Journal of Adolescent Health 52 (2013) S39–S42

ELSEVIER

JOURNAL OF ADOLESCENT HEALTH

www.jahonline.org

Review article

Neurobiology of Food Addiction and Adolescent Obesity Prevention in Low- and Middle-Income Countries

Albert Lee, M.B.B.S., M.P.H., M.D.[a,b,*], and Susannah E. Gibbs[c]

[a] *Centre for Health Education and Health Promotion, JC School of Public Health and Primary Care the Chinese University of Hong Kong, Hong Kong*
[b] *Department of Applied Health Science, School of Health, Physical Education and Recreation Management, Indiana University, Indiana*
[c] *Department of Population, Family and Reproductive Health, Johns Hopkins Bloomberg School of Public Health, Baltimore, Maryland*

Article history: Received January 22, 2010; Accepted June 12, 2012
Keywords: Adolescent obesity prevention; Low- and middle-income countries; Neurobiology; Multilevel interventions; Healthy setting

A B S T R A C T

Adolescent obesity has become an increasingly urgent issue in low- and middle-income countries. Recent relevant advances include the application of the neurobiology of addiction to food addiction and obesity. The biochemistry of the etiology of obesity indicates the need for multilevel interventions that go beyond simple behavioral approaches. Additional research on the neurobiology of food addiction and adolescent obesity in low- and middle-income countries, as well as program evaluations that examine the biochemical effects of complex interventions, is urgently needed.

The prevalence of overweight and obesity among adolescents has increased in many low- and middle-income countries (LMICs) [1,2]. For example, in Brazil, the prevalence of overweight and obesity among adolescents increased from 3.7% in 1974 to 12.6% in 1997 [3]. These countries are now faced with the challenge of addressing this growing obesity epidemic while also contending with issues of food scarcity and undernutrition [4]. Overconsumption of high-calorie low-nutrient foods in LMICs is due, in part, to changes in global food production. To save money and time, many consumers in LMICs have shifted from consuming meals prepared at home to consuming mass-produced processed foods that are high in sugar, salt, and artificial ingredients [4–7]. The addictive nature of these ingredients has led to patterns of overeating [8]. This change in diet along with increasingly sedentary lifestyles is contributing to increase in obesity. Obesity outcomes are influenced by genetics [9] and epigenetic mechanisms [10], biological bases for food preferences [11], and biological mechanism for exercise [12]. However, these individual-level factors are not sufficient to explain the recent trends in obesity. Macro-level stressors that contribute to obesity outcomes include national wealth, government policy, cultural norms, and the built environment [4]. A systematic review has shown that evidence was not strong enough to demonstrate the effectiveness of physical activity and/or healthier diet to prevent childhood obesity [13], and a single approach is unlikely to be effective without addressing the environmental and psychosocial perspective [1,13,14]. A comprehensive understanding of both individual- and macro-level factors is necessary to formulate urgently needed evidence-based policies and programs. Advances in understanding the neurobiology of addiction may provide important guidance in shaping how obesity is addressed in LMICs.

Neurobiology of Addiction

Investigation into the neurobiology of addiction offers new insight into the biological mechanisms of food addiction and obesity. The extensive review conducted by Potenza et al elaborates on the biological models for addiction and suggests linkages between the pathways of substance abuse and food addictions [15,16]. A network of interconnected brain regions is related to food rewards [17,18]. When people are exposed to images of appetizing food, their brains automatically and reflexively secrete dopamine, which activates the same reward circuits

* Address correspondence to: Albert Lee, M.B.B.S., M.P.H., M.D., Centre for Health Education and Health Promotion, the Chinese University of Hong Kong, 4th Floor, Lek Yuen Health Centre, Shatin, New Territory Hong Kong, Hong Kong.
E-mail address: alee@cuhk.edu.hk (A. Lee).

1054-139X/$ - see front matter © 2013 Society for Adolescent Health and Medicine. All rights reserved.
http://dx.doi.org/10.1016/j.jadohealth.2012.06.008

that are involved in substance abuse [19,20]. Palatable food activates the brain rewards circuitry through fast sensory inputs and slow postingestive consequences as glucose concentrations rise in the blood and in the brain [8]. The repeated supraphysiological stimulation of these reward pathways triggers a neurobiological adaptation that leads to compulsive food consumption and lowers the control of food intake in a manner analogous to biological mechanisms involved in substance abuse [8].

This neurobiology of food addiction has been examined in rat models that have shown that when the animals are exposed to a palatable high-fat diet, they increase their caloric intake and experience a decrease in stimulation of these brain reward circuits [21]. Furthermore, rats exposed to a palatable high-fat diet experience downregulation of striatal dopamine D2 receptors, which can lead to subsequent cycles of overconsumption [21]. Evidence for food addiction has also been established in studies using human subjects. The specific regions of the brain stimulated during anticipation and consumption of palatable foods have been identified in human subjects and confirm the similarities of food addiction to processes associated with other addictions [22]. Low distress tolerance, which has previously been studied in the context of cigarette addiction and substance abuse, has been shown to have an association with overeating among college students [23]. Adults with food addiction are more likely to have other conditions typically associated with drug addiction including severe depression and a history of childhood attention-deficit hyperactivity disorder [24]. Those with food addiction also may exhibit greater impulsivity and stronger food cravings [24].

A study of women with a history of emotional eating found that participants preferentially selected a carbohydrate beverage over a protein-containing beverage in response to induced dysphoria [25]. Enjoyment of the beverage increased on repeated consumption but the mood-enhancing effect diminished, which indicates the abuse potential of foods high in carbohydrates [25]. Although studies of food addiction among adolescents are scarce, Merlo et al found that 15.2% of a group of children aged 8–19 years who were seeking treatment for overweight and obesity indicated that they struggled with food addiction [24]. Children with symptoms of food addiction exhibited greater impulsivity and stronger food cravings [26].

Investigation into the mechanisms of food addiction so far has largely neglected to focus on adolescents as a subject group of interest. Adolescents differ from children and adults in key ways that warrant targeted research attention to determine whether the adolescent experience of food addiction differs significantly from that of children or adults. For example, Spear highlights the differences in the way that adolescents and adults experience rewarding and aversive stimuli [27]. Adolescents have an enhanced sensitivity to rewards and attenuated sensitivity to aversive stimuli. Sensitivity to basic rewards such as sweet substances is higher in youth aged 11 to 15 years than for individuals in late adolescence or early adulthood [28]. These differences suggest that adolescents may be particularly prone to developing food addictions when given access to high-calorie low-nutrient foods.

The neurobiology of food addiction contributes to a larger ecological model of obesity and underweight, with implications ranging from micro to macro. On the individual level, risk of developing obesity or overweight during adolescence is influenced by biological characteristics including epigenetic programming during fetal development [29–31], as well as the specific neurobiological responses to palatable foods. On the macro level, social norms about food consumption and exercise, as well as policies regulating access to healthy and unhealthy foods, can influence individual consumption behaviors.

Implication for Interventions

The prevalence of overweight and obesity in many LMICs has not yet reached the level of some of the most affected high-income countries, but it will be necessary to act quickly and decisively to prevent overweight and obesity from expanding further. The current body of knowledge about food addiction suggests that lifestyle- or willpower-based models of prevention and intervention may not be effective, particularly among adolescents, who may be at a higher risk than adults for developing food addictions. The need to implement effective interventions to curb and reverse the growing epidemic of obesity among adolescents in LMICs is urgent. Furthermore, interventions must be evaluated in light of their impact on the neurobiology of individuals.

Adolescents' sensitivity to sweet substances and propensity to take risks pose a challenge to policy makers. An enhanced understanding of the influence of epigenetics and possibilities of restoring the normal epigenome [30], the plasticity of adolescent brain [27], and the biological basis of addiction [15] may provide a foundation for effective antiobesity programming. For example, the potential reversibility of epigenetic alterations holds promise for multifactorial interventions that include attention to the individual [31].

An ecological model for understanding factors associated with obesity and overweight takes into account the complex etiology of the condition including the biological factors and stresses the importance of taking advantage of health-promoting settings for prevention [32–35]. In addition to focusing on the individual, it is critical to target interventions in settings such as schools, communities, and workplaces to reach the target population [32–35]. A Cochrane review of 55 studies found that interventions in a wide variety of settings can successfully address overweight and obesity among children and adolescents. However, the size of the reduction in body mass index detected in the review was greater among 6–12 year olds than among 13–18 year olds, indicating the need to develop more effective interventions for adolescents. Furthermore, only 5 of the 55 studies were conducted in LMICs [36]. The health-promoting school model espouses a holistic school-based approach to create a healthy setting at the school, as well as in the community [34,37–39]. Studies of the Hong Kong Healthy Schools Award Scheme showed that the health-promoting school model can be effective in improving eating behaviors and preventing obesity among students, with a 60% increase in vegetable consumption during school lunch and a 10% decrease in purchasing fast food [35,39]. Interventions are also needed outside the school setting in LMICs. Policies to improve the environment in which adolescents make choices have shown promising results [40]. Promising policy solutions include levying taxes on unhealthy foods and regulating advertising of unhealthy foods targeted at children and adolescents [40]. Therefore, synergistic actions between home, school, and community are needed to enhance positive adolescent neurodevelopment on healthy eating and regular physical exercise (Figure 1). Additional rigorous evaluations that measure neurobiological, behavioral, and physiological changes are

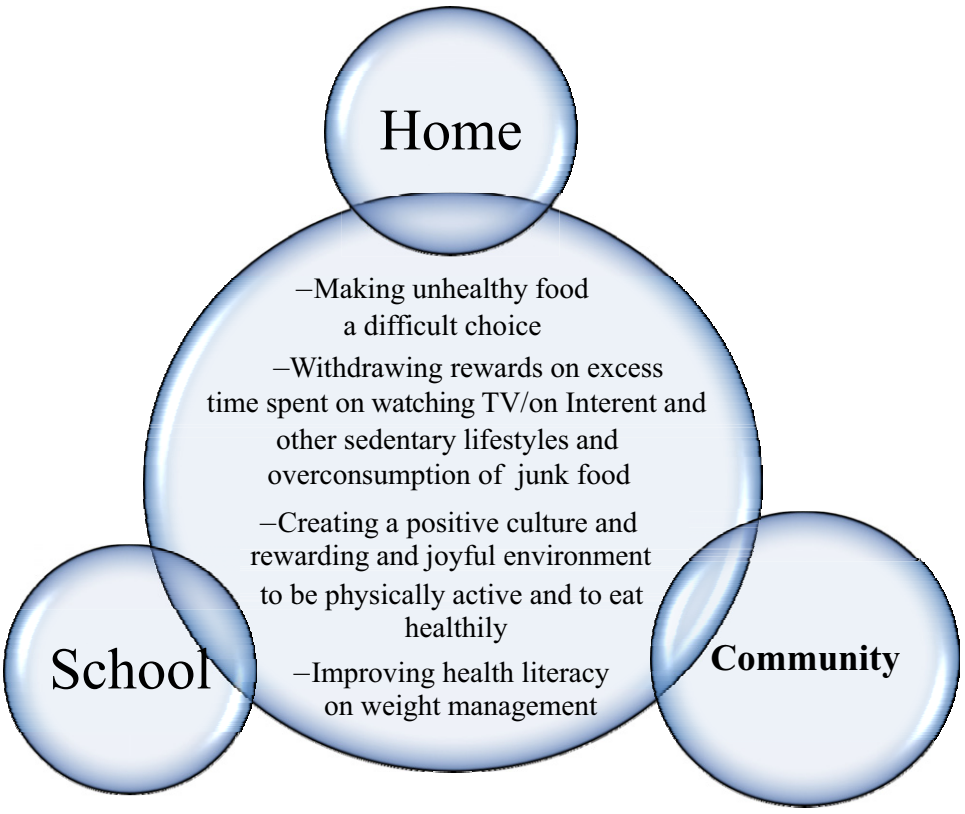

Figure 1. Enhancing positive adolescent neurodevelopment on diet and exercise through home-school-community actions.

needed for interventions implemented in a variety of settings in LMICs.

Conclusions

Solutions to address obesity among adolescents in LMICs must approach this public health priority on a variety of levels from national policy to individual-level interventions and should take into account what is known about the neurobiology of food addiction. Policies must be made to discourage consumption of high-calorie low-nutrient foods and encourage consumption of healthy foods. Physical exercise must be encouraged throughout childhood and adolescence in the home, school, and community settings. Furthermore, interventions must take into account the behavioral implications of the neurobiology of food addiction and should incorporate biochemical changes as part of intervention evaluations.

Successful interventions require a multi-level approach: education, behavioral, environmental and policy changes along with a change in the culture in approaching food, health and economics. Future research on adolescent obesity should be cross cutting, linking public health science and biomedical science with outcome measurements including neurobiological changes as well as behavioral and physiological changes.

References

[1] Lobstein T, Baur L, Jackson-Leach R. In: Waters E, Swinburn B, Seidell J, Uauy R, eds. Preventing childhood obesity. Oxford, UK: Wiley Blackwell, 2010.
[2] Wu Y. Overweight and obesity in China. BMJ 2006;333:362–3.
[3] Wang Y, Monteiro C, Popkin BM. Trends of obesity and underweight in older children and adolescents in the United States, Brazil, China, and Russia. Am J Clin Nutr 2002;75:971–7.
[4] Swinburn BA, Sacks G, Hall KD, et al. The global obesity pandemic: Shaped by global drivers and local environments. Lancet 2011;378:804–15.
[5] Monteiro CA, Moura EC, Conde WL, et al. Socioeconomic status and obesity in adult population of developing countries. Bull World Health Organ 2004; 82:940–6.
[6] Mendez MA, Monteiro CA, Popkin BM. Overweight exceeds underweight among women in most developing countries. Am J Clin Nutr 2005;81: 714–21.
[7] Irizarry LM, Rivera JA. Developing countries perspective on interventions to prevent overweight and obesity in children. In: Waters E, Swinburn B, Seidell J, Uauy R, eds. Preventing Childhood Obesity: Evidence, Policy and Practice. Hoboken, NJ: Wiley-Blackwell, 2010:147–54.
[8] Volkow ND, Wise RA. How can drug addiction help us to understand obesity? Nat Neurosci 2005;26:5160–6.
[9] Bouchard C. Gene-environment interactions in the etiology of obesity: Defining the fundamentals. Obesity (Silver Spring) 2008;16(Suppl 3): S5–10.
[10] Ahmed F. Epigenetics: Tales of adversity. Nature 2010;468(Suppl):S20.
[11] Kessler D. The End of overeating: Taking control of the insatiable American appetite. Emaus, PA: Rhodale Books, 2009.
[12] Sallis JF. Age-related decline in physical activity: A synthesis of human and animal studies. Med Sci Sports Exerc 2000;32:1598–600.
[13] Summerbell CD, Waters E, Edmunds LD, et al. Interventions for preventing obesity in children. Cochrane Database Syst Rev 2005;3:CD001871. http://dx.doi.org/10.1002/14651858.CD001871.pub2.
[14] Ward DS, Ries AV, Tabak R. Environment and policy interventions to prevent obesity in children. In: O'Dea J, Eriksen M, eds. Childhood Obesity Prevention: International Research, Controversies, and Intervention. New York, NY: Oxford University Press, 2010.
[15] Potenza MN. Biological contributions to addictions in adolescents and adults: Prevention, treatment, and policy implications. J Adolesc Health 2013;52(suppl 2):S22-S32.
[16] Potenza MN. Should addictive disorders include non-substance related conditions? Addiction 2006;101:142–51.

[17] Di Chiara G. Dopamine in disturbances of food and drug motivated behavior: A case of homology? Physiol Behav 2005;86:9–10.
[18] Kelley AE, Baldo BA, Pratt WE, et al. Corticostriatal-hypothalamic circuitry and food motivation: Integration of energy, action and reward. Physiol Behav 2005;86:773–95.
[19] Beaver JD, Lawrence AD, van Ditzhuijzen J, et al. Individual differences in reward drive predict neural responses to images of food. J Neurosci 2006; 26:5160–6.
[20] Killgore WD, Young AD, Femia LA, et al. Cortical and limbic activation during viewing of high- versus low-calorie foods. NeuroImage 2003;19:1381–91.
[21] Johnson PM, Kenny PJ. Dopamine D2 receptors in addiction-like reward dysfunction and compulsive eating in obese rats. Nat Neurosci 2010;13: 635–41.
[22] Gearhardt AN, Yokum S, Orr PT, et al. Neural correlates of food addiction. Arch Gen Psychiatry 2011;68:808–16.
[23] Kozak AT, Fought A. Beyond alcohol and drug addiction. Does the negative trait of low distress tolerance have an association with overeating? Appetite 2011;57:578–81.
[24] Davis C, Curtis C, Levitan RD, et al. Evidence that "food addiction" is a valid phenotype of obesity. Appetite 2011;57:711–7.
[25] Spring B, Schneider K, Smith M, et al. Abuse potential of carbohydrates for overweight carbohydrate cravers. Psychopharmacology (Berl) 2008;197: 637–47.
[26] Merlo LJ, Klingman C, Malasanos TH, Silverstein JH. Exploration of food addiction in pediatric patients: A preliminary investigation. J Addict Med 2009;3:26–32.
[27] Spear LP. Adolescent neurodevelopment. J Adolesc Health 2013;52(suppl 2):S7-S13.
[28] Desor JA, Beauchamp GK. Longitudinal changes in sweat preferences in humans. Physiol Behav 1987;52:216–24.
[29] Bjornsson HT, Sigurdsson MI, Fallin MD, et al. Intra-individual change over time in DNA methylation with familial clustering. JAMA 2008;299: 2877–83.
[30] Petronis A, Gottesman KP, II, Kan P, et al. Monozygotic twins exhibit numerous epigenetic differences: Clues to twin discordance? Schizophr Bull 2003; 29:169–78.
[31] Wang G, Walker SO, Hong X, et al. Epigenetics and early life origins of chronic noncommunicable diseases. J Adolesc Health 2013;52(suppl 2): S14-S21.
[32] Dooris M. Holistic and sustainable health improvement: The contribution of the settings-based approach to health promotion. Perspect Public Health 2009;129:29–36.
[33] Dooris M, Poland B, Kolbe L, et al. Healthy settings: Building evidence for the effectiveness of whole-system health promotion—challenges and future directions. In: McQueen D, Jones C, eds. Global Perspectives on Health Promotion Effectiveness. New York, NY: Springer Science and Business Media, 2007:327–52.
[34] Lobstein T, Swinburn B. Health promotion to prevent obesity: Evidence and policy needs. In: McQueen D, Jones C, eds. Global Perspectives on Health Promotion Effectiveness. New York, NY: Springer Science and Business Media, 2007;132:137–9.
[35] Lee A, Ho M, Keung V. Healthy setting as an ecological model for prevention of childhood obesity. Res Sports Med An Int J 2010;18:49–61.
[36] Waters E, de Silva-Sanigorski A, Hall BJ, et al. Interventions for preventing obesity in children. Cochrane Database Syst Rev 2011;12:CD001871.
[37] Bell AC, Swinburn BA. What are the key food groups to target for preventing obesity and improving nutrition in schools? Eur J Clin Nutr 2004;58: 258–63.
[38] Shaya FT, Flores D, Gbarayor CM, et al. School-based obesity interventions: A literature review. J Sch Health 2008;78:189–96.
[39] Lee A, Cheng FF, Fung Y, et al. Can Health Promoting Schools contribute to the better health and well-being of young people: The Hong Kong experience? J Epidemiol Community Health 2006;60:530–6.
[40] Gortmaker SL, Swinburn BA, Levy D, et al. Changing the future of obesity: Science, policy, and action. Lancet 2011;378:838–47.