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Review

Order and disorder: Temporal organization of eating

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ABSTRACT

Feeding behavior is described from an evolutionary perspective, and implications for modern neurobiological studies are suggested. In particular, it is argued that meals may have evolved more for sociocultural reasons than physiological imperatives, and that biological approaches to the study of feeding episodes should adopt a more flexible model that is founded in economic or cost-benefit considerations. Specific examples of flexibility in mouse feeding behavior are given. It is further argued that the modern human food environment is so immoderate that physiological manipulations designed to restrain eating have little hope of achieving this goal.

Contents

1. Introduction

The early 1950s were formative years for the physiological psychology of feeding behavior. In 1954, our honoree Philip Teitelbaum defended his Ph.D. dissertation entitled "Sensory control of hypothalamic hyperphagia" and in the same year his mentor, Eliot Stellar, published his seminal paper on the physiology of motivation [1]. The previous year, Gordon Kennedy had introduced his lipostatic theory of the control food intake [2], to be followed a few months later by Jean Mayer's glucostatic hypothesis [3]. Phil continued to work on the neural basis of feeding for another decade before moving to other problems in physiological psychology. The panorama of feeding behavior today differs in many ways from when Phil was in the field, and I will mention just two. First, there have been immense technology-driven advances including an ever-expanding appreciation of neurons, genes, epigenetics, and signal molecules. Second, there has been a substantial increase in obesity, usually measured by population mean body mass index (BMI), in humans living in most affluent societies.

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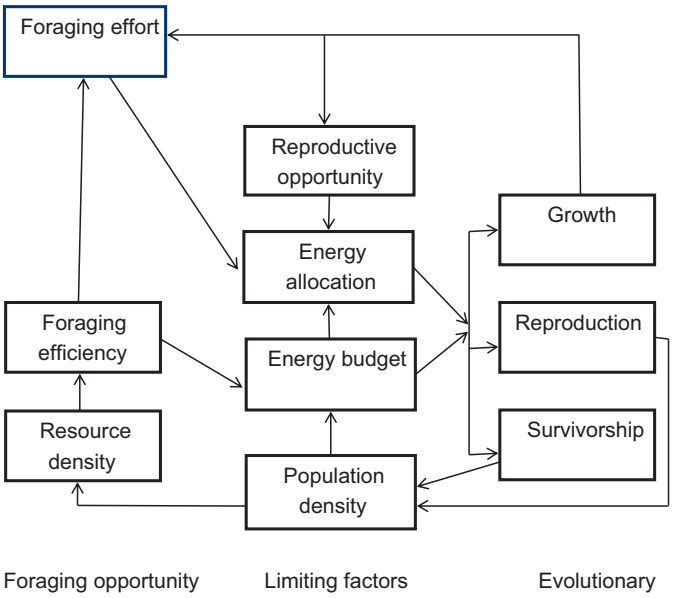

Fig. 1. The nutritional ecology of feeding (adapted from [24]).

One of our scientific Zeitgeists is translational research. There is strong public interest to identify biological factors linked with obesity because therapeutic manipulation of those factors might prevent or reverse obesity. For example, several genetic polymorphisms have been correlated with human obesity in genome-wide association studies [4], and these results are driving research on how those gene products affect food intake or energy expenditure, and whether they might be viable therapeutic targets. If we look beyond the allure of biomedical application, I suspect that scientists in related fields are less enthusiastic about this approach.

Behavioral ecologists and anthropologists adopt a view of feeding and energy flux through organisms (Fig. 1) in which the animal and its behavior are a complex interaction among many environmental variables. Every living species, in particular hominids, has survived periodic food shortage and famine [5,6], so our species as a whole is endowed with "thrifty" genes in relation to energy balance. The formal models from these fields view eating as opportunistic and often de-emphasize organismic or physiological factors. Given a modern human environment with many opportunities to eat, together with high quality food, a model such as that in Fig. 1 leads to the conclusion that elevated intake leading to fat storage is inevitable. In our past, it was a necessity for survival: "natural selection. . .. favors individuals who can effectively store calories in times of surplus" [7]. These authors add that "Selection could not provide for the eventuality of continuous surplus because, since it had never existed, there could be no obesity and no adaptive disadvantage for those tending to become obese".

The pace with which the incidence of obesity, including childhood obesity, has occurred in the past 25–50 years may be too rapid to be associated with genomic change, although epigenetic factors may work over a shorter time-course and some polymorphisms will underlie individual differences. These changes in gene expression could affect either physiological or behavioral traits, or both. Obesity and/or the overeating that produces it, with the exception of very rare genetic mutations, is enabled entirely by an "obesigenic" food environment. Indeed, the behavior of some individuals toward food has been likened to addiction [8]. While modern physiological and neurobiological analysis of feeding remains founded on a classic principle of homeostasis or resistance to change, there is now an appreciation that "reward" system(s) can be over-activated by highly palatable food and overwhelm intrinsic homeostatic mechanisms; instead, allostasis [9,10] may be a more appropriate descriptor.

Homeostatic systems at their core are a regulated set of physiological variables or derivatives such as body weight. These systems can be revealed in controlled laboratory experiments in animals. One of the key behavioral concepts behind homeostasis is the meal: Le Magnen [11] was the first to measure and make physiological interpretation of free feeding patterns in rats. Observations like this led Smith [12] to advocate that the study of meals (*viz* their size and frequency) is *the* fundamental problem facing the behavioral neuroscience of feeding. Smith further proposed that there are both direct and indirect controls of meal size. Direct controls are the physiological changes that occur as food is consumed, such as changes in a variety of hormonal and visceral nerve signaling to the brain, whereas indirect controls are factors that modulate the sensitivity of the brain to those direct signals. These latter include environmental and organismic (e.g. deprivation state) variables. If that is true, then why has average portion size increased as people have become more obese? Although focus on when and how much is eaten at meals is an important area for analysis, Fischler and Masson [13] present an alternative perspective, namely that the disintegration of structured meals is a contributing factor to obesity. The purpose of this paper is to examine from empirical and evolutionary perspectives the significance of meals compared with non-meals. For convenience, we will treat non-meals as a single category – snacking – although this is almost certainly an oversimplification.

2. Organization of feeding in non-human primates and pre-agrarian humans

2.1. Non-human primates

Relatively detailed analysis of feeding has been performed in non-human primates in the wild as well as under laboratory conditions. In the wild, non-human primates forage extensively in groups, use primitive tools (e.g. to open nuts), engage in food sharing, but do not accumulate food. Most of their food items are small (e.g. fruits, leaves, insects, small mammals): they have no technology for transporting these items. All of their feeding is opportunistic – an immediate consequence of locating food – and is literally hand-to-mouth. Social hierarchies may in part determine access to food [9,14,15].

Altmann [16] analyzed feeding bouts of wild howler monkeys, where a bout was defined as continuous physical contact with one food type. For all foods examined, eating within bouts was well-described by two parameter Weibull functions which describe the probability of continuing physical contact with food in time sampling interval $(n+1)$ given that it occurred in interval (n). As n increases, the probability declines in an orderly manner. This does not differentiate meals from snacks without imposing an arbitrary criterion. Whenever an animal stops a bout of eating of commodity A, the latency with which it may start a bout of eating commodity B will depend not only on the attractiveness (palatability) of B relative to A but also on the proximity of the two food sources and factors such as conspecific competition and risk of predation. Thus, the inter-bout interval (eventually an inter-meal interval) may be determined primarily by the environment. In the laboratory, most of these factors usually are absent or highly controlled/predictable.

Foltin [17] measured meal patterns of young adult baboons in conditions designed to emulate foraging. They were housed individually and performed operant responses (on two levers) to obtain food. One lever was appetitive so that a minimum of 30 min elapsed between the first response and a final response that activated a second (consummatory) lever. A single food item (1 g banana flavored