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VOLUME 6

ACADEMIC PRESS New York San Francisco London
A Subsidiary of Harcourt Brace Jovanovich, Publishers

The Selection of Foods by Rats, Humans, and Other Animals

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I. Solutions to the Food Selection Problem 21
A. The Specialists 24
B. The Generalists or Omnivores 27
II. Rats: An example of Successful Generalists 27
A. The Specialist within the Generalist 28
B. The Rat as a Generalist 34
III. Food Selection in Humans 52
A. Biological Factors in Human Food Selection 53
B. Specific Hungers in Adult Humans 56
C. Biological Basis of Ethnic-Racial Dietary Differences 58
D. Culture and Cuisine 62
References 67

I. SOLUTIONS TO THE FOOD SELECTION PROBLEM

Feeding and the search for food are probably the predominant activities of most animals. For some, clams or cattle, for instance, feeding occupies almost all waking time. Through adaptive radiation, animals have managed to exploit just about every source of nutrition in the world. The pressure for survival is too great to leave a potential food source untouched. Even the most inaccessible nutritional riches have been compromised. Consider the clam, its rich meat so well protected by a thick shell that can seal tight. Even such an impregnable beast has been compromised in remarkably diverse ways: certain mollusks gain access to the clam by slowly drilling a hole through the shell with a raspy organ, the radula; starfish do the job by attaching their feet to both halves of the shell and exerting a steady but powerful force to separate the halves; herring gulls fly

up with a clam in their beak and drop it over rocks, shattering the shell; otters find a rock and break the shell with it; and man often makes a mess of it, but ends up with the meat. A paper such as this could easily succumb to the "gee whizzery" of adaptive radiation of feeding mechanisms, but the task at hand is quite different. It is to describe the mechanisms of food selection—how food is recognized and how choices are made.

Food selection implies food ingestion. Food ingestion implies the presence of food. Therefore, background for the study of food selection includes the food search process: search images and search mechanisms for finding appropriate food stimuli in the environment. Honey bees (von Frisch, 1967) provide fine examples of a highly developed food search system. Food selection also implies the ability to obtain or capture food, and to assimilate it, for which many often exotic mechanisms have been evolved (Jennings, 1965).

The presence of food, however, is not a sufficient condition for food ingestion. Food must ordinarily be accompanied by the organism's inclination to eat it, at any particular time. In the absence of strong competing stimuli or drives, it is normally assumed that some aspect of the internal state of the organism determines whether or not it will eat a particular food. This state can be described as a "detector" that facilitates or inhibits ingestion. If the internal state or detector controls ingestion rather tightly, so that the internal signal is held within a narrow range, the process can be described as a regulation of food intake. In this sense, most animals seem to have some internal system, directly or indirectly responsive to energy balance, that modulates food intake (Rozin, 1964).

Given the presence of potential food, ingestion then usually depends on an internal state or *detector* indicating a "need" for the particular food or class of foods, and a *recognition* of the potential food as food. In a few cases, such as some filter feeders (for other examples, see Rozin, 1964), internal state may play a minimum role; feeding always occurs in the presence of adequate stimuli. In many cases, the issue is simplified, because a species may consume only a rather small set of nutritionally exchangeable foods, such as the larger fauna of the African savannah which serve as food for lions. In this case, only one detector system is in principle necessary: any food source will serve to correct the internal, presumably energy-deficient state. In some cases, the single detector may be linked to a simple food recognition system, if the class of foods can be easily categorized (e.g., small moving things). As the range of food for a species is enlarged, as it approaches omnivory, the problems of both detection and recognition are vastly increased. There is no simple way of separating the class of potential foods from inedible or harmful substances. Also, it is likely that the numerous acceptable foods are not nutritionally equivalent, so that a single detector mechanism will not suffice.

The specialists in the animal kingdom, who eat a narrowly circumscribed group of foods, are likely to have detection and recognition of foods under tight genetic control. The generalists or omnivores require a much more plastic

system. This paper focuses on the complex problems, especially in food recognition and choice, in the omnivores or generalists. The complex interaction of genetically determined and experiential influences on food selection will be constantly in view, since there are both clear advantages and disadvantages to a heavy reliance on either nature or nurture in solving the problem. Omnivores, such as rats and humans, faced with an enormous number of potential foods, must choose wisely. They are always in danger of eating something harmful or eating too much of a good thing. Although there are some helpful internal mechanisms, such as poison detoxification, nutrient biosynthesis, and nutrient storage, the major share of the burden for maintaining nutritional balance must of necessity come from incorporation of appropriate nutrients in the environment and, hence, behavior. Curt Richter, the great Hopkins psychobiologist, demonstrated in the 1930s and 1940s that behavior was equal to this task and that, in rats, metabolic homeostasis could be maintained by adaptive self-selection of nutrients (Fig. 1). The concern here is to describe and extend Richter's work, by looking further into mechanisms, and looking at the food selection of man.

When omnivores are examined closely, their resemblance to specialists becomes greater. In some respects, an omnivore is simply a number of specialists combined in one organism. To explore the "specialist within the generalist" and the role of built-in programming in food selection, the specialists are considered

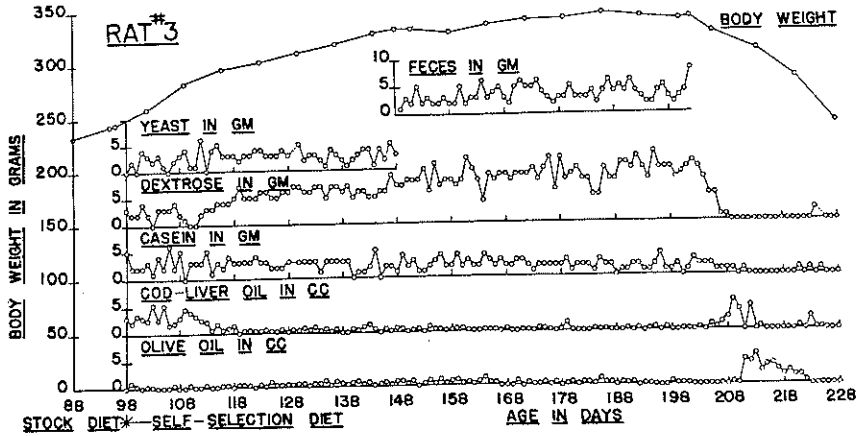


FIG. 1. Example of self-selection behavior of rats on a "cafeteria" regime. Selections from among a variety of mineral sources are not included in this figure. The left portion of the figure shows self-selection with fully adequate diet components available. At about day 148, yeast, a source of B complex vitamins is removed, but rats are allowed to consume feces, which normally contain B complex vitamins. Selection of feces averts a vitamin deficiency. When the feces are removed, the rats gradually become B vitamin-deficient. Note the marked decrease in carbohydrate intake with B vitamin deficiency and the predominant role of fats as a calorie source. Note also the stability in day-to-day selections prior to deficiency. (From Richter and Rice, 1945.)

first. This approach leads to clear parallels with food selection in rats and, possibly, in humans.

A. THE SPECIALISTS

The specialists survive by being especially good at finding, catching, and eating their special food. The price is complete dependence on one or a family of foods. Since the food category is limited and usually homogeneous, the problem of food recognition is easily solved with fixed circuitry i.e., easily programmed genetically. The problem of food choice rarely arises since the world is pretty well categorized as food and not food; natural selection rather than the individual organism makes the significant food choices. The problem of deficiency is not a behavioral one, since each individual food in the narrow range of foods is ordinarily nutritionally complete in itself. Carnivores, for example, rely on their prey to regulate intake qualitatively: only vitamin-deficient zebras can produce vitamin-deficient lions.

The extreme form of specialists are those that consume only one type of food (monophages). Examples are the koala bear, surviving on eucalyptus leaves, and the caterpillar of the monarch butterfly, which eats only milkweed. Omnivores may become monophagous—as almost happened to the Irish peasant who relied almost completely on the potato. The dangers of monophagy are clearly illustrated here by the disastrous potato crop failures in the mid-nineteenth century and the resultant famine.

Animals that restrict their food intake to a rather well-defined category of foods can "solve" the food selection problem at the receptor level. Certain patterns or sets of patterns of receptor responses can define acceptable foods (Dethier, 1967, 1973). This linkage can be permanent and unmodifiable by experience. For example, in some species of frogs that limit their fare to insects, there is a special visual receptor and central nervous system processing system that responds to small, convex, dark moving objects—bugs to be sure (Lettvin *et al.*, 1959). Such a recognition system could be easily wired into the motor side of the feeding system.

Among plant-eating insects, which often rely on chemical stimuli for food identification, approach is often guided via the olfactory system, whereas ingestion per se is under the control of contact chemoreception. Although there is some evidence for control of behavior by specific "token" substances in acceptable or unacceptable foods, in most cases feeding seems to be controlled by the combined response of several receptors to multiple constituents (Dethier, 1973). The chemicals and receptors may be described with some precision (Dethier, 1967, 1973), and identification by the experimenter of acceptable foods by electrophysiological response in olfactory or contact chemoreceptor nerves is a real possibility.

A limited definable food category, then, is associated with a characteristic receptor response pattern, probably connected directly to the motor side of the feeding system, with some sort of control over the linkage by what may often be a simple, single, internal detector system. Such systems are often genetically programmed and also unmodifiable. All that is necessary is a way of locating the food, which can be accomplished by a hierarchy of "search images," often olfactory or visual to contact chemoreceptor (gustatory) (Dethier, 1967, 1973). For virtually all animals, the mouth is the final checkpoint before entry into the sacred precincts of the body. The final criterion for the acceptable-unacceptable "judgment" is made here, mediated by the taste response. These taste judgments may override the previously acceptable food signals coming from other receptors (Dethier, 1969). Vomiting is one of the few available defenses once food is in the stomach, and rats, at least are incapable of vomiting.

Staying within the range of specialists, one can see increasing levels of complexity by the concatenation of specialized systems. The best example I know of this, which is also the best example I know of the detailed analysis of any significant behavior, is the work by Dethier and his colleagues (Dethier, 1969) on feeding in the blowfly. The adult blowfly, in its rather brief life, needs only an energy source (e.g. carbohydrate) and water, except for the female's need for protein during the stages of egg development. Flies identify foods primarily on a chemical basis, ultimately by contact chemoreception. The chemosensory hairs contain four to five nerve fibers—one particularly sensitive to sugars, one to water, one to salt, with the remaining one or two difficult to categorize. High-protein foods lead to a characteristic response pattern across receptors. Potential food encounters a hierarchy of chemoreceptors: first olfactory receptors, then chemoreceptors on the legs when the fly alights on the food, and, subsequently, two sets of receptors in the oral area, if the receptors previously stimulated indicate acceptable food.

The energy control system seems to be built around the sugar receptor. A fly with an empty gut is particularly responsive to sugar solutions. When the insect steps in such a solution, receptor discharge leads to proboscis extension and sucking. Sucking continues, with a gradual rise in the sugar concentration that will maintain it, until there is sufficient adaptation so that no significant signal is received by the critical part of the central nervous system. A sort of regulation occurs, since the receptor input is attenuated (probably centrally) by the presence of solutions sensed by interoceptors (probably mechanoreceptors) in the foregut (Gelperin, 1966). The supply of sugar solution in the foregut is maintained over modest periods by periodic squirting of stored, recently ingested sugar solutions, from the crop into the foregut. When the crop is empty and, hence, there is no sugar in the foregut, input from "sugar" receptors again drives feeding effectively; there is an effective drop in the sugar response threshold (Dethier, 1969). Note that this energy control system does not directly regulate

the amount of energy intake, since the prime determiners of the amount ingested are sweetness of solution and amount of solution in the gut.

In the presence of dehydration, sensed probably as a reduction in blood volume (Dethier, 1969), flies show an increased tendency to ingest water, i.e. water receptor input effectively drives sucking. The sodium receptor activity seems to turn off sucking; that is, it seems to be a food-avoidance mechanism.

Gravid females show an adaptive increase in protein and decrease in carbohydrate intake during the period of egg development (Dethier, 1969). The explanation of this protein-specific hunger in terms of internal metabolic and/or hormonal changes is not yet at hand.

The blowfly solves the problem of ingesting three types of substances with three specific systems, involving specific receptors or receptor complexes and detectors. Each system is quite inflexible and not susceptible to change via learning. The blowfly is, in effect, a small bundle of specialists.

Even among species with a rather narrow set of potential foods, there is quite a bit of evidence for experiential influences on food selection. Unlike intraspecies recognition, where imprinting seems to be a common mechanism for determining selection, irreversible effects of early contact with a particular food are not common. However, there is a general tendency for animals to prefer familiar foods. Thus, Jermy *et al.* (1968) demonstrated preferences for feeding on particular plants in lepidopteroid larvae selectively exposed to these plants 1 or 2 instars prior to testing. Similarly, Fuchs and Burghardt (1971) showed that within the narrow set of potentially acceptable food stimuli, young garter snakes would develop a selective preference for fish or worms on the basis of prior exposure. Unlike imprinting, this familiarity effect was reversible. Similarly, Burghardt (1967) reported a preference effect lasting over 1 week for a food (meat or worms) offered to snapping turtles in their first meal, indicating both a familiarity and primacy effect. Finally, Hess (1964: see also Hogan, 1973a) reported evidence for a critical period for chicks in acquisition of preferences for stimuli that, when pecked, led to food reward. Chicks receiving such an experience on days 3 and 4 of life showed a continued preference for this stimulus, whereas, if the critical reinforced experience occurred before day 3 or on day 7, little effect was seen.

The role of familiarity in food acceptance seems almost universal. It is clearly present in primates. Weiskrantz and Cowey (1963) studied the response of rhesus monkeys to new foods in the laboratory. They found that though monkeys tended to sample new foods immediately (e.g., black currant juice or a chocolate malted drink), many would consume very little on the first few days, and later increase their intake considerably over a period of weeks. They noted that visual exposure to other monkeys ingesting the new food facilitated increased acceptance by "nonconsumer" monkeys. Imitation or observation also seemed critical in acceptance of new foods by free-living Japanese macaques (Itani, 1958). Some new foods were rapidly accepted by the troop studied, e.g.,

wheat, summer oranges, whereas others, such as apples, were accepted more gradually. In a detailed study of the acceptance of candy, to which the monkeys did not initially respond with enthusiasm, Itani showed a clear pattern of social transmission of candy-eating from young and infant monkeys to older siblings and mothers to the rest of the troop. The key role (resulting from minimal neophobia) of the young animals was indicated by the fact that, although less than 10% of adults ate candy on the initial presentation, 50% of 3-year-old and younger monkeys did. Furthermore, a year after initial introduction, 100% of 1-year-olds were candy eaters, compared to 51% of adult and young females and 32% of adult and young males. We shall again see the critical importance of this novel-familiar dichotomy as we consider selection in rats and humans.

B. THE GENERALISTS OR OMNIVORES

Versatility and flexibility in choice of foods is a great asset. In a changing environment, it is a much less Spartan solution than massive deaths resulting from natural selection against specialists whose food is on the decline. Omnivory allows a change in preferred basic food to occur within an organism's lifetime rather than over many generations. A true omnivore such as cockroach, man, or the rat, considers anything of potential nutritional value as a possible food.

The problem is that experimentation with new foods can be dangerous, since such substances can be harmful. On the one hand, the omnivore should be familiar with and in touch with the various food sources in its environment; on the other hand, this involves risks, particularly needless risks, if there is already adequate familiar food. One sees in some omnivores, particularly the Norway rat (*Rattus norvegicus*) a fascinating conflict arising from these opposite forces: a distinct exploratory tendency, coupled with an often powerful avoidance of new things (neophobia). The optimal solution to the omnivoral problem involves devoting quite a bit of brain circuitry to the food problem, and employing multiple mechanisms. Thus, we see instances of built-in programming, modification through "general experience," more traditional learning, imprinting, social interactions, and culture or tradition all playing a role in food selection. The object is to explain the great success of at least some omnivores such as roaches, rats, and humans as indicated by their incredible numbers and resistance to annihilation. After building up a picture of food selection in rats, I shall turn to man, to see what common and what new principles are needed to account for man's diverse food habits and cuisines.

II. RATS: AN EXAMPLE OF SUCCESSFUL GENERALISTS

Richter's classic demonstration (Richter, 1942–1943, 1955) (see Fig. 1) of the "cafeteria" seems a most effective starting point. Left to their own devices, with

a large variety of purified ingredients, most rats self-select wisely and grow about as well as rats on nutritionally balanced laboratory diets. Some of this might be accounted for as a generally broad sampling pattern since acceptable ranges for most nutrients are quite wide. Whatever contribution such nonspecific factors make, the basic validity of Richter's work is demonstrated by the challenges he presented to the rats, within the cafeteria self-selection situation. Induction of an increased need for sodium via deficient diets or adrenalectomy led to appropriate adaptive changes in sodium intake. Similarly, vitamin deficiencies resulted in increases in intake of the appropriate vitamin; parathyroidectomy, with the induced Ca^{2+} loss, led to increased Ca^{2+} intake; and diabetes mellitus resulted in a shift from reliance on carbohydrate to greater reliance on fat and protein. We must attempt to explain these behaviors, often called specific hungers. Moreover, we need to examine how, in the wild, rats discover and test new foods and how they strike the balance between exploration and neophobia. Most of the work discussed involves domesticated rats as subjects, but the major phenomena are present in both wild and domesticated animals.

A. THE SPECIALIST WITHIN THE GENERALIST

The list of substances required by the Norway rat (or, with little modification, man) is large and impressive. It consists of thirty to forty different components, including water, nine amino acids, a few fatty acids, at least ten vitamins, and at least thirteen minerals, and involves, in some instances, critical levels of these (National Academy of Sciences, 1962). For three required "nutrients," each of extraordinary importance, rats behave as specialists, with a rather fixed, largely genetically determined, selection system. These three substances are oxygen, water, and sodium. I will not discuss oxygen intake here, since it is not quite a form of ingestion, but it does share important features with the other two. The main difference is that breathing virtually guarantees adequate oxygen, since oxygen is quite uniformly distributed in air, so that no specific recognition system is needed: only a detector hooked into the motor side. A fourth special partly built-in system regulates calories or energy intake. It is more complex than the other systems because it involves, in varying degrees, almost the full range of acceptable foods. All four of these systems have the characteristic of being absolutely basic and of representing "substances" that must be present in the organism within a limited density range. In short, all four are rather precisely regulated. Compared to other essentials, such as vitamins, a severe lack, imbalance, or cutoff of these four components can lead rather quickly to death. [A fifth possible candidate might be protein. There is evidence for regulation of protein intake, although little is known about recognition of protein sources (Rozin, 1968b).]

No wonder, then, that relatively foolproof, rather fixed solutions have been found. The importance of these substances to survival is such that much of internal metabolic physiology is devoted to their defense—hence the existence of liver and kidney. However, since all four components are dissipated over time, internal homeostasis cannot do the job by itself, and ingestion and, hence, behavior, must be harnessed to the system. Richter was the first scientist to clearly demonstrate this link. To illustrate the specialist within the generalist, I will describe briefly the systems involved in water, sodium, and calorie selection and regulation.

1. Water Hunger

Rats and probably most other mammals come equipped with detector systems that indicate the state of body fluids vis-à-vis the need to ingest water. There appear to be two detector systems, that sense some aspects of the tonicity of intracellular fluid and the volume of the intravascular space (Stricker, 1973; Epstein, 1973). The result of a signal indicating water deficit is presumably a unique sensation that we call thirst. Of course, other factors contribute to the sensation, such as temperature or dryness in the throat. The thirst sensation arouses exploratory behavior. The question is whether the target for this search, water itself, is prewired into the organism or whether water's ability to reduce the thirst sensation is discovered through experience. In other words, Is there built-in specificity both in terms of unique internal state with its own detectors and in terms of a system for identifying the target substance?

Surprisingly, we know very little about water recognition in rats. Rats do not drink water until just before the time of weaning (Teitelbaum *et al.*, 1969), suggesting that if it is built in, water recognition is late in maturing. At the moment, there is no simple way of distinguishing between absence of thirst sensation (e.g., absence of functioning internal state detectors) or failure to recognize water. However, the existence of a specific water-recognition mechanism is suggested by reports of a characteristic water response in taste receptors (Zotterman, 1956; Bartoshuk, 1972), although the response seems to vary markedly depending on the state of adaptation (Bartoshuk, 1972). Whatever the specificity for recognition of water in the mouth, it is hard to imagine how visual recognition of water could be prewired, given the various visual forms that water may take. (It is anecdotally reported that following removal of congenital cataracts, a human patient was unable to immediately recognize water.) There is almost certainly an important role for experience in the "distal" recognition of water. The relative simplicity of chemical as opposed to visual recognition of water is indicated by the fact that chicks seem to have built-in water-taste recognition, but must rapidly learn its appearance (Morgan, 1894; see also discussion in Section II, B, 6).

2. *Sodium Hunger*

Sodium hunger is the example, par excellence, of a genetically determined specific hunger. Its properties were first described by Richter in the late 1930s (Richter, 1936; for reviews, see Richter, 1956; Denton, 1967; Nachman and Cole, 1971). Richter showed that rats normally have a preference for low concentrations of NaCl in water. This preference was enhanced, and extended to both lower and higher concentrations, by induced sodium deficiency. Similarly, in natural periods of increased sodium utilization, such as pregnancy, increases in intake and preferences occurred (Richter and Barelare, 1938; for a review, see Richter, 1956). Richter believed the increased sodium preference in the face of increased sodium need was innate, expressed in part by a drop in the absolute threshold for detection of NaCl, since the minimum preferred NaCl concentration was lower in deficient animals (Richter, 1939). The preference was specific to sodium: it appeared with various sodium salts (e.g., the chloride, phosphate, or lactate) but not for the equivalent salts of other anions (e.g., potassium) (Richter and Eckert, 1938; Nachman, 1962). Richter's theorizing about both the innateness and the sensory threshold drop has formed the focus of research in the field since his early work.

On the issue of innateness, Richter's original explanation has been strongly confirmed. The detection of sodium deficit seems to be a part of the prewired, body fluid regulation system. Since sodium is the major extracellular electrolyte, this should not be too surprising. Changes in electrolyte concentration, including hyponatremia, hypovolemia, and changes in mineralocorticoid levels have all been implicated as triggers of sodium hunger (see Stricker, 1973, for a discussion of the physiological conditions necessary and sufficient to release sodium appetite).

It is the innateness of sodium recognition, rather than internal detection, that is of particular interest here, and the data are impressive. The evidence indicates that sodium recognition is mediated by the sense of taste (Richter, 1956). I will describe here only three of many lines of evidence indicating that the increased, taste-mediated sodium appetite accompanying sodium deficiency is innate.

First, there are taste receptors that are especially sensitive to sodium salts. Sodium ions, and sodium chloride in particular, seem to define a basic taste modality and produce characteristic electrophysiological responses (Pfaffman, 1959; Bartoshuk, 1972). What could the function of these receptors be except to signal the presence of this critical element? Sodium specificity of taste receptors must be related to the ability of sodium-deficient rats to show a preference for a variety of different sodium salts. And given the existence of sodium receptors, how easy it would be to connect them into an existing internal sodium-detection system.

Second, sodium hunger appears immediately upon exposure to solutions containing sodium. The critical point here is that the first time a rat is made

sodium-deficient, it shows a preference for sodium salts in solution in less than a minute (Nachman, 1962; Handal, 1965; Quartermain *et al.*, 1967). Since the deficiency and the solutions were not experienced before and since the preference occurs before postingestional effects could occur, the argument for innateness is strong.

Third is the exception that really proves the rule. There is one way to fool sodium-deficient rats and that is to offer them lithium salts. Rats behave toward these salts as they do toward sodium salts. In an elegant series of experiments, Nachman (1963a,b) has turned this exception into a telling proof of the innateness of sodium hunger. Sodium-deficient rats cannot prefer lithium for its effects, since, although it has similarities to sodium as an electrolyte, it produces rapid toxic effects. In fact, LiCl has emerged as the poison of choice in experiments on rat poison avoidance (see below). To humans, LiCl and NaCl taste almost identical. For this reason, LiCl was used for a while as a salt substitute. If lithium salts tasted like sodium salts to the rat, then an innate mechanism could be triggered into making a dreadful mistake by consuming a poison that tastes like the built-in "target" substance. Nachman was able to demonstrate that lithium and sodium salts do taste almost the same to rats. He poisoned rats after they consumed a sodium salt solution, so that they avoided this solution on future encounters. He now tested for generalization of the avoidance or aversion by measuring intake of other solutions in brief exposures. The results were that sodium chloride aversion generalizes completely to lithium chloride, and much less so to potassium, ammonium, or other chloride salts. [Under appropriate circumstances, rats can discriminate sodium from lithium salts (Harriman and Kare, 1964; Balagura *et al.*, 1972).]

There is plenty of opportunity for experience to supplement and modulate sodium hunger. Rats must still learn where to find sodium. Krieckhaus and Wolf (1968; Wolf, 1969) trained normal rats in a two-lever box, where one lever delivered a low-concentration sodium solution as a reward and the other water. These rats had never been sodium-deficient. After establishing baseline pressing rates for the two levers, rats were made sodium-deficient. On returning to the box, under extinction conditions (no fluid delivery), an enhanced preference for the sodium lever was shown. The rats had learned where sodium taste could be found. With deficiency and enhanced preference, they applied their learning immediately. Rats can also learn to avoid foods that are sodium-deficient. They will reduce their food intake rather than continue to consume a sodium-deficient diet and will show an immediate preference for a new food over the sodium-deficient food they had previously been eating (Rodgers, 1967a,b).

When sodium hunger is pitted against training to avoid sodium, the strength of sodium hunger becomes apparent. Sodium hunger appears even in rats that had a previously established sodium aversion by association of sodium solution intake with poison (Stricker and Wilson, 1970). Furthermore, sodium-deficient rats will

not abandon their sodium preference even when poisoned after drinking it (Frumkin, 1971).

Since I shall argue that the majority of what are called specific hungers are basically acquired, the question arises as to why sodium (but not calcium, vitamin B_1, etc.) hunger should be innate. I can offer four somewhat related reasons.

1. Sodium is of particular importance in body fluid homeostasis, and a foolproof mechanism of regulated ingestion would have high adaptive value.

2. There are significant shortages of sodium in some geographic areas, so the likelihood of deficiency is rather high. The existence of animal salt licks and salt mines exploited by man testify to the unequal distribution of salt in the environment.

3. Probably as a consequence of reasons 1 and 2, there exist both sodium-sensitive taste receptors and internal detectors, which would neatly serve an innate recognition system.

4. It is just possible that learning about the positive effects of sodium would be difficult, since it might be that the initial effects of sodium ingestion by a sodium-deficient animal might be negative. Rodgers (1967b) was unable to induce a preference for a neutral substance in sodium-deficient rats, when ingestion of this substance was immediately followed by intragastric intubation of NaCl. Since intragastric delivery of food or water can serve as a reinforcement (Miller and Kessen, 1952; Epstein and Teitelbaum, 1962), this failure suggests that the concentrations of NaCl used may produce negative effects in the gut.

Richter's second supposition, that sodium hunger was mediated by lower absolute threshold, has met a more uncertain fate. This mechanism, if it did exist, would only explain part of the phenomenon. It would not, of itself, account for increased preferences for higher concentrations. The upshot of a number of experiments on the existence of lowered absolute thresholds has been that it is the preference rather than the absolute threshold that is lowered in sodium deficiency. Normal and sodium-deficient rats, when tested appropriately, show the same absolute thresholds for NaCl (Carr, 1952; Harriman and MacLeod, 1953; Koh and Teitelbaum, 1961). There was also no difference in the absolute thresholds determined electrophysiologically (Pfaffman and Bare, 1950). The difference between them was that deficient rats start preferring sodium soltuions at the absolute threshold level, whereas normals are indifferent to the lowest sodium levels. However, there is evidence that there may, indeed, be increased sensitivity to low concentration of sodium in deficient rats or humans (Yensen, 1959; McBurney and Pfaffman, 1963; Henkin *et al.*, 1963). This apparent increase appears to be related to the drop in sodium levels in the saliva during deficiency. This changes the adaptation level to sodium and increases sensitivity (McBurney and Pfaffman, 1963).

The adaptation level notion has interesting implications for the detection of needed substances, because it implies that solutions will taste salty only when they have a higher concentration of salt than the existing body level, as represented in the saliva. Thus, the adaptation would neatly provide a characterization of all those concentrations of biological utility at the moment (Desor, personal communication). It is suggested that it is not so much a change of threshold as a change in "classification" of the stimuli: concentrations of sodium below those in normal saliva, which might have been discriminable but were not "salty" in taste to normals, would taste salty in the sodium-deficient organism, since they would represent higher sodium levels than those in deficient saliva. All of these interesting matters may have little to do with sodium hunger. As pointed out above, changes in threshold or classification for very low concentrations cannot account for most of the characteristics of sodium hunger, which are clearly present in reactions to high concentrations.

In summary, in the case of sodium hunger or appetite, there is a built-in sodium receptor, a built-in system for detecting the body's state of need for sodium, and some built-in linkage between them (presently not understood), in which the motivational value (or preference for) the incoming sodium signal is modulated by the report of the internal detector.

3. *Calorie Hunger*

Calorie hunger, or what is usually called just plain hunger, has a rather anomalous position as a specific hunger. There is an elaborate machinery, only partly understood, to detect energy imbalance in mammals, and evidence that similar control systems are at work in other groups, such as fish (Rozin and Mayer, 1961, 1964; Rozin, 1964). There have been suggestions that this system operates by detecting levels or amount of utilization of specific substances, such as glucose (Mayer, 1955). The presumably prewired regulation system is modified in very significant ways by experience; otherwise, it would be difficult to explain, for example, adjustments in meal size made when caloric density of food is varied. It is also possible that some eating may be viewed as a way of avoiding a hunger signal (see Le Magnen, 1971, for a general discussion of the role of experience in the regulation of food intake). The major significance of regulation of energy balance is indicated by the fact that it takes precedence over acquisition of sufficient amounts of many other essentials; for example, rats will not overeat calories in order to obtain adequate amounts of protein (Andik *et al.*, 1963) or water (Bruce and Kennedy, 1951).

The peculiar feature of the specific calorie system is that there is no simple way of specifying what substances in the environment are adequate sources of calories, i.e., no possible equivalent to a sodium or water receptor. Given a limited range of foods, as with the great cats, specification of the class of foods can be accomplished and, hence, preprogrammed. However, true omnivores base their success on their ability to tap the widest range or sources of nutrition.

Thus, omnivores must basically learn what is food, which usually means calories, and what is not food. Undoubtedly, there are biases in exteroceptors, most especially in chemoreceptors, that relate to food recognition. Sugar-sensitive receptors, common in a wide variety of animals, undoubtedly serve such a function (although their presence in carnivores is a bit puzzling). Thus, the complexity and variability of the food (calorie)-recognition problem requires a great deal of plasticity. In this case, then, there is an elaborate and substantially prewired regulation-detection mechanism and a rather loosely constrained, plastic recognition component.

B. THE RAT AS A GENERALIST

Extension of specific innate mechanisms to handle the full range of food selection in rats is unthinkable, both in fact and in principle. One should have to postulate the equivalent of a full table of nutritional essentials in the rat's head. For each component, there would have to be a unique specific sensory message and a unique central state characterizing the deficiency and sensed by a specific detector. The incredible amount of machinery needed for this would, for the most part, remain unused during the lifetime of the animal, since it is probable that a given animal does not experience most specific nutritional deficiencies in its lifetime. Moreover, the selection behavior of animals deficient in most dietary essentials does not show the certainty, directedness, and rapidity seen in sodium or water deficiency.

In particular, poison avoidance cannot, in principle, be explained with a specific, built-in mechanism, since some poisons successfully avoided by rats are man-made and were synthesized or made available for the first time during the to-be-poisoned rat's very own lifetime. Clearly, here we must assume an ability to learn about dangerous foods.

In the past, the main argument in favor of innate-type mechanisms for most specific hungers was the inability to find a reasonable way of explaining how they *could* be learned. In principle, according to this argument, these specific hungers could not be learned because the interval between ingestion and consequences was too long (at least 30 minutes) and violated the basic law of temporal contiguity for associations. Of course, the great advantage of a mechanism through which a rat learns what foods make it sick and what foods make it feel better is that with one basic system, the whole host of nutritional and poison-avoidance problems can be solved. This simplicity apparently appealed to mother nature.

1. *Poison Avoidance – Background*

Rats and man have been locked in a fierce battle of wits probably since the earliest days of civilization. In the twentieth century, on man's side, has been an

experimental attempt to find ways to eradicate local rat populations through poisoning. A critical problem in the field has been to get rats to ingest enough poison. A wide variety of poisons has been tried (see Chitty and Southern, 1954, for a general review). Workers in this field, nonpsychologists on the whole, have assumed that rats learn to avoid poisons. Their evidence was overwhelming in amount, although critical experiments were not done. Wild rats (Rzoska, 1953), when faced with a new food, become extremely "shy" and "suspicious" and may avoid it for long periods. When they finally ingest it, they take a very small amount and go away, as it were, to "test" the food. Should the food contain poison, the mild effects of the small amount of poison lead to a learned aversion to the food, and the rat does not return. For this reason, rat exterminators often use a procedure called "prebaiting." The rat is first offered the vehicle (diet) in which the poison will later be placed. The rat's initial neophobia to the vehicle thus dissipates, and it becomes an accepted part of the diet. Now, a poison is added. To the extent that the poison is potent, tasteless, and odorless and does not change the texture or appearance of the food, it may be accepted in large quantities and be successful. The method has had fair success, but the eradication of local rat populations is still outside man's capabilities.

The considerable literature on poisoning has been unknown to most psychologists, until very recently. Ironically, the "father" of specific hungers, Curt Richter, is one of the few psychologists who made contact with it. Richter has done some of the finest experiments in the area and participated in development of a major rat poison (Richter, 1950, 1953). He also showed (Richter, 1953) the extent to which the wild rat's aversion to new things, especially foods (neophobia) can be augmented by poisoning experiences. He produced some rats so suspicious of new foods, on account of successive poisonings, that they starved to death rather than try additional new foods. The work of Richter (1950, 1953), Barnett (1956, 1963), and Rzoska (1953), all with some significant exposure to the psychological community, raised fundamental issues regarding the psychology of learning and suggested the critical importance of neophobia and responses to novel vs. familiar stimuli in the rat world. The work was not assimilated into psychology (as indicated by its absence from texts through the 1960s and into the 1970s). I believe this occurred (*a*) because the work dealt primarily with wild rats, organisms avoided by psychologists (the feeling was probably mutual), (*b*) because the work challenged some dearly held beliefs in the psychology of learning, and (*c*) because the work was not presented in "mainstream" psychology journals. However, the situation has now changed, as two lines of research within psychology have converged on the major issues raised by poison avoidance: the existence of special learning processes and the importance of the novelty-familiarity dimension. These two approaches—the further explanation of specific hungers and the analysis of poison avoidance within experimental psychology, together have led to reconsideration of some previously accepted views in the psychology of learning.

2. *Poison Avoidance – Recent Work*

Parallel to the rat poisoning studies, and closer to the center of experimental psychology, were a series of investigations on the effects of X-irradiation on behavior. This work included reference to the fact that rats tended to avoid foods whose consumption was followed by X-irradiation. [see Garcia *et al.* (1961) and Smith (1971) for reviews]. It was from the basic work of Garcia, Smith, and their colleagues that the ground was laid for findings which would have vastly more generality than the confines of X-rays and their effects on food preferences.

Two critical experiments by Garcia and his colleagues solved the basic problem of how learning principles could explain X-ray- or poison-induced aversions. The experiments were simple and incredibly brief (a total of 4 pages in all), which is fitting for studies of major importance. One problem for a learning interpretation of poison avoidance is that foods and feeding must be selectively associated with poisons, even though other behaviors (running, sleeping) and their consequences were as closely associated in time with poisoning as the feeding. How could the rat "know" what was relevant? On the basis of what was known in the psychology of learning, the answer was that the rat could not and that poison avoidance was therefore a true mystery.

Garcia and Koelling (1966) did a simple but powerful experiment demonstrating that rats do "know" what is relevant (although it was not explained how they knew). Thirsty rats were given "bright, noisy, and tasty" water to drink; that is, they were given flavored water, and each time they licked it a light flashed, a buzz occurred, and, of course, the taste was experienced. Following a brief drinking session, half the rats were poisoned by injection or X-irradiation and the other half were punished by strong electric shock to the feet. On a subsequent day, rats were tested to see whether they had developed an aversion to the taste and/or the light or sound. Rats that had received poison or X-irradiation would not drink the flavored water, but would drink plain water, when licks of this were accompanied by the light and sound. Conversely, the shocked rats avoided "bright, noisy" water but did not avoid the taste. Garcia and Koelling described this important finding as an instance of "belongingness." [This idea along with relevant data are present in a less clearly defined form in prior work by Capretta (1961) and Braveman and Capretta (1965).] According to this notion, certain stimuli preferentially associate with certain others; in particular, tastes and possibly smells associate selectively with a set of internal visceral events that include gastrointestinal disturbances. This particular linkage is obviously perfect to handle food selection, since food enters by the mouth (hence taste) and produces gastrointestinal and metabolic consequences. The important taste-visceral link and the more general "belongingness" principle have been further amplified and extended (Garcia and Ervin, 1968). (Shettleworth (1972) has reviewed a wide variety of examples of specificity in associations,

which she calls "constraints on learning." Seligman (1970) has extended the notion to a general principle of learning, called "preparedness" [see also the recent volumes: "Constraints on Learning" (edited by Hinde and Stevenson-Hinde, 1973) and "Biological Boundaries of Learning" (edited by Seligman and Hager, 1972)].)

To date, the limits of the taste-visceral system have not been defined. There is very little work on the relevant visceral field [e.g., Would pain in the chest be an unconditioned stimulus (US) for a taste conditioned stimulus, (CS)?]. Tastes appear more associable with gastrointestinal events than smells, although there is clear evidence for "smell-aversion learning" (Garcia and Koelling, 1967; Pain and Booth, 1968; Lorden *et al.*, 1970; Domjan, 1973). It is possible, however, to "associate" exteroceptive cues with internal malaise, but the conditioning process is much longer and the results less impressive (Garcia *et al.*, 1961; Rozin, 1969a). Widening the horizon beyond rodents, it appears that the general principle here is that animals tend to associate food-related stimuli with the type of consequences that foods produce (Rozin and Kalat, 1971, 1972). In the case of rats, this leads naturally to the taste-gastrointestinal linkage; in the case of quail, which identify food visually, it leads to preferential association of visual characteristics of food with gastrointestinal consequences (Wilcoxon *et al.*, 1971).

Coupled with the major finding on belongingness and taste-visceral linkages, Garcia *et al.* (1966) published another paper, at about the same time, which is leading to major revision in our view of learning and to great advances in our understanding of mechanisms of food selection. They showed clearly for the first time that rats could learn to avoid a solution (CS) if its gastrointestinal consequences (US) occurred an hour of more after ingestion. This long-delay learning was only demonstrable with taste (or smell) CSs and gastrointestinal or metabolic USs (Garcia *et al.*, 1972; Rozin, 1969a). This is, of course, as it should be, since only in the feeding system are the initial events (tastes) separated significantly in time from their (metabolic) consequences; the gut induces an inherent delay. In the rest of life, e.g., predator avoidance, causes and consequences follow rapidly in time.

Garcia, Ervin, and Koelling originally demonstrated the long-delay learning by using saccharine solutions as a CS and apomorphine injection as a US. Previous studies by J. C. Smith and his colleagues (reviewed in Smith, 1971) and Garcia's group had all flirted with this fundamental new phenomenon. However, Garcia, Ervin, and Koelling were the first to physically separate CS and US by long time intervals, so that it could not be argued that there were early, subtle immediate effects of the US (as when a poisoned solution is drunk) that provided temporal contiguity.

The long-delay finding is of such significance for both learning theory in general and food selection that it has been subject to rather intensive scrutiny and study, particularly by Revusky and Garcia (1970; Revusky, 1971) and by

Rozin and Kalat (1971, 1972; Kalat and Rozin, 1973). The following is a summary of what is known about this system.

1. Long-delay learning has been clearly demonstrated for intervals as long as 8 (Revusky, 1968) or 12 hours (Smith and Roll, 1967). There is evidence that with anesthesia administered for the period between CS and US, the CS–US interval could be extended indefinitely (Rozin and Ree, 1972).

2. Long-delay learning is limited to taste and smell CSs and an unknown class of visceral USs that include gastrointestinal stimuli.

3. Long-delay learning occurs rapidly—in most cases in one trial.

4. Long-delay learning lasts a long time but can be extinguished rather easily. The adaptive fit of this and the previously stated features with the problems of poison avoidance and food selection should be obvious.

5. Long-delay learning cannot be explained as a peripheral phenomenon, e.g., aftertaste contiguous with sickness. Evidence against this view has been reviewed (Rozin and Kalat, 1971; Revusky and Garcia, 1970) and includes the following: (*a*) aftertastes would hardly be likely 6 hours or more after drinking (Revusky, 1968; Smith and Roll, 1967); (*b*) rats can quickly learn to avoid a particular concentration of a solution, which would be hard to do on the basis of aftertastes (Rozin, 1969a); (*c*) quail show long-delay learning for food-related visual stimuli, in which aftertaste cannot be involved (Wilcoxon *et al.*, 1971).

There is at present no clearly correct explanation of the mechanism of long-delay learning. Three theories have been suggested. In brief, a *trace decay* notion, asserting, simply, that the associability of taste memory traces decays less rapidly than other memory traces (Rozin and Kalat, 1971, 1972); an *interference theory* (Revusky and Garcia, 1970) asserting that limits on the length of the CS–US interval are always produced by retroactively interfering stimuli (in the taste-visceral system, with only tastes as relevant stimuli, very little taste interference occurs); and a *learned safety* approach (Rozin and Kalat, 1971, 1972; Kalat and Rozin, 1973), asserting that what rats really learn is what is safe and that they learn this gradually over time. There is some evidence for each of these views, which are, in fact, mutually compatible.

3. *Specific Hungers*

a. Thiamine-Specific Hunger. I shall now describe the mechanisms of specific hungers and relate them to poison avoidance.

Specific hungers exist in two forms: (*1*) the self-selection of adequate diets by healthy rats and (*2*) the adaptive selection of specific nutrients by animals deficient in those nutrients. I will concentrate on the latter, as it is better understood and may help to explain selection in healthy rats.

The prototypical, simple specific hunger experiment involves raising an animal on a diet, D, deficient in element X, and, when deficiency signs appear, offering the animal a choice between diet D and diet D + X. More complex versions involve using more choices.

I will focus on the simple setting, and discuss the specific hunger for vitamin

B_1 (thiamine), as the best-investigated example. Thiamine deficiency, in young rats, produces clear deficiency signs of anorexia and weight loss within a few weeks after thiamine is removed from the diet. Classic studies by Richter *et al.* (1937) and Harris *et al.* (1933) clearly demonstrated a preference for thiamine-rich foods by thiamine-deficient rats (Fig. 2). These studies were subsequently

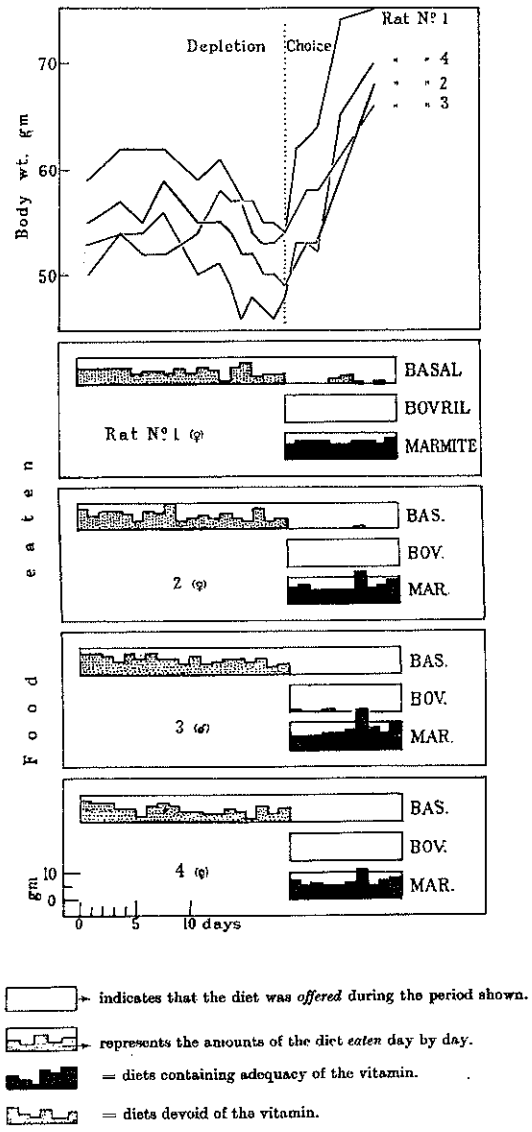


FIG. 2. Preference of 4 vitamin B complex-deficient rats for a diet (Marmite) containing B vitamins. (From Harris *et al.*, 1933.)

confirmed and extended (Scott and Quint, 1946a; Scott and Verney, 1947; Rozin *et al.*, 1964). Work by Scott and his colleagues (Scott and Verney, 1947) suggested that there was no specific recognition of thiamine per se. When they added a distinctive anise flavor to the diet with thiamine, the rats developed a preference for the thiamine-anise diet. The anise flavor was then switched to the deficient diet, and the rats now preferred the anise-deficient diet. The upshot of the early work is that thiamine hunger appears reliably and rather rapidly (within 1 day).

In its simplest and historically most commonly accepted form, a learning explanation of specific hungers assumes the following sequence. A rat is deficient in X and, presumably, feels sick. It encounters, among other foods, a food containing X, and eats some. It starts feeling better and is, thus, reinforced for eating X. Hence, a preference for X develops. In spite of experiments by Harris *et al.* (1933) and Scott and Verney (1947) demonstrating something like this, the conflict with basic learning principles was too great to convince psychologists that some specific hungers were learned. Specific hungers, when discussed at all in elementary textbooks were mentioned only under the heading of motivation. In addition to the serious long-delay problem, there was a problem in explaining how foods (as opposed to light or sounds, grooming, etc.) would specifically be associated with their consequences. The notion that the positive reinforcement of beneficial consequences following on ingestion of enriched food explains specific hungers had two other serious shortcomings: (*a*) rats failed to show a vitamin B_1-specific hunger when the choice was water vs. vitamin-enriched and flavored water, even though vitamin-enriched water produced the same recovery as vitamin-enriched food (Rozin *et al.*, 1964); and (*b*) rats that had recovered from deficiency by injection of thiamine showed a preference for thiamine-rich foods when they were presented for the first time *after* recovery. Under these circumstances, the vitamin in the preferred choice should have no particular positive effects (Rozin, 1965). Thus, we have absence of specific hunger when the positive reinforcement condition is fulfilled (*a*), and presence of the specific hunger in the absence of need, i.e., no positive reinforcement (*b*).

Observation of rats while they became deficient and in subsequent choice situations led to a resolution of this puzzle (Rozin, 1967a). In the deficiency period, when the standard deficient food is presented after a period of food privation, rats avidly approach the food cup, sniff at it, and then either spill the food with their paws or walk away and chew on something inedible in the cage. This is the same type of behavior as is shown by normal rats when they are offered a highly unpalatable, quinine-adulterated diet. This "displacement" or "redirected" behavior suggests that the deficient diet is *aversive* to the rats. If given a new diet, these same rats consume it avidly. The aversion conception gains force with the observation that when rats made deficient on diet A, and

recovered on diet B, are offered diet A again, they will not eat it, even if food deprived. When a hungry rat prefers eating nothing to a particular diet, it seems fair to call that diet aversive.

But, if there are aversions to deficient diets, then the following conclusions may be drawn.

1. Vitamin-deficient diets are like slow poisons, and the literature on poisoning is relevant to specific hungers [in fact, it was this specific aversion experiment that connected diet deficiency to the researches on poisoning (Rozin, 1967a,b)].

2. The critical learning apparently takes place during exposure to the deficient diet, not at the time of choice.

3. The preference first manifested after recovery (Rozin, 1965) is not a problem, since it can be seen as a retained aversion to deficient diet (Rozin and Rodgers, 1967).

4. Given that rats do not stop eating per se, but rather stop eating a particular diet, what has been learned appears to fit better into a classical-aversive paradigm than into the suggested operant-positive reinforcement scheme. This is supported by the fact that taste-aversion learning may occur by association of taste or smell with gastrointestinal upset in the absence of ingestion (Domjan and Wilson, 1972a,b; Bradley and Mistretta, 1971).

5. With points 1-4, the Garcia experiments (which appeared concurrently with and independently of these specific hunger experiments) seem to provide a basis for a learning explanation. The belongingness principle takes care of the food–illness association. In fact, independently of Garcia, Rozin (1967a) showed that deficient rats, although avoiding their deficient food, did not avoid the food cup or its location—only its contents. In essence this is another, but less elegant, version of the Garcia belongingness effect.

Thus to some extent at least, thiamine-specific hunger can be described as an aversion to thiamine-deficient diet, learned with the special belongingness and long-delay abilities of the feeding system. A full understanding of thiamine and related specific hungers involves considerably more than this, however. We must yet consider (*1*) the critical novel–familiar distinction—its potency as a food classifier and importance in determining specific hungers; (*2*) the possibility that, in addition to learning what is bad for them, rats can learn what is good; (*3*) mechanisms through which (moving toward cafeteria situations) rats select enriched food in complex multichoice situations; and (*4*) the extent to which what we have described for thiamine deficiency also holds for other essential nutrients.

b. The Novel–Familiar Dimension. Research on specific hungers (Rodgers and Rozin, 1966; Rozin, 1968a) has clearly highlighted the importance of past experiences with foods on rat behavior. In a very real way, the food world of a rat consists of those foods never before tried (novel) and those previously

sampled (familiar). The familiar category subdivides into three subcategories: harmful, neutral, and "beneficial" foods. The marked negative response of wild rats to new events or objects in their environment, especially foods, is well known among rat exterminators. Rats, most especially wild rats, tend to stick with familiar foods. The way to get a clear preference for a new food, in rats, is to offer a choice between a familiar aversive diet and a new food (Rodgers and Rozin, 1966; Rodgers, 1967a). Poisoning (or deficiency) experiences are almost by definition situations in which a new food (new by virtue of presence of poison or absence of essential nutrient) is associated with aversive consequences. Rats appear to become more and more neophobic, the more experiences of this type they have (Richter, 1953; Rozin, 1968a). Rats that have been poisoned or deficient show an increased preference for old "safe" familiar foods, and an increased avoidance of new ones (Rozin, 1968a). This is true for both wild and domestic rats, the difference between them being simply a more generally neophobic base line for wild rats (Rozin, 1968a).

The novel-familiar dichotomy allows an important stimulus selection principle to operate: rats tend to associate new events (e.g., new CSs) with new consequences (e.g., new USs). Hence, in a confounded situation in which a new and a familiar food are both consumed prior to poisoning, only the new food acquires a significant aversion (Revusky and Bedarf, 1967; Wittlin and Brookshire, 1968; Kalat and Rozin, 1973). Only one prior experience with a previously new food, followed by neutral or positive consequences, suffices to make that food strongly resistant to becoming aversive (Kalat and Rozin, 1973). However, a few minutes of exposure to a food is required for it to become effectively familiar (Domjan, 1973). I might add, parenthetically, that the novelty effects make obvious sense in an adaptive framework, as do the belongingness and long-delay capacities.

c. Learning Preferences as Well as Aversions. The original formulation of specific hungers was in terms of learning about the beneficial consequences of an enriched food. We have seen that, in fact, the major phenomenon appears to be learning about aversive consequences of deficient foods. The question remains: Can rats learn about the positive effects of foods? The answer seems to be yes. A number of investigators (Garcia *et al.*, 1967; Zahorik and Maier, 1969; Revusky, 1967) have reported enhanced preferences for substances whose ingestion is followed by an improvement in "physiological state" (thiamine or caloric repletion). The effect is usually relatively small and appears only over a number of conditioning trials in contrast to poison avoidance. One study (Seward and Greathouse, 1973) directly comparing "positive" learning about recovery from thiamine deficiency with aversion learning when thiamine deficiency is the aversive event found much more rapid and marked learning in the aversion paradigm. Recent evidence (Simson and Booth, 1973) also suggests that long delay intervals may be more characteristic of learned aversions to toxins than to learning with "nutritional reinforcers." These authors raise the important question of whether the fundamental distinction should be between positive and negative reinforcers, or "nutritional" (part of normal metabolic function) and toxic events.

The question is whether or not and under what circumstances rats discriminate between safe and particularly beneficial foods. In other words, does the food world of rats consist of three categories—novel, familiar-dangerous, and familiar-safe—or four, with the last category bifurcating into familiar-safe and familiar-beneficial. To distinguish between safe or neutral and beneficial foods, the food that is a candidate for positive preference (e.g., associated with recovery from thiamine deficiency) must be tested against a familiar-safe food. Until very recently the only study supporting such a positive preference has involved caloric repletion as the reward (Revusky, 1967). However, Zahorik *et al.* (1974) have just demonstrated a clear preference for foods associated with recovery from thiamine deficiency over familiar safe foods, indicating four functional food categories. The positive preference has obvious adaptive value. However, there is no doubt that rats, at least, are strongly biased toward learning effectively and rapidly what makes them sick, and rather poor at learning what makes them well. This makes sense for an animal whom everyone is trying to poison—paranoia in wild rats is consistent with contact with reality.

d. Selection among Foods in Complex Situations. Returning to our thiamine-deficient rat faced with a new garbage can, we must ask (with an obvious eye toward cafeteria-type experiments), how he uses the capacities described up to this point to, as it were, "find the good stuff." Long-delay learning makes it possible, belongingness effectively limits the candidates for dangerous or beneficial things to foods, and the novel-familiar dimension further restricts the number of "suspect" foods to the new ones. But, faced with a variety of new foods, how does the rat solve the problem? The first thing to realize is that under such circumstances, rats are often not successful (Harris *et al.*, 1933; Rozin, 1969b). Harris *et al.* (1933) showed that, if vitamin B-deficient rats were offered a choice among ten new foods, only one of which contained the vitamin, they typically did not show a selective preference for the enriched source. It was usually necessary to "educate" them by exposing them exclusively to the enriched choice for a period of days, following which they would show a maintained enriched food preference. However, such educational guidance is not to be expected in the real world. In the absence of educational guidance, and with a smaller number of new food choices, many rats do develop a preference for a single enriched food source (Harris *et al.*, 1933; Rozin, 1969b). How, then, does the rat learn which of the variety of foods present produces significant positive, or at least, nonnegative effects? How, if the rat consumes a number of foods at a time, can it specifically learn about the consequences of a particular food?

First of all, as mentioned above, the novelty principle simplifies the situation to some extent. Some of the available choices may have already been associated with deficiency, and will not be ingested. If there are, in addition, some familiar,

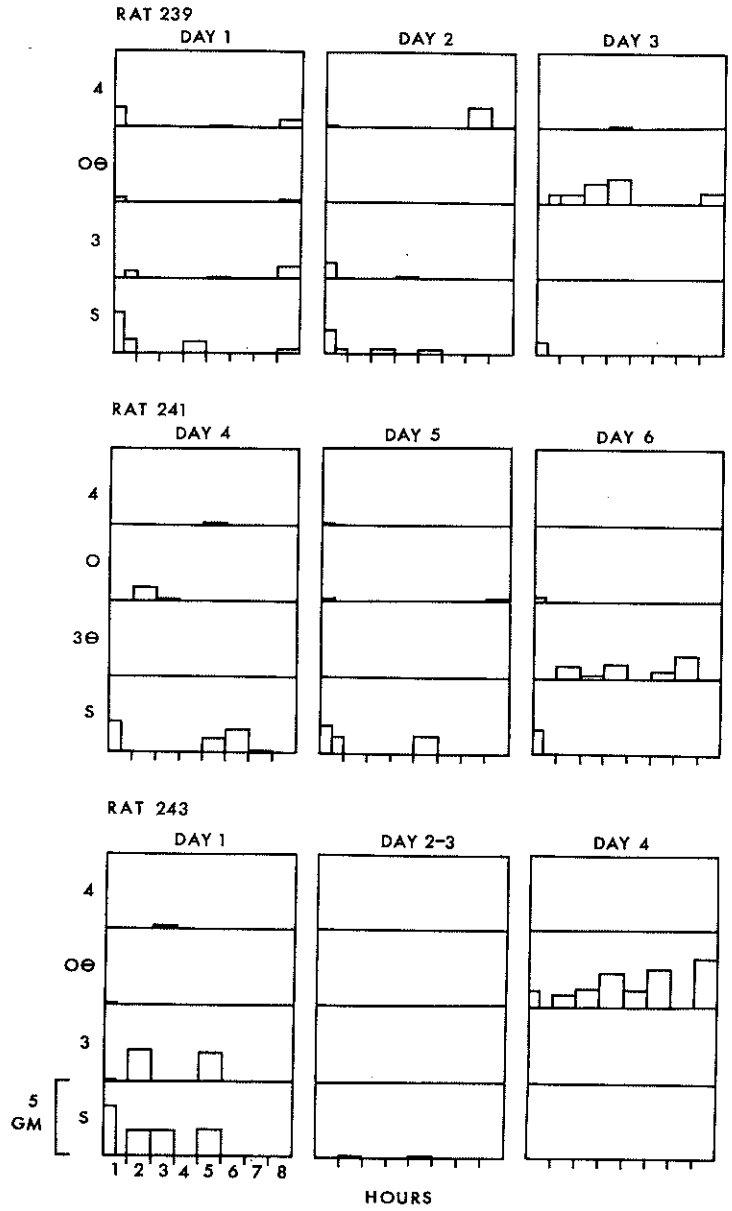
neutral (safe) or beneficial foods, say from the days before the deficiency, rats will explicitly prefer these. In their absence, the rat is faced with novel foods, among which it must choose. Another factor, which may simplify the task, is that all novel foods do not have an equal probability of being associated with toxic consequences. Some tastes are more likely to become aversive by conditioning than others (Kalat and Rozin, 1970; Brackbill *et al.*, 1971). We do not know, at this time, what characteristics make a particular taste more salient than others vis-à-vis poison association. There is evidence (Kalat, 1974) that novelty per se is an important determiner of salience. Salience could be of significant adaptive value if it was tied to the real world probabilities of given tastes being associated with natural poisons. For example, it appears that bitter tastes are often characteristic of poisonous plants, and the general negative response toward bitter tastes may well be, in fact, a built-in danger recognition mechanism.

The primary burden of selection among multiple food sources is borne by the rat's natural feeding pattern itself. Rats do not eat randomly in time. Their feeding is clustered into short bouts, appropriately dubbed meals, separated from one another by periods of 30 minutes or more. Observation of rats faced with multiple new food sources (Rozin, 1969b) (Fig. 3) indicates that any given meal tends to involve only one food source. In other words, rats seem to sample new foods one at a time. This pattern is exaggerated in deficient animals. The consequence is that each food can be evaluated in an uncontaminated fashion. The rat's natural feeding behavior simplifies a complex situation (see also Barnett, 1956). Observation of deficient rats faced with a number of new food choices suggests that it may be a period of days before a rat samples a single enriched source, but one or two meals from this source seem to be sufficient to establish a stable preference for it (Fig. 3).

In nature, rats are social animals. There are numerous possible avenues of social interaction which could facilitate some transfer of information about foods in the environment from one animal to another. Galef (see first chapter in this volume) has done some elegant studies demonstrating parent-child interaction in domestic rat colonies. In outline, if parent rats learn to avoid diet A and to eat diet B, their infants, at the time of weaning and separation from the parents will show the same preference pattern (Galef and Clark, 1971).

FIG. 3. Meal patterns of 3 thiamine-deficient rats faced with a choice of four foods. The rats were allowed to feed freely for 8 hours each day, with intake recorded every half hour or hour. Each rat, following a deficiency period on one diet, was offered a choice of this diet and three new diets. One of the new diets (indicated by a θ on the figure) was enriched with thiamine. The figure shows the intakes for 3 rats over a 3–4 day period, during which they "discovered" the vitamin-rich diet. This occurred during the first 3–4 days of testing for rats 239 and 243, and during days 4–6 for rat 241. Subsequent to these days, each rat ate the enriched choice almost exclusively for a number of days. Note the tendency for the rats to consume only one food at a time, and to show a maintained preference for the enriched food once they have eaten an isolated meal of it. (From Rozin, 1969b.) Copyright 1969 by the American Psychological Association. Reprinted by permission.

The preference for B over A in the young weanling rats has been shown to be explainable in terms of neophobia. Young rats will tend to eat familiar safe foods, and for them, diet B is familiar and safe for two reasons. First, before total weaning, young rats go out and feed in the environment along with their



parents. Since their parents feed on diet B, they will also and, thus, this diet becomes familiar (Galef and Clark, 1971, 1972). Second, salient characteristics of the diet eaten by the mother appear in the mother's milk (LeMagnen and Tallon, 1968; Galef and Sherry, 1973). Nursing pups experience chemical stimulation that resembles parental food, making it familiar and safe and, hence, preferable (Galef and Henderson, 1972). No doubt there are other social interactions among adults as well as between parent and child that are of great significance (Barnett, 1956).

e. Other Adaptations to Thiamine Deficiency. We have yet to exhaust the multiple mechanisms available to handle thiamine shortage. Vitamin B_1-deficient rats, as well as rats deficient in some other substances, show a marked increase in feces ingestion or coprophagy (Richter and Rice, 1945; Barnes, 1962; Rozin, 1967b). This has adaptive value, since the flora of the hindgut synthesize many vitamins, which can only be utilized by the host through feces ingestion. This was first demonstrated, it should by now be no surprise, by Richter and Rice (1945). They showed that rats on a cafeteria regime would ingest the fecal output of 2–4 rats per day, when this was their only source of B vitamins, and would remain healthy on this regimen (see Fig. 1). Removal of the feces led to onset of deficiency symptoms. Rats normally consume about 35–50% of their feces, even when kept on screen floors. Feces ingestion frequently rises to 100% in the case of vitamin deficiency (Barnes *et al.*, 1957, 1960; Barnes, 1962).

Rats that are B complex-deficient develop specific dietary habits which have the effect of prolonging survival. Fats spare thiamine; rats survive longer on a high fat than on a high carbohydrate or protein diet when placed on a thiamine-free diet (Scott *et al.*, 1950a). Clearly, a shift away from the normally high carbohydrate diet toward more fat would spare thiamine, and just such a shift has been observed in B complex deficiency (Richter *et al.*, 1938; Richter and Hawkes, 1941) (see Fig. 1) and with pure thiamine deficiency (Scott *et al.*, 1950a). The avoidance of sucrose could well be accomplished through the mechanisms already described, with fat emerging as the primary calorie source on the grounds that it produced the least aversive consequences.

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4. *Nature of Taste-Aversion Learning*

Taste-aversion learning appears to be a very low-level phenomenon—its impressive characteristics notwithstanding. It is probably widespread among the vertebrates. Its fundamental importance and high reliability suggest rather tight wiring. Seligman (1970) has described it as "prepared" learning and, hence, "primitive" or subcortical (see also Rozin and Kalat, 1971; Garcia *et al.*, 1970; Seligman and Hager, 1972); the evidence supports this view. Most notably, taste-aversion learning can occur in an anesthetized animal (Roll and Smith, 1972), where, presumably higher centers are selectively depressed. Furthermore, the Kamin ("blocking") effect, what might be considered a higher-order learning effect seen with exteroceptive stimuli, is difficult to obtain with taste-aversion learning (Kalat and Rozin, 1972). Since common sense and human experience seem, retrospectively, to have been very good guides in this field, we might risk a prospective look in this direction. The verdict here is clear: human taste aversions seem to be independent of cognitive control. I have heard of a number of cases in which the situation leading to a specific food aversion is known by the "affected" individuals and it is known that the illness following ingestion of the now aversive food was not caused by the food (for example, often others ate the same food and did not become sick). Yet, a deep-seated aversion remains, uninfluenced by a contrary cognitive "overlay." This again suggests a low-level system.

parents. Since their parents feed on diet B, they will also and, thus, this diet becomes familiar (Galef and Clark, 1971, 1972). Second, salient characteristics of the diet eaten by the mother appear in the mother's milk (LeMagnen and Tallon, 1968; Galef and Sherry, 1973). Nursing pups experience chemical stimulation that resembles parental food, making it familiar and safe and, hence, preferable (Galef and Henderson, 1972). No doubt there are other social interactions among adults as well as between parent and child that are of great significance (Barnett, 1956).

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Furthermore, there is a question as to whether the classical conditioning paradigm is appropriate for taste-aversion learning. Most modern investigators of exteroceptive conditioning consider the CS as conveying information about the probability of a US. "Stimulus substitution" is not the preferred formulation. But the behavior of animals and humans toward aversive foods suggests that the foods themselves may arouse strong emotions of disgust. Food aversions have an immediacy of affect usually lacking in exteroceptive CSs. Pfaffman (1960) has pointed out that the taste system itself has an unusual affective loading compared to other systems (see also Young, 1948), and this may be carried over to the taste-aversion paradigm. The recent demonstration of clear taste projections in the rat into the hypothalamus as well as the traditional projection pathways supports this view (Norgren and Leonard, 1973). At any rate, we may be dealing with a new (or rather, very old) kind of learning here. It seems quite likely that the tastes in taste-aversion learning acquire some of the affective qualities of the US, in contrast to the usually employed exteroceptive CSs, which serve as signals for USs. [Gleitman (1974) has recently suggested methods to determine in animals whether a CS serves as a signal or acquires the properties of a US. As he points out, these methods could usefully be applied to this problem.]

5. *Domestication*

The great majority of specific hunger experiments have been done on domestic rats. The adaptive food selections shown by these creatures is remarkable, given that they have been raised for fifty or so generations on laboratory chow—without any selection pressure to maintain their exquisite food-selection abilities. Indeed, great changes in appearance, physiology, anatomy, and behavior have occurred during the domestication process (e.g., see Barnett, 1963; Kavanau, 1964; Richter, 1954, 1959). Some may directly affect food choices, such as decreased neophobia in domesticated rats (Richter, 1953; Rozin, 1968a; Galef, 1970) or changes in the adrenal gland and salt tolerance, which might account for the changes in salt preference (Richter and Mosier, 1959; Richter, 1959). Yet, in spite of all this, the domesticated rat seems surprisingly able to deal behaviorally with nutritional stresses. Surely, there must have been some basic changes in food preferences. With this in mind (and with tongue in cheek), some years ago I initiated a search into the food habits of domestic rats for some sign of the decadence and frailties of so many generations in what Richter (1959) has called a "welfare state." The results from actual experiments are clear, as shown in Fig. 4. When 4 domestic male rats were offered binary choices between Hebrew National and Genoa salami, they clearly preferred the former (Fig. 4, left). Subsequently, when offered a choice between gefülte fish and shrimp, they showed a massive preference for the gefülte fish, averaging around 50 gm a day of this delicacy (Fig. 4, center). To further consolidate the view of an emerging preference for kosher foods, a final test choice between Mogen

David and Virginia Dare wines was arranged. As can be seen in Fig. 4 (right) the kosher hypothesis was strongly supported. I can only hope that such preferences are not present in wild rats—I have not put this to the test.

6. *The Chicken — Some Interesting Parallels*

Some very old and very recent studies on the genesis of food and water recognition in chicks, omnivores with a somewhat limited food range, provide an instructive comparison with the work on rats described above. Classic work by C. Lloyd Morgan (1894), extended and confirmed by Hunt and Smith (1967), showed that chickens had to learn to identify water visually. The taste of water and the regulation of water intake (Stricker and Sterritt, 1967) are apparently preprogrammed, but neither can come into play until water is visually recognized in the outside world and ingested. Young chicks, virgin with respect to water and rather dehydrated, would run through water puddles without recognizing them. Chicks have a built-in tendency to peck at small irregular objects (e.g., grains). When this happened to occur at an irregularity in water, the association between visual water and "prewired" water taste was rapidly made (Morgan, 1894), so that the chicks immediately began drinking and drank an amount approximately equal to their water deficit (Stricker and Sterritt, 1967). From that moment on, water was recognized visually. Thus, everything but the visual recognition of water is prewired here.

The same tendency to peck at small irregularities initiates the development of

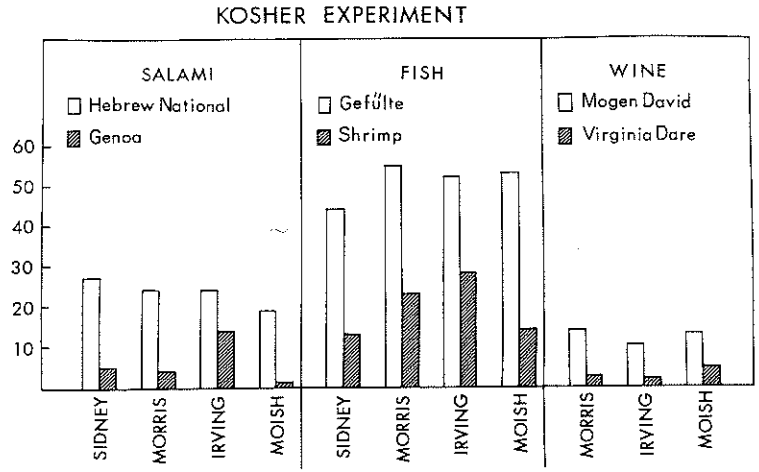

FIG. 4. Preferences of 4 albino male rats for kosher versus nonkosher foods. The intakes plotted represent the mean intake in grams per day, with each pairing of foods being represented exclusively over a period of a few days. Sidney died between the fish and the wine. (From P. Rozin, unpublished observations.)

food preferences which have been analyzed by Hogan (1973a,b,c). He notes that, given certain innate stimulus constraints on peckable items, young chicks are directed to useful foods by the following mechanisms.

1. Chicks tend to eat in proximity to the mother hen, whose food call has a directing effect on their pecks.
2. Chicks learn quickly to reject foods with bad tastes or other irritating properties.
3. Chicks can learn about the consequences of food ingestion by a long-delay learning mechanism similar to that described for rats.
4. Such learning commences on day 3, when yolk sac reserves are down to one-half and the need for an environmental food source is becoming imminent. Long-delay learning does not occur prior to day 3.
5. Initially, what the chick seems to learn is that pecking is desirable, in that pecking followed by metabolic repletion leads to increased pecking. But this pecking increase is also shown for nonnutritive sand, even though edible grains provided the initial repletion experience.
6. Later on, specific acquisition of pecking to edible stimuli occurs. In other words, the first effect of delayed reinforcement is a generalized increase in pecking, which is later discriminated. This may involve further maturation of learning abilities and/or be related to development of sampling patterns, where foods are tried one at a time. Hogan reports that separate experiences with a nutritive and nonnutritive source facilitate discrimination, possibly through a combination of delayed learned aversion and preference.

This work is interesting not only for comparative purposes, but for its emphasis on the ontogeny of food recognition, a subject little studied in mammals. Very little is known about how mammals come to recognize food and water.

III. FOOD SELECTION IN HUMANS

The most striking parallel between human and rat feeding is in the neophobia seen in both. The mouth is the final voluntary checkpoint on the route into the body and, thus, the last opportunity (other than vomiting and diarrhea) to reject dangerous foods. Probably for this reason, strong likes and strong aversions to tastes or smells under minimal higher or cognitive control appear in rats and humans. Appropriately, as mentioned above, both taste and smell systems have rather direct projections to the hypothalamus, and/or limbic system, which mediate emotional responses (Norgren and Leonard, 1973; Pfaffman, 1960). Even with the enormous overlay of culture in humans, one can clearly see this neophobia at work: indeed, it is at the heart of the conservatism of cuisines. Many observers have remarked that ethnic food habits in minority groups are the last vestiges of the old culture to disappear. The best way to tell the ethnic origins of a particular minority ethnic group is to go into the kitchen. Long after accents and costumes are assimilated in the culture, food habits remain.

I shall return to this issue in the later discussion of cuisine. For the moment I would like to discuss the multiple determinants of food selection in man. These can be divided into biological factors and effects of individual experience, on the one hand, and cultural influences, on the other.

A. BIOLOGICAL FACTORS IN HUMAN FOOD SELECTION

There is compelling reason to believe that we have descended from apelike forest dwellers with an omnivorous, but primarily vegetarian diet (Pfeiffer, 1969). Somewhere around 3 million years ago, our apelike ancestors ventured gradually out of the jungle and onto the savannah, motivated very likely, by the possibility of exploiting new food sources. Thus began a shift from a diet probably dominated by fruits and other plant materials, with occasional insects or very small game, to a primarily carnivorous pattern (Pfeiffer, 1969; Morris, 1967). If we assume, as seems reasonable, that our ancestors (Reynolds, 1967; Jolly, 1972) had food habits similar to present-day chimpanzees, then we can assume a marked preference for sweet things such as fruits. [The sweet preference is strikingly illustrated by fruit-eating spider monkeys, which are reported to bite Yocoyena fruit skin when unripe, but not consume the fruit. This accelerates ripening, so that the monkey can return in a day or two to eat the ripe fruit (Jolly, 1972). However, van Lawick-Goodall's (1971) recent observations suggest that a substantial portion of the chimp's diet is made up of insects, meat, and nonfruit plant material.] In spite of millions of years on a substantially carnivorous existence, possibly supplemented by a modest ingestion of seeds, the sweet taste is clearly with us.

Our taste system, both psychologically and physiologically, to some extent (Pfaffman *et al.*, 1971) consists of the four basic submodalities: sweet, salt, bitter, sour. The sweet system seems to be tied directly into an acceptance or pleasure system, appropriately given the sweetness of mother's milk and our ancient beginnings as fruit eaters. Infants prefer sweet solutions to water (Desor *et al.*, 1973) and show characteristic "positive" facial expressions on their first contact with sweet substances (Steiner, 1973). Conversely, the bitter system seems to have the opposite affective loading. Bitter tastes lead to body and facial movements of rejection (Steiner, 1973) and, in at least some studies of infants, appear to be avoided (see Maller and Desor, 1973, for a summary). Adaptively, this is probably related to the bitter tastes in many naturally occurring poisons, e.g., alkaloids and glycosides (Shallenberger and Acree, 1971; Richter, 1950). How this basic bitter aversion becomes transformed in adults of some societies, into a strong preference for bitter substances such as quinine water or coffee remains a mystery. Possibly, bitter sensitivity decreases around the time of puberty. With these minimal biological constraints (and possibly some additional

taste or olfactory tendencies), the human infant begins life on its all-milk diet. The question is, Are the various special recognition or general learning mechanisms described above present in the human and can they be studied in the face of the vast cultural overlay? After all, the smells in the home and taste and smell of the mother's milk reflect the food eaten by adults in the family.

The possibilities for definitive research on the uncontaminated basic biological system are limited, but we are fortunate that one remarkable series of experiments on self-selection in human infants, free from most sources of contamination, is available. These are the classic experiments of Clara Davis in the late 1920s and 1930s (Davis, 1928, 1935, 1939). In the original and most thoroughly documented study, 3 children, weaned in the hospital, were immediately placed on a cafeteria diet in the hospital for the following 6 months to 1 year. Prior to weaning, 1 had only had milk, 1 milk and orange juice, and the third milk, orange juice, and cod-liver oil. Only natural foods, raw or simply cooked without seasoning, were used. Children were presented with a tray containing about twenty different foods. They indicated selections by pointing to a food, which was then offered by a nurse. Children rotated through three different meal selections each day, with milk, lactic milk, and sea salt available at all meals, as well as two cereals, some meats (including organ meats), and fruits and/or vegetables. Results with these children, plus an additional group (Davis, 1939), were extremely successful. Children sampled rather broadly at first but later narrowed down to a rather stable selection of a narrower range of foods in the 1939 study, and higher variability in 1928. Binges (self-terminating) were reported in 1928. Davis reports that appetite nicely anticipated state of health–dropping 24–48 hours before signs of frank illness and picking up 12–24 hours before other signs of recovery. In the second group of 15, in which the children were on the regime for 1 to 4½ years, on the average 17% of calories were taken as protein (range 9–20%)–just in the recommended range.

In the initial study ($N = 3$), Davis (1928) reports greater than average weight gains. No deficiencies appeared on the self-selection regimen. It is hard to evaluate the possibility that deficiencies could have developed, with nutritious foods such as milk, whole-grain cereals, sea salt, and meat making up the majority of the choices, and the absence of highly sweet artificial foods, such as candy or cake. Nonetheless, the reported stability of choices and thriving on the regime is impressive. Davis (1928) provides some sample daily intakes, from which one can estimate relative food preferences. By weight, from the samples provided, milk was most preferred, accounting for 26 to 53% of the total weight of ingested food, with fruits next (14–49%) and then cereals (10–16%). Meats (organ and muscle) were eaten in smaller quantities, as were vegetables (3–6%). One can make a case for sweetness being the prime determiner of infant choice, with milk and fruit the most popular sources. Furthermore, the preferred food by all children, at the one meal a day in which it was offered, was prototypical chimpanzee food–raw bananas. The percent of calories taken as fruit is underestimated, since one meal of the three did not contain any fruit, so that milk was the only sweet choice. Even with this limitation, one child took 41% of calories as fruit.

If we can discount subtle influences from the cooperating nurses, the culturally more-or-less unspoiled human infant does seem to show an adaptive pattern of food preferences. Davis (1935) was sufficiently encouraged that she operated an orthopedic ward for 3- to 12-year-olds on the self-selection principle with good results. It would be highly desirable to repeat these classic studies with some control for "demand" characteristics and some less nutritionally adequate choices. However, the studies stand as a major contribution.

Turning now to the correction of nutritional imbalance (specific hungers), there are two striking studies on children. One of the children in the Davis (1928) study had ricketts (vitamin D deficiency) on admission. Cod-liver oil was offered as a choice, along with an addition of cod-liver oil to the milk. Over a period of 101 days, the child consumed 178 cc of pure cod-liver oil (plus 80 cc more in milk). Davis reported that when the child's blood calcium and phosphorus returned to normal and X-rays were normal, the cod-liver oil appetite ceased. These are suggestive data at best, especially since recovery from vitamin D deficiency is slow, and vitamin D hunger has been hard to demonstrate in animals. Again, one can worry about demand chracteristics and the effects on cod-liver oil preference of mixing it with milk.

The classic example of specific hungers in children comes, not surprisingly, from the work of Wilkins and Richter (1940). It concerns a 3½-year-old boy with the primary symptom of marked development of secondary sexual organs. He was admitted to the hospital, ate very little of the food, and died suddenly 7 days after admission. Postmortem revealed that death was due to adrenal cortical insufficiency. The child had had a great craving for salt and had eaten salt in large quantities from the age of 12 months. The hospital diet did not give him the opportunity to ingest enough sodium to maintain electrolyte balance and probably caused his death. The following is part of the remarkable letter written to Wilkins and Richter by the parents some time after the child's death (Wilkins and Richter, 1940):

> When he was around a year old he started licking all the salt off the crackers and always asked for more. He didn't say any words at this time, but he had a certain sound for everything and a way of letting us know what he wanted. This was the first we had noticed his wanting the crackers or salt. Finally he started chewing the crackers; but he only chewed them until he got the salt off, then he would spit them out. He did the same with bacon, but he didn't swallow the pieces. When he was about sixteen months old, crackers were the first food he chewed and swallowed; but it was quite a while after that before he would chew up and eat a whole cracker. He would usually just make a mess of them eating the salt off.
>
> In an effort to try to find a food that he would like well enough to chew up and swallow, we gave him a taste of practically everything. So, one evening during

supper, when he was about eighteen months old, we used some salt out of the shaker on some food. He wanted some, too. We gave him just a few grains to taste, thinking he wouldn't like it: but he ate it and asked for more. This was the beginning of his showing that he really craved salt, because this one time was all it took for him to learn what was in the shaker. For a few days after that, when I would feed him his dinner alone at noon, he would keep crying for something that wasn't on the table and always pointed to the cupboard. I didn't think of the salt, so I held him up in front of the cupboard to see what he wanted. He picked out the salt at once; and in order to see what he would do with it, I let him have it. He poured some out and ate it by dipping his finger in it. After this he wouldn't eat any food without having the salt, too. I would purposely let it off the table and even hide it from him until I could ask the doctor about it. For it seemed to us like he ate a terrible lot of plain salt. But when I asked Dr. ——— about it, he said, "Let him have it. It won't hurt him." So we gave it to him and never tried to stop it altogether. After we gave it to him all the time he usually didn't ask for it with his dinner; but he wouldn't eat his breakfast or supper without it. He really cried for it and acted like he had to have it. Foods that he ordinarily wouldn't touch he would eat all right if I added more salt to them. He would take the shaker and pour some out on his plate and eat it with his finger, but we always tried to keep him from getting what we thought would be too much for him. He never did care much for zwieback, toast or bread or for cooked potatoes, but he did like raw potatoes, raw carrots, celery, tomatoes, lettuce and different other foods if he could dip them in salt. If I didn't give it to him, he always asked for it. At eighteen months he was just starting to say a few words, and salt was among the first ones. We had found that practically everything he liked real well was salty, such as crackers, pretzels, potato chips, olives, pickles, fresh fish, salt mackerel, crisp bacon and most foods and vegetables if I added more salt.

B. SPECIFIC HUNGERS IN ADULT HUMANS

There is abundant folklore, or anecdotal evidence, and few hard data on changes in human food habits in response to nutritional deficiencies or excesses. The best that can be done here is to give an impression of the kind of information available, realizing full well that we may sometimes be in a situation in which cultural traditions operate in opposition to sound nutritional practice.

There is an association between pica (clay, starch, or earth eating) and iron-deficiency anemia (Cooper, 1957). This widespread practice is most common in children and pregnant women, i.e., during periods of high nutritional demands. We may be dealing here with a mix of deficiences, often including iron, and a cultural tradition which, under some circumstances, has adaptive value, since some of the clays eaten are high in essential minerals. A puzzling increased frequency in ice eating (pagophagia) is also seen in association with iron deficiency in rats and humans (Woods and Weisinger, 1970). The high calcium demands of lactation present special problems in cultures where the prime sources of calcium, milk and milk products, are not consumed. China is a prime example. In this light, the Chinese custom (de Castro, 1952; Simoons, 1961) of

chewing on sweet and sour spareribs by lactating mothers makes great sense. The acidity (vinegar) tends to render some of the calcium in the bones utilizable.

Humans have a sorry record in adjusting food intake to alleviate vitamin deficiencies. In most cases, in Western civilization at least, it was the scientific segment of culture that ultimately brought to light the proper corrective measures. In many cases this did not occur until the development of modern nutritional sciences in this century. Beri-beri has been endemic in many parts of Southeast Asia because of the custom of milling off the vitamin-rich coating of rice before storing and ingesting it. During the age of exploration, vast numbers of sailors died of scurvy, because the simple nutritional cure, vitamin C, ordinarily via fresh fruit, was not understood. Columbus left some men (at their request) on a Caribbean Island to die of severe scurvy (de Castro, 1952). They ate fruit, recovered, and were picked up by a boat some months later. The island received the name Curacao—meaning cure in Portuguese—from this happening. Cartier, in 1535, was told by the Indians of the cure but didn't believe them (Lowenberg *et al.*, 1968). Lind in 1753 did some experiments on scurvy treatment with fresh fruit on his ship with striking results. However, it took the Navy 50 years to accept and implement these results, finally putting to an end massive deaths on the long sea routes. The fresh fruit treatment of scurvy is the origin of the term "limey," applied to British seamen. The discovery of the treatment of other vitamin deficiencies occurred even later—the wisdom of the body appears surprisingly fragile.

There is spotty evidence for at least interesting diet changes during periods of nutritional stress produced by pregnancy and lactation or disease. Most striking, though utterly puzzling, are the strange cravings and aversions associated with pregnancy. In studies in England (Trethowan and Dickens, 1972), cravings or aversions were found in one-half to two-thirds of the pregnancies, with most cravings directed toward fruits (30% of the cases). These cravings and aversions tend to appear in the first trimester. At the moment, it is difficult to map these onto the orderly adaptive behavior seen in rats in Richter's (1955) work. One can only speculate that hormonal-metabolic changes of pregnancy, which, in "simpler" organisms trigger adaptive behavior sequences, in humans interact with habits and culture in a peculiar way. All we see in the human food habits is that there is a change inside the organism.

We have established at least some biological basis for human appetites. In addition to the weak evidence for biological constraints influencing food choice, we can be quite confident that taste-aversion learning over long delays occurs in humans. The anecdotal evidence in this case is overwhelming and there are also some data from the food-poisoning literature. Garb and Stunkard (1974) in a questionnaire given to over 600 American subjects of varying ages, found reports of aversions in 36% of subjects, 88% of these associated with gastrointestinal upset.

C. BIOLOGICAL BASIS OF ETHNIC-RACIAL DIETARY DIFFERENCES

Human food habits are characterized by their diversity in different parts of the world. This diversity is explainable in large part, by variations in the availability of foods on different parts of the earth and, of course, by cultural influences. Nonetheless, one may ask whether any of the substantial genetic differences among races or ethnic groups contribute a biological determinant to food practices or vice versa. I will discuss three examples here: carbohydrate metabolism in Eskimos, phenylthiocarbamide tasting, and lactase deficiency. I expect that there are many more examples to be discovered in the existing literature or through research.

The high-protein and -fat and extremely low-carbohydrate diet of Eskimos is unique in the world. Going along with this dietary pattern of long standing is a parallel metabolic adaptation. Schaeffer (1969a,b) has noted a very high incidence of abnormal glucose tolerance curves to orally administered glucose in Canadian Eskimos. Twenty-five percent of normal Eskimos showed abnormal curves, but most had no other signs of diabetes. Tolerance to intravenously administered glucose was much higher. Schaeffer postulates that a gastrointestinal hormone that stimulates insulin secretion in the presence of carbohydrate in the gut is often absent in Eskimos. He suggests that this is a genetic adaptation, although the possibility remains that it is produced within the lifetime by the very low-carbohydrate diet itself. Alternatively, he suggests, release of the hormone in Eskimos may be under the control of amino acids in the gut. In either case, the suggestion is that a particularly unusual diet is associated with major metabolic changes.

1. Tasting of Phenylthiocarbamide

The ability to taste phenylthiocarbamide (PTC) represents a more widespread trait, which has been studied as one of the best examples of a single-allele effect with behavioral implications. Phenylthiocarbamide is a synthetic thyroid antagonist, closely related biochemically and physiologically to natural goitrogens such as thiourea. Natural goitrogens are found in a number of edible plants, including cabbage, turnips, and peas. Phenylthicarbamide and related compounds taste bitter to most persons, but a significant number of people are unable to taste it at concentrations producing a strong bitter sensation in tasters. The nontaster trait is under the control of a single recessive allele (Kalmus, 1971). There is a polymorphism for the trait, with different frequencies at the two modes, depending on the racial-ethnic population studied. Richter and Clisby (1941) were among the first to point to the bimodality in PTC thresholds, in both rats and man.

Thirty percent of Caucasians (West European and North American) are nontasters, compared to 10.6% of Chinese, 6.4% of American Negroes, and 1.9% of natives of highland Peru (Greene, 1974). The general notion is that nontasting is maladaptive in areas where there is a combination of the presence of natural

goitrogens in the diet and shortage of iodine in the environment (Fischer, 1967; Greene, 1974). The incidence of nontasters is higher in some thyroid diseases, suggesting either a link between the defect producing thyroid dysfunction and the taste defect or that undiscriminating ingestion of bitter goitrogens produces thyroid pathology (Fischer, 1967). (There is no direct evidence of thyroid pathology produced by ingesting natural goitrogens in normal quantities.) Greene (1974) has recently reported work on 6- to 15-year-olds in two Andean communities in Equador which suggests a behavioral (goitrogen-avoidance) interpretation. Both communities have endemic goiter, a level of cretinism around 7%, and presence of natural goitrogens in foods normally included in the diet. In one of the communities, all youngsters were given iodine supplementation, in the other they were not. Greene reports a significant correlation between PTC taste sensitivity and "neurological maturation" (Bender Gestalt) only in the population not protected with iodine. He suggests that the PTC sensitivity has protective value since tasters are less likely to ingest bitter-tasting foods containing goitrogens. This is likely to be a direct behavioral effect rather than a direct effect of the allele on thyroid function (with taste changes as a pleiotropic effect) since PTC sensitivity is correlated with sensitivity to other bitter substances (quinine), suggesting taste as the salient selective factor. In order to explain the polymorphism, Greene points out some advantages accruing to nontasters—including a significant tendency toward hyperthyroidism in adulthood in some tasters. In summary, the hypothesis is that tasters can successfully avoid bitter tasting goitrogens, which has adaptive value in geographic areas where goitrogens are potentially significant in the diet and iodine is in short supply. Greene (1974) claims that the distribution of nontasters in the world is consistent with this hypothesis.

2. Lactose Intolerance

There is one biological factor that has profoundly affected ethnic-racial food pattern differences. This is the substantial decrease in levels of the enzyme lactase, which digests milk sugar, after infancy. As shall be seen shortly, the incidence of this enzyme deficiency that relates to a very basic food has had a significant impact on some world cuisines, especially oriental cuisines.

Lactose, a disaccharide made from galactose and glucose, is the only carbohydrate in milk (Kretchmer, 1972). It constitutes 6.5–7% of fresh human milk, and 4.5% of cow's milk by weight, and accounts for 40% of the calories in human milk (McCracken, 1971; Nelson *et al.*, 1969). Lactose is broken into absorbable constituents—galactose and glucose, by the intestinal enzyme lactase. Lactase is obviously present in the gut of virtually all human infants—otherwise breast or other milk feedings would be disastrous. In very rare cases, the enzyme is absent at birth (genetically controlled, primary lactase deficiency), requiring a milk substitute or specially processed milk diet from birth. We are concerned

here with secondary lactase deficiency, a notable decrease of lactase in early childhood. This decrease usually occurs in the age range of 2 to 4 years for humans (Kretchmer, 1972). Lactase levels drop off rapidly in other mammals that have been studied, the sharp drop in the rat being in the range of 2 to 4 weeks of age. Adult lactose-tolerant humans seem to have the same lactase as infants, so that the difference between deficient and nondeficient subjects appears to be whether the lactase produced in infancy continues to be produced in significant amounts.

Lactose cannot be efficiently digested and absorbed by intolerant adults. When eaten in significant amounts, it draws excess water into the gut and produces gas by a fermentation process in the gut. Characteristic symptoms are thus gastrointestinal upset, bloating, and diarrhea (Kretchmer, 1972; McCracken, 1971). Note that the response to lactose is *not* an allergic type reaction, so that a substantial intake is required to produce significant symptoms. Typically, one or more glasses of milk produce symptoms in 30 to 90 minutes (Simoons, 1969). These gastrointestinal symptoms must form the basis for a learned aversion.

There is extensive data on the incidence of secondary lactase deficiency in groups all over the world. Lactose tolerance is determined either by report of symptoms following lactose ingestion or measurement of blood glucose after orally administered lactose. Blood glucose will rise only if the glucose breakdown product of lactose gets into the blood. There is an enormous range in tolerance across different racial or ethnic groups. Only 10–20% of Caucasians of North European origins are lactose intolerant, compared to high levels in the range of 80 to 99% intolerance for many Oriental and African groups. It is not surprising that there is a relationship between lactose tolerance and amount of milk in the diet. Thus, in Nigeria (Kretchmer, 1972), the Yoruba, originating in the Congo, and without a tradition of keeping cattle are 99% intolerant. In contrast, the Fulani, a pastoral nomadic group with a long history of milk drinking show only 22% intolerance. The general relationship between milk-drinking habits and lactose intolerance is displayed in Table I.

Given the subject under discussion, it seems appropriate that we should have to ask, Does milk drinking induce lactase production or does lactase deficiency lead to milk avoidance? The weight of evidence appears to favor the latter—a genetic control of lactase inactivation (Simoons, 1969; Kretchmer, 1972; McCracken, 1971). There is a suggestion (McCracken, 1971) that lactase is under the control of a single locus, with possibly two separate alleles—one programming production of lactase in infancy and the other throughout the lifetime. Although it would at present be hard to establish this point, the following facts strongly support genetic determination in general, with implications regarding the underlying mechanism.

1. With one lactose-intolerant parent, 45% of offspring are intolerant (McCracken, 1971).

2. In marriages between Ibos or Yorubas (almost entirely intolerant) with Europeans, most children are tolerant (Kretchmer, 1972).

3. There are other documented, genetically based, disaccharide-splitting enzyme deficiencies.

4. Most animal studies have shown little or no success in inducing or markedly prolonging lactose tolerance in young animals by milk or lactose administration (Simoons, 1969; Leichter, 1973).

5. Thai children in institutions in which they were regularly fed milk over the first 2 years became intolerant at about 2 years (Simoons, 1970).

6. Members of ethnic groups with high intolerance, when transplanted to milk-drinking cultures, continue to show high intolerance (e.g., American Blacks). However, this is not entirely compelling, given the apparent popularity of milk among current-day Japanese and, on the animal side, the frequent ingestion of milk by pets such as cats.

Assuming a genetic basis, we now ask, What came first, adult lactase deficiency or adult lactase production? Which trait is primitive? The answer here is fairly clear, with the arguments mustered by Frederick Simoons (1969, 1970), an outstanding cultural geographer. Data and common sense support lactase deficiency as primitive, since there is no need for an ability to digest milk in any adult mammal other than man—and for man only in the last eight or nine thousand years. The argument is, then, that with domestication, there would be an adaptive value for adult lactase, and thus an appropriate selection pressure. What is known about the origin of dairying and its dissemination fits, in general, with the lactose-intolerance incidence: nonmilking groups today show low tolerance, along with migrants from these areas (Simoons, 1970).

TABLE I

SUMMARY OF LACTASE DEFICIENCY BY CULTURE[a]

Table No.	Table title	No. subjects	No. deficient	% Deficient
6	Herders	56	21	38
7	Hunters and Gatherers	76	61	80
8	Cultures without Dairy Animals	171	144	84
9	Cultures with Dairy Animals but No Adult Milk Consumption	694	566	82
9	(Omitting children)	(403)	(351)	(87)
10	Cultures with Low Milk Production, Variable Consumption	88	58	66
11	Cultures with High Milk Production, Generally High Consumption	1,810	247	14
12	North American Caucasian	582	91	16
	Total	3,477	1,188	34

[a] Data from McCracken (1971).

One might ask why, in the primitive state, lactase production was inactivated. I can think of two reasons. First, production of a useless enzyme is a waste of energy—there are a number of examples of metabolic processes that are phased out after infancy, presumably on these same grounds. Second, it is possible that lactase phase-out is a means of promoting weaning in mammals (Marcia Levin, personal communication). The phase-out seems to occur around weaning time in the species studied, and it might be of considerable adaptive value to facilitate this difficult but necessary separation by imparting some aversive character to the milk. Some contemporary mothers do this with quinine on the nipple.

The serious problem remaining is to suggest a sequence that would have led to strong selection for adult lactase in some populations. Simoons (1970) hypothesizes that milk was first drunk, in small nontoxic amounts, as a cult offering, and that this formed a base for a gradually increasing milk-drinking habit, coevolving with milk tolerance. I think it is also possible that milk just tasted good and may have been sampled by either adults or very young children prematurely weaned. But whatever the initial scenario, there are serious problems. First, note that many milk products, such as yoghurt or many cheeses, contain very little lactose (Simoons, 1970). Thus, a milk culture could develop before raw milk tolerance. It is hard to imagine a culture that would not have discovered souring or fermenting of milk, since both techniques are widespread in the world, and milk, by its nature, is likely to end up that way. And what would be the selective advantage for a cheese- or yoghurt-based culture to move toward milk? Second, assuming either unavailability of cheeses or yoghurt, or a culture based on them, why would lactose tolerance have a clear selective value? Surely, if a small minority of a group was able to digest lactose as adults, it would not be in a small society's interest to stop killing animals in order to get milk for a few.

Going along with Simoons, we would have to argue that strong selection for lactose tolerance would occur in what must have been a relatively rare group that had not accidentally discovered cheese, butter, yoghurt, or fermented milk and that was under some special food privation such that milk became a particularly valuable food source. Under these circumstances, families who were tolerant (and, of course, it would run in families) would have a much greater chance for survival.

The lactase story is obviously of fundamental importance to the understanding of man's food habits. Many questions, especially pertaining to the origin of lactose tolerance, remain. However, it is already clear that it is a major shaper of man's food habits: the total absence of milk products from Chinese cuisine, in spite of the fact that the Chinese have been heavily exposed to a culture using milk in the form of the neighboring and invading Mongols, must be in part attributable to lactase deficiency.

D. CULTURE AND CUISINE

We finally arrive at what is distinctively human about food selection—organized bodies of knowledge and tradition. But it is not quite uniquely human. Recent work by a number of Japanese zoologists has introduced examples of cultural tradition concerning food in nonhuman primates (*Macaca fuscata*) (Kawai, 1965). A troop of these monkeys, living on Kashima Island in Japan, has been under observation for many years.

In 1953, a 1½-year-old female (Imo) initiated a new feeding practice called sweet potato washing by the observers. A sweet potato was held under the water with one hand, and brushed (presumably to remove sand) with the other hand. Over the next 4 or 5 years, this behavior spread as a characteristic way of treating sweet potatoes prior to eating. The route of transmission was primarily through lineage and playmate relationships. By 1958 about 80% of monkeys aged 2–7 had acquired it and 18% of the adults. All monkeys over 5 that acquired it in this early stage were females. Starting about 1958, the mode of transmission of this "tradition" shifted to a mother-to-child pattern. Young monkeys, eating while their mother was eating, would be directly exposed to the potato-washing ritual, and pick it up quite naturally. Almost all children born after 1958 acquired sweet potato washing. Sweet potato washing took an interesting turn around 1958, when some monkeys began washing the potatoes in the ocean, rather than the brook previously used. By 1961, all monkeys that washed potatoes did at least some in salt water; this probably resulted both from the easier availability of salt water and a preferred taste for the salted potatoes. Subsequent to the shift toward salt water, some monkeys commenced a new variant of washing, called "seasoning." In this case, between bites, the potato was continually dipped into the salt water. This behavior appears to be supported by taste enhancement, although there is no direct evidence for this. As of 1965, some monkeys did a substantial amount of "seasoning" while others remained straight "washers."

We have in this fascinating work unique observations of the origin and propagation of food traditions. Ritualized food preparation and seasoning have already been demonstrated. In addition, in another case involving the same monkey troop, a trait called "wheat washing" has spread through a population. This involves taking a handful of wheat and sand (the wheat having been thrown on the sand by the observers) and throwing it in the water. This rapidly separates the wheat and sand, making sorting of the wheat much quicker than would otherwise be possible. Imo, again, was the originator of this practice. One can only wait with anticipation for further developments from this exciting line of investigation.

Most of man's daily feeding choices are made within rather severe constraints, which can be defined as the cuisine of the culture in question. In fact the cuisine, coupled with limited availability of types of foods at particular times, forces the eater's hand, or mouth as it were, making the individual choice for a meal in a nonaffluent society rather meager. However, we should not be too hasty in ascribing this lack of choice to limited basic ingredients. As my wife discovered when we were going through graduate school together on a low budget, there are lots of ways of preparing tuna fish (masquerading under such

appetizing names as "tuna dream" or "tuna whiz"). The fact is, with a small number of ingredients, some utensils, and fire, one can make a wide variety of dishes. Yet only two of the world's major cuisines, French and Chinese, seem to be almost limitless in tapping very many combinations, but selection is highly constrained even within these two cuisines. The fact is that man himself has introduced constraints that severely limit acceptable dishes. Recipes are just such constraints, whether written or passed on, appropriately enough, by mouth. I propose, in this last portion of the paper, to try to describe briefly the types of rules or constraints that characterize man's cuisines and, then, in keeping with earlier material, to try to relate the nature of cuisine to some basic determiners of food selection. Many of the ideas and examples I will discuss come from Elisabeth Rozin's (my wife's) work in attempting to extract the principles or distinctive features of cuisines and understand their nature (Rozin, 1973, 1975).

Are there any universals of man's cuisines? The answer would seem to be no–but this should not discourage an attempt at understanding general underlying processes. For the unique species that can make do near the Arctic circle, in forbidding deserts, on coral reefs, at elevations over 10,000 ft, it is not surprising that geographic or ecological constraints may overwhelm natural tendencies. The fact remains that when cultures highly deviant in this sense are sometimes excluded (Eskimos being common candidates for exclusion), certain characteristics emerge: (*a*) almost all cultures practice cooking; (*b*) almost all cultures either prepare an alcoholic beverage or explicitly prohibit it (the exception that proves the rule); (*c*) almost all cultures have a cuisine, i.e., a set of rules about what to eat and how to prepare it; (*d*) almost all cultures have characteristic staple foods, characteristic methods of preparation, and characteristic flavors used with their foods.

It is surprising how simply one can define a particular cuisine, if the purpose is to get at the core of the cuisine and encompass its principal dishes. E. Rozin (1973) has analyzed cuisine into four components.

1. Basic foods–the basic nutrient sources. These are/were clearly selected largely on the basis of local availability and accessibility of nutrients. Thus, in southern China, rice, eggs, chicken, pork, fish, shellfish, and a few vegetables dominate the cuisine. On the other hand, in the Middle East, wheat, barley, lamb, and goat meat are dominant.

2. Manner of preparation. Again, with a wide variety of possibilities, in this case not so limited by nature, each cuisine selects a few methods for repeated use–both simplifying and giving character to the cuisine. Thus, for southern Chinese cuisine, brief rapid heating (stir fry) is most common, with steaming and deep frying also common, whereas Middle Easterners rely primarily upon stewing (see Rozin, 1973, for further examples).

3. Flavor principles. E. Rozin has argued that the most distinctive definer of the character of a cuisine is the characteristic flavor combinations regularly placed on (in a sauce) or mixed with the basic foods. She calls these combinations flavor principles, and attempts to abstract them, as a set of distinctive features, from the corpus of typical foods in a cuisine. The proof of the pudding for the centrality of these principles is that, when applied to a basic food not characteristic of a particular cuisine, the product turns out to seem to be an "instance" of that cuisine. Thus, potatoes, which are rarely used in China, if stir-fried with soy sauce and gingerroot will taste Chinese, since this is the basic flavor principle of Chinese cuisine (supplemented with garlic, sesame oil, sugar, vinegar, and a few other ingredients on occasion). By contrast, the Middle Eastern flavor principles include a lemon–parsley combination and an olive oil, tomato, and cinnamon combination. Each of the world's major cuisines has been so characterized (Rozin, 1973).

4. Cuisines also involve a host of rules having to do with who can eat when, what foods can or cannot be mixed with particular other foods, and so on.

The points I wish to make here are that cuisines can be defined in rather simple terms and that they are extremely stable and resistant to change. E. Rozin's research (personal communication) shows that some cuisines–Mexican, Indian (subcontinent), Chinese, Middle Eastern–have remained basically the same over many thousands of years. Simplicity and stability surely suggest basic important underlying determinants at work. What are they? Why do we have cuisines?

I would like to suggest four answers, and spend some time on the two that have direct relevance to the subject of this paper.

Let me begin with a brief mention of two functions of cuisine that are *extrinsic* to the fundamental nature of food per se and, hence, of only peripheral interest here.

First, cuisines can become an art form and, hence, a means of expression and esthetic satisfaction for man, in the same sense as music or art.

Second, cuisine as a characteristic of a culturally coherent group, serves as one of many means of identifying that group, setting it off from others, and also making social distinctions within members of the group, or distinctions among occasions within the group. All this may involve overt or symbolic relationships (Levi-Strauss, 1964, 1966). The racial identity function of cuisine may itself be a major determiner of food practices. In part, it may explain some of the conservatism of cuisine. It may account for the origin of some kosher food practices as ways for the ancient Jews to set themselves apart from surrounding tribes.

The other two functions of cuisines are *intrinsic*, that is, directly related to eating and nutrition. Thus, as a third function, cuisines embody some of a culture's accumulated wisdom about foods. This is not to say that some traditional food practices are not nutritionally maladaptive; however, many practices are clearly functional. The institutionalization of cooking in virtually all cuisines would be such an advantage. Cooking serves to kill some potentially dangerous

microorganisms, render food easier to chew, and in many cases increase its nutritional value by making a larger proportion digestible (see Renner, 1944, for a discussion of this and related issues). Whether any of these factors directly contributed to the *origin* of cooking is problematical. An improvement in taste may be the critical factor. But the *maintenance* of cooking practices is almost certainly related to some of these advantages.

The avoidance of milk institutionalized in Chinese cuisine is no doubt in part a cultural adaptation to high lactose intolerance. In this case, cultural mechanisms may have overresponded, since Chinese cuisine also rejects milk products (e.g., cheese) low in lactose.

Manioc (cassava) is a staple carbohydrate source in many parts of tropical South America and West Africa. A principal variety is quite toxic, containing what can be dangerous levels of cyanide. Tradition among Brazilian natives in preparation of manioc for ingestion includes crushing, rinsing, and pressing. In this way, most cyanide is washed away, and a rich, easy to grow and resistant carbohydrate source is made available by a traditional preparation procedure (Jones, 1959). Interestingly, when manioc was introduced to Africa by Portuguese trades in the late 1500s, they brought the preparation techniques with them and, hence, the detoxification procedure. Cassava is now a staple in parts of Africa, and in many areas the toxic variety is the preferred form (Jones, 1959).

Many groups, undoubtedly through trial and error, have developed combinations of staples that complement each other and form the basis of an adequate diet. Thus, for example, corn and beans form the basic protein sources in traditional Mexican cuisine. Although the proteins of corn and beans are each deficient in a different essential amino acid, together they provide a reasonable amino acid balance. We can be quite confident that, in general, there is strong selection in cuisines to provide adequate nutrients, since literal survival is at stake. Again, as with Southeast Asian habits of removing the vitamin-rich rice hull, strong tradition can often act in opposition to nutritional wisdom on occasion, but the pressures for nutritional adequacy of cuisine are strong and must usually have the upper hand. Now, of course, with synthetic or highly refined vitamins and other nutrients, the pressure for nutritional adequacy of cuisine per se is relaxed.

A fourth, somewhat speculative, function of cuisines concerns their role as modulators of food neophobia. I have mentioned the drama of ingestion—allowing a foreign substance to pass into the body. There is a real and present danger in the act of eating, and the stranger the food the more frightening is the experience. Eating can be both nerve-racking and satisfying. One way, in accordance with our animal heritage, of reducing the tension of ingestion, is to add a characteristic, distinctive, and familiar taste to one's food. In this respect, familiarity breeds *content*.

Flavor principles are then seen as ways for clothing foods in familiarity. And, paradoxically, the characteristic sauce(s) of a cuisine may become the vehicle for successfully incorporating new staple foods, as may happen occasionally, into a cuisine. The familiar flavors blunt the neophobic edge. If this view is true, the flavor principles should be the most conservative aspect of cuisine. This seems true, on the whole, although the wholesale adoption of the tomato as a flavoring element in Mediterranean cuisines would be a glaring exception.

Man's food habits, selections, and cuisines have just been "braised" in this discussion. We have yet to understand the basic forces behind the evolution of man's foods over the course of the history of civilization. That food has been a potent enough force to have caused wars and formed a basis for the wealth or poverty of nations is unquestioned (Tannahill, 1973). That, in the form of the spice trade, that mysterious search for seasonings, it was a major force in the history of the world over a period of many hundreds of years cannot be denied. That food selection plays a significant role in health, and especially obesity, is certain. I have tried here only to raise some questions, suggest some solutions, and whet my own and hopefully the reader's palate for more answers.

Acknowledgments

I thank Elisabeth Rozin for development of some of the ideas presented in the latter part of this paper, and my former students, Willard Rodgers, Bennett Galef and James W. Kalat for contributing to some of the formulations presented in the earlier part of this paper. I also thank Jeanette DeSor, Elisabeth Rozin, and Edward M. Stricker for valuable comments on this manuscript and Curt Richter for opening up this field and setting an example of the highest quality of research. Much of the author's research described herein was supported by the National Science Foundation. A collection of a number of articles relevant to the issues raised in this paper may be found in Kare and Maller (1967).

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