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SCIENTIFIC TRACKS & ABSTRACTS

Katarina T. Borer, J Obes Weight Loss Ther 2022, Volume 12

Why we eat too much, have easier time gaining than losing weight, and do not expend enough energy: What to do about it

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In the USA, obesity has tripled since 1970's and is associated with a number of pathologies ranging from insulin resistance and type 2 diabetes to cardiovascular disease. Reducing obesity has been difficult whether by caloric restriction, pharmacological approaches, or bariatric surgery. Difficulties in preventing, ameliorating , and reducing obesity stem from our general lack of understanding the limitations of human physiology and psychology and challenges of societal obstacles. Five poorly understood human physiological limitations that increase the risk of gaining, and difficulty of losing, weight are : 1. Human genetic burden of almost limitless capacity to increase fat mass; 2. Absence of a mechanism that restrains overeating and increases energy expenditure to maintain healthy weight; 3. Capacity to increase stomach size with binging and weight gain; 4. Hormonal changes that promote fat gain after weight loss; and 5. Reductions in metabolic rate and physical activity that limit energy expenditure during negative energy balance. Psychological limitations include: 1. Capacity to register hunger mostly to volume, rather than energy content, of food, and fullness or satiation registering only meals eaten by mouth and processed through the gastrointestinal tract; 2. Social facilitation of overeating; 3. Trigger to overeat by the size or availability of food, and 4. Chaotic snacking causing protracted postingestive effects that may extend to 19 hours. Societal barriers include 1. Mechanisation of transportation and household chores; 2. Convenient availability of relatively inexpensive food; 3.

Promotion of energy dense palatable foods by food and restaurant industries; and 4. Urban planning limiting the opportunities for walking. Solutions include 1. Deliberate restricting of eating to an 8 to 10 hour window within waking hours; 2. Using gastrointestinal signals of hunger and fullness to eat appropriate volumes of moderate-to low-caloric density healthy foods ; 3. Using activity tracking devices to sustain motivation for higher activity levels; 4. Daily weight monitoring to provide necessary body- weight feedback in the absence of a physiological feedback counterpart.

Biography

Katarina T. Borer, PhD, is a professor in the School of Kinesiology at the University of Michigan in Ann Arbor, where she has spent over 35 years teaching and researching the hormonal control of metabolism, particularly in response to exercise. She has spent 40 years researching endocrine mechanisms operating in acceleration of growth by exercise and regulation of energy balance. Borer also developed and validated radioimmunoassay for hamster growth hormone and prolactin.

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A comparative analysis of shoes designed for subjects with obesity using a single inertial sensor: Preliminary results

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Walking remains a highly recommended form of exercise for the management of obesity. Thus, comfortable and adequate shoes represent, together with the prescription of a safe adapted physical activity, an important means to achieve the recommended physical activity target volume. However, the literature on shoes specific for obese individuals is inadequate. The aim of the present study was to compare the performance of shoes specifically designed for subjects with obesity with everyday sneakers during instrumented 6-min walking test and outdoor 30-min ambulation in a group of subjects with obesity using a single wearable device. Twenty-three obese individuals (mean age 58.96 years) were recruited and classified into two groups: deconditioned (n = 13) and nondeconditioned patients (n = 10). Each participant was evaluated with his/her daily sneakers and the day after with shoes specifically designed for people with obesity by means of a questionnaire related to the comfort related to each model of shoes and instrumentally during the i6MWT and an outdoor walking test. The results showed that the specifically designed shoes displayed the higher score as for comfort, in particular in the deconditioned group. During the i6MWT, the distance walked, and step length significantly increased in the deconditioned group when specifically designed shoes were worn; no significant changes were observed in the nondeconditioned individuals. The deconditioned group displayed longer step length during the outdoor 30-min ambulation test. In the non-deconditioned group, the use of specific shoes correlated to better performance in terms of gait speed and cadence. These data, although preliminary, seem to support the hypothesis that shoes specifically conceived and designed for counteracting some of the known functional limitations in subjects with obesity allow for a smoother, more stable and possibly less fatiguing gait schema over time.

Biography

Dr. Camillo Buratto, graduated in orthoprosthetics in 1986 and 1989, has experience in the field of biomechanics of at-risk patients (diabetic and rheumatics). He participated as speaker in numerous conferences in Europe (Milan, Madrid, Paris) and outside Europe (Sydney, Tokyo, Dubai).

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Xia Guo, J Obes Weight Loss Ther 2022, Volume 12

Novel role of DOCK2 in diet-induced obesity and lung injury

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Obesity is a significant heath burden and is involved in the development of various lung diseases. However, little is known about the effects of chronic high-fat and high-fructose (HFHF) diet-induced obesity on lung inflammatory/injury. We previously showed that dedicator of cytokinesis 2 (DOCK2) is critical for high-fat diet (HFD)-induced obesity and adipose tissue inflammation. DOCK2 deficient mice were protected from HFD induced body weight gain, insulin resistance, and increased proinflammatory cytokines in the adipose tissue and peripheral circulation. However, it remains elusive whether DOCK2 plays a role in lung injury associated with chronic HFHF diet-induced obesity. In this study, we showed that chronic HFHF diet (20 weeks) induced lung inflammatory infiltration and collagen expression in the wild-type (WT) C57BL/6 mice. Macrophage marker CD68 and monocyte chemoattractant protein-1 (MCP-1) expression were notably increased in the lungs of WT mice fed a HFHF diet. Importantly, HFHF diet increased lung DOCK2 expression that co-localized with fibroblast marker, fibroblast-specific protein 1. These data suggest a potential role of DOCK2 in regulating proinflammatory phenotype of lung fibroblasts. Further, DOCK2 deficiency attenuated lung inflammation and fibrosis induced by chronic HFHF diet. In primary normal human lung fibroblast cells, TNF-α and IL-1β induced DOCK2 expression concurrent with MCP-1, IL-6, and matrix metalloproteinase-2. DOCK2 knockdown also suppressed TNF-α induced increase of these inflammatory mediators. Taken together, these findings suggest a previously unrecognized role of DOCK2 in mediating diet-induced obesity, and lung inflammation/fibrosis in chronic HFHF diet caused obesity.

Biography

Dr. Guo completed her PhD in 2014 from the University of Georgia (UGA) where she continued as a postodoc scholar and a research scientist. During her study at UGA, Dr. Guo received both pre-doctoral and postdoctorl fellowships from American Heart Association. She joined the University of Texas Health Science Center at Tyler as an Assistant Professor in 2019 with a NIH K99/R00 grant. She has published more than 30 papers in reputed journals (e.g., Circ. Res., ATVB, J Lipid Res., J. Hepatol., and Am. J. Pathol.). She also serves in the reviewer committee for the American Heart Association.

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Milton D. Chiang, J Obes Weight Loss Ther 2022, Volume 12

Endotoxin induces pulmonary inflammation in obese mice

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Obesity is a global public health concern that has been related to an increased risk of several chronic diseases, such as cardiovascular diseases, type II diabetes mellitus, obstructive sleep apnea. As overweight has become more prevalent, the effect of obesity on acute lung injury incidence and outcome has gained more attention in recent years. Despite this negative correlation, some investigations have revealed a controversial correlation, termed "obesity paradox" in which overweight with established cardiovascular disease have a better prognosis. To elucidate further on this issue, we conducted this study with a diet-induced obesity murine model. Obese mice, adult C57BL/6J mice fed a high-fat diet for 12 weeks, received normal saline or endotoxin (lipopolysaccharide, 10 mg/kg, intraperitoneally administered) (denoted as the Obese and LPS group, respectively). After 48 hours of administration of normal saline or endotoxin, mice were euthanized. The level of lung injury (injury score, tissue water content, and leukocyte infiltration in lung tissues) in the LPS group was significantly higher than in the Obese group (p=0.0002; =0.02; and =0.0001, respectively; Figure 1). The levels of pulmonary cytokines (tumor necrosis factor-α [TNF-α], interleukin-6 [IL-6], and interleukin-1β [IL-1 β]) in the LPS group were also significantly higher than in the Obese group (p=0.03;=0.0003; =0.0007; respectively; Figure 1D-F). Moreover, the level ofpulmonary inducible nitric oxide synthase (iNOS, indicator of pro- inflammatory M1 phase macrophage polarization) in the LPS group was significantly higher than in the Obese group (p=0.0087; Figure 1G). The expression levels of nuclear factor-κB (NF-κB) and hypoxia-inducible factor-1α(HIF-1α) in lung tissues in the LPS group were significantly higher than in the Obese group (p=0.003 and <0.0001, respectively; Figure 2A), too. Similar pictures were observed in the levels of oxidation and apoptosis in lung tissues, as the levels of malondialdehyde (MDA) and DNA fragmentation (assayed using the terminal deoxynucleotidyl transferase dUTP nick end labeling [TUNEL] method and the count of TUNEL-positive cells) in the LPS group were significantly higher than in the Obese group (p<0.0001 and =0.001, respectively; Figure 2B-C)

In conclusion, data from this study collectively demonstrate that endotoxin induces significant inflammation in obesity mice.

Biography

Milton Chiang currently is a Ph.D. candidate in the department of the International Master/Ph.D. program in Medicine, Taipei Medical University, Taiwan. He got his specialty in Internal Medicine at Francisco Marroquin University, Guatemala, and Medical Degree in Rafael Landivar University, Guatemala. His research focuses on obesity, inflammation, and its therapeutic.

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