, but two. Let us pause for a minute and look at the system that is taking shape (Figure 3-6). Remember that a motivational state has *multiple outputs*—the various responses it controls, like lapping, running, or bar-pressing. Now we see that it also may have *multiple inputs.* Shrinkage of the extracellular fluid can produce thirst; shrinkage of the intracellular fluid can also do so. Either one can act alone, or both can act together and sum their effects. This kind of multiple-input, multiple-output organization is characteristic of motivational states.

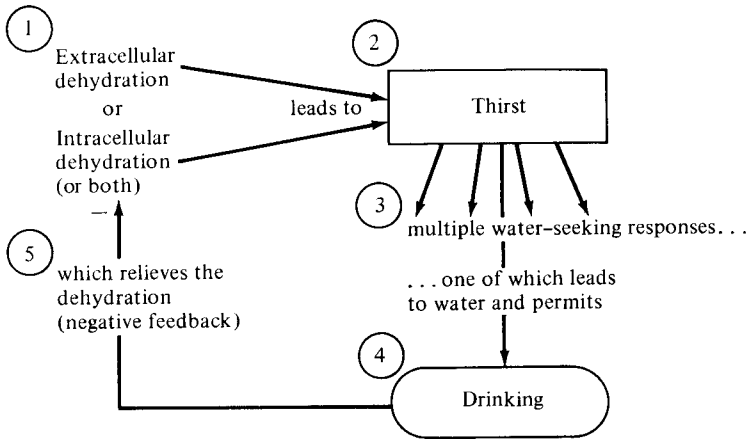


**Figure 3-6.**
Thirst as a motivational state with multiple response outputs, and two inputs. It is a negative-feedback loop inasmuch as the result of its action—drinking—takes away the dehydration(s) that acted as input.

## Satiation of Thirst

Now let us turn to another question. If either or both of these two inputs can turn thirst on, what turns it off?

### Voluntary Dehydration

Matters would be simple if drinking continued until the body's fluid loss was made good. Satiation of thirst, in that case, would be just the absence of the conditions that initiate it. But this is not so. A water-deprived rat, when offered water, stops drinking well before its water deficit is made good. The extracellular compartment, in particular, may remain shrunken. This phenomenon is called *voluntary dehydration.*[7]

There is an excellent reason for it. When a rat drinks water, most of the water gets back to the body fluids eventually; but this takes time, and during that time the rat would load too much water into its stomach if it continued to drink. If the rat did not stop drinking until it was fully hydrated, then the water still remaining in its stomach would move on from there to *over-hydrate* the body later. That could seriously disrupt the body's functions. Indeed, the state known as *water intoxication*—disruption of cellular functioning by drinking too much water—can actually cause death in rats or humans.*

The animal, then, must drink the right amount of water and then stop, even though its body is still dehydrated, especially the extracellular compartment. There are two solutions to that problem. First, the rat (or human) might *learn* to calibrate its intake, so that it drinks appropriate amounts and no more. There is evidence that this can in fact happen, but we will defer it till later (pp. 224–225).

Second, the controlling system may have an *inhibitory* component. Some short-term consequence of drinking water might shut down, or *inhibit,* the seeking and drinking of water even though a fluid deficit remains. That also happens, and we turn to it now.

### Inhibition of Thirst in the Rat

Consider what happens when a rat drinks. Water passes through the mouth; it is swallowed and enters the stomach; then it passes to the intestine, from which it is absorbed into the extra-cellular fluid; and finally, most of it (about two-thirds of the water ingested) passes into the cells. Somewhere in that chain of events must lie the inhibitory conditions that switch off thirst. A series of experiments by Elliott Blass and Warren Hall[8] explored the possibilities one by one (see Figure 3-7).

What contribution do mouth factors alone make to satiety?† To determine this, the experimenters equipped their rats with gastric fistulas, permanently implanted tubes that drain the stomach contents to the outside. When these fistulas were closed, the rats were in effect drinking normally (Figure 3-7A); but when the fistulas were open, water drained out of the stomach as fast as it entered it (Figure 3-7B). The water drunk,

[7]Adolph, 1943.

*It is true that the kidney can get rid of excess water by producing urine, but that too takes time, and a serious excess could occur before it happened.

[8]Blass and Hall, 1976.

†Satiety simply means the state of being satiated, or not inclined to drink any more.

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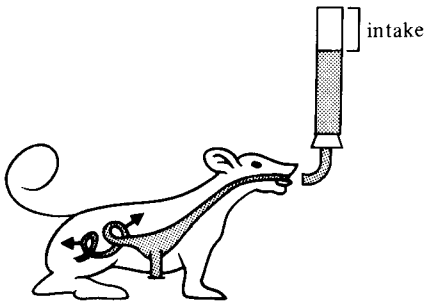
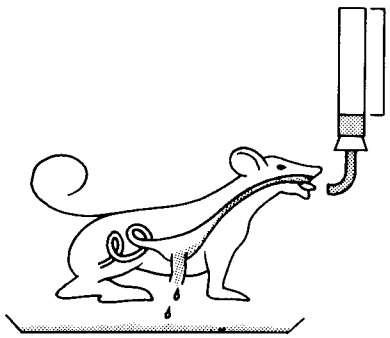
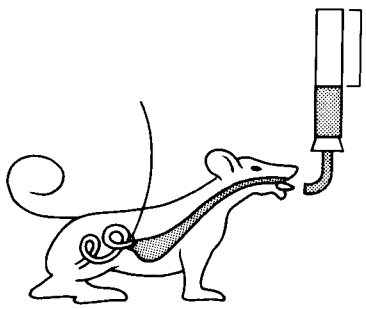


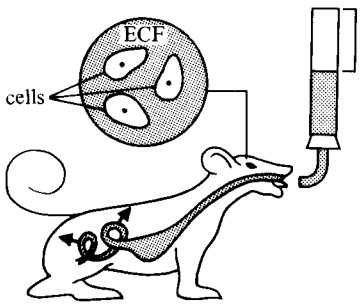

A. Normal drinking with gastric fistula closed. Water enters the stomach and passes from there to the intestine (represented by loops), from which it is absorbed into the body fluids. A moderate amount of water is drunk.



B. Sham drinking. When the ingested water drains from the stomach through the open gastric fistula, the amount drunk is very much greater than normal.



C. Drinking without stomach emptying. The entrance to the intestine is clamped off, so that water is held in the stomach. Intake is lower than with sham drinking, but still higher than normal.




D. Drinking without cellular hydration. An appropriate salt solution, after it is absorbed from the intestine, expands the extracellular fluid (ECF) without supplying water to the cells. Intake is still higher than normal. It is only when water enters the cells, as happens in (A) above, that the rat's intake is limited to the normal amount.

**Figure 3-7.**
Inhibition of thirst by the consequences of drinking.

therefore, passed through the mouth but did not fill the stomach or, of course, reach the body fluids. This is known as *sham drinking.*

When the rats drank normally (with fistulas closed), the amounts drunk varied with how severely deprived of water they were, as one might expect. When the rats sham-drank (with fistulas open), the amount passed through the mouth still varied directly with deprivation. These amounts were much higher throughout, however, than those drunk under normal conditions.

This tells us two things. (1) Just passing water through the mouth makes *some* contribution to satiety. Sham drinking stops eventually, even though the water hasn't gone anywhere in the body. And the amount sham-drunk varies with deprivation, as if a thirsty rat needs more of that oral stimulation to satiate thirst than a less thirsty rat does. (2) However, passing a normal amount of water through the mouth is *not* enough to satiate thirst. Sham intake was much higher than normal intake.

If mouth factors alone are insufficient, what if we add stomach filling? Now the rats were permitted to drink with fistulas closed, so that the stomach filled; but the passage from the stomach to the intestine was pinched off with an ingenious "noose" arrangement, so that the water did not pass into the intestine and from there to the cells. As Figure 3-7C shows, intake was still higher than normal. Also, under these conditions, the amount drunk did *not* vary with severity of deprivation. Perhaps stomach filling sets an upper limit on intake, so that the rat could drink only so much and no more. Clearly, though, a *normal* amount of stomach filling is not enough to satiate the rat.

Well, let us add still more. Suppose the water empties from the stomach and is absorbed from the intestine, but does not enter the cells? That too can be arranged. Suppose a rat drinks a salt solution rather than water. Then the water, the sodium, and the chloride will all be absorbed into the extracellular fluid. But sodium and chloride do not enter the cells; and since they are confined to the extracellular fluid compartment, osmotic action holds the water in which they are dissolved in that compartment. By using an appropriate salt concentration, one can produce expansion of the extracellular fluid with no effect on the intracellular fluid—neither expansion nor shrinkage.

Drinking such a solution, then, sets up the conditions shown in Figure 3-7D. All the other consequences of normal water drinking take place, but the cells are not hydrated. The result? Intake remains abnormally high.*

In short, nothing that water does in the body reduces intake to normal levels *unless the water enters the cells and hydrates them.* It appears that cellular hydration provides an inhibitory stimulus for thirst, shutting off water intake even though the rat is not yet fully rehydrated by its drinking.

*Of course a salt solution also tastes different from plain water, but it has been shown that taste makes no contribution to this high intake (see Mook, 1963; Mook and Kozub, 1968).

## A Look Backward: The Organization of Thirst

Figure 3-8 shows the system as we have developed it thus far. It has come quite a way, as we see, from the simple negative-feedback loop we began with. It has its two inputs (and there may be more). It has its multiple outputs, so that the rat can take whatever means its environment and its training make available to get the water it seeks. The rat drinks, and the water it drinks activates an inhibitory system that switches off thirst, though the initial dehydration of the body is still there. And the inhibitory system too has multiple inputs—three at least, from the mouth, from the stomach, and from the cells.

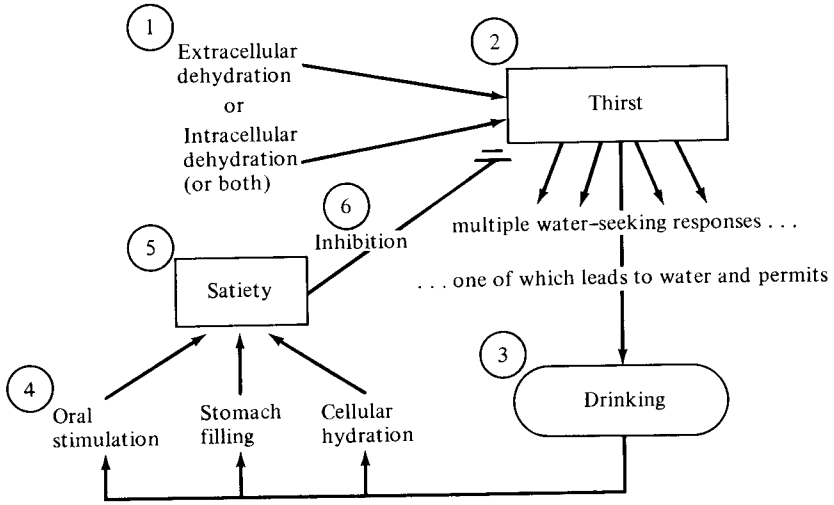



**Figure 3-8.**
The role of satiety in thirst. Oral, gastric, and cellular consequences of drinking activate an inhibitory mechanism—satiety—that shuts off thirst and drinking *before* the body's dehydration is alleviated.

That is still only a little of it. But before we take on any more complications, let us look at another basic homeostatic system—hunger. We will find that hunger and thirst have a great deal in common.

## HUNGER

In contrast to thirst, the physiological signal(s) for **hunger** remain to be clearly identified. Even the perennial candidate, the availability of glucose (blood sugar) to the cells, remains a controversial one.

### Arousal of Hunger: The Glucostatic Hypothesis

Cannon's gastric-pangs theory was coupled to the homeostatic control of food intake by the suggestion that a drop in blood sugar level was

what started the whole process going. When blood sugar level drops, the stomach begins to contract, and that is what we feel as hunger.[9] As Cannon's theory faded, the possibility gained ground that a low blood sugar level could stimulate hunger directly. That is the **glucostatic hypothesis.**

In this simple form, the idea runs into a major difficulty. The disease known as *diabetes* is characterized by insufficiency of insulin, a hormone that is required for the passage of glucose from the blood into the cells. As a result, diabetic people may have very high blood sugar levels. If the theory were correct, they should seldom get hungry. In fact, however, many of them report feeling hungry nearly all the time. Obesity is frequently a complication of diabetes.

The physiologist Jean Mayer[10] suggested a way out of the difficulty.* Perhaps what is important is not how much glucose is in the blood, but how much gets into the cells. If so, the diabetic's hunger would make sense; the sugar is in the blood all right, but it doesn't go from there into the cells. As a result the cells are literally starved for glucose, and hunger results.

If this is so, then preventing the cells from obtaining and using glucose should elicit hunger. One way of doing that is to inject a chemical known as 2-deoxy-D-glucose, or 2-DG. This compound interferes with the uptake of glucose by the cells. That causes glucose to accumulate in the blood; but the cells are not receiving that fuel. The body is like a car with a full tank but a block in the fuel line. If Mayer's idea is correct, then this elevated blood sugar level should be accompanied by hunger and ravenous feeding. And that is exactly what happens, in rats and monkeys[11] and in humans.[12]

## Are There Other Stimuli for Hunger?

Things are not as simple as that, however, for two reasons. First, some investigators believe that whereas decreased use of glucose by the cells may be *a* stimulus for hunger, it cannot be *the* stimulus. Rats whose brains have been selectively damaged may no longer feed in response to decreased glucose availability. Yet such rats may eat voluntarily in a perfectly normal way, and maintain themselves in good health. If they eat, but no longer respond to 2-DG or related manipulations, then they must get hungry for other reasons than the unavailability of glucose to the cells. Other stimuli for hunger must exist.[13]

Second, there is the question whether decreased use of glucose is a

[9]Carlson, 1916.
[10]Mayer, 1955.
*"Jean" is the French form of "John."
[11]Smith and Epstein, 1969.
[12]Thompson and Campbell, 1977.
[13]Epstein and Teitelbaum, 1967; Blass and Kraly, 1974.

stimulus that comes into play under normal conditions. Some think it is,[14] but others believe that the drop in glucose availability necessary to produce hunger is so extreme that it would occur in nature only as an emergency, not as a normal stimulus for hunger.[15]

What other such stimuli might there be? Unfortunately, if we turn our backs on stomach contractions and sugar use by the cells, we are pretty much reduced to guesswork. We just do not know.

## How Many Hungers Are There?

Part of the problem may be that the question we began with is wrong. Asking "What causes hunger?" may be like asking "What causes catastrophes?" Obviously, earthquakes and tornadoes have different causes, and maybe different hungers have different causes, too.

As one example, rats that are severely deficient in sodium are usually sluggish eaters of standard rat food; they reduce their intake of such food and lose weight. But they eat or drink vigorously when offered salt.[16] It is not that rats learn to do this, in order to feel better; they do it even under sham-drinking conditions, where the salt they taste never does the body any good.[17] Rather there is a separate "salt hunger," which can be high at a time when hunger for other commodities is low.

How many other such *specific hungers* might there be? We do know that at least in the rat, protein intake is regulated separately from the ingestion of other nutrients. One experimenter's rats[18] lived in a "cafeteria," with separate sources of protein, carbohydrate, fat, and vitamins and minerals. When the protein solution was diluted with water, the rats promptly increased their intake *of the protein solution*—but of nothing else—in compensation. And this was not just because they *liked* the dilute solution better; the increase occurred even if the solution was less preferred when diluted.* Rather, protein and carbohydrate intake may be responsive to separate "hungers."

Perhaps, rather than a hunger system per se, we have separate systems controlling hunger for *this* and hunger for *that.* It makes eminent sense that both humans and rats should have evolved that way. We need not only energy, but fats, proteins, vitamins, and minerals, and we could easily eat enough of a particular food to satisfy one need while others persist. So, we have developed a system in which hunger for *this* is satiated, while hunger for *that* persists. Of course, this also means that the

[14]See LeMagnen, 1981.
[15]See Geiselman and Novin, 1982; Smith, 1982.
[16]Richter, 1942–43.
[17]Mook, 1969.
[18]Rozin, 1968.
*One might say, "Well, if a rat takes a meal from a more dilute protein solution, he will get fewer calories for his efforts and get hungry again sooner. And so he will eat more." True; but if that were all, then the rat should increase his intake of *all* dietary components when the protein is diluted. Nothing of the sort happened; only protein intake increased.

organism must have ways of identifying, and responding to, *this* rather than *that*—a problem that looks forward to the topic of stimulus factors in ingestion.

## Satiation of Hunger

As with drinking, neither rat nor human can afford to go on eating until the food is digested, absorbed, and brought to the cells by the blood. This simply takes too long. There must be an inhibitory system here too. So, as with water, we can follow the progress of food, from the mouth through the body. Somewhere along that journey, ingested food must have the effect of shutting feeding down (Figure 3-9).*

### Mouth Factors

Sham feeding, like sham drinking, is sensitive to the state of need that was there when the feeding bout began. A rat that has gone without food for only 4 hours sham-feeds only slightly more than he would eat if the fistula were closed and he were eating normally. But if more severely deprived, such a rat may sham-feed literally for hours (Figure 3-9A).[19] So, in feeding as in drinking, passing food through the mouth can make *some* contribution to satiety. However, again as in drinking, the passage of normal amounts of food through the mouth alone does not suffice to turn ingestion off.

### Stomach Factors

One can study the role of the stomach using a technique similar to that of Blass and Hall, preventing stomach emptying by means of a noose (Figure 3-9B). When this was done, rats 3 hours without food (a period within the rats' normal inter-meal interval) ate meals of normal size. Therefore, filling of the stomach with food, even if the food does not move on from there, *can* produce satiety.[20]

### Intestinal Factors

On the other hand, presentation of food to the duodenum, bypassing the stomach, *can* also produce satiety.[21] (The duodenum is the portion of the small intestine that is nearest the stomach.) One team of experimenters infused liquid food into the duodenum while the rats were sham-feeding the same liquid food through an open gastric fistula (Figure 3-9C). (That

*Many of these studies were conducted using liquid diets such as fortified milk or eggnog, because, especially in rats, sham-feeding experiments are more easily conducted that way. Thus the motor act is one of drinking or lapping. However, since the rats are deprived of food but not of water before the experiment, we assume that they are actually "eating" the liquid diets in response to hunger, rather than drinking them in response to thirst. There are various lines of evidence that support that assumption (see Teitelbaum and Epstein, 1962).

[19]Young et al., 1974.

[20]Kraly and Smith, 1978; Deutsch, Young, and Kalogeris, 1978.

[21]Liebling, Eisner, Gibbs, and Smith, 1975.

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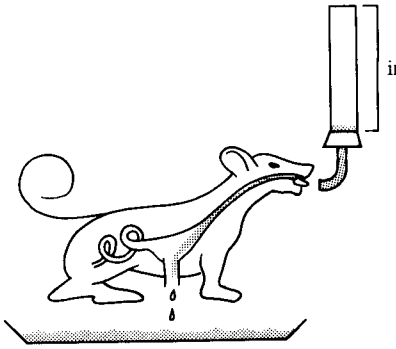
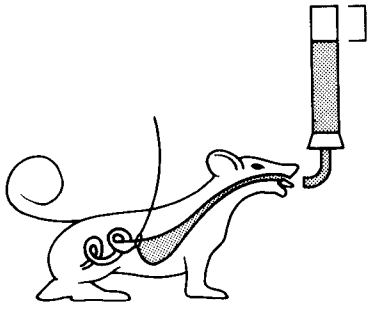
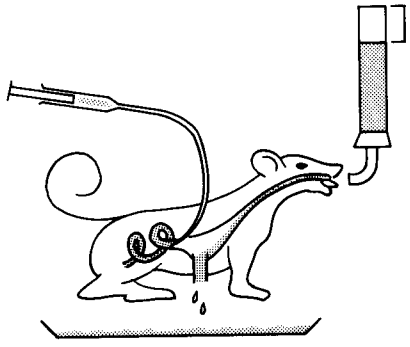



A. Sham feeding. If the liquid diet drains from the stomach as the rat feeds, then food intake, like water intake, is very much higher than normal.



B. Satiation of feeding by the stomach. If the food ingested enters the stomach, even though it goes no further, intake is limited to normal amounts.



C. Satiation of feeding by the duodenum. When food is injected into the duodenum through flexible tubing while the rat sham drinks, the amount drunk is limited to normal amounts. Therefore, food in the stomach or in the duodenum can inhibit feeding after a meal of normal size has been ingested.

**Figure 3-9.**
Inhibition of feeding by the consequences of eating a liquid diet.

way, intestinal contents were prevented from mixing with the food the animal was ingesting through its mouth.) The infusions depressed sham feeding, to produce a meal of normal size. Moreover, the volumes required to do it were about the same as the volumes that were found in the intestines of rats that were satiated after feeding normally.

### Blood Factors

The *glucostatic theory* implies that an increase in blood sugar, *if* the sugar enters and is used by the cells, ought to terminate hunger. In fact, however, injection of glucose directly into the blood typically has little effect.

If a hungry animal is going to eat anyway, it will eat about the same amount even after such an injection.

However, there is a part of the circulatory system that may be special. When nutrients leave the intestine for the bloodstream, they pass first into the *portal vein* that conveys them to the liver. When glucose was injected directly into the portal vein, it inhibited feeding in rats. Injections into the jugular vein, bypassing the liver, had no such effect.[22] In other words, artificial elevation of blood sugar depressed feeding only if the elevation was in the blood going directly to the liver. Perhaps there are receptor cells in the liver that detect elevated nutrient levels in the blood that bathes them, and respond by saying to the rat, "Eat less!"[23]

If this is so, it would make good sense, for two reasons. First, the liver is a major storage site for glycogen. If blood sugar level is high, it withdraws glucose from the blood and stores it as glycogen. If blood sugar level is low, it releases glucose into the blood. Second, as just noted, it is the first place nutrients go after they are absorbed from the intestine. The liver therefore receives information about how much fuel is required, and how much is coming in. If it has that information anyway, it might as well tell the brain about it—as apparently it does.

As we see, there are a large number of internal factors that *can* inhibit food intake, each by itself if need be. Inhibition of feeding is another multiple-input system. Which input is first to operate under normal conditions, or whether they all work together, we are not sure. Moreover, there is no particular reason to think that the same mechanism is most important under all conditions. That takes us to the next question.

## How Many Satieties Are There?

The discussion thus far has treated satiety as if it were a single state. But if there are multiple hungers, there must be multiple satieties as well, so that a creature may be satiated for one commodity but still hungry for another.

A rat that has drunk a glucose solution to satiety, and will drink no more of it, may eat vigorously if offered standard rat chow.[24] Indeed, it may eat as much as if the just-prior "meal" of glucose had never occurred. The same thing happens in the opposite direction: Offered glucose after a chow meal, the rat may drink as much glucose as if the meal of chow had never occurred. It is clear that a rat or a human can be satiated for one food and be ravenously hungry for another, at the same time. These specific satieties are only beginning to be investigated and we know little about them. The response to *variety* may play a role (p. 87),[25] but it probably is not the whole story.[26]

[22]Campbell and Davis, 1974.
[23]See Russek, 1971.
[24]Mook, Brane, Kushner, and Whitt, 1983.
[25]Rolls, Rowe, and Rolls, 1982.
[26]Mook, Brane, and Whitt, 1983.

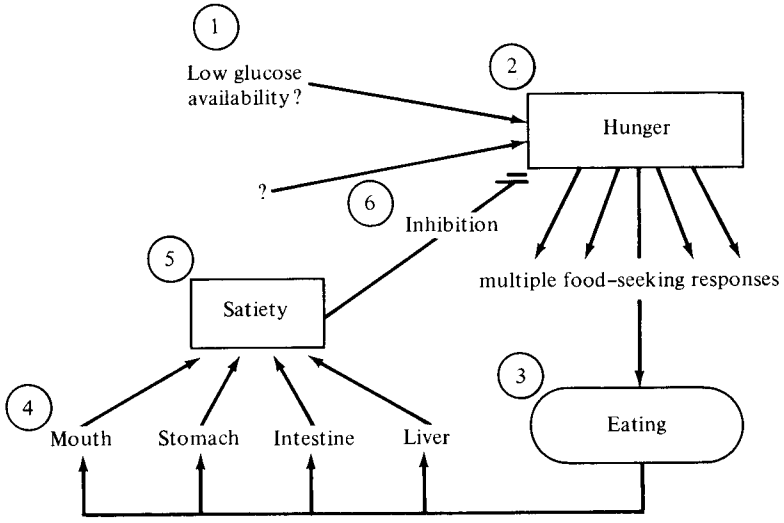



**Figure 3-10.**
Hunger as a multiple-output, multiple-input negative-feedback loop. Like thirst, it has a satiety component that shuts off ingestion even before the inputs are removed.

## A Look Backward and a New Complication: Lattice Hierarchies

Figure 3-10 shows the system controlling hunger in the rat as we have thus far developed it.

### Parallels with Thirst

The overall organization of our hunger system parallels thirst quite closely. There are almost certainly multiple inputs. There are certainly multiple outputs: A hungry rat will chew or lap food, or it can be trained to press a lever or run a maze to get food. There is an inhibitory mechanism—satiety—that shuts the system down, and it in turn has multiple inputs from various places in the body.

### Converging Outputs: Lattice Hierarchies

Notice something else as well. A hungry rat, in the experiments we described, was often offered a liquid diet such as eggnog or a glucose solution. Offered such a liquid food, a rat will lap, much as it would lap water. Therefore, the lapping response can act as a component of a hunger system *or* of a thirst system. So can approaching, or running a maze, or pressing a lever (Figure 3-11A). A hungry rat can be trained to perform the action to get food; a thirsty rat, to get water.

Think again of a chain of command, and imagine an officer with specialized skills that can be made available to whichever superior officer requires them. Here, it could be the officer in charge of lapping. General

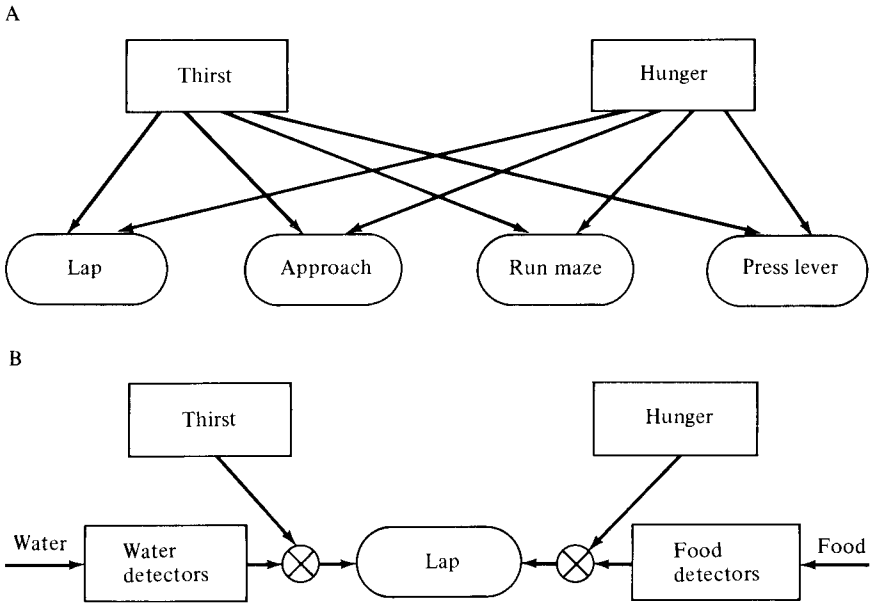



**Figure 3-11.**
(A) Hunger and thirst, and the responses that express them, form a lattice hierarchy. Both are hierarchically organized, and they converge to control responses in common. Either system can call the lapping response into play —or the bar-press response or the running response if appropriate training has been given. (B) A closer look at part of Panel A. Hunger and thirst call the lapping response into play, not directly, but by making it responsive to the appropriate stimuli—water in the one case, or liquid food in the other. The symbol ⊗ represents a point where converging inputs are combined.

Thirst can give orders to that officer when she needs his skills. General Hunger can give orders to that officer when *he* needs his skills.

This kind of organization is very often found in motivated actions. We can eat because we are hungry (a physiological motive), or to be sociable (a social motive), or because we are acting in a play and the script calls for it (a more complex social motive). If we mate, it may be because we are sexually aroused, or because we wish to please the partner—or, for that matter, the motive may have nothing to do with sex. It may be strictly economic, as in prostitution.

The principle in all these cases is the same. A given action may be called into play by different motivational systems, higher up in the hierarchy, on different occasions.

A hierarchical organization like this, in which lower components can be controlled by different higher-level systems at different times, is called

a **lattice hierarchy**.[27] Figure 3-11 shows why: The lines of influence from systems to actions cross each other to form a lattice. Such organization provides for a great deal of the flexibility of behavior. It makes specific actions into general-purpose tools or modules, which can be put to different uses at different times. We will have much more to say about it in the following pages.

### A Look Forward: Internal Effects on Response to Stimuli

But let's return to ingestion. Even for the rat, all this is still too simple. A hungry, non-thirsty rat is not just ready to lap at any old liquid. It will ignore water altogether. It is ready to lap *food*—a liquid diet. So in Figure 3-11B we have added a complication. The effect of hunger is not to drive the lapping response directly, but to make it responsive to the stimuli that indicate food.

Conversely, a thirsty rat is reluctant to chew dry food. But it will chew wet lettuce and obtain water that way. Just as it will eat by lapping, so it will drink by chewing.

Thus in these simple cases, *the actual behavior is a response to stimuli in the environment*—their learned or unlearned signal properties that identify them as water on the one hand or food on the other.[28]

We could say the same sort of thing about the arbitrary learned acts we require of an animal. A thirsty rat will press a lever, *if* (1) there is a lever present and (2) lever-pressing has led to water in the past. If these conditions are met, then the rat responds to the *stimuli* the lever provides, by approaching the lever and pressing it. These considerations are not shown in the diagram, for they would complicate it tremendously—even if our understanding were complete enough to draw such a diagram, and it isn't—but we will address them later (see Chapters 8 and 9).

## FEEDING IN HUMANS

The experimental analysis of human feeding has been guided by our developing understanding of animal mechanisms. Once specific questions are defined by animal research, one can often find a way to ask those questions of humans. Moreover, humans do have this advantage: They can tell you how their bodies feel. Rats cannot.

### Inhibition in Human Feeding

One experimenter,[29] for instance, invited human volunteers to have breakfast with him in his laboratory over a number of sessions. He fed

[27]Gallistel, 1980.
[28]Ibid.
[29]Jordan, 1969, 1975.

them a standard liquid diet for breakfast, under controlled conditions where the distractions of the everyday could not interfere with their eating. Moreover, the better to examine biological controls over feeding, he took steps to minimize cognitive controls, such as the subjects' knowledge of how much they were eating. Subjects sipped the liquid diet through an opaque straw, from a hidden reservoir, so that they could not see how much they were taking in. And, in fact, they proved very poor at judging the amount they had eaten when asked to do so.

What does feeding look like under these conditions? It is rapid at first, then slows down gradually until it stops. If the subjects drink a large "pre-load" glass of liquid diet from a cup before the sipping session starts, then the meal is depressed—but usually not quite enough to compensate for the calories in the pre-load.

But why is feeding depressed at all? What inhibits it? Is it the volume of the pre-load in the stomach, or the nutrients it contains, that depresses feeding? Here, you will be happy to know, is a distinct difference between humans and rats. Rats respond to the nutrients in a pre-load, but humans do not; they respond to its volume. When the pre-load is diluted with water, reducing the amount of nutrients it contains, this makes no difference to human subjects. A given pre-load volume causes about the same depression of feeding, whether it is very concentrated and supplies many calories, or very dilute, supplying few.

Let us look further. In humans as in rats, food passes from the mouth to the stomach to the intestine, and from there to the blood. Let us ask the question we asked earlier of animals: Which of these events can play a role in controlling how much is eaten?

First of all, these subjects could not see the food they were eating, but of course they tasted it and felt themselves swallowing it. Can intake be controlled without oral cues at all? Yes, it can. In another experiment the subject swallowed a tube, which led directly from a pump into the stomach. By pressing a button whenever he wanted some food, the subject activated the pump which gave him a direct intragastric infusion. Result? The meals the subjects "ate" by such intragastric injections were of normal size. And again, it was the volume of the meal and not its nutrient density that was controlled. When the diet was diluted with water, intake was no greater than when it was full strength.

Well, then, what if the whole digestive tract is bypassed? The experimenter turned to a sample of hospitalized volunteers who were being fed intravenously, receiving a mixture of glucose and amino acids—the building blocks of proteins—directly into the bloodstream.[30] All of these patients had lost considerable weight before intravenous feeding began, and they were receiving very substantial amounts of nourishment now. Yet most of the patients reported that they still felt hungry. Apparently car-

[30]Jordan et al., 1974.

bohydrates and protein circulating in the blood fail to suppress human hunger, just as carbohydrates in the blood fail to suppress rat hunger.

However, an intriguing finding was obtained in four patients, to whose diets fats were added to the intravenous infusions. When this was done, hunger *was* suppressed. We noted earlier the evidence that there may be separate hungers for protein and carbohydrate. Is fat intake also regulated separately, by a system sensitive to fat constituents in the blood? Perhaps so.

Finally, let's look at a human feeding study that came *directly* from animal research.

When food enters the intestine, it causes the release into the blood of a hormone called *cholecystokinin,* mercifully abbreviated *CCK.* This hormone normally is liberated into the blood by the passage of food through the duodenum; but when injected by itself, it inhibits sham-feeding in the rat, just as infusion of food into the duodenum does. Perhaps CCK is a step in the chain of events by which food in the intestine shuts down feeding.

Once we know that, it is natural to ask: Would injection of this hormone inhibit hunger in other species, humans as well? It does.* Human beings ate less after CCK injection than after control injections, though they could not tell the difference between them. In particular, they reported no feelings of sickness or nausea—something hard to determine in animal research, but humans can tell us! Thus this hormone may play a role in the suppression of feeding, in humans as well as rats.

## Weight Regulation and Its Failures

So far we have focused on the short-term, meal-by-meal control of hunger and feeding. There is another regulatory system, however, that operates more slowly over much longer time periods.

Suppose you are an average woman, twenty years old. You can expect to gain about 11 kilograms—roughly 24 pounds—over the next forty years. That means that you are eating more than your energy needs require. How much more? If we do the arithmetic—so many calories in an average meal, so many calories expended in living and working, so many days—we find that you are eating more food than you need by an average of about 300 *milligrams* of food a day![31] (A milligram is very, very small.)

Obviously you don't adjust your meal-by-meal intake to your energy needs with anything like that kind of precision. Something must operate over a longer time, so that deficits or surfeits in intake are slowly and gently corrected to make long-term energy intake about equal to long-term energy expenditure.

*This is the study referred to in Chapter 1, pp. 17–18.
[31]Hervey, 1959.

That assumes that you are average. Not everyone is so fortunate. You surely know people who, despite continued efforts, weigh more than they should and more than they want. If obesity is severe, then its costs are also severe—to health, to social acceptance, and to one's acceptance of oneself.

Are obese people simply weak-willed and self-indulgent? It doesn't seem likely, for one simple reason—*most people avoid becoming obese without having to give the matter any thought.* They don't *need* "willpower" or to exercise "self control." They avoid getting fat without even trying. Why?

### Is Body Fat Regulated?

Let us see how a long-term weight-regulating system might operate (Figure 3-12A). We need a mechanism that keeps a kind of running total of the difference between energy intake and energy output. And we have tissue that keeps just such a running total. It is the *adipose tissue,* or fat stored in the body.

We mentioned earlier that as fuel in the blood gets low, glycogen is drawn upon to keep the cells supplied with fuel. This "checking account," however, is quickly depleted. Then overdrafts are covered by the breakdown of fat, which in turn is converted to usable fuel. Thus, the fat stores are our long-term savings account. If output exceeds intake for any length

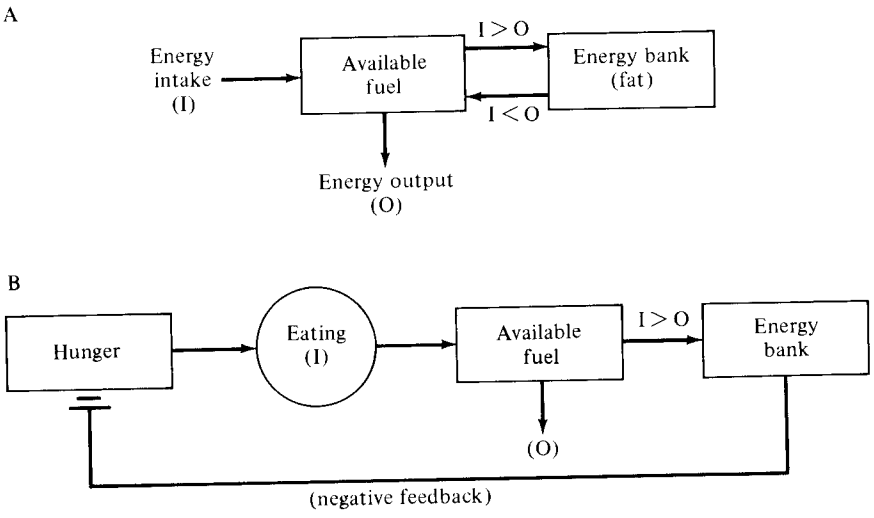



**Figure 3-12.**
(A) The relations between energy intake, energy output, and energy storage as fat. (B) Accumulation of fat in the energy bank may provide a signal, not yet identified, that inhibits hunger (or augments satiety; not shown). If so, it is part of a negative-feedback loop by which amount of fat in the body is regulated.

of time, fat will be lost. Conversely, if intake exceeds output for any length of time, the excess fuel will be saved in the fat stores.

But how does this help us to regulate weight? In 1950, Gordon Kennedy[32] suggested that it is not weight per se, but body fat, that is regulated. There may be a *feedback loop* such that as fat accumulates in the body, appetite is depressed so that meals become smaller, fewer, or both; and excess fat is lost as a result (Figure 3-12B).

In normal rats, this does happen. Rats can be made artificially fat by force-feeding. Then, after force-feeding is stopped, the rat (a creature not noted for willpower, by the way) goes on a diet. It reduces its meal size and frequency until the excess weight is lost over the next few days or weeks.[33]

Such a rat does continue to eat meals, even while dieting. The short-term cycle of hunger and satiety continues to cycle along. Therefore, the effect of excess fatness must be to modulate these short-term controls, re-setting them so that meals are slower to begin, quicker to end, or both, as Figure 3-12B shows.

Such a feedback loop would keep the body's fat stores from getting too high (barring force-feeding or other interferences with the system). Another feedback loop could keep them from getting too low; as fat is lost, meals may be biased in the direction of greater size and/or frequency. Over the long term, then, gains or losses in weight (i.e., fat) would be corrected and weight would be held nearly constant, just as a furnace and an air conditioner, controlled by a thermostat, would hold room temperature constant in summer and winter.

### Obesity: An Elevated Setting?

That analogy suggests an interesting possibility. Might the obese person be one whose fat thermostat, or body-weight *set point,* is set too high? Some researchers think so, and the idea has much to recommend it.

People who have lost weight have a distressing tendency to gain it back again, and to return to about the weights they were at before the loss occurred. This tendency is shown dramatically in Figure 3-13. Here the therapeutic book was thrown at a series of patients: behavior modification aimed at eating habits, an exercise program, and appetite-suppressant drugs besides. The patients lost, on average, about 15 kg (roughly 30 pounds); but 12 months after treatment stopped, the *average* patient had gained back about two thirds of the weight lost. This return toward the starting point does suggest a negative-feedback system; and if the starting point is too high, then such a system must be "set" to keep it there.

The notion of an elevated set point for body weight also may help us understand the other reactions of the body, besides loss of fat, that accompany dieting. The body seems to resist loss of weight by every means

[32]Kennedy, 1950.
[33]Cohn and Joseph, 1962; Hoebel and Teitelbaum, 1966.

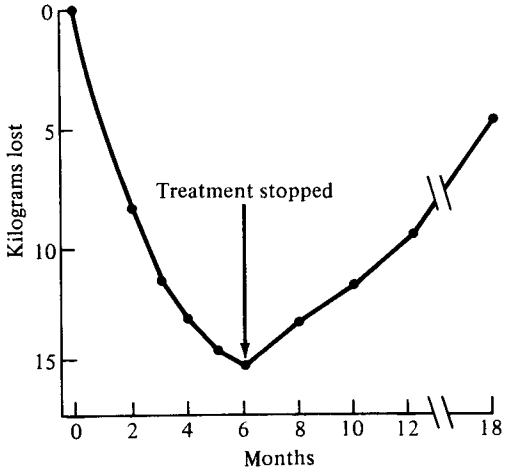



**Figure 3-13.**
Patients receiving multiple treatments for obesity lost weight during treatment, but, on average, regained most of it within 12 months. (From Brownell, 1982.)

available, just as it resists a drop in blood sugar in multiple ways. Metabolic rate goes down in dieters, so that less energy is wasted and less fat is broken down.[34] In other words, energy output as well as intake is affected. In addition, the chemical machinery that pulls fat constituents out of the blood, and converts them into stored fat molecules, goes into high gear—and it stays that way in persistent dieters.[35] To lose weight, one must persist in the face of these treacheries by the very body one is trying to make healthier.

Further evidence for a body-weight set point comes from the converse case. If people find it hard to take off weight and keep it off, they also find it hard to put on weight and keep it on! Normal-weight volunteer subjects have forced themselves to overeat, day after day. By doing so, they gain weight; but they gain less and less weight as time goes on, even though the high caloric intake is maintained. One reason is that in this case, energy output rises. More heat is generated, so that many of the excess calories are simply wasted rather than being stored as fat. The body resists gaining weight, just as fiercely as it resists losing it.[36]

Taken together, these findings may explain a paradoxical but vitally important fact: *Obese people simply do not overeat that much.*[37] Many studies, in both laboratory and natural settings, have found them not to overeat at all. Because the body has other means of resisting fat loss, it

[34]Garrow, 1978.
[35]Schwartz and Brunzell, 1981.
[36]For review see Keesey, 1980.
[37]See Spitzer and Rodin, 1981.

may take surprisingly few calories to keep a fat person fat.[38] And as a result, even Spartan restriction of food intake may have effects on weight that are discouragingly small.

Excess fat, then, may be very hard to take off and keep off once it is gained. But why is it gained in the first place? If a body-weight set point is set too high, why? There are many theories about that. They could all be correct, for there is no reason whatever to think that a single cause underlies all cases of obesity.

One commentator has divided obesities into two kinds.[39] In one, overeating comes first, in response to *non-regulatory* influences of the kind we'll consider later. For example, some obese individuals may be overresponsive to good-tasting foods (pp. 86–87) or may eat in response to arousal or stress (Chapter 6). In the second type, the tendency to obesity may actually precede the overeating. Metabolic abnormalities, for instance, could drive fuels out of the blood and into the fat cells, which turn them into more fat. That leaves the other cells starved for fuel, and chronic hunger could result.

If this is true, we just might have to turn our thinking right around. Such people may not get fat because they overeat. They may overeat because they are getting fat!

That doesn't mean that obese people are doomed to obesity forever. People *do* take off weight and keep it off—perhaps more people than we think, because the ones who do it by themselves may not come to clinicians' attention.[40] It may mean, however, that for some people the struggle against obesity will be a lifelong battle, whereas others stay lean without even trying.

## STIMULUS FACTORS IN INGESTIVE BEHAVIOR

Thus far, we have considered the role of influences from *inside* the body on hunger and thirst. However, these motives are powerfully affected by *external* factors as well.

Think about the last beverage you drank. A cup of coffee, maybe, or a Coke, or a mug of beer. Were you really thirsty? Or was it that you wanted the *taste* of what you drank? How much of our eating and drinking is really *pulled* to the commodity by its sensory properties, rather than *pushed* by a state of need or drive? Quite a bit.

### Taste-evoked Drinking

Consider a rat sitting placidly in its cage, with food and water freely available. We clip a bottle of sweet fluid to the cage—it may be sugar, which is nutritive, or saccharin, which is not—and the rat will rush to the

[38]See Rodin, 1981.
[39]Stricker, 1978.
[40]Schachter, 1982.

bottle and drink phenomenal amounts, as much as two or three times his total blood volume.[41]

These rats are *not* hungry or thirsty in the physiological sense. They have access to food and water all the time. Yet at a time when feeding and drinking would otherwise be minimal, the presence of a sweet solution evokes a huge bout of ingestion that *would not occur if the tasty solution were not there.* The effect occurs with a variety of sweet solutions.

Now a special treat of this kind does not trigger just lapping behavior. It evokes a *motivational state.* We know that because the behavior itself is not fixed. Non-deprived rats will run down a runway to get a few drops of sweetened water.[42] Or they will work vigorously at lever-pressing for a few laps of glucose solution. In short, the rat will take *whatever action is available* to obtain the sweet fluid. And that gives us our familiar multiple-output system, or motivational state, expressed by any of several responses—running, bar-pressing, or lapping.

Clearly, *not all motivational states are evoked by drives.* The word *drive* connotes a physiological condition, a push from within. But here, we have a clear case of a motivational state *evoked by a stimulus from outside the body*—not a push from within, but a pull from without.*

## Dietary Obesity

The prevalence of obesity in modern societies may very well depend in part on the availability of rich, high-calorie, *tasty* foods. Of course there are many factors that promote and maintain obesity in humans, and it is hard to prove that what foods are available is really an important influence. So, once again, we turn to the rat, where at least we can see what effects the properties of food *can* have, in the absence of all these complications.

Normal rats, fed normal rat food, regulate their weights very well, gaining weight through their adult lives only very slowly. But offer them special treats, and a perfectly normal rat will overeat and get fat. High-fat diets are especially good stimuli for this dietary obesity,[43] but a sweet, high-carbohydrate eggnog will also do.[44] We could look at this result as a kind of long-term analogue of the taste-evoked drinking phenomenon. As the sweet taste triggers a single meal that would not occur without it, so a special diet can trigger a persisting elevation in daily caloric intake that would not occur without it.

An especially worrisome case of this phenomenon has been reported

[41]See Ernits and Corbit, 1973.
[42]Young and Shuford, 1954.
*Some writers refer to such a pull from without as *incentive motivation,* to distinguish these cases from *drives.*
[43]Corbit and Stellar, 1964.
[44]Keesey, 1980.

in rats.[45] The animals were maintained cafeteria style on a variety of common, commercially available supermarket foods: chocolate chip cookies, cheese, bananas, salami, milk chocolate, peanut butter . . . These rats gained nearly three times as much weight in 2 months as their rat-food-fed controls! Inasmuch as these are not rat foods but people foods, no farther away than the grocery store, one must wonder what pressure their ready availability exerts on the weight-regulating systems even of perfectly normal human beings in this society.

## The Effect of Variety

The supermarket diet that made rats fat offered not just one special treat, but a *variety* of special treats. That may be important in its own right.

To see this, consider the converse case. Few things are worse than having to eat the same food meal after meal, day after day—however nourishing and tasty it may be. We come to feel that we never want to *look* at another X—where X is whatever we have eaten again and again. This can have important clinical implications, too. It is not hard to get hospitalized obese patients to lose weight on a bland liquid diet offered as their only food. The problem is that patients refuse to stick with such a regimen, simply because it is killingly monotonous.[46]

The powerful influence of variety has been demonstrated by direct experiment in both rats and humans. In one study, hungry rats were offered either a series of four different foods, each for 30 minutes; or a single one of the foods for the full 2 hours. They ate about 30 percent more when the varied menu was offered, even though the foods were about the same in caloric density. Humans (nursing students) also ate more when offered a variety of sandwiches than when offered only one.[47]

Later explorations[48] showed the effects of more restricted variations in the sensory properties of foods. Even varying the *shape* of the food had some effect: Subjects ate more cooked pasta if it was offered sometimes as spaghetti, sometimes shaped as half hoops, and sometimes shaped as bow ties. The three of course were nutritionally identical.

In short, simply changing the stimulus properties of the food can keep ingestion going longer. This effect may have evolved for good reason. It may be one of the ways in which an animal, satiated for one kind of food, can still eat another kind and thus get the various nutrients it needs (pp. 76–77). But, like the effect of a good taste, it could work against the precise regulation of ingestion. Both good taste and variety can encourage intake in excess of need, during a single meal, or day after day.

[45]Sclafani and Springer, 1976.
[46]Hashim and van Itallie, 1965.
[47]Rolls, 1979.
[48]Rolls, Rowe, and Rolls, 1982.

## Obesity and the "Externality" Controversy

This leads us to another idea. Earlier, we emphasized the role of *internal* signals in maintaining homeostasis—permitting regulation of food and water intake, and body weight. Now we are discussing motivational influences that arise from *outside* the body and, therefore, *are not responsive to its needs.* These sources of motivation may not aid in the maintenance of homeostasis. Indeed, they may actively threaten it.

Psychologist Stanley Schachter[49] developed this idea into an influential theory of obesity. Perhaps feeding in normal-weight people is controlled primarily by internal (homeostatic) factors, but feeding in the obese is more responsive to external stimulus factors. Such responsiveness to external factors is called **externality**.

If so, then in the environment we live in, the profusion of external stimuli that say "Eat, eat!" might play an important role in producing and maintaining obesity. Schachter backed up that idea with an ingenious series of experiments.

For example, Schachter and his colleagues[50] compared the effect of a full stomach on amount eaten in normal-weight and obese human subjects. Within each weight group, some subjects, but not others, were fed a roast-beef sandwich lunch. After that, they were allowed to eat as many crackers of varying flavors as they wished. The experiment was disguised as a taste-rating task; that is, the subjects were told that they were to judge the quality of the crackers. This was done so that the subjects would not be self-conscious about how much they ate. But in fact, that is what the experimenters were interested in—how many crackers would each subject eat?

The results are shown in Figure 3-14. As we might expect, the normal-weight subjects ate less if their stomachs were full. But the obese subjects did not. The internal state of their bodies (empty, or full of roast beef) did not affect their intake at all.

Just as the obese are less responsive to internal cues, so, some found, they are more than normally responsive to external ones. One experimenter[51] found that obese subjects consumed more of a good-tasting milkshake than subjects of normal weight; but when the milkshake was made slightly bitter, obese subjects actually drank *less* than those of normal weight.

Schachter's idea stimulated a large and controversial research literature.[52] As it now stands, the evidence suggests that externality may play some role in some cases of human obesity. But it cannot be the whole story. Externality is found in both lean and obese people, and not all obese people show it. Besides, as we saw earlier, there are many things

[49]Schachter, 1971.
[50]Schachter, Goldman, and Gordon, 1968.
[51]Nisbett, 1968.
[52]See Rodin, 1981; Polivy and Herman, 1983.

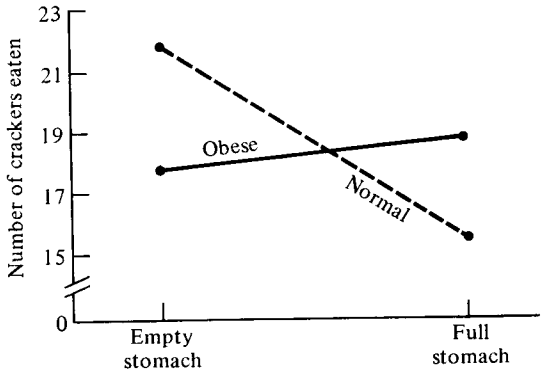



**Figure 3-14.**
A full stomach inhibits food intake in normal-weight subjects, but not in obese ones. (From Schachter, 1971.)

besides *overeating* that can promote and maintain obesity. And at best, overeating is all that the theory can account for.

We cannot go further into these complexities here. For our purposes, the important things to see are that (1) external stimuli as well as internal states can trigger motivational states; (2) the effects of these external stimuli may actually pose a threat to homeostasis; and (3) individuals may differ in sensitivity to them.

### A Look Backward: External Influences on Feeding

We have seen several ways in which ingestion can be initiated and maintained from the outside, not the inside. To see one way of thinking about this, look back at Figure 3-11B. We show internal drive factors as priming or, as we say, **potentiating** the responsiveness to external stimuli.[53] But what if the stimuli need no potentiation? Very powerful stimuli might trigger ingestion even without input from motivational states. Perhaps taste-evoked drinking and dietary obesity are such cases.

Conversely, simple exposure, producing boredom or perhaps **habituation** (see pp. 216–217), can reduce the effectiveness of stimuli. The variety effect may reflect such a process—a process which, once again, is quite independent of the state of the body.

## INTERACTION OF INTERNAL AND EXTERNAL INFLUENCES

To this point, we have divided factors affecting hunger and thirst into external and internal ones, in an either/or way. But they are not just two

[53]Gallistel, 1980.

independent influences; they affect each other. In this section, we look first at some of the influences of the stimulus on the internal mechanisms that in turn affect what we do. Then we will consider the converse case: Internal states of the body can affect how we respond to stimuli from outside.

## Stimulus Effects on Internal State: Can Food Make the Body Hungry?

External stimuli can evoke motivated behavior directly, as we have seen. However, they can also affect it indirectly by modifying the state of the body, which then in turn calls motivational systems into play.

In both animals and humans, the sweet taste alone can trigger the release of insulin into the blood. Insulin drives blood sugar level down, so that it is unavailable to the cells; and a drop in available sugar, we recall, can in turn promote eating and food-seeking.

In humans, just the sight and smell of a crackling, charcoal-broiled steak are sufficient to elicit insulin release, especially in "external" individuals.[54] Whether these effects are sufficient to influence feeding behavior in their own right is not clear, but some writers are beginning to wonder seriously whether food may actually increase hunger in just such ways.[55] Perhaps that is why it is easier to avoid the cake altogether than to have "just a bit."

## Internal Modulation of Responses to Stimuli

Input from the environment, then, can modify the body's internal states in ways that in turn affect what the person or animal does. Conversely, internal factors can affect how one responds to stimuli coming in from the outside world.

### The Identification of Commodities

The first such influence we have seen before. A thirsty rat responds to water; a hungry rat responds to food. It must identify these commodities, and it identifies them by their stimulus properties.

A rat deprived of food, but not of water, will drink very little water.[56] But put a bit of sugar or saccharin in the water, and the rat will drink with gusto; and the hungrier it is, the more sweetened water it will drink.[57] The sweet taste labels the commodity as food. It is as if the stimulus properties of the water say to the rat, "I am water; if you're thirsty, drink me," whereas the sweet taste says, "If you're hungry, drink me; I'm a source of nourishment." (In the case of saccharin that is a lie, but that is not the rat's fault.)

[54]Rodin, 1981.
[55]For review see Geiselman and Novin, 1982.
[56]Adolph, 1947.
[57]Mook and Cseh, 1981; Teitelbaum and Epstein, 1962.

These messages from the environment are received by systems primed, from inside the body, to react to them. It is as if the thirst general says to the lapping officer, "Be ready to lap if you encounter *these* stimuli"—ones that identify water. The hunger general says, "Be ready to lap if you encounter *those* stimuli"—ones that identify liquid food. The salt-hunger general says, "Be ready to lap if you encounter *the other* stimuli"—those that identify salt. Such priming of responses to specific stimuli would provide one way for the rat to identify specific needed commodities, and thus meet its multiple nutritional needs.*

### The Range of Effective Stimuli

Internal states also modify the *range* of stimuli to which the organism is responsive. If we get thirsty enough—say, after a few days in a lifeboat under a broiling sun—we will be willing to drink almost anything, even urine, or the sea water that actually dehydrates us further and will kill us all the sooner if no help arrives.

It is the same for food. When hunger is severe enough, people have eaten grass, or shoe leather, or each other.† In short, as the body's state of need becomes more and more severe, there is an *increase in the range of stimulus objects* that are treated as edible or drinkable.

The same thing can be seen under less drastic laboratory conditions. As we saw earlier, rats will drink sweet solutions even if they are not hungry. But as we lower the concentration of sweetener, we soon reach a point at which the sweetness is too weak to support drinking in a nondeprived rat. Make the rat hungry, though, and it will drink the same weak solution with avidity.[58]

This is what we would expect if states of hunger and thirst potentiate the responses to relevant stimuli. The more potentiation from inside, the *less* effective the external stimulus has to be to trigger ingestion.§

### The Pleasantness of Stimuli: Alliesthesia

*They surfeited with honey, and began*
*To loathe the taste of sweetness, whereof a little*
*More than a little*
*Is by much too much.*
—SHAKESPEARE, *Henry IV,* PART I: ACT III, SCENE 2

*Even that is too simple, as we will see later (pp. 305–307).

†A particularly dramatic case occurred in the winter of 1874. A certain Alferd Packer, a prospector, was trapped by snow in the mountains of Colorado. He survived by killing and eating his five companions. Legends quickly grew up around the antisocial ramifications of this action. The story is told that the judge, before passing sentence of death, said to Mr. Packer: "There were only seven Democrats in Hinsdale County, and you, you ----, you ate five of them." That part of the story, I'm afraid, is not true; but it *is* true that in recent years the students at a nearby university named a dining hall after Mr. Packer, and so his name lives on.

[58]Campbell, 1958; Mook and Cseh, 1981.

§For a different view, see Jacobs and Sharma, 1969.

Michel Cabanac, in 1971, reported that the nutritional status of the body affected the *pleasantness* of sweet solutions. When first allowed to taste a series of sugar solutions, human subjects rated them more pleasant as the sugar concentration increased. The sweeter the solution, the better it tasted. But after the subjects had swallowed appreciable amounts of solution, the ratings were reversed: The sweeter the solution, the worse it tasted.

This effect, which Cabanac called **alliesthesia**[59] (literally, "changed sensation"), was not just an effect of too much taste input. If the subjects simply rinsed their mouths with the solutions and spat them out again, there was no such effect. It required that the solutions enter the stomach. It is therefore an internal effect on the pleasantness of taste inputs.

Cabanac and others have suggested that *satiety* may be mediated by such a change in the pleasantness of food. Perhaps, as the stomach (or duodenum, or portal vein, or whatever) is stimulated by food, that internal stimulation is reported back to the brain to make the food taste less good.

Later, Cabanac and his colleagues[60] extended their work to the long-term regulation of body weight. In some parts of Africa, there still survives an ancient custom: Young women deliberately overeat and make themselves fat prior to their weddings. Taking advantage of this natural experiment, the investigators asked the women to rate the pleasantness of sugar solutions, before and after the voluntary weight gain. Sure enough: After gaining weight, the women rated the solutions less pleasant. Perhaps excess fat, like a stomach full of glucose, makes a sweet taste no longer a good taste.

The idea is attractive, but the experimental evidence is mixed. Some people have had trouble replicating Cabanac's findings.[61] Moreover, it is not clear that satiety is *necessarily* accompanied by diminished pleasantness of the food. Surely we can sometimes say, "It's delicious, but I really don't want any more," and mean it. Nor is it clear, if we think about it, that the good taste of food has much to do with keeping eating going in the usual course of events. C. S. Lewis reminds us: ". . . anyone who has watched gluttons shovelling down the most exquisite foods as if they did not know what they were eating, will admit that we can ignore even pleasure."[62]

On the other hand, there is another and quite different line of evidence that the sort of thing Cabanac described can occur. Experimenters have videotaped rats, close up, when the rats were drinking various solutions.[63] They find that rats make faces—and quite different faces depending on what they are offered to drink. Sweet solutions are accepted with protru-

[59]Cabanac, 1971.
[60]Fantino, Baigts, Cabanac, and Apfelbaum, 1983.
[61]See for example Stellar, 1977.
[62]Lewis, 1962, p. 93.
[63]Grill and Norgren, 1978.

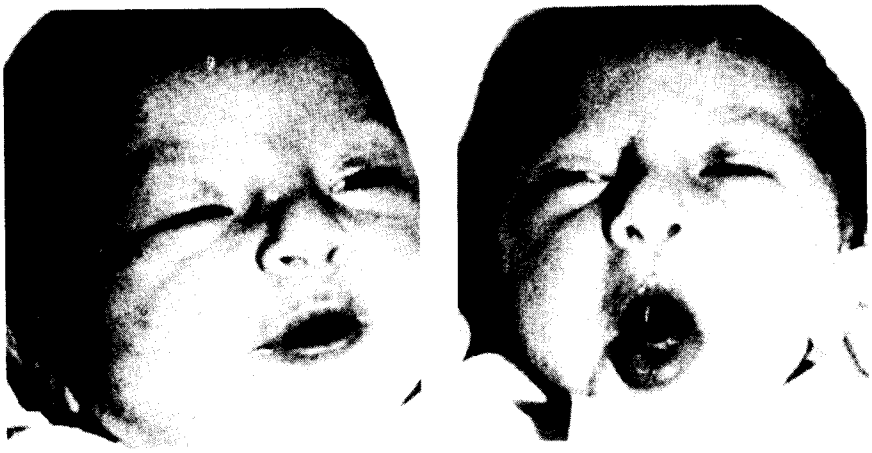


**Figure 3-15.**
Facial expressions in a newborn, when a sugar solution (left) or a bitter quinine solution (right) is applied to the tongue. Note protrusion of the tongue in response to sugar, and the wide gaping response to quinine, with turning of the head and clenching of the eyes. (From Steiner, 1977.)

sion and lateral movements of the tongue, as if seeking further contact. Bitter solutions, or intensely salty ones—the kind we rate "unpleasant"—are reacted to instead with a peculiar kind of yawn or gape. Human infants make faces similar to the ones rats make; and, like rats, they greet nice and nasty tastes with very different expressions (Figure 3-15).

How does alliesthesia come in? It develops that the characteristic facial response to sweet solutions can be altered by manipulating internal state. If a rat's stomach has just been loaded with a large amount of glucose solution, then the taste of that solution evokes the facial expressions usually made to unpleasant tastes. It is as if glucose in the body changes the taste of sugar water from nice to nasty.

Of course this evidence is indirect. We do not really know what a rat finds nice or nasty (more on that in a minute). However, we can say this much: (1) Rats react in different ways to acceptable and unacceptable tastes, and (2) a fully fed rat treats a sweet taste as if it were unacceptable. Whether this mechanism plays an important role in normal satiety for a normally eaten meal, we are not yet sure.

## A Look Backward: Internal and External Controls

We have seen how stimuli can evoke motivated behavior, how they can affect the body's internal state, and how that internal state can affect responses to them. Can we bring these ideas together, and link them to our discussion of internal factors, negative feedback, and homeostasis?

We can try. But note well that the following attempt is *highly speculative;* it is not established truth by any means.

In Figure 3-16, we see two kinds of systems that lead to *commerce with the object*—which simply means finding food and eating it, finding water and drinking it, or the like. In Figure 3-16A, the pleasantness of the stimulus leads the organism to seek such commerce, as in taste-evoked ingestion and perhaps dietary obesity. That commerce, or its consequences inside the body, reduces the pleasantness of the stimulus (allies-thesia), and the system shuts down.

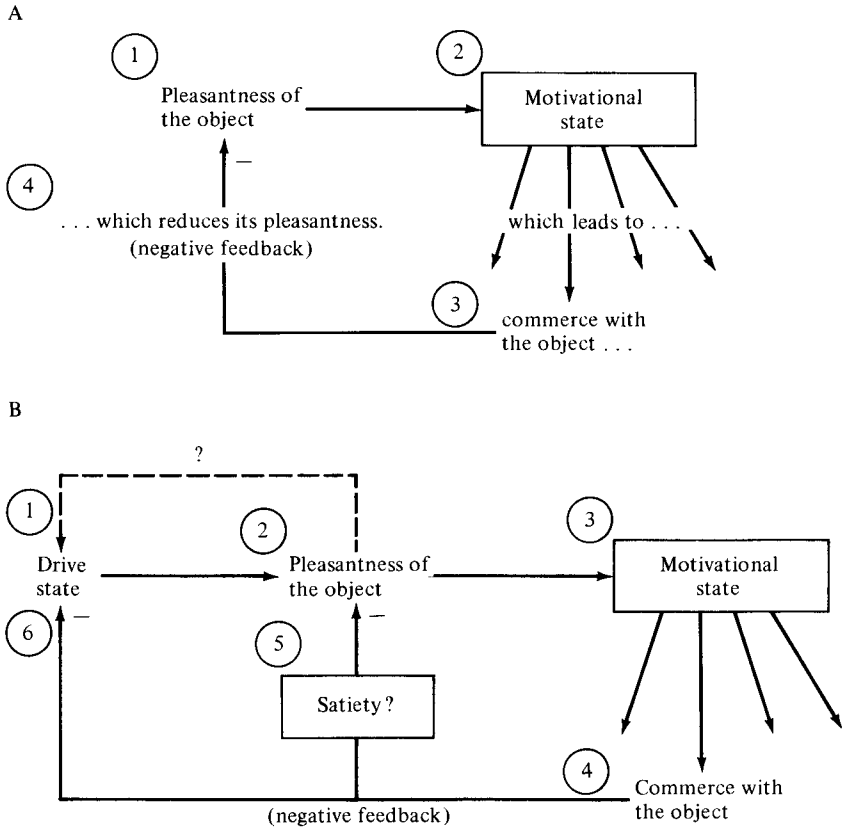



**Figure 3-16.**
How pleasantness might affect motivated behavior. In A, the object—tasty food or a sugar solution—is pleasant in its own right, and evokes a motivational state directed toward obtaining commerce with it. In B, the system is the same, except that an internal drive state produces the pleasantness. Alleviation of that state, or its inhibition or satiety, reduces pleasantness and the system shuts down.

In Figure 3-16B, the process begins not with the pleasantness of the object itself, but with a drive state that *makes* it pleasant. Such a state might extend the range of acceptable objects, or it might enhance respon-

siveness to all of them.* If the state is one of need, such as dehydration or a low level of nutrients in the blood, then commerce with the object would reduce the need and restore homeostasis, and again the system would shut down. Or the effects of that commerce could reduce the pleasantness of the object (alliesthesia again), even *before* homeostasis was restored. The inhibitory mechanisms of satiety might work that way.

Even a system like B, however, could be activated by stimuli. For example, perhaps some foods can make our bodies "need" more food (p. 90). This is shown by the dashed line with question mark in the figure.

Notice that both these speculative systems are negative-feedback loops. But *only B is a homeostatic system*—and then only if the dashed line is not in the picture. System A is not triggered by the body's needs. Therefore, it could lead a person or animal to drink more delicious fluid than the body requires. Or it could lead the person or animal to overeat and (if this happens often) get fat.

For that matter, there is no reason why individuals might not differ from one another as to which kind of system is operative. Schachter's internal/external division amounts to this suggestion: In some people (externals), feeding is controlled by an A system; in others (internals), by a B system.

Now all this speculation does tie some threads together, and it makes a good story. But it *is* speculative; and some of the links in the chain of evidence—for example, the alliesthesia experiments in humans—are weaker than we would like.

Then too, in giving *pleasantness* and *unpleasantness* a central role in our overview, we are taking on a difficult problem of method—a problem so difficult that it is worth our stepping back to look at it specifically.

## A Note of Caution: The Problem of Self-Report

At several points in this chapter I have made use of data that purport to show us how subjects *feel* about stimuli. How pleasant is this taste? Are you hungry? Do you feel nauseous? And so on.

These are descriptions by the subject of his or her own consciousness. They are precisely the kind of data that John B. Watson would have thrown out of court (see Chapter 2). And yet they are the kind of data that only a human can provide, and they offer information we would like to have. The question is how to interpret them.

Consider: If a rat tastes a solution and makes a wide-mouthed gape, that is an objective *fact* for all to see. But when we call it a "nasty face," we are already going beyond the data. Nice faces and nasty faces may just be external indicators of an intention to accept or reject; we do not know what, if anything, the rat feels. Humans, we say, can tell us. But then, we

*Or of course these two possibilities might amount to the same thing. If the responsiveness to *all* potential edibles were enhanced, then, all else equal, the number of them that meet our standards of good enough to eat would necessarily increase.

don't really know what a fellow human feels, either—even if she tells us. If a person *says,* "This tastes pleasant," that too is a fact. But what does it mean? Do you and I mean the same thing by "pleasant," or even "sweet"? Is your red the same as my red?

The question is: Do we move on from the facts to make inferences about our subjects' *feelings* of pleasure or displeasure, hunger or satiety? A mediationist may be willing to do so. A behaviorist will remind us, with some justice, that we really don't know what we're talking about when we do.

To see the problem clearly, consider that our objective measures may go in one direction, subjective reports in another. Quite a few investigators have asked human subjects to rate how hungry they were, before eating a meal—and have found no correlation at all between how hungry the subjects said they were, and how much they ate.[64]

Now there are reasons why that might be so. Perhaps as eating progresses, satiety develops at a rate that is independent of how hungry we are when the meal begins. But there are other possibilities too:

1. We might not really *know* how hungry we are. Surely we have all said at some time, "Gosh, look how much I ate. I must have been hungrier than I thought."
2. Maybe our *feelings* of hunger arise from some internal signals that really have nothing to do with the signals that control the starting and stopping of feeding behavior.
3. Maybe "hunger" means different things to different people. Are my hunger sensations the same as yours?

This example brings the problem into focus, because objective and subjective measures do not agree. But the same problems arise even when they *do* agree. In the alliesthesia experiments, people refrained from eating any more of the food offered, and they said that it didn't taste good any more. That seems to make sense, but does it? Are the subjects confusing how good the food tastes with how much they want to eat it? Are they confusing a bad taste with the onset of nausea? Are they reporting what they think *ought to happen* to the pleasantness of tastes? And so on.[65]

It would be wonderful if it were clear that in *these* cases we can take subjects' self-reports at face value, and in *those* cases we cannot. But no such rules exist.

Perhaps the safest way to look at it is to realize that self-report data are just that—data, to be considered along with others. We take subjective data when we think they may tell us something; they are not to be dismissed automatically. *But they have no special privileged status either.* They are not automatically the last word. We should remember their

[64]Reviewed by Spitzer and Rodin, 1981.
[65]Stellar, 1977.

ambiguities, and keep a bottle of salt tablets handy when we make inferences about our subjects' feelings on the basis of them.

That means in turn that all our knowledge about the role of sensations, feelings, and pleasure in behavior must have that salty flavor. For it rests squarely upon just such inferences, and upon nothing else.

## A LOOK FORWARD: THE ROLE OF CULTURE

Before ending this chapter, we should note that our discussion of hunger and thirst has still only stroked the surface. If we look again at the human case, we find at every step that our feeding and drinking fit into the context of what the society we live in—our culture—has taught us to do, or to expect, or to value. Consider:

1. In a hunting society, whether or not we eat will depend on the hunting skills we have learned—*or on the skills of our fellows,* who will share their food with us. Food use articulates with concepts of division of labor, mutual aid, and sharing that are transmitted from one generation to the next within a society.
2. Cultures constrain our feeding and drinking as to what is permitted under what circumstances. We don't munch potato chips at a funeral, however hungry we may feel. In some societies, people have starved rather than accept a food that is taboo.
3. What is food, anyway? In our society, most of us would not consider ants as food. Moreover, we don't just refrain from eating ants because they are forbidden; many of us would be revolted by the very idea of ants as food. Yet in some other societies, ants are considered a great delicacy and are eaten with gusto.

All this means that to understand even feeding and drinking as they occur in nature, we need to understand how we make decisions and set priorities, how the environment takes control of our actions, how we make a society's values our own, and other matters that later chapters will address. In a word, regulatory biology is not the whole story—even for biological drives.

## SUMMARY

The concept of drive arose from the observations (1) that behavior varies even in a constant situation and (2) that animals will make various responses, including arbitrary learned ones, to achieve a goal. That implies a hierarchical organization of behavior, so that a single motivational state can call into play any of a number of specific behaviors. When such a motivational state is evoked by a change or condition inside the body, we call it a *drive.*

Drives link behavior to *homeostasis,* or the regulation of physiological

variables within the narrow limits the body requires. A drop in body temperature can be corrected by physiological means (increased metabolic rate) or by behavioral means (building a fire). A drop in blood sugar can be corrected by physiological means (release of stored sugar into the blood) or by behavioral means (eating). All these are examples of *negative feedback,* in which a system operates to reduce or remove the input—low temperature, low blood sugar—that produced the operation. Homeostasis is maintained by negative-feedback loops that correct deviations from optimal values.

The early local-sign theories thought of hunger as an unpleasant sensation from the stomach, and thirst as unpleasant dryness of the mouth. However, these theories tell at most only part of the story; other inputs must exist as well.

There are two independent stimuli for thirst: reduction of fluid inside the cells, and reduction of fluid outside the cells. Either can occur without the other, or both can occur and add their effects. Thus thirst, a multiple-output motivational state, is also a multiple-input system called into play by different stimuli at different times. Inhibition of thirst, or *satiety,* is also a multiple-input system; it receives some input from the mouth and the stomach, though hydration of the cells is required to limit intake to normal levels.

A drop in the availability of glucose (blood sugar) to the cells may trigger hunger, but this probably is only one of many stimuli that can do so. Moreover, there may be several hungers rather than only one. Protein intake is regulated separately from carbohydrate intake, and sodium is regulated separately again. Satiety for hunger is another multiple-input system; stimuli from the mouth, the stomach, the liver, and the intestine can act to limit feeding. In humans, blood-borne fat constituents can also suppress hunger. However, just as there are multiple hungers, so there may be multiple satieties; a rat can be satiated for one food, but hungry for another, at the same time.

Feeding and drinking may use the same responses, as when a rat laps a liquid diet when hungry, or laps water when thirsty. The same lower-level movements may be called into play by different higher levels at different times. Such a *lattice hierarchy* suggests that the effect of internal states is to prime, or *potentiate,* ingestive responses to specific external stimuli.

The short-term cycle of hunger and satiety is superimposed on a longer-term regulatory system that holds body weight remarkably constant over long periods. This may mean that the amount of fat in the body is regulated. Obesity might result from an elevated set point for body fat, so that the "normal" weight, which the body regulates, is set too high. The body does defend its normal weight vigorously; it is difficult to gain or to lose very much weight, for adjustments in body chemistry and metabolic rate oppose these attempts. As a result, overeating may have little to do with what we call excess weight.

Besides being *pushed from within* by physiological needs, ingestion may be *pulled from without* by stimulus factors. A sweet taste can evoke ingestion. Access to tasty and varied foods can make even normal rats fat. Even simple variety can increase intake, in both rats and humans. Some writers have suggested that obese people may be more affected than those of "normal" weight by such external controls. But the idea is controversial, and at best it could account only for overeating, whereas obesity is maintained largely by physiological defense against weight loss.

Internal and external influences interact with each other. Food-related stimuli may trigger internal changes that in turn promote feeding. Conversely, internal state affects how external stimuli are responded to. Thirst makes us responsive to water; salt deficiency makes us responsive to the salt taste. Hunger may greatly extend the range of commodities we are willing to eat. Short-term satiety or long-term excess weight gain may make food taste less good, to humans and even to rats as judged by the faces they make. This effect is called *alliesthesia.*

We might conceive of food-seeking behavior as responsive to the attractiveness of food. That attractiveness could arise either because the food is attractive in itself, or because internal states of need enhance its attractiveness. In the former case we would have taste-evoked ingestion; in the latter, homeostatic control of ingestion. Such theorizing, however, depends on self-reports of pleasure or displeasure which are not always easy to interpret.