6

FEEDING REGULATION: A MODEL MOTIVATIONAL SYSTEM

The operant behavior of higher animals depends on their motivation, the value of the reward or punishment. If we ignore for the moment the problem of competition among motivational systems (hunger vs. sex, vs. thirst, etc.) there are two aspects to motivation: "How hungry am I?" and "How hard should I work to get food?" The answer to the first question depends entirely on the animal's internal state; it is a *regulatory* question. But the answer to the "How hard should I work?" question depends both on the animal's state *and* on the opportunities offered by the environment: It's worth working hard for food if much food is to be got. It's not worth working hard if the probability of payoff is low. This aspect of motivation is known as *incentive*.

This chapter deals with regulation and incentive. I take feeding as a typical motivational system and show how taste, body weight, and even drugs and brain lesions, combine to affect how much rats will eat and how hard they will work for food. The aim of the discussion is to dissect a single motivational system, and show how a few relatively simple principles underlie diverse effects. The next chapter deals with interactions among motivational systems.

REINFORCEMENT AND HOMEOSTASIS

Animals must eat to live and, on an operant food schedule, must work to eat. It would be astonishing, therefore, if schedule performance had nothing to do with the regulation of nutritional balance.[1] The molar (as opposed to molecular) properties of behavior on reinforcement schedules — for example, the average rate of responding over a period of several minutes — are intimately related to energy regulation.

Eating can be looked at from two points of view: diet selection and energy balance. Diet selection deals with the mechanisms that allow omnivores such as rats, dogs, and people under normal circumstances to pick out a diet not deficient in essential vitamins or trace minerals. Energy balance deals with how much animals eat in relation to their energy expenditures: If they eat more than they expend, their body weight goes up; if they eat less, their weight goes down. Performance on simple reinforcement schedules relates most obviously to energy balance. Performance on more complex procedures involving choice between different foods also brings in mechanisms of diet selection. I deal here with energy balance; later chapters say something about diet selection.

The great French physiologist Claude Bernard more than 100 years ago pointed out the importance to organisms of a constant *milieu interne* ("internal environment"): constant or narrowly varying core temperature, adequate cellular oxygen and energy supply, constant blood pH, and so on. In the 20th century, Walter Cannon coined the term *homeostasis* for the processes by which this internal constancy is maintained. Homeostatic mechanisms are of two kinds: internal, acting only within the body; and external, acting on the environment. Examples of internal mechanisms are vasoconstriction and vasodilation, to conserve or shed heat or direct blood flow to active organs; the release from the liver into the blood of glucose and ketone bodies, which maintain the nutritional needs of other organs; the release to the liver of fatty acids and glycerol from cellular fat stores, which provide the raw materials from which glucose and ketone bodies are obtained.

Operant behavior has several functions, but in all species a major and essential one is to serve as the external process of homeostasis. In simple animals such as *Paramecium* and *Stentor* all operant behavior can perhaps be explained in regulatory terms, as Jennings proposed. For these animals, which have limited sexual behavior and usually reproduce by fission, fitness is the same as growth and survival. In more intelligent, sexually reproducing animals, fitness requires more

than mere survival, and operant behavior subserves a wider range of social, sexual, communicative, and exploratory functions. Regulation is a prerequisite to everything else, however. One does not compose a sonnet, solve an equation, or even seek a mate, on an empty stomach.

If operant behavior is essential to energy regulation, then to understand reinforcement, which is the guide of operant behavior, we need to know something about how energy regulation works. Regulation implies feedback. Accordingly, in recent years much attention has been devoted to so-called set-point theories, which attempt to explain various aspects of eating, drinking, and even sexual behavior, in terms of feedback loops guided by internal reference levels.

A key feature in all such accounts is the critical variable with respect to which behavior is regulated. In the study of hunger, the spotlight has historically been on physiological measures such as body-fat stores, blood glucose, or free fatty acids and the like. "Lipostats" or "glucostats," sensitive to the levels of these substances, were postulated, and deviations from optimal levels were hypothesized to act as the feedback signals that urge the organism into action, to seek food. Diligent search for these various "-stats" has failed to find them, however. Perhaps more important, no single physiological measure has been found adequate to predict the full range of behavior related to eating. Quite apart from the complications associated with specific hungers for particular dietary ingredients such as salt or vitamin B, there appear to be serious difficulties in the way of a simple feedback model of eating with a single physiological variable as its set point.[2]

Alfred Hitchcock used to refer to the thing everybody is looking for in a mystery story as the "McGuffin" — good McGuffins are the Ark of the Covenant, the secret formula, Harry's corpse, and so on. It seems that there is no physiological McGuffin for feeding. Failure to find one has led some to urge the abandonment of the set-point idea. This suggestion misses the point of theoretical models in general, and feedback models in particular. It is an instance of the dangers (discussed in Chapter 3) of interpreting feedback diagrams literally in terms of neural structures. The equations generated by a set-point model may provide a useful description of a system such as a capacitor even though no physical entity corresponds to any of the elements on the diagram, including the comparator and set point. All sorts of expedients, including block diagrams, can be helpful in arriving at a useful description of a system. But these are to thought as scaffolding to a building, and can often be discarded once the objective — a working model — is attained. Any theoretical account contains nonessential features, as well as ingredients that are critical — not that it is ever easy to tell the difference. The block diagrams in feedback accounts are often superfluous.

There are, in fact, good reasons why operant behavior should not be tightly determined by physiological variables, even if maintaining the constancy of the internal environment is, in an evolutionary sense, the function of the behavior. As a familiar analogy, consider the strategy of a traveler driving across country who must keep enough gas in the tank to carry him where he needs to go. Obviously he will not wait until the fuel needle hovers on empty before looking for more. Nor will he pass up a bargain, even if his tank is still half full. His decision to look for gas will be determined by what he knows, has learned, or guesses, of the typical spacing between gas stations along his route, as well as the *variance* in spacing. If stations are far apart, or are spaced unpredictably, he is likely to seize any opportunity, even if the price is high or the mixture not quite right. Conversely, if stations are close together and predictable, he is likely to look for a good price and let his tank run down low.

The general point is that an adaptive organism will usually be guided in such matters by the best predictors available. Deciduous trees shed their leaves because the benefit from weak winter sunlight is outweighed by the dangers of frost. But the tree does not wait until the first frost to begin to shed. Instead it responds to a weighted average of temperature and day length as a better and safer predictor than the first frost. In arboreal evolution we must presume that trees that waited for frost were more often damaged than those that shed at the appropriate time of year, even if they

thereby passed up a few warm and sunny days in some years. Tissue need is the "bottom line" for food-motivated behavior, but only a loser waits until it's starving to look for food.

The distribution of gas stations for the traveler is analogous to the distribution of meal availability for an organism. The evolutionary history of each species provides it with some information on this distribution and, presumably, provides each with a rough clock or clocks that partially determine the spacing between meals and the times of day when eating is likely. The period of the intermeal clock will depend both on the expected spacing of food availability, and on the size of the gas tank; that is, the internal food stores in fat, stomach, intestine, and cheek pouch. Many species also have external stores of hoarded food that can extend the time over which they can survive without fresh food sources. The size of internal stores will be constrained by factors such as predator-avoidance that may limit the animal's size or the proportion of its body weight devoted to fat versus muscle and other non-storage tissues. The size of external stores is limited by the availability of free time for foraging and the pattern of fluctuations in available food density.

In "simple" animals, the temporal pattern of food seeking is presumably determined almost entirely by ancestral information; that is, by the seasonal, daily, and hourly regularities in the availability of food encountered by successful ancestors. In "higher" animals, in addition, discrimination learning allows each individual to take some advantage of the peculiarities of its own particular environment to learn the times or places when food is more or less likely to be available. In all species there is likely to be a preferred tempo of eating — of meal spacing — attuned to the animal's storage capacity, size, and metabolic expenditure.

Taste (food palatability) is like the price of gas in the automotive analogy. A satiated animal can be induced to eat by a tasty food, just as most motorists will stop to tank up during a "gas war," whether they need more fuel or not. If the price is low enough, additional containers may be found and filled. So also, normal animals will become obese if given ad libitum access to a highly palatable diet.[3]

Overriding all these local considerations is the veto power exercised by tissue needs. The longer the time period we are considering, the more closely the animal's rate of eating, in calories per unit time, must conform to the minimum needed to sustain life.

Organisms are thus designed by their evolution to cope with an uncertain food supply that nevertheless has predictable spatiotemporal features. The level of fat stores, hence body weight, is presumably determined by the maximum periods of deprivation that can be anticipated. The frequency with which the animal seeks food is presumably jointly determined by the current level of fat stores and the clocks that determine the initiation of feeding episodes. These clocks are themselves determined by the temporal distribution (current and ancestral) of food availability and the overall metabolic needs of the animal.

There are adequate functional reasons, therefore, why eating in most species is unlikely to be determined by any single internal or external factor. Nevertheless, despite the probable complexity of the system, it is not necessary either to despair, or to give up the possibility of a behavioral analysis until the physiologists have mapped every internal feedback loop. There are four factors likely to be important no matter what the physiological details, namely, *body weight* (the size of the energy store), *activity level* (energy expenditure), *eating frequency* (energy income), and *taste* (the evolutionary predictor of food quality). I now discuss a simple model of how these four factors combine to guide operant behavior.

OBESITY AND SCHEDULE PERFORMANCE: A STATIC ANALYSIS[1]

It is rare to find something interesting to both science and fashion, but obesity is just such a topic. The study of obesity, like research on cancer, death rays, and the internal combustion engine, is a

[1] A dynamic version of this model for feeding is presented in Staddon & Zanutto (1997); see also Staddon (2001b), Chapters 7-9.

topic that needs no defense before the general public. Its interest is palpable, its potential benefits large. In matters of design, "less" may or may not be "more," as the Bauhaus decreed, but in fashion there is no room for doubt: Thin is in and fat is out.

Experimental studies of obesity provide a convenient arena where the interactions among body weight, rate of eating, activity level, and taste have been extensively explored, at least in rats. More limited information is available on other species, including people, but the broad conclusions that can be drawn probably apply to many omnivores.

Insight into the workings of a system can often be gained from the ways in which it breaks down (its *failure modes,* in engineering jargon). Think of the ways that a TV set can fail. Typical picture defects include various forms of "tearing" and loss of frame hold, impairment of video but not sound, and the reverse, changes in color balance attributable to loss of a single color input, and so on. Even without prior knowledge of the system, or access to its innards, one could nevertheless deduce such things as the separate coding of audio and video channels, the three-color picture code, the line-by-line raster scan, and frame-by- frame synchronization. Experimentally induced aberrations in the normal eating pattern provide similar opportunities to understand the regulation of energy balance.

Animals that eat either less, *aphagia,* or more, *hyperphagia,* than normal can be produced either genetically, or by means of appropriate brain or other lesions. Hyperphagic rats that become chronically obese if given adequate food can be produced by gonadectomy or by electrolytic lesions or knife cuts in the ventromedial region of the hypothalamus (VMH).[4] Lesions in the lateral hypothalamus (LH) produce animals that eat less than normal and settle down to a lower than normal weight even when given unlimited access to food. Comparable results have been produced in cats and monkeys. Clinical data point to a similar role for the hypothalamus in man. Strains of rats and mice are available that are chronically obese. Animals of these different types behave differently when provided with especially tasty or unappetizing food, or when required to work for food on a schedule of reinforcement.

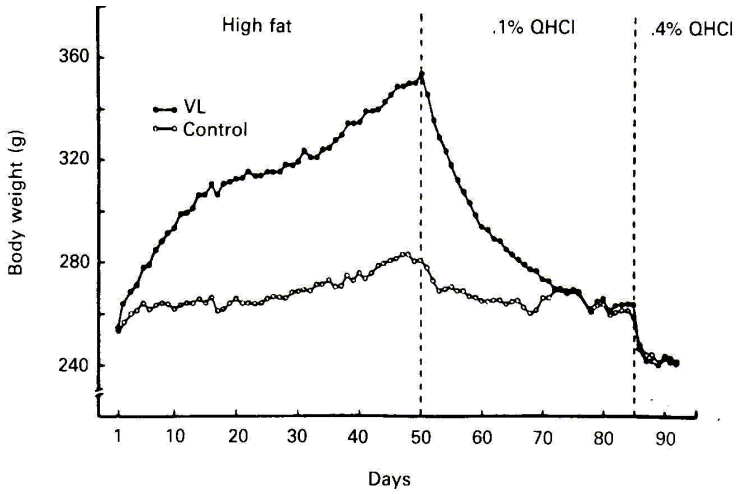

Figure 6.1. Mean body weights of hypothalamic hyperphagic (filled circles) and control rats (open circles) maintained on high-fat, .1% and .4% quinine-hydrochloride-adulterated diets. (From Sclafani, Springer, & Kluge, 1976.)

The most revealing effects are those produced by VMH lesions. In the mid- nineteenth century, chronic weight gain in some human patients was traced to tumors of the hypothalamic-pituitary complex. The modern study of this effect began with a series of classic studies by Hetherington and Ranson (1940, 1942), who showed that electrolytic lesions in rat VMH are sufficient to produce voracious postoperative eating and chronic obesity. Subsequent work has identified several reliable differences between VMH-lesioned and normal rats:[5]

1. VMH animals are "finicky," that is, they eat more of a palatable food than normals, but less of an unpalatable one. This is eventually reflected in their body weight, as illustrated in Figure 6.1, which shows body-weight changes in normal and VMH animals in response to successive adulterations of their diet with .1% and .4% quinine hydrochloride (QHCl) — a harmless, but bitter-tasting substance. The VMH rats eat much less of the adulterated food and show precipitous

declines in their body weights, whereas the normals are much less affected. VMH rats become extremely fat on palatable diets (reaching weights two to three times normal), but may show no weight gain or even a loss on an unpalatable diet. This excessive reaction to taste extends to other stimuli as well. VMH rats are more distractible and react more readily to extraneous stimuli than normals. Psychophysical tests show that the difference is one of reactivity, since VMH animals are not more sensitive (in the sense of having lower thresholds) than normals.

The sensitivity to taste shown by VMH animals is of the same type as that displayed by normal animals, but quantitatively greater. Even normal rats will become obese if given unlimited access to a varied and highly palatable diet, and normals sustain some loss of body weight if restricted to a severely adulterated diet. VMH animals show comparable, but much larger, effects.[6]

2. VMH animals are less able than normals to adjust their food intake to compensate for dilution of their food. For example, if 50% or 75% of the diet is nonnutritive kaolin, normal rats soon increase their total intake so that they show little permanent loss in body weight. VMH animals take much longer to show any compensatory response, and sustain a considerable loss below their table (obese) weight.[7]

3. VMH animals weakly defend their obese settling weight. For example, immediately after the operation, in the so-called dynamic phase, they eat voraciously and are relatively insensitive to taste. As their weight increases, however, they become more finicky and, eventually, maintain the higher weight "indefinitely on a hardly detectable excess of food intake" (Kennedy, 1950, p. 541). If the animals' weight is reduced by food deprivation, when ad lib. food is once again available they go through a second dynamic phase and recover their obese weight. A similar recovery occurs when the normal diet is restored following a period of weight loss induced by quinine adulteration. If obese VMH animals are made even fatter by force-feeding, they lose weight when normal feeding is resumed. If normal rats are made obese by force-feeding before receiving VMH lesions, they show no postoperative dynamic phase, but rather maintain the increased weight by an intake that is only slightly greater than normal.

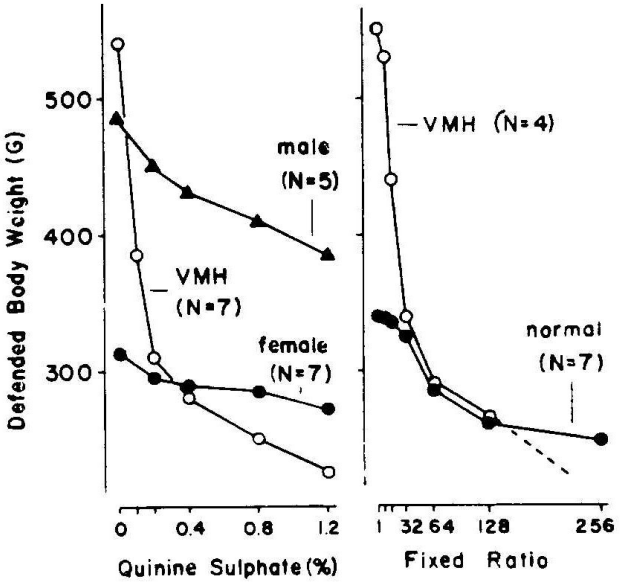

Figure 6.2. Mean stable body weights successively reached by rats exposed to diets adulterated with quinine sulphate (left panel), or available only on fixed-ratio schedules of reinforcement (right panel). VMH rats were females. Both food intake and body weights were stable for at least eight days for each schedule or diet plotted. (From Peck, 1976.)

4. VMH animals are less finicky, and will work harder for food, if their body weight is reduced, particularly if it is reduced below their preoperative weight. For example, the left panel of Figure 6.2 shows the stable weights attained by groups of female, normal and VMH rats on diets adulterated with increasing percentages of quinine sulphate. Initially, the VMH animals ate much less of the adulterated diet than normals, but as their weight dropped they became less sensitive to the adulteration. Over the range of concentrations from .2% to 1.2% the VMH animals defended their lowered weight about as well as the normals. A similar result can be seen on the far right of Figure 6. 1. The right-hand panel of Figure 6.2 shows the results of a similar experiment in which food was available only on a ratio schedule.

Once again, the VMH animals at first sustained a substantial weight loss, but then defended their lowered weight almost as well as the normals.

5. Obese VMH animals work much less hard for food than normal rats deprived of food for the same period of time. If food is available on a ratio schedule, VMH rats respond as rapidly as normals at low ratios, but fail to show compensatory increases in response rate at high ratios.

A regulatory model

The effects of VMH lesions on sensitivity to taste, diet dilution, and work requirements, as well as other effects to be described in a moment, can be explained by three simple feedback assumptions:

1. That *eating rate* is a regulated variable, defended by operant means.
2. That incentive factors (palatability) have an additive effect (positive or negative) on operant behavior related to eating.
3. That eating-rate regulation is inversely related to body weight; that is, animals tend to regulate better as their weight declines.

There is empirical evidence for each of these assumptions. For example, several studies have looked at the behavior of rats or guinea pigs responding for food or water on ratio schedules with different amounts of access to each reinforcer (Allison, Miller, & Wozny, 1979; Hirsch & Collier, 1974; Kelsey & Allison, 1976; and Timberlake & Allison, 1974). The results show that the relation between the amount of access to food or water (e.g., seconds of access, or grams, per hour of exposure to the procedure) and the rate of instrumental responding is largely independent of the size of the "meal" the animal receives at the completion of each ratio: if the animal will respond 20 times a minute on FR 30 for a 45-mg pellet, he will respond at approximately the same rate for a 90-mg pellet on FR 60 or a 180-mg pellet on FR 120. These findings imply that eating rate (rather than meal rate, say) is a regulated variable.

It is hardly surprising that animals try and maintain their eating rate when they must obtain all their food or water via the schedule — if not they would soon lose weight under some schedules. However, they also seem to defend eating rate even for short experimental sessions where weight loss is eliminated via post-session feeding. Perhaps they don't trust the experimenter! More likely, their evolution has programmed them for a short *time horizon:* present, certain, benefits are weighted much more than distant (and perhaps uncertain) ones. Henry Ford, pithily capturing a sentiment still to be found in the business community, once commented, "In the long run, we're all dead." Rats seem to echo this sentiment.

The second assumption has two parts: that taste affects food intake, and that the effect is additive. The first part is almost self-evident: Common experience, and numerous experiments, shows that animals eat more of a palatable diet than an unpalatable one. Davis and his associates have shown that meal size is proportional to the concentration of a palatable substance such as glucose.

Many experiments have shown that taste has an independent effect and may sometimes override regulatory considerations. For example, even normal animals learn only with great difficulty to feed themselves through an intragastric fistula, that is, by a method that bypasses the usual taste inputs (Holman, 1969; Snowdon, 1969). Learning is much aided by giving the animals something pleasant to taste as the intragastric meal is delivered. Rats can learn to maintain themselves on food delivered intravenously, but long-term intake is much below normal and they lose 2040% of their body weight (Nicolaidis & Rowland, 1977). VMH animals are even more sensitive to the loss of taste cues than normals: Many fail to feed themselves at all on an intragastric regimen, and all achieve settling (equilibrium) weights much below their weights when feeding normally (McGinty, Epstein, & Teitelbaum, 1965). VMH animals that fail to feed

themselves can be induced to do so if allowed to lick saccharin solution with each gastric load. Taste is obviously important. I show in a moment that it is additive.

The third assumption, that body weight affects degree of regulation, is required to account for the improvement in regulatory performance that is always found as body weight declines. It also makes functional sense: A loss in weight is much more costly to a lean animal than a fat one, hence a fat animal should defend its weight less vigorously than a lean.

A convenient way to represent the defense of eating rate by means of operant responding is shown in Figure 6.3. The abscissa units are eating rate (in seconds- of-access-to-food per unit time) and the ordinate is the rate of the instrumental response (e.g., lever presses/time). A ratio schedule constrains the point representing response and reinforcement rates to a straight line through the origin (ratio feedback function; see Chapter 5). Feedback functions for FR 1 and FR 10 are shown in the figure. On a given schedule the point representing performance will settle down somewhere on the feedback function. The equilibrium point, B_1, on FR 1 is shown in the figure.

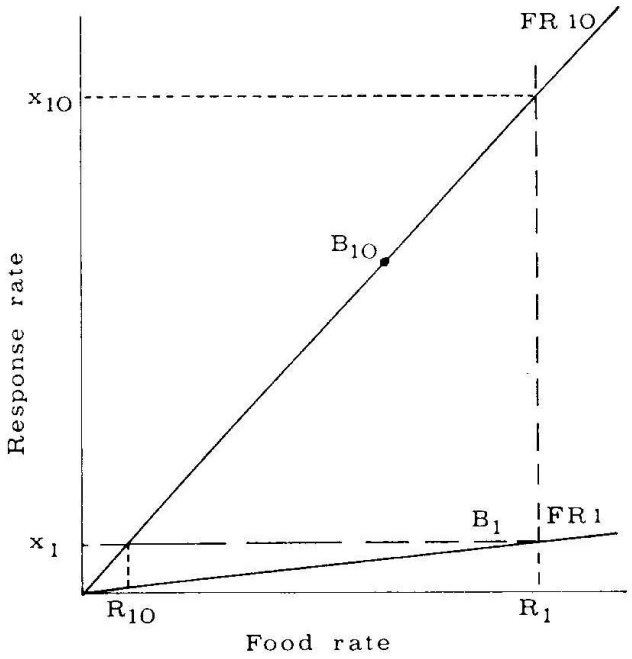

Figure 6.3. Regulatory options on ratio schedules.

The two dashed lines show the two extreme options available to the animal when the ratio size is increased from one to ten. The vertical dashed line represents the option of perfect regulation: Food rate is maintained at R_1 by a tenfold increase in response rate, from x_1 to x_{10}. The horizontal dashed line is the option of no regulation: Response rate is held constant and food rate declines tenfold from R_1 to R_{10}. The point labeled B_{10} on the FR 10 feedback function represents a possible compromise adaptation to the schedule shift. A set of such points can be obtained for a range of ratio values. The line through these points is termed a *response function;* its shape is a measure of the degree of regulation that the animal achieves. For example, if the response function is vertical, then the animal regulates perfectly, adjusting his response rate exactly in proportion to the demands of the ratio schedule. If the response function is horizontal, the animal does not regulate at all. A function of negative slope represents partial regulation, some negative feedback. A function of positive slope represents the opposite of regulation, positive feedback.

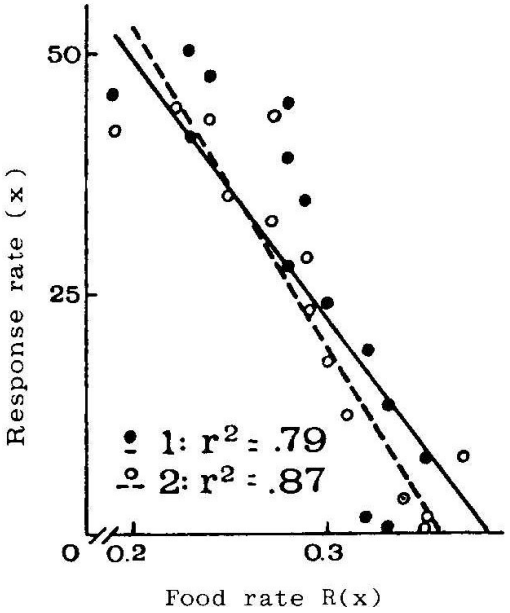

Figure 6.4. Regulatory ratio-schedule responding by two rats that obtained all their food on ratio schedules. The straight lines were fitted by least squares regression. Response rate is in lever presses/min and food rate in pellets/min. (Redrawn from Collier, Hirsch, & Hamlin, 1972.)

Figure 6.4 shows response functions from two rats that obtained all their food on ratio schedules; the points are the equilibria established on a range of ratio values from 1 through 240. The functions for both rats are approximately linear, with negative slope, representing partial regulation. This result is typical of experiments with rats responding for food. At very high ratio values (low food rates), the instrumental responding itself, and the time taken away from other activities, begin to exert some cost and the function begins to tip over (positive feedback instead of negative). Since this tip over represents a severe drop in food rate at higher ratios, it is not a viable pattern in chronic experiments.[8] Tip over is seen in brief-session experiments only with a wide range of ratio values. In this chapter, I deal just with the regulatory linear segment that is typical of long-term responding and short-term responding with a limited range of ratio values.

It is not possible to see the implications of even the simplest feedback process without making some quantitative assumptions. Fortunately, linear response functions, and many of the other properties of feeding behavior, can be derived from a very simple static model based on the three assumptions stated earlier. The model is illustrated in Figure 6.5. The elements within the dashed lines are inside the animal; the box outside represents the reinforcement schedule. The model assumes that the actual eating rate, $R(x)$, is subtracted from a set point for eating rate, R_0. In turn, $R(x)$ is jointly determined by the rate of the instrumental response, x, and the value of the ratio schedule, m = l/M. In most experiments, the animal's state of deprivation is such that $R(x)$ will be less than R_0. G represents the transfer function by which the animal translates a given eating-rate deficit, $R_0 - R(x)$, into a given rate of instrumental responding, x. G is assumed to be a function of body weight, W (in which case, it is written as $G(W)$), and perhaps of other things as well. I represents incentive factors, which make an additive contribution to response rate. I is assumed to depend upon taste, T (in which case it is written as $I(T)$), and perhaps on other things such as time of day or temperature. There may also be a small effect of body weight on I: When body weight is very high, I may be reduced. R_0, the set point, is assumed to be constant, although this is not essential to the model. Function G may be as complex as we please. For simplicity, and because it is sufficient, I assume G is just a multiplier.

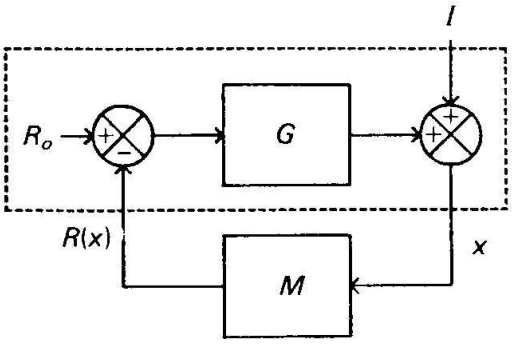

Figure 6.5. Feedback model for the regulation of eating.

The system in Figure 6.5 can now be analyzed in a similar way to the fundamental feedback equation in Chapter 3 (Figure 3.12, Equations 3.11-3.13). At equilibrium the value of $R(x)$ must be such as to produce a response rate x that, by the terms of the schedule, produces the same eating rate, $R(x)$. In other words, at equilibrium the system is defined by two simultaneous, linear equations: the feedback function, and the hypothetical control function (here a simple multiplier). Thus,

$$R(x) = Mx \quad \text{(the feedback function)} \qquad (6.1)$$

$$x = G(R_0 - R(x)) + I \text{(the control function)}. \qquad (6.2)$$

On a given schedule, G. R_0, and I are constants; hence Equation 6.2 defines a relation between x and $R(x)$ that is independent of the schedule value — the response function — which is here obviously linear, with negative slope. It can be rewritten more conveniently as

$$x = GR_0 + 1 - GR(x), \qquad (6.3)$$

which is a straight line with slope $-G$ and x-intercept equal to $GR_0 + I$. I shall refer to Equation 6.3 as the *linear model.*[9]

As before, x can be eliminated from Equations 6.1 and 6.2 to yield the obtained food rate, $R(x)$, as a function of the "loop gain," MG:

$$R(x) = MGR_0/(1 + MG) + MI(1 + MG). \quad (6.4)$$

The first term on the right-hand side of Equation 6.4 is the familiar fundamental feedback equation first encountered in Chapter 3 (Equation 3.13), with set point R_0 and gain MG. The second term represents the additive contribution of incentive factors.

Before turning the effects of other variables, such as brain lesions, notice that Equation 6.4 has the properties we might expect from the earlier discussion of normal regulation: The steady-state rate of eating, $R(x)$, although regulated is not absolutely constant. It depends on both incentive, I and G, regulation. If I is high (as it might be with an especially palatable diet), steady-state eating rate will be higher than if I is low. The effect of I depends on the regulatory factor, G, however: Because I appears only in the second term, divided by $(1+ MG)$, the higher the value of G, the smaller the effect of variation in I. Hence incentive factors will have relatively little effect on the eating rate of normal (high-G) animals.

Body weight appears implicitly in these equations as a factor that affects G (and to a lesser extent, I). There is also a reciprocal relation, of course: between steady-state eating rate and settling body weight. This relation is not well understood. All we are entitled to assume is that a higher eating rate will lead to a higher body weight. Fortunately, no more is needed for the present analysis.

Consider now the possible failure modes of the system in Figure 6.5. There are three simple ones: (a) impairment of the set-point input; (b) alteration of the regulatory function; and (c) alteration of the incentive input. Hypothalamic lesions seem to have effects mainly of the second type. VMH lesions clearly act as if they just impair regulation — a decrease in the value of G in the present model. Other variables, such as a taste or reduced oxygen (hypoxia), have other effects. The experimental effect of VMH lesions can be explained as follows:

Finickiness

Obese VMH rats are more sensitive than normal rats to taste factors; that is, they will eat more of a highly palatable food, less of a less palatable one. If one were to graph their eating rate, $R(x)$, as a function of some measure, T, of taste, the function for VMH animals would be steeper than for normals: A given change in taste would produce a larger change in eating rate. The effect on the slope of this function of varying G can be derived as follows. Incentive is directly related to taste: $I(T)$ is an increasing function. If M, C, and R_0 are constant, then from Equation 6.4, rate of eating, $R(x)$, is a linear function of I, with slope $M/(1 + MG)$. Clearly if G decreases, this slope will increase: The effects of a change in incentive (taste) will be greater. Hence, if VMH lesions reduce the regulatory parameter G, one effect will be an increase in finickiness.

VMH, and normal, animals become more finicky as they become more obese. But we assume that G is inversely related to body weight; hence the higher the weight, the smaller the value of G, the larger the relative effect of incentive, and the more finicky animals should become.

Response to dilution of diet

VMH animals adjust less well than normals to dilution of their diet with a nonnutritive substance such as kaolin. Rats cannot sense directly that kaolin is nonnutritive. Its effects must therefore be via a small loss in body weight: When kaolin is first added, the animal either maintains or reduces (because the dilution does not improve the taste of the food) its volumetric intake, so that after a few days it loses some weight. Hence the reduced sensitivity of VMH animals to dietary dilution implies that these animals are less sensitive than normals to small changes in body weight.

I assume that weight loss increases G, which, on a neutral or unpalatable diet, causes an increase in eating rate (according to Equation 6.4) that partially compensates for the loss in weight. The steady-state result is a small loss in weight and an increase in eating rate that almost completely compensates for the kaolin dilution. On a highly palatable diet, however, dilution

should cause an actual *decrease* in eating rate, according to this model, because the amount by which the animal's actual eating rate exceeds its set point for eating, R_0, is inversely related to *G*.

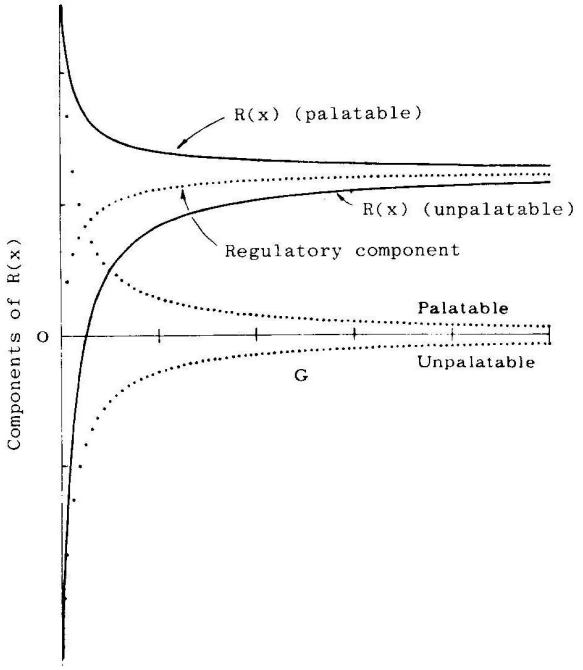

Figure 6.6. The separate and combined effects on eating rate ($R(x)$) of regulatory feedback and taste factors, as a function of gain (G), according to Equation 6.4 in the text. Curves are shown for relatively palatable and relatively unpalatable diets.

No one seems to have looked at how the effects of dilution interact with diet palatability, so this prediction remains unchecked. (The prediction may not be easy to test, given that a kaolin-diluted diet is probably not very palatable.)

The hypothesized effect of VMH lesions is to reduce *G*. The first question, therefore, is: How much does a given change in *G* affect eating rate, $R(x)$? If a given change in *G* has a larger effect on eating rate in VMH animals than in normals, the differential effect of kaolin dilution is explained.

The answer can be deduced from Figure 6.6, which is a plot of Equation 6.4, showing $R(x)$ as a function of *G* (i.e., with everything else held constant). The dashed lines represent the two components of Equation 6.4: The upper dashed line is for the first (regulatory) term; the lower two lines represent the second term — the upper one for a palatable diet (*I* positive), the lower for an unpalatable diet (*I* negative). The upper solid line shows $R(x)$ as a function of *G* for the palatable diet; the lower solid line shows $R(x)$ as a function of *G* for the unpalatable diet.

Just look at the upper solid line (I return to the others in a moment). The *slope* of this line tells us how much R(x) changes for a fixed change in *G*. The curve is negatively accelerated, and it is easy to see that the amount of change in $R(x)$ for a given change in *G depends on the value of G:* When *G* is small, a given change in *G* causes a large change in *R(x),* because the slope of the line is steep; but when *G* is large, the same small change in *G* has little effect, because the line is almost horizontal. Exactly the same thing is true of the lower solid line, except that the changes in *R(x)* are in the opposite direction. In both cases, the smaller the value of *G,* the larger the effect on *R(x)* of a given change in *G*. Thus, the effect of VMH lesions in reducing sensitivity to diet dilution follows from the assumption that VMH lesions reduce the regulatory parameter *G*.

Weak defense of settling weight

Given a relatively palatable diet, VMH animals stabilize at a weight that is higher than normal, and return to it when free feeding is resumed after a period of deprivation. The equilibrium rate of eating at a given body weight can be derived directly from Equation 6.4. It is obvious that if *G* is decreased, the contribution of taste factors, the second term, is increased, and the regulatory component, the first term, is somewhat decreased. The net effect depends on the balance between palatability and regulation, as shown in Figure 6.6. Since normal animals are relatively insensitive to variations in palatability, they can be presumed to lie toward the right, high-*G*, side of the graph, with stable $R(x)$ values close to the asymptote of these functions. As *G* decreases, the equilibrium rate of eating, *R(x),* on the palatable diet rises. Hence the finding that VMH animals eat more than normals, and therefore gain weight, on palatable diets follows from the hypothesis that the have lower *G* values. Conversely, when *I* is negative, a decrease in *G* invariably produces a decrease in eating rate, which is consistent with the finding that VMH animals may actually lose weight on relatively unpalatable diets. Stability of body weight at each

palatability level is a consequence of the presumed fixed, monotonic relation between mean eating rate and settling weight: Since each eating rate defines a settling weight, an increase in eating rate entails an increase in body weight; a decrease, a loss of weight.

Improved defense of low settling weights

VMH animals seem to defend their weight about as well as normals, providing body weight is low enough, although systematic tests with different work schedules do not appear to have been done. The present analysis predicts improved defense at low weights because of the presumed inverse relation between *G* and body weight.

Poor adaptation to work requirements

VMH animals respond for food on low ratio schedules as well or better than normals, but fail to increase response rate appropriately on larger ratios. This result follows naturally from the linear model. Equation 6.3, the response function, is a straight line with slope -*G*. It is easy to show that as *G* varies the equation defines a family of straight lines converging on the point $R(x) = R_0$ and $x = T$. Figure 6.7 shows two response functions of this sort, together with feedback functions for low- and high-valued ratio schedules. The two response functions that intersect at point *P* are termed "VMH" and "normal" and differ only in the value of *G*. The dashed response function is not part of the same family; it has the same slope, hence the same value of *G*, as the "normal" function, but a higher value for the $R(x)$- and x-intercepts. If all three curves were obtained from animals on the same diet, one could infer that the dashed curve differs from the others in having a higher value for R_0.

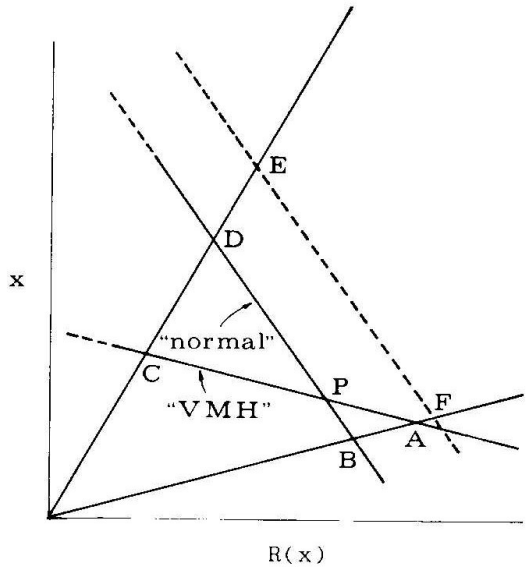

Figure 6.7. Theoretical response functions for VMH, normal, and genetically obese rats exposed to low and high ratio schedules. See text for details.

This diagram raises some questions and makes several testable predictions. Consider first *P*, the point of intersection of the two solid response functions: Why place *P* above the equilibrium points, *A* and *B*, for responding on the low ratio schedule? (The answer to this question is critical, because the predictions are not right if *P* is below points *A* and *B*.) If for simplicity we let the low ratio equal unity, then the equilibrium value for $R(x)$ can be obtained at once from Equation 6.4. It is

$$R(x) = GR_0/(1 + G) + I/(1 + G). \quad (6.5)$$

For normal animals *G* is large ($G >> 1$), hence the first term is approximately equal to R_0. If *I* is positive (a relatively palatable diet), $R(x)$ on FR 1 will therefore be larger than the $R(x)$ value at point *P*, which is R_0, the amount of the difference being inversely related to *G*. The graph illustrates these relations, since the $R(x)$ value of point *A* (equilibrium for the "VMH" curve on FR 1) is greater than the $R(x)$ value of point *B* (equilibrium value for the "normal" curve on FR 1).

The situation is reversed at high ratio values. Here the "normal" equilibrium at point *D* is higher than the "VMH" equilibrium at point C. Thus, the assumption the VMH animals have a lower feedback gain than normals accounts both for their excessive eating rate on low ratios and their failure to adapt to high ratios.

The dashed response function in Figure 6.7 illustrates the case of an animal that simply has a higher set point, R_0, or a lower weight, *W*, but is otherwise normal, that is, has a normal value of

G. This animal defends its eating rate as well as the normal animal, but responds faster at all ratio values, hence has a higher eating rate. Some strains of genetically obese rats fit this picture.

Figure 6.8 shows data that conform over much of the ratio range to the predictions in Figure 6.7. The filled circles are the response function for a group of normal animals on fixed-ratio schedules ranging from 1 to 128. The declining limb of the function is approximately linear with a high negative slope, representing good regulation of eating rate. The open triangles represent a group of VMH rats in the dynamic phase. On FR 1 and FR 4 these animals respond, and eat, more than the normals. At higher ratios they fail to increase their response rate appropriately, however, and therefore eat much less than the normal animals, showing very poor regulation of eating rate.

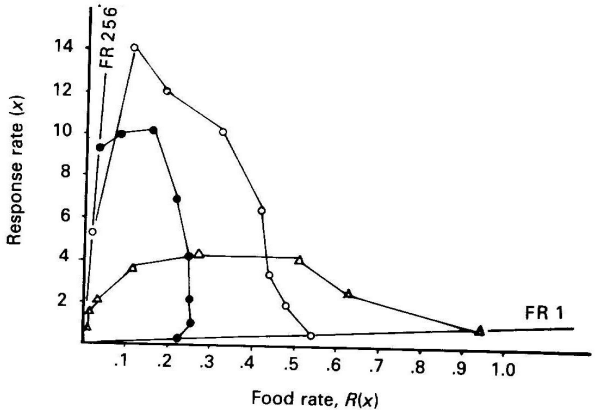

Figure 6.8. Response functions for groups of normal, VMH-lesioned, and genetically obese rats exposed to ratio schedules from FR 1 through FR 128. Response rate is in lever presses/min; food rate in pellets/min. (Redrawn from Greenwood et al., 1974.)

The open circles represent a group of genetically obese rats, also in their dynamic phase. The difference between this response function and that for the VMH animals in striking. The genetically obese animals regulate almost as well as normals (i.e., have a similar value for *G),* but respond, and eat, more at each schedule value. This difference, as well as their lack of finickiness and normal adaptation to dietary dilution, is consistent with a higher value for R_0 for these animals.

The effects of VMH lesions thus correspond very closely to an effect on regulation alone, with no effect on set point or incentive.

What of lateral hypothalamic lesions? Substantial lesions in the lateral hypothalamus (LH) produce a loss in weight accompanied by extreme finickiness.[10] I have not been able to find satisfactory information either on the finickiness of these animals once they have recovered from the severe initial effects of the lesions, or on their ability to defend body weight against dietary dilution and work requirements. The simplest interpretation of what is known is that LH lesions have an even more severe effect on regulatory feedback, *G,* than VMH lesions. One objection is that LH animals do not become obese, even on very palatable diets. One possible answer is that severe LH lesions produce a generalized impairment that interferes with the simple processes of eating and making lever-press responses. There is little doubt that these animals are severely impaired, and perhaps a model that deals solely with the regulation of feeding should not be expected to deal with a lesion that affects many other functions as well.

Thus, the effects of the classic hypothalamic syndromes, as well as other syndromes associated with obesity, appear to be consequences of a system for the regulation of feeding in which eating rate is affected in an additive way by two factors: a regulatory input that is determined by the difference between the actual eating rate and a "natural" rate for that body weight, and taste factors: by an endogenous regulatory process and an exogenous non-regulatory one. The defects of the VMH animal are traceable to impairment of the endogenous regulatory system, with an inevitable gain in the relative effect of exogenous factors. In consequence the animals become finicky, distractible, and "emotional" and cannot adequately defend a body weight that is excessively dependent on the palatability of their diet. The behavior of the classic LH animal is less clearly defined, but may just be a more extreme version of the VMH syndrome. Genetically obese animals are the converse case: Their reference level is elevated and they defend it adequately, without the excessive reactivity to taste of the VMH animal.

This system for feeding regulation has the properties one would expect from the gas-tank analogy. Each body weight defines a certain regulated rate of eating (the first term in Equation 6.4), but that rate can be increased or decreased by incentive (the second term in Equation 6.4). One might also postulate additional additive factors such as temperature and circadian and circumannual periodicities.

Effects of taste and body weight on work schedules

The response functions shown in Figures 6.4 and 6.6 take a long time to obtain: Animals are usually allowed at least one session at each ratio value, and sometimes many more. The whole response function may therefore take a week, or even several weeks, to get. This makes it difficult to measure directly the effects of changes in body weight, diet palatability, or other manipulations, such as appetite-affecting drugs, on response functions. It turns out that there is a quicker way to do these experiments. Rats are very good at tracking changes in the spacing of food over time — so good, in fact, that they will quickly learn to follow a sequence of ratios that changes in a cyclic fashion. For example, suppose that when the rat is first put in the Skinner box, he gets a pellet. Then after two lever presses (FR 2) he gets another; then after four more (FR 4) he gets the next; the next requires 8, and so on up to 64. Rats very quickly learn to anticipate a *cyclic-ratio* schedule of this form: 2, 4, 8, 16, 32, 64, 64, 32, 16, 8, 4, 2, 4,… where they get six or so cycles per session each day. A plot of response rate (ratio size/interfood time) versus food rate (1/interfood time) at each ratio then gives the response function.

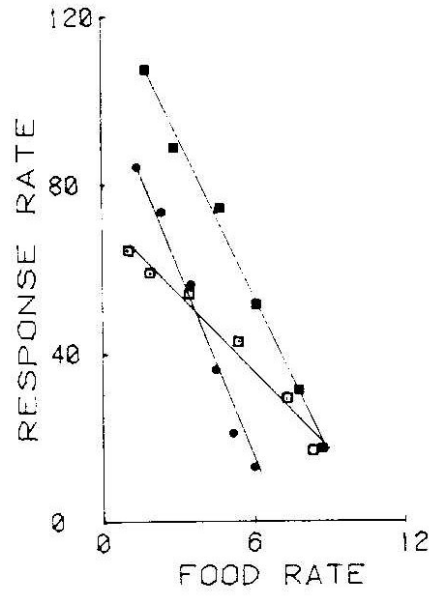

Figure 6.9. The effects of body weight and diet palatability on cyclic-ratio response functions. Points show data from a group of rats at either 80% of normal weight (filled squares), 95% of normal weight (open squares), or 80% weight with quinine-adulterated food pellet reward (filled circles). Ordinate is lever presses/min, abscissa is 45-mg pellets/min. Lines were fitted by linear regression. (From Ettinger & Staddon, 1983.)

Both response functions, and more molecular properties of ratio behavior, correspond quite closely to what is found in the standard procedure where the rat contends with just one ratio value in each experimental session. For example, the effect of ratio size on time-to-first-response (postfood pause) and response rate after the first response (running rate) is similar in both. But the cyclic method makes it easy to look at the effects of such things as body weight and diet palatability on response functions.

Figure 6.9 shows cyclic-ratio response functions obtained from a group of four rats given three treatments: (a) run at 80% of their normal body weight (hungry); (b) at 95% of their normal body weight (not so hungry); and (c) run at 80% weight with quinine-sulphate-adulterated (bad-tasting) food pellets. The effects of the three treatments are just about what we would have anticipated: The effect of the bad taste (filled circles) is to displace the response function inward from the control (80% wt.) function (filled squares); this is an effect on the incentive parameter, *I*. The effect of satiation (95% wt., open squares) is to decrease the slope of the line. A comparison of response functions across body weights between 80% and 95% showed that only parameter *G* in Equation 6.3 is affected by changes in body weight. In other experiments (Ettinger & Staddon, 1983) the anorectic (appetite-reducing) drug amphetamine has been shown to affect mainly parameter *G,* while reduced atmospheric oxygen (hypoxia) affects mainly parameter *I* — confirming the reports of mountain climbers that food loses much of its taste at high altitude.

Thus, the effects of both regulatory variables, such as body-weight changes (food deprivation), and incentive variables (taste) seem to affect ratio response functions in the simple way suggested by the linear model: Body weight affects slope, taste affects intercept.

Other motivational effects

The precise quantitative form of the present analysis should not be taken too seriously: The static linear model is not the last word. I introduced some equations only because without them, it is impossible to see how all the factors that affect eating interact. The strong point of the linear model is that it is simple enough to allow one to see pretty clearly how these various factors combine to affect eating rate and work rate on an operant schedule. It suggests simple experiments to check out the effects of physiological manipulations. For example, factors such as taste that enter in additively to the control function (Equation 6.3) appear as terms of the form $MB/(1 + MG)$ in the eating- rate equation (Equation 6.4), where B is the factor in question. Hence the effects of changes in B are inversely related to G. Temperature is known to have a linear effect on eating rate of the sort predicted by this interpretation.[11] VMH animals appear to be characterized by a lower-than-normal value for G; hence their temperature function should also have a lower slope. As far as I am aware, this experiment has not been done.

Another experimental manipulation that has a comprehensible effect is sectioning the vagus nerve below the level of the diaphragm.[12] Vagotomy abolishes input to the CNS from the gut and liver and suppresses eating in both normal and VMH rats. The effect on the VMH animals is much greater, however, and generally sufficient to eliminate obesity on the usual diets. Finickiness remains, which suggests that the vagus is involved in a separate additive term in the eating-rate equation. Since such additive effects are inversely related to G, the effect of vagotomy should be, and is, smaller not only in normal animals, but also in genetically obese animals and animals made obese by ovariectomy — because the obesity of these animals does not reflect impaired regulation.

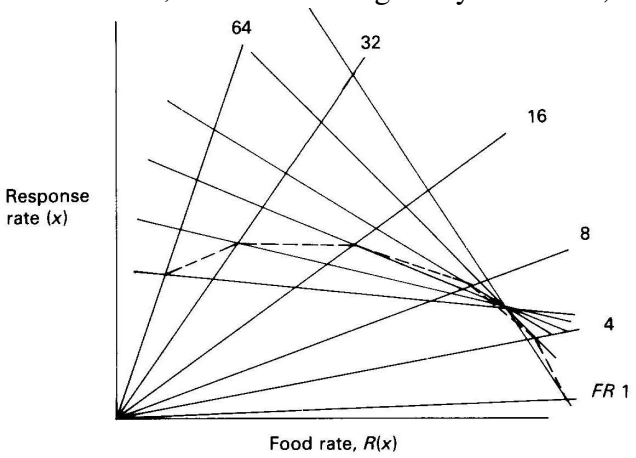

Figure 6.10. Theoretical effects of changing body weight on the ratio-schedule response function. Lines passing through the origin are feedback functions for ratios of 1,4, 8, 16, etc. used successively in the experiment by Greenwood et al. (1974). Radial lines of negative slope are response functions computed from the linear model on the assumption that body weight is increasing each day. The dots connected by a dashed curve represent the daily equilibria.

Limitations of the linear model

It is probably asking too much of the linear analysis to expect it to provide precise quantitative predictions; and, of course, it is a *static* analysis and can give no account of the time course of the various regulatory mechanisms. Response functions are only linear over part of their range, not all as the linear model implies. The curvature in the VMH function in Figure 6.8 is substantial, for example. Most workers in this area have not thought much about quantitative matters, however, so that few data of the precision necessary to test quantitative models are yet available. Moreover, commonly used procedures sometimes involve confounding factors. For example, the data in Figure 6.8 were gathered under conditions where the animals obtained all their food in the experiment. After three days on FR 1, ascending ratios of 4, 8, 16, and so forth, were introduced in succeeding 24-hour periods. Body weight was not controlled, so that the linear model does not in fact predict that the successive daily data points should all lie on the same straight line. If, as seems likely, body weight was increasing

over days, the points should lie on a curved line, as illustrated in Figure 6.10. The linear model may therefore do a better quantitative job than is apparent at first sight.

SUMMARY

Feeding, a typical motivational system, involves both internal and external factors. The endogenous factor is regulatory and tends to oppose anything that forces eating rate to be reduced below a preferred value, the set point. The exogenous factors are approximately additive, and cause eating rate to be higher or lower than the set point, according as they are positive or negative. The degree of feeding regulation appears to be inversely related to weight: the more food-deprived the animal (the lower its body weight), the more vigorously it defends its eating rate. Feeding regulation is impaired by ventromedial hypothalamic lesions. Since the endogenous factors are reduced, exogenous factors become relatively more important, and VMH animals are "finicky" and will not work hard for food.[13]

Joint determination by exogenous and endogenous factors is just what would be expected from functional considerations: Feeding behavior is guided in sensible fashion by all the information available to the animal, both internal (signaled by body weight), and external (signaled by taste, temperature, and discriminative stimuli).

These regulatory and incentive effects can be described by a linear, static model. The linear model is simple and useful, but it is also limited in a fundamental way: It predicts the same response function (Equation 6.3) no matter what form is taken by the feedback function (Equation 6.1). This is contrary to fact. For example, interval schedules have a nonlinear feedback function, which can be approximated by a hyperbola (see Chapter 5); response functions on such schedules are also nonlinear, with positive, rather than negative, slope over most of their range. When extreme ratio values are considered, the response function on ratio schedules appears to be an inverted-U shape. Response functions on avoidance schedules are often not linear and never of negative slope. The most successful attempts to bring these varied effects together in a unified way involve *optimality analyses;* that is, explanations of behavior as the outcome of processes that act to maximize or minimize some quantity such as utility, cost, or Darwinian fitness. These approaches are the topic of the next chapter.

NOTES

1. Astonishing or not, the study of reinforcement schedules was — and to a large extent, still is — pursued in isolation from regulatory considerations. For example, *open-economy* experiments, where the animal is maintained at a reduced weight throughout brief daily experimental sessions show a positive relation between the rate of an operant response and the rate of food delivery. *Closed economies*, where the animal lives in the experimental apparatus and gets all its food from the reinforcement schedule, rate of operant responding is inversely related to rate of food delivery. This difference should not surprise. In an open economy the animal cannot regulate his food intake (the experimenter does it for him); in a closed open economy, he must. In the first case, responding is driven by incentive processes: the more the better. In the latter, it is driven by regulation: enough is enough.

2. The difficulties in finding a single critical variable as the trigger for eating are well described in a paper by Friedman and Stricker (1976), which also gives an excellent account of the physiology of hunger. Uses and abuses of the set- point idea are discussed by Mrosovsky and Powley (1977). Wirtshafter and Davis (1977) show by example that regulation implies negative feedback, but does not require any identifiable set point. Chapters by Booth, Bolles and Davis, and Hogan in the book

edited by Toates and Halliday (1980) also discuss this issue. See Staddon (2001b) for more recent references and a dynamic analysis of feeding behavior.

3. The evolutionary significance of taste is not well understood. On general grounds one might expect taste to be an evolutionary predictor of nutritional value: "Sweet" signifies high caloric value, "bitter" potential poison danger, and so on. But the correlations are obviously weak in many cases, and obscure in others. So little is known about the food available to ancestral species that arguments of this sort have few facts to restrain them.

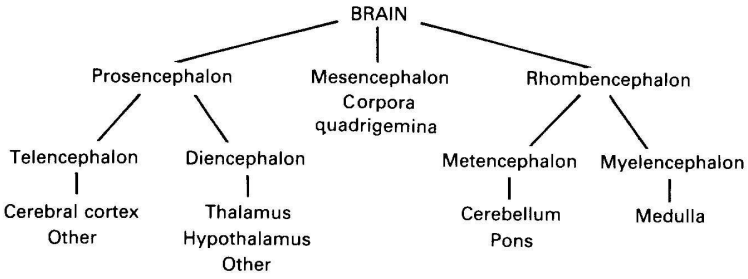

Figure 6.11. Mammalian brain structures, organized in terms of development and anatomy. (Modified from Ransom & Clark, 1958.)

4. The mammalian brain is conventionally classified into a hierarchy of structures, based on anatomical arrangement and pattern of development, as shown in Figure 6.11. The vertical axis of the figure corresponds to the pattern of development: The primitive brain first differentiates into pros-, mes-, and rhombencephalon, then these structures differentiate further into telencephalon, diencephalon, and so on. The left-right axis corresponds to anatomical arrangement: The medulla and cerebellum are closest to the spinal chord, the cerebral cortex further away.

The hypothalamus, thalamus, and epithalamus are the three main divisions of the diencephalon. Experiments in which the hypothalamus has been lesioned, or stimulated electrically or with chemicals, have shown it to be involved in a wide variety of motivational and regulatory functions.

5. *The meaning of lesion experiments.* What is the significance of brain-lesion experiments? It was once assumed that if destruction of a brain area caused loss of some function, than that area was a "center" for that function. A bit more thought soon showed that this inference is unwarranted: Cutting the wire to the speaker will silence a TV set, but the wire is hardly the source of music. The whole notion of "centers" had to be given up. The brain is a system at least as complicated as a digital computer, and the effects of interference with it are at least as hard to interpret.

If the interference is sufficiently subtle — the neural equivalent of disabling a single computer-memory element, for example — then very specific effects might be expected. But lesions are not subtle, and even the much more delicate pharmacological and electrophysiological interventions now commonly applied to single neurons or small groups probably represent relatively crude assaults. Lesions therefore probably tell us more about the broad principles of brain organization than about the function of specific neural structures. In other words, these physiological manipulations may tell us more about the functional properties of the brain — the topic of this book — than about structure-function (brain-behavior) relations — the topic of main interest to neuroscientists.

This point is hard to prove, but I can suggest an analogy. Consider the TV-set problem again. Imagine that you have an indefinite number of the things (all the same, or a related, model) and want to find out how they work. But your instruments are limited to a small collection of woodworker's tools: a ball-peen hammer of sturdy design, some screwdrivers, a bit brace, drill press, and a few chisels. Undaunted by the crudeness of the tools, you set to work in methodical fashion. Placing TV set #1 in the drill press (stereotaxic instrument) you drill into it for a precisely measured distance. When the machine has recovered from the surgery, you turn it on and try to

tune in a few test patterns. No doubt some drastic defect, from a longer version of the list mentioned earlier, will appear. Encouraged by your success, you take TV set #2, repeat the experiment with some slight variation, and measure the effects. Dogged application of the experimental method continues until you have studied perhaps 1,000 sets with all sorts of assaults, drilling, striking, and chipping in carefully recorded and replicable fashion. What will you find? What can you conclude?

I suggest that you will find much the same kind of thing as from a study of the natural history of defective TV sets: a few common defects, some rare ones, an often-loose correlation between the precise site of the lesion and the exact nature of the defect.

Nevertheless, the defects are likely to fall into a few major groups, defined by the basic functional categories of the machine: video effects, sound effects, effects on picture synchronization, and so on. These will be the common failure modes of the machine. They tell little of the circuit details, but much about functional organization. All is not likely to be perfectly clear, however. Many lesions, as carefully done as the rest, will produce odd defects that fail to fit into any pattern. Perhaps more than one functional subsystem has been damaged, probably because the functionally different structures overlap physically. These effects are much less informative than the common, simple ones.

I suggest that the hypothalamic-lesion studies should be interpreted as this analogy suggests. The common effects are most informative, but there are likely to be many intermediate cases that muddy the picture. It is not surprising that although most VMH-lesion studies report essentially the list of properties described in the text, there are several that present conflicting evidence. A number of these point to the location of the lesion and possible involvement of other structures as the cause. All this is to be expected. Electrolytic lesions, although somewhat more precise than our assault on the TV sets, are still crude when measured by the precision of the neural structures affected. Common, simple effects therefore mean more than rare, complicated ones. The major effects of VMH lesions are the ones I have described.

The experimental literature on the behavioral effects of hypothalamic lesions is voluminous and impossible to more than touch on here. Articles I have found especially helpful are Kennedy (1950), Hoebel and Teitelbaum (1966), Panksepp (1974), Keesey and Powley (1975), and Collier, Hirsch, and Kanarek (1977). The book edited by Novin, Wyrwicka, and Bray (1976) contains a number of useful reviews and theoretical contributions. Particularly relevant to the present topic are chapters by Booth, Toates and Platt, Sclafani, Peck, Panksepp, and Davis, Collins and Levine. A search on the web will reveal many recent studies.

The model I describe, although not identical to any in the literature, is closest to the proposal of Panksepp (1974).

6. Nisbett (1972) in a theoretical review has pointed out that hungry people and rats are in one sense *more* (rather than less) sensitive to taste than non-deprived individuals: When given a choice between tasty and neutral foods, the hungry animal increasingly chooses the more palatable alternative. This apparent finickiness of deprived animals is *not* shown in the single-choice situation, however. When a mediocre food is the only one available, normal rats will eat enough of it to maintain their body weight — but obese VMH animals will not. It is therefore wrong to infer from the choice results that VMH animals resemble food-deprived animals. Moreover, the behavior of the hungry normal animals is perfectly sensible from a regulatory point of view, granted the hypothesis that palatability is an evolutionary predictor of nutritional quality. The undeprived animals can afford to sample alternatives, but the hungry animal, given a choice, would do well to concentrate on the richest payoff.

Theoretical and experimental work in behavioral ecology has expanded this commonsense analysis to include probabilistic food sources. For example, suppose that the animal has a guaranteed, but lean, source of food, and an alternative, chancier, but occasionally richer, source;

which should he choose? The answer depends on the animal's estimate of expected food rate: If the rate from the certain, lean, source is high enough to sustain life, then he should concentrate on it (be *risk-averse,* in decision-theory terminology). This corresponds to the Nisbett situation. But if the expected rate is too low to sustain life, then he should go for the rich, but chancy, source, since only that provides any chance of survival. Surprisingly, perhaps, birds seem able to assess the necessary probabilities and behave as theory suggests they should (Caraco, Martindale, & Whitham, 1980), although more recent studies have muddied the picture somewhat (see, for example, Kacelnik & Bateson, 1997).

7. I assume that the effects of kaolin are largely due to its nonnutritive, diluent properties, but later arguments suggest that the relative un-palatability of Kaolin-diluted diets may also be a factor.

8. The data in Figure 6.4 were obtained with an unusual ratio schedule in which the ratio value was fixed, but the meal length was under the control of the animal: At the end of each completed ratio, the feeder remained available until 10 min after the animal removed its head from the feeder opening. This was necessary to ensure that the animal (which obtained all its foods from the schedule in round-the-clock sessions) would not starve, no matter how high the ratio setting. The effect of this procedure is that the animal selects meal lengths appropriate to the ratio value (large meals if the ratio is large, smaller if it is small) and always remains on the linear part of the response function. Similar data can be obtained with the conventional procedure (short sessions and a fixed meal length), providing the ratio values (gram/response or seconds-of-access/response) are kept within the same range. (See Staddon, 1979b, for a fuller discussion.)

In subsequent discussion the term *eating rate* has two meanings: as measured it refers to grams of food (or seconds of access) per hour in relatively brief experimental sessions. However, I assume that eating rate measured in this way is also a valid measure of the free rate of eating in 24-hour sessions, either unrestrained, or with the animal-determined-meal-size ratio-schedule procedure.

9. Equation 6.2, a good approximation to the declining limb of ratio response function, is equivalent to the *conservation model* for schedule performance proposed by Allison (e.g., Allison, Miller, & Wozny 1979; Staddon, l979a) — although the number of parameters, and their interpretation, is different.

10. This is true of the classical LH syndrome discussed by Keesey and Powley (1975), Teitelbaum and Epstein (1962), and others. More recently, Peck (1976) has identified two other LH syndromes, types I and III, in addition to the classical one, which he terms type II. For the reasons discussed in the text, the commoner syndrome is likely to provide simpler insights into the nature of the feeding system than exceptional cases, and I do not consider them here.

11. For a recent review of temperature effects see Kraly and Blass (1976). Data from Brobeck (1945) fit linear functions; see also Russek (1976).

12. Studies of the effects of vagotomy have a long history. Recent findings are summarized by Powley and Opsahl (1976).

13. *Human obesity.* What are the implications of animal studies for the problem of human obesity? IS there, in fact, a problem? If not, the innumerable recipes for weight loss may, like swine-flu vaccine, be a cure for which there is no disease. It is easy to get the impression that even moderate obesity is a terminal ailment, even though the evidence for its unhealthiness is mixed at best. Charles Darwin's father died after 82 years of vigorous life at a weight in excess of 300 lbs. A

250-lb. Tongan is regarded as a lightweight, yet the islanders live as long and as well as their slimmer neighbors. Recent medical work seems to concede that "standard" weight tables err on the light side: People thus deemed overweight seem to live as long or longer than their leaner brethren. And there are disadvantages to looking like a *Vogue* ad: thin women are more likely to be infertile than their ampler sisters, for example. Jogging may be an involuntary birth-control device. Is it really impossible to be too thin or too rich, as Dorothy Parker contended? Objective evidence on the virtues of slenderness, at least in middle age and beyond, is in fact…slim.

Most of the problems of obesity are probably social rather than physiological. Still, since physiology may be easier to change than fashion, an inquiry into the parallels with animal studies has some appeal. Schachter and his associates (e.g., Schachter & Rodin, 1974) have suggested that obese people are like VMH rats, and both are characterized by excessive reactivity to taste and other stimuli. A number of ingenious experiments with people tend to support this view, although there are some dissenting arguments (e.g., Milich, 1975; Kolata, 1977). My analysis suggests that the reactivity of VMH animals is in fact a secondary effect of dietary dilution and work schedules, as well as finickiness. The same analysis also accounts for the fact that VMH animals *do* regulate, albeit at a higher-than- normal weight, a fact which, as Schachter points out, is ". .. so troubling to ours as well as all other theories about the obese animal, that they do stop eating" (1974, p. 72). Moreover, there are several possible animal models for human obesity, such as genetic obesity (of several varieties) and obesity caused by gonadectomy. Simple as the linear model is, it nevertheless suggests several ways for obesity or weight loss to occur, and animal examples exist for most of them. The one constant in biological populations is individual variation, and it is unlikely that all human obesity conforms to a single pattern.

The fact of negative feedback means that obesity by itself is uninterpretable. Tests of the sort described in the chapter — dietary dilution and adulteration, work schedules, temperature changes — are essential before the factors maintaining a given body weight can be properly identified.

Unfortunately, even if the causes are identified, altering body weight will still be difficult. Suppose that regulatory feedback is impaired. Body weight will then be more sensitive than usual to dietary palatability and other stimulus factors. But an unpalatable diet tastes bad, and who will insist that the would-be sylph sticks to it after she leaves the Spartan regimen of the fat farm? Theory predicts that food intake can also be reduced by imposing a work schedule, but again who will impose it? If someone is obese because of an elevated set point, things are even worse, because regulatory efforts will be vigorous: Sensitivity to taste and work will be relatively low and only enforced deprivation will reduce eating.

My conclusion is that even for VMH-like obese people, a study of the feeding regulation system by itself provides no real answer to the problem of weight regulation. If there is a solution, it lies in other motivational systems that compete with the feeding system for access to the behavioral final common path. Fat people often lose weight when they acquire a new interest, which may be either positive, such as a new job, companion, or religion; or negative, such as a personal or career crisis. Rats housed in activity wheels gain less weight on a palatable diet than animals without this opportunity to run, and the difference is not attributable just to increased energy expenditure — rats decrease, rather than increase, their food consumption when provided with this opportunity for a competing, highly favored activity (Sclafani & Springer, 1976; Premack & Premack, 1963). Opportunities for desirable but drastic changes in life style of this sort are obviously limited in practice. Nevertheless, they may provide the only effective means of producing permanent changes in body weight. The existence of negative feedback means that the only reliable way of controlling one regulatory system is with the aid of another, competing one.