Pharmacology, Biochemistry and Behavior 97 (2010) 1–2

Contents lists available at ScienceDirect

Pharmacology, Biochemistry and Behavior

journal homepage: www.elsevier.com/locate/pharmbiochembeh

Editorial

Pharmacology, Biochemistry and Behavior: Special issue on the psychopharmacology of feeding, obesity and body weight regulation

The impact of the current obesity epidemic on the individual and society is publicised on an almost daily basis. This serves to remind governments, regulators, the scientific community, and health professionals of the need for innovative science and public policy with the potential to translate into behavioural, psychological, and pharmacological therapeutic interventions that will deliver meaningful and sustainable weight loss and/or prevent weight gain. This collection of reviews and original articles bring together data from animal models of overeating and body weight regulation with human clinical data providing a snapshot of current thinking and trends in ingestive science and its relevance to these broader problems.

We eat not only in response to appetite and in sufficient quantities to maintain homeostasis but also because we are attracted to palatable, high calorie, foods through learned rewarding consequences. Figlewicz and Sipols comprehensively review the burgeoning literature on the integration of homeostatic control of food intake with reward systems. Insulin, leptin and ghrelin provide a hormonal input into hindbrain and hypothalamic systems to integrate signals of nutrient status and adiposity with short term meal-by-meal controls (serotonin, NPY, etc). However, it is also becoming clear that these hormones have a direct input on opioid, endocannabinoid and dopamine mediated reward systems throughout the brain and are capable of altering both food intake and the rewarding effects of self-stimulation and drugs of abuse. Figlewicz and Sipols put forward the idea that understanding how the reward system underpins eating and overeating might reveal potential therapeutic targets, a theme which is also taken up by Bello and Hajnal as well as Pecina and Smith in this special issue.

Original articles by Currie and colleagues and Rowland and colleagues explore further the nature of the interplay between satiety and energy balance signals. Currie and colleagues examine the modulation of the orexigenic and metabolic action ghrelin by serotonergic agents and identify the hypothalamic PVN as a critical site of action. Rowland and colleagues use MC3 and MC4 knockout mice as a tool to investigate the recruitment of melanocortin systems in the hypophagic effect of 5-HT2C agonists and, controversially, suggest that MC3 receptors are the more likely candidate. This theme is dealt with in a concise review by Lam and Heisler who consider recent research on serotonin systems and energy regulation in the hypothalamus. An original article by Fletcher and colleagues examines the effects of serotonergic ligands on instrumental responding for food. They show that although 5-HT2C agonists reduce responding, 5-HT2C antagonists have no effect. This strongly contrasts with their earlier studies using cocaine reinforcement in which 5-HT2C antagonists enhanced such responding. Olszweski and colleagues draw to our attention the role of oxytocin as an anorexigenic signal that acts as a satiety signal within hypothalamic systems and has the ability to alter the strength of satiety signals when additional nutritional needs are present, such as during lactation and pregnancy.

Bello and Hajnal focus on the role of dopamine in relation to compulsive eating typically seen in bulimia nervosa and binge eating disorder, combining both human clinical data and data from animal models of bingeing. They put forward the argument that progressive dysregulation of dopamine systems occurs because of repeated overconsumption of palatable foods. Furthermore, Bello and Hajnal stress the importance of understanding genetic traits that predispose individuals to weight gain and seem to cluster in populations with binge eating disorder (e.g. polymorphisms of the DA2 receptor, DA metabolism or the DAT mechanism). Learning is also likely to play an important role in individual responses to foods and dopamine signalling is also critical here. The review by Sclafani and colleagues outlines recent findings related to the neurochemical basis of learned flavor preferences and highlights the involvement of a distributed DA network in food preference learning.

Pecina and Smith review the role of opioid systems in food "wanting" and "liking", presenting a wide range of behavioural paradigms combined with microinjection mapping and the possibilities these hold for translation. Pecina and Smith assert that the mu-opioid system in critical limbic hotspots is responsible for the pleasurable experience of palatable food exposure and works to consolidate the incentive value of foods. Of note is the extent to which an induced motivational need state can alter the neural firing in response to previously learned cues that were perceived as aversive in a non-need state.

The reviews of Pecina & Smith, Bello & Hajnal and Pecina & Smith suggest that overconsumption of the highly palatable energy dense foods freely available in a 21st century obesogenic environment can directly affect feeding control and reward systems. Several original articles presented here speak to this issue. Bocarsly and colleagues present data from several studies indicating that consumption of beverages sweetened with high-fructose corn syrup (as compared to sucrose sweetened liquids) can promote adiposity and increase levels of triglycerides in rats. This paper suggests that some foods promote body weight gain more than others. This paper is a timely addition to the current debate in both the USA and UK on the issue of taxation as a means of controlling intake of specific food and beverage types (Roehr 2009). In a similar vein, Giraudo and colleagues address the impact of high fat diet consumption during pregnancy in an animal model demonstrating that maternal diet affects the body weight of offspring over several generations. Both these papers should be of interest to policy makers as they underline the societal impact of obesity and point to population interventions.

0091-3057/$ – see front matter
doi:10.1016/j.pbb.2010.07.012

All of the reviews and original articles contributing to this special issue emphasise the complexity of systems that control eating behaviour through homeostatic and reward pathways. In the light of a growing understanding of genetics and the impact of the environment on these systems there is a substantial challenge for drug discovery enterprises. Kennett and Clifton provide a timely review of anti-obesity agents that are at a late pre-clinical stage, undergoing clinical trials, or awaiting a decision from the regulatory authorities. Taking a novel approach Kennett and Clifton assess the efficacy of current and potential treatments against the stringent criteria adhered to by the Federal Drugs Administration (FDA) and European Medicines Agency (EMA). Rodgers puts forward a strong argument for the use of precise behavioural characterisation of potential anorectic agents at an early stage of development using the behavioural satiety sequence. Rodgers argues that this tool is useful in elucidating promising treatments, and may be particularly helpful in assessing the behavioural specificity of poly-drug therapy.

We thank all the authors and reviewers for their contributions to this Special Issue and the Editors Dai Stephens and Larry Young. Finally, a special thank you to Eve Naughton, at Elsevier, for her support, professionalism and patience.

References

Roehr B. "Soda tax" could help tackle obesity, says US director of public health. British Medical Journal 2009;339:b3176.

We dedicate this special issue to the memory of our colleague, collaborator and friend Steve Cooper.

Guest Editors

Pete Clifton*
Suzanne Higgs
Michelle Lee

*Corresponding author.
School of Psychology, Sussex University, Brighton BN1 9QH, UK
E-mail address: p.g.clifton@sussex.ac.uk (P. Clifton).