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Review

When do we eat? Ingestive behavior, survival, and reproductive success ☆

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ABSTRACT

The neuroendocrinology of ingestive behavior is a topic central to human health, particularly in light of the prevalence of obesity, eating disorders, and diabetes. The study of food intake in laboratory rats and mice has yielded some useful hypotheses, but there are still many gaps in our knowledge. Ingestive behavior is more complex than the consummatory act of eating, and decisions about when and how much to eat usually take place in the context of potential mating partners, competitors, predators, and environmental fluctuations that are not present in the laboratory. We emphasize appetitive behaviors, actions that bring animals in contact with a goal object, precede consummatory behaviors, and provide a window into motivation. Appetitive ingestive behaviors are under the control of neural circuits and neuropeptide systems that control appetitive sex behaviors and differ from those that control consummatory ingestive behaviors. Decreases in the availability of oxidizable metabolic fuels enhance the stimulatory effects of peripheral hormones on appetitive ingestive behavior and the inhibitory effects on appetitive sex behavior, putting a new twist on the notion of leptin, insulin, and ghrelin "resistance." The ratio of hormone concentrations to the availability of oxidizable metabolic fuels may generate a critical signal that schedules conflicting behaviors, e.g., mate searching vs. foraging, food hoarding vs. courtship, and fat accumulation vs. parental care. In species representing every vertebrate taxa and even in some invertebrates, many putative "satiety" or "hunger" hormones function to schedule ingestive behavior in order to optimize reproductive success in environments where energy availability fluctuates.

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Introduction

A clinical perspective on human obesity suggests that we are eating ourselves "to death" (Shelton and Miller, 2010). According to this perspective, some combination of genes, diet, and lifestyle promotes overeating and/or low energy expenditure, which leads to a putative disease known as obesity and its myriad adverse health consequences (Vetter et al., 2010). A biological perspective, however, suggests that many phenotypes we see today have been molded by natural selection; they were maintained in populations over generations because they were adaptive, i.e., they increased reproductive success. If natural selection was a primary architect of the mechanisms that control ingestive behavior, we might gain key insights by studying their link to reproductive success. In other words, we need to know how we eat ourselves to life. We need to determine how putative orexigenic and anorectic hormones schedule ingestive behavior in order to optimize reproductive success.

Most often, controls of food intake are assumed to function primarily to maintain homeostasis. As noted by Friedman (2008), homeostasis is from the Latin words for staying "similar to," not "the same as," and thus it implies both fluctuation and maintenance. The variable that must be maintained in equilibrium is metabolic fuel availability, not body weight, adiposity, or food intake. Food intake, body fat storage, and energy expenditure often fluctuate wildly in order to maintain *energy* homeostasis, i.e., homeostasis in the availability and flux of oxidizable metabolic fuels (Friedman, 2008).

Energy for cellular processes and organismal activity comes from eating food. In typical laboratory environments, animal subjects have ad libitum access to readily available food. In most natural habitats, however, food supplies fluctuate, and animals must expend energy to find and procure food (Bronson, 1989). In the wild, both survival and reproduction require anticipatory overeating, food storage, and metabolic adaptations to fasting. It is not surprising then that most vertebrate species show metabolic transformations that allow the breakdown of bodily tissue in order to fuel activity during fasting and starvation (Wang et al., 2006). Reproductive processes, including gametogenesis, vitellogenesis, fetal and embryonic development, lactation (in mammals), and parental care are energetically expensive, and these processes can be delayed to conserve energy for survival under harsh energetic conditions (Cooke et al., 2006; Gittleman and Thompson, 1988; Sibly et al., 2012; Van Dyke and Beaupre, 2011). Behavioral phenotypes that were molded by natural selection, including the controls of ingestive behavior, likely increased reproductive success or helped individuals survive to realize their reproductive potential (Darwin, 1859).

Behaviors, including sex and ingestive behaviors, are critical in energy homeostasis. Experiments designed to measure the metabolic costs of reproductive processes revealed that the most important adaptation to fluctuating energy availability is behavioral compensation (reviewed by Gittleman and Thompson, 1988). Examples include those species that overeat and store energy on the body as adipose tissue prior to mating so as to meet the future energetic demands of gestation; species in which pregnant females conserve energy for their growing conceptus by reducing flight, locomotion and/or general activity; species of birds that relax their homeothermy during incubation; and species in which lactating female mammals increase food intake or draw upon a food cache or their own adipose tissue (reviewed by Gittleman and Thompson, 1988). Thus, behavioral flexibility allows adaptation to a labile energetic environment, and this idea is central to the behavioral neuroendocrinology of ingestive behavior. We suggest that a primary role of hormones is to orchestrate these changes in behavioral priorities. Anorectic, catabolic hormones often stimulate or permit reproductive behaviors and orexigenic, anabolic hormones inhibit reproductive behaviors. The idea that so-called hunger and satiety hormones function to maintain a particular level of body weight or adiposity is incomplete without the understanding that reproductive success depends upon fluctuations in adiposity and the ability to gain access to stored energy in adipose tissue (Schneider and Watts, 2002, 2009; Wade and Schneider, 1992).

We begin with a brief overview of chemical messengers that influence food intake in laboratory rats and mice studied in isolation from opposite-sex conspecifics and without behavioral options. Next, we consider the perspective developed from experiments using other mammalian species and a few from other vertebrate taxa. We emphasize a small number of experiments in which energetic variables are manipulated, thereby calling attention to the need for more such experiments. Specifically, it would be instructive to examine ingestive and reproductive processes under conditions in which the following variables are manipulated: food availability, ambient temperature, the amount of energy expended on exercise, and the presence or absence of competitors and potential mating partners.

In addition, we emphasize the need to consider appetitive as well as consummatory behaviors. These behaviors provide a window into motivation, and, in many cases, motivation is under the control of neural circuits and neuropeptide systems that differ from those that control performance. So-called anorectic peptides enhance sexual motivation whereas orexigenic peptides inhibit sexual motivation, often with no effect on sexual performance. Chemical messengers control moment-to-moment decisions about whether to engage in courtship, parental behavior, ingestive behavior, migration, territoriality, and/or aggression. Furthermore, the effects of these chemical messengers on behavior differ according to subtle changes in food availability, body fat content, and the availability of oxidizable metabolic fuels. When energetic demands are low, there is ample food available to eat, and animals can access metabolic fuels from their adipose tissue stores, there is a pool of oxidizable metabolic fuels that can support cellular process, activity, immune function and reproduction. This is depicted by the full tank in Fig. 1A. Frank starvation (e.g., total food deprivation in lean animals or in fattened animals deprived for prolonged periods) inhibits the hypothalamic–pituitary–gonadal (HPG) system, leaving enough metabolic fuels for survival (depicted by the drained liquid in Fig. 1B and reviewed by l'Anson et al., 1991; Schneider, 2004; Wade and Jones, 2004; Wade and Schneider, 1992). In contrast to severe energetic challenges, mild energetic challenges (e.g., less than 25% food restriction in fattened animals) interact with hormone and neuropeptide action to produce fluctuations in the motivation to engage in sex and ingestive *behavior*, depicted in Fig. 1C. Effects of mild restriction can change sensitivity to sex hormones without impact on steroid secretion (Klingerman et al., 2010, 2011a, 2011b; Schneider, 2006; Schneider et al., 2007, 2012). If most of these chemical messengers evolved to orchestrate conflicting appetites on a moment-to-moment basis, it is not surprising that any one of these alone fails to prevent or reverse obesity. Understanding neuroendocrine control of behavior in a dynamic energetic environment will be crucial as we face the problems of global climate change, endocrine disruptors in the environment, and rising obesity in human beings, pets, and wildlife (Humphries et al., 2004; Wingfield, 2008).

Neuroendocrinology of food intake in laboratory rats and mice

In the vast majority of experiments on ingestive behavior, the model system is either laboratory rats or mice (*Rattus norvegicus* and *Mus musculus*), and the behavioral end point is food intake, the amount of