Journal of Veterinary Behavior 8 (2013) 376–385

Contents lists available at SciVerse ScienceDirect

Journal of Veterinary Behavior

journal homepage: www.journalvetbehavior.com

Review

Stress-induced and emotional eating in animals: A review of the experimental evidence and implications for companion animal obesity

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ARTICLE INFO

Article history:
Received 5 May 2012
Received in revised form 29 October 2012
Accepted 2 November 2012
Available online 28 December 2012

Keywords:
stress-induced eating
emotional eating
overweight
obesity
companion animal

ABSTRACT

Eating in response to stress or negative emotional states is well-documented in humans as well as animals in experimental settings and has been shown to work by alleviating the unpleasant emotional experience. This type of eating behavior, termed stress-induced or emotional eating, is linked to the development of obesity. Standard approaches to companion animal obesity have failed to incorporate this concept. Not every animal given more food than they need will become overweight, which raises the critical question: Why does the animal that overeats do so? If it is to help alleviate stress or negative emotional states, then the standard obesity management approach of restricting food intake without alleviating the emotional distress may actually exacerbate the distress by removing one of the animal's coping mechanisms. Moreover, because emotional eating is a coping mechanism, overeating may be a sign that an animal's psychological well-being is impaired.

Introduction

Obesity in humans (Burton and Foster, 1985) and numerous nonhuman mammalian species (Klimentidis et al., 2011) has increased worldwide to epidemic proportions and continues to grow at an alarming rate. The causes of obesity are both diverse and complex, but it is widely agreed that the fundamental component underlying the development of obesity is a prolonged imbalance between the level of energy intake and the level of energy expenditure, with the resultant surplus being stored as body lipids, predominantly in adipose tissue (Rutters et al., 2008; Speakman et al., 2008). Understanding the factors that regulate both energy intake and expenditure is consequently a crucial step toward developing effective approaches to the prevention and treatment of obesity (Speakman et al., 2008).

A key focus of obesity research in humans is eating behavior, specifically, the causes of eating in excess of one's caloric needs. One area that has received increasing attention is the influence of stress. It had long been supported anecdotally that a variety of stressors could disrupt normal eating patterns (Oliver and Wardle, 1999; Wansink et al., 2003), but evidence accumulating over the past several decades has demonstrated convincingly that both acute and chronic stress exert a strong influence on food choice and eating behavior (Greeno and Wing, 1994; Macht, 1999; Dube et al., 2005). Acute stress has been demonstrated in several studies in laboratory settings involving adult human subjects to influence energy intake by altering food preference, eating frequency, and amount of energy intake (Heatherton et al., 1991; Zellner et al., 2006). Chronic stress appears to alter overall food intake in 2 ways, resulting in undereating or overeating, which may be influenced by a number of factors, including type and severity of the stressor, arousal level, affect valence, psychological makeup of the individual involved, and type of food available (Heatherton et al., 1991; Greeno and Wing, 1994; Torres and Nowson, 2007; Macht, 2008; Rutters et al., 2008). Studies have been remarkably consistent in demonstrating that only a relatively small minority of people do not change feeding behaviors during stressful periods; of the roughly 80% who do, approximately 50% increase and 50% decrease caloric intake when stressed

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http://dx.doi.org/10.1016/j.jveb.2012.11.001

(Oliver and Wardle, 1999; Gibson, 2006; Kandiah et al., 2006; Torres and Nowson, 2007; Macht, 2008).

The broad disparity of eating responses to stress—enhanced, suppressed, and unchanged—has been the impetus to a more recent research interest on the individual nature of obesity development (Ganley, 1989; Greeno and Wing, 1994; Nguyen-Rodriguez et al., 2008). The Individual Differences Model, proposed by Greeno and Wing (1994), maintains that eating responses to stress and negative emotions vary considerably between, but remain consistent within, individuals (Torres and Nowson, 2007). The questions that the individual approach to obesity is intended to address include why some individuals appear to be more susceptible to unhealthy shifts in food choice when under stress; do individuals who are overweight or obese lack appropriate mechanisms to cope with daily stressors and are their existing coping mechanisms ineffective; do stress-prone individuals have different brain pathways influencing mood and stress coping; and, why in the same individual may different emotions, even when of the same valence, affect appetite differently (Macht, 2008; Ozier et al., 2008).

Obesity in companion animals

Obesity in companion animals is now considered an epidemic, which some researchers (Speakman et al., 2008) have suggested may be more severe than that observed in humans. The prevalence of combined overweight and obesity has been reported to range from approximately 25% to approximately 45% in both domestic canine and feline populations (Edney and Smith, 1986; Scarlett et al., 1994; Robertson, 2003; Lund et al., 2006).

The causes and predisposing risk factors for obesity in companion animals have been discussed widely in the literature. As with human obesity, there is a consensus that excessive weight gain and obesity result from a positive energy balance of intake relative to expenditure (German, 2006; Bland et al., 2009; Zoran, 2009). As presented in the veterinary literature, specific factors contributing to the development of obesity can be subdivided into several broad categories: medical-related, genetic predisposition, perinatal influences, dietary factors, lifestyle (environment and exercise), and reproductive and gender factors (Bland et al., 2009).

Some medical disorders, such as hypothyroidism and hyperadrenocortism, have been associated with weight gain (German, 2006). Iatrogenic causes of polyphagia and weight gain caused by certain drugs such as glucocorticoids and anticonvulsants are also well recognized (German, 2006). A genetic contribution to obesity in dogs and cats is suggested by several lines of research. In dogs, certain breeds are known to have an increased risk of obesity, for example, Labrador retriever, cairn terrier, Cavalier King Charles spaniel, Scottish terrier, cocker spaniel; in cats, the domestic shorthair has been shown to be at increased risk (Edney and Smith, 1986; Zoran, 2009; Degeling et al., 2011).

Dietary factors that can contribute to the development of obesity in dogs and cats include feeding regimen, quantity of food, and type of food (Degeling et al., 2011; Zoran and Buffington, 2011). Obesity in dogs is associated with the number of meals and snacks fed, the feeding of treats and table scraps, and the dog's presence when its owners prepared or ate their own meal (Chauvet et al., 2011). Results of another study (Bland et al., 2009) suggested that there was a greater frequency of obesity in dogs offered 1 meal or offered more than 3 meals daily, especially when provided treats of high caloric content or palatability. In cats, ad libitum feeding and highly palatable, energy-dense commercial diets appear to increase the risk of obesity (Buffington, 2005; Zoran and Buffington, 2011).

Lifestyle factors, including amount of physical activity and exercise, human–animal interrelationship, and living environment, have all been shown to have an effect on food intake, weight gain, and obesity (Edney and Smith, 1986; Bland et al., 2009; Degeling et al., 2011). For example, an indoor lifestyle and middle age are risk factors for obesity in dogs (Edney and Smith, 1986) and cats (Scarlett et al., 1994; Robertson, 1999). In addition, overweight cats are more likely to be living in houses with only 1 or 2 cats (Robertson, 1999), and dogs in single-dog households were more likely to be obese than dogs in households with 2 or more dogs (Robertson, 2003).

Neutering is an important risk factor for weight gain in dogs and cats (Edney and Smith, 1986; Robertson, 2003). Sex itself is also a predisposing factor in some canine studies, with females overrepresented (Edney and Smith, 1986).

Limitations of the veterinary literature

When comparing the discussions of contributing factors and causes of overeating, it is evident that a prominent part of the human literature—stress-induced eating—is virtually absent in the veterinary literature. The vast majority of recent literature reports of obesity in companion animals (Edney and Smith, 1986; Robertson, 2003; Lund et al., 2005, 2006; Roudebush et al., 2008; Speakman et al., 2008; Bland et al., 2009; Zoran, 2009; Degeling et al., 2011; German et al., 2011) include no mention of psychological, emotional, or stress-related factors as a contributing cause. Importantly, for all the statements of obesity in companion animals being attributable to excessive dietary intake, a crucial question is left unasked and unanswered: Why does the animal overeat? This is the central question currently being addressed by the individual approach to obesity in people (e.g., the Individual Differences Model). In the companion animal literature, overfeeding and the reasons behind it are often discussed (Degeling et al., 2011); however, overfeeding does not cause obesity—overeating does. A large body of literature, reviewed by Speakman et al. (2008), now exists demonstrating in a wide variety of species of animals that when free-choice feeding is offered, including with high-fat diets, not all animals become overweight and obese. Mugford (1977) reported that when offered a highly palatable diet-free choice, some beagles gained only a little weight, but others ate very large amounts and their body weight increased markedly. Zoran and Buffington (2011) wrote that some cats (even neutered) are able to self-regulate effectively their food intake and not become obese.

Consideration of stress and emotion as a contributing factor for companion animal obesity has not been entirely omitted from the veterinary literature. Buffington (2002) wrote that obesity is exacerbated by stressors, and in