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EDITORIAL

An American Physiological Society cross-journal Call for Papers on "The Physiology of Obesity"

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INTRODUCTION

Obesity is a medical condition described as excess body weight in the form of fat. Although the first recorded use of the word "obesity" was by Randle Cotgrave in 1611 (1), obesity (in earlier times, "corpulence") has been recognized at least since prehistory. The first known depiction of an obese person is the Venus of Willendorf, an 11-cm-long limestone figurine discovered in Wallendorf, Austria, in 1908, which has been dated to the Upper Paleolithic period, ~25,000 years ago (2).

Being overweight has been in the past (and sometimes still is) taken as an indicator of prosperity, power, and fertility (3), since—as highlighted by Dr. Robert Fogel, recipient of the 1993 Nobel Memorial Prize in Economic Sciences—throughout most of human history, "chronic malnutrition has been the norm" (4). It was only after progressive technological advances in crop production, starting in Britain during the Second Agricultural Revolution (circa mid-17th to late 19th centuries) (5), and continuing up to and beyond the Third Agricultural Revolution (The Green Revolution, circa 1950s and 1960s) (6), that food became more plentiful in selected regions of the globe. That coincided with the emergence of reports in the mainstream medical literature in the 18th century about the medical consequences of being overweight (7). However, a connection between being overweight and having poor health had already been recognized long before, in antiquity, by Hippocrates of Kos (circa 460–370 BC), who noted that obese individuals had shorter life-spans and suffered from reduced fertility (8).

Fat deposition is a natural adaptive physiologic process that facilitates energy storage during times of food abundance (9). This process effectively becomes maladaptive when the balance between food consumption (energy intake) versus physical activity (energy expenditure) is perturbed, where more food and less activity create a positive energy balance that results in a gain in body weight, ultimately culminating in obesity. There is increasing

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recognition of other factors beyond nutrition and physical activity that might influence body weight gain (9), and behavioral, environmental, and genetic risk factors now also constitute components of integrated models of obesity pathogenesis (10).

Today, both "overweight" and "obese" classifications are numerically defined, using what was originally termed the Quetelet Index, first described in 1832 (11) by Belgian mathematician, astronomer, and statistician Dr. Adolphe Quetelet (1796–1874), the founder of the science of anthropometry (12). The Quetelet Index was renamed "body mass index" (BMI) in 1972 by American physiologist Dr. Ancel Keys (1904–2004) and coworkers (13) during Dr. Keys' extensive work on nutrition and human starvation, some of which has been published in the pages of the journals of our American Physiological Society (APS) (14–16). BMI is defined as the body mass (in kg) divided by the square of the body height (in m), and is expressed in units of kg/m^2 (11). Persons with a BMI between 25 and <30 kg/m^2 are considered overweight, and persons with a BMI ≥ 30 kg/m^2 are considered obese. Based on those criteria, the World Health Organization reported in 2016 that the incidence of obesity worldwide had tripled since 1975, and estimated the 2016 worldwide incidence of adults (persons $\geq$18 yr of age) being overweight at 39% (1.9 billion adults), of which 13% (650 million adults) were obese, with over 340 million children and adolescents aged 5–19 also overweight or obese. In addition, 39 million children under the age of 5 were overweight or obese in 2020 (17).

Obesity was recognized by the World Health Organization as a global epidemic in 1997 (18), and obesity is now one of the leading preventable causes of death worldwide (19). The global epidemic of obesity has also been accompanied by a surge in obesity-related disease, particularly cardiovascular (including cerebrovascular) diseases, type 2 diabetes, asthma, obstructive sleep apnea, some types of cancer, osteoarthritis, and depression (20). The dramatic increase in the incidence of obesity and obesity-related disease over the past four decades has led to a surge of interest in basic, translational, clinical, public health, and epidemiological studies, as is evident from the published literature. As of July 27, 2022, for the calendar year 1991, 2,900 published articles were retrievable from the MEDLINE database via PubMed using the keyword "obesity." At 10-year intervals after that, the number of retrievable articles increased to 7,521 for the year 2001, 23,259 articles for the year 2011, and 33,853 articles for the year 2021. Some of those studies emphasized a pressing need to elucidate mechanisms that are responsible for obesity and its comorbidities (21) to facilitate provision of the best care for patients with obesity. The current high level of interest in this topical area of physiology continues to be reflected by the diverse array of obesity-related topics that have recently been the subject of comprehensive reviews in the three review journals of the APS: *Physiology*, *Comprehensive Physiology*, and *Physiological Reviews*, which have included consideration of neuronal regulation of brown/beige versus white adipose tissue (22), sex differences in obesity-related cardiovascular disease risk (23), and mechanisms of obesity-related cardiomyopathy (24, 25).

To support the current widespread interest in the physiology of obesity across the spectrum of affected tissues, organs, and systems, 13 journals that publish Research Articles within the APS publications portfolio have teamed together to issue a cross-journal Call for Papers on "The Physiology of Obesity." This Call for Papers aims to attract Research Articles that report original research studies on the physiological processes responsible for obesity or consequences of (and comorbidities associated with) obesity including cardiovascular, lung, kidney, and bladder disease, as well as interorgan cross talk between adipose tissue and the heart or vasculature, disruption of the gut-liver axis and onset of diet-induced liver and kidney inflammation, consideration of adipose tissue as an endocrine organ, and adipocyte and adipokine biology. Manuscripts that consider multi-organ/multi-system cross talk that identify novel interorgan causes or effects of obesity are particularly encouraged. From an educational perspective, the submission of Research Articles addressing how obesity and its consequences are effectively taught to a certain type(s) of student/learner in outreach situations is encouraged. This Call for Papers also aims to attract Reviews and Mini-Reviews that provide new insights or address specific gaps in our knowledge. Journal-specific information is provided below for each of the 13 participating journals.

For the submission of their manuscript, authors should select from the list below the journal that best fits the focus of their manuscript. During the submission process, authors should select "Call for Papers: The Physiology of Obesity" from the "Category" drop-down menu listed under the "Keywords & Special Sections" heading at eJPress. The deadline for submissions is June 30, 2023. A cross-journal collection at https://journals.physiology.org will showcase all articles published in the 13 participating journals that participate in this Call for Papers.

ADVANCES IN PHYSIOLOGY EDUCATION

Advances in Physiology Education (*Advances*) is requesting submissions for a collection of teaching about Lifestyle Medicine concepts (obesity, nutrition, exercise, and metabolism) to a specific learner population (K-12 outreach, undergraduate, graduate, professional, and lay public) (26, 27). A collection of "how to do it" papers similar to the *Advances* article type Illuminations would be appropriate. The physiology of obesity can be addressed as a risk factor or as a chronic disease. Accompanying laboratory activities would also be welcome.

Please submit to *Advances* via the eJPress portal at https://advances.msubmit.net. Please address any questions related to this Call for Papers to *Advances* Editor-in-Chief, Dr. Barbara E. Goodman, at barb.goodman@usd.edu.

AMERICAN JOURNAL OF PHYSIOLOGY-CELL PHYSIOLOGY

The *American Journal of Physiology-Cell Physiology* (*AJP-Cell*) is dedicated to the study of cell and molecular physiology in all tissues and model systems. Manuscripts dealing with the structure and function of cell membranes, contractile systems, cellular organelles, and membrane channels, transporters, and pumps are encouraged. Studies dealing with integrated regulation of cellular function, including

mechanisms of signal transduction, development, reproduction, metabolism, adaptation, gene expression, cell-to-cell, cell-microbe, and extracellular matrix interactions are also eagerly sought. Interdisciplinary studies that apply the approaches of biochemistry, biophysics, molecular biology, morphology, epigenetics, redox biology, and immunology to the determination of new principles in cell physiology, cell pathophysiology, and therapy are especially welcome. Recent work related to obesity that has been published in our journal include reports that question whether thermogenic T cells represent a cell therapy for obesity (28), a review on in vitro and in vivo models of adipocytes (29), and consideration of adipose tissue inflammation in obesity (30).

Please submit your Research Article, Rapid Report, Perspective (after approval of a pre-submission enquiry), Method, Mini-Review, or Systematic Review (presubmission enquiries) to the *AJP-Cell* via the eJPress portal at https://ajpcell.msubmit.net/. Please address any questions related to this Call for Papers to *AJP-Cell* Editor-in-Chief, Dr. Liliana Schaefer, at lschaefer@physiology.org and *AJP-Cell* Managing Editor, Mr. Michael Pogachar, at mpogachar@physiology.org.

AMERICAN JOURNAL OF PHYSIOLOGY-ENDOCRINOLOGY AND METABOLISM

The *American Journal of Physiology-Endocrinology and Metabolism* (*AJP-Endo*) publishes original, mechanistic studies on the physiology of endocrine and metabolic systems, namely those relating to body weight homeostasis. Physiological, cellular, and molecular studies in whole animals or humans will be considered. Given the focus of our journal on metabolism, *AJP-Endo* is the home to a diverse array of reports on metabolic syndrome and obesity, having recently considered, for example, the sex-dependence of transcriptomic responses in the skeletal muscle and liver in the offspring of obese mice (31) and correlations between the metabolomic signatures of low- and high-adiposity neonates and maternal BMI (32).

Specific themes addressed in *AJP-Endo* include, but are not limited to, mechanisms of hormone and growth factor action; neuronal, hormonal, and nutritional regulation of metabolism, inflammation, microbiome and energy balance; integrative organ cross talk; paracrine and autocrine control of endocrine cells; function and activation of hormone receptors; endocrine or metabolic control of channels, transporters, and membrane function; temporal analysis of hormone secretion and metabolism; and mathematical/kinetic modeling of metabolism. Novel molecular, immunological, or biophysical studies of hormone action are also welcome. If your manuscript addresses the impact of obesity of any of these systems, submit it to *AJP-Endo* via the eJPress portal at https://ajpendo.msubmit.net/ and address any questions related to this Call for Papers to *AJP-Endo* Associate Managing Editor, Ms. Teki Best, at tbest@physiology.org.

AMERICAN JOURNAL OF PHYSIOLOGY-GASTROINTESTINAL AND LIVER PHYSIOLOGY

The *American Journal of Physiology-Gastrointestinal and Liver Physiology* (*AJP-GI*) publishes papers on all aspects of the physiology and pathophysiology of the digestive organs. For this call, we encourage submissions detailing mechanisms through which obesogenic processes alter, and are in turn altered by, the physiology of the gastrointestinal tract, gut microbiome, exocrine pancreas, and liver. By example, recent papers in the journal relevant to this call include studies on altered nutrient uptake driven by the pathophysiology of obesity (33), effects of obesity on digestive physiology (34, 35), and microbiota-gut-liver communication in obesity (36).

We in particular encourage submissions related to how metabolic dysfunction and digestive pathophysiology promote a vicious cycle of disease, the importance of understanding and targeting inter-organ communication to restore homeostatic regulation, and the role of the microbiome in obesity-driven or obesity-inducing digestive pathophysiology. Benchmark methodologies for modeling these processes, critical datasets that will drive ongoing understanding, and studies that effectively connect laboratory studies to clinical observations are also most welcome.

Please submit your manuscript to *AJP-GI* via the eJPress portal at https://ajpgi.msubmit.net/. Any questions related to this Call for Papers can be addressed to *AJP-GI* Editor-in-Chief, Dr. Mark R. Frey, at mfrey@physiology.org and *AJP-GI* Assistant Managing Editor, Ms. Jami Jones, at jjones@physiology.org.

AMERICAN JOURNAL OF PHYSIOLOGY-HEART AND CIRCULATORY PHYSIOLOGY

The *American Journal of Physiology-Heart and Circulatory Physiology* (*AJP-Heart*) is especially interested in Research Articles and Rapid Reports on mechanisms whereby obesity alters the cardiovascular response to injury. We encourage submissions related to the effects of obesity on cardiac cross talk with other organs, including kidney, spleen, liver, and lung. How obesity impairs blood vessel physiology is relevant, as is effects of obesity on the immune system and how immunity influences development of cardiovascular diseases (e.g., hypertension, heart failure, atherosclerosis, and aneurysm formation), both as instigating mechanisms and as an influencer of the response to pathology. In addition, the editors encourage submissions on the interplay between obesity, diabetes, and cardiovascular disease.

Examples of recent articles published in *AJP-Heart* that would fit into this call include articles that evaluate the effects of obesity on the ability of the cardiovascular system to respond to COVID-19 (37), and articles that study the effects of interventions such as exercise on cardiovascular responses (38). The interplay between diabetes, obesity, and cardiovascular disease have clear relevance (39, 40), as does the role of obesity in pregnancy-related cardiovascular disease (41). Review Articles and Systematic Review Articles related to obesity influence on cardiovascular physiology are welcome, particularly those that summarize transcriptomic, proteomic, or metabolomic evaluations that reveal mechanisms of obesity regulation of cardiac or vascular physiological and pathophysiological processes. Submission of novel methodological advances to study obesity in the cardiovascular setting are also encouraged.

Please submit your manuscript to *AJP-Heart* via the eJPress portal at https://ajpheart.msubmit.net. Any questions related to this Call for Papers can be addressed to

AJP-Heart Executive Editor, Ms. Kara Hansell Keehan, at ajphearteditor@gmail.com.

AMERICAN JOURNAL OF PHYSIOLOGY-LUNG CELLULAR AND MOLECULAR PHYSIOLOGY

Probably the most well-known historical reference to the impact of obesity on the respiratory system comes from Charles Dickens' first novel, *The Posthumous Papers of the Pickwick Club* (1837), in which the markedly obese character "Joe" fell asleep uncontrollably during the day. Subsequent to that, both obstructive sleep apnea syndrome (42, 43) and alveolar hypoventilation syndrome (44) have been called Pickwickian Syndrome, much to the chagrin of Dr. Julius H. Comroe (1911–1984), 33rd President of our APS, who took issue with the terminology (45). This terminology has now fallen out of favor, as it does not distinguish these two (sometimes co-existing syndromes) as separate disorders. Since that time, in addition to sleep-disordered breathing and obesity-related alveolar hypoventilation, obesity has been linked with a number of respiratory disorders, notably asthma, chronic obstructive lung disease (COPD), and acute respiratory distress syndrome (ARDS) (46).

Obesity is a major risk factor and also a disease modifier of asthma, both in children and in adults (47). Most preclinical work addressing obesity in asthma has focused on the role of metabolism in airway hyperreactivity, and the *American Journal of Physiology-Lung Cellular and Molecular Physiology* (*AJP-Lung*) is home to some of those studies, including recent reports on respiratory function in patients with diabetes with and without diabetes-associated obesity (48). Subsequent recent reports provided an explanation for why obese individuals with hyperinsulinemia are prone to airway hyperreactivity (49, 50), and, building on those studies, a preclinical demonstration that medications that target insulin may be effective treatments for obesity-related asthma (51). Our *Journal* welcomes the submission of any reports addressing the physiological link between obesity and asthma.

The impact of obesity on respiratory disease also presents us with two paradoxes: the first of these relates to obese patients with COPD (52). Although it is not known whether obesity is a risk factor for COPD or vice versa (or both), obesity is associated with increased morbidity in moderate to severe chronic COPD, and, paradoxically, at the same time obesity is associated with decreased risk of acute COPD exacerbations (53) and decreased mortality in patients with COPD (54). A second paradox is found in a critical care setting in patients with ARDS, where obesity is associated with increased risk of ARDS, and worse in-hospital morbidity (55), but, paradoxically, obesity is associated with a lower mortality in patients with ARDS compared with nonobese or underweight patients with ARDS (56, 57). It is not known why obesity has a protective effect in patients with COPD and ARDS. It has been speculated that higher energy reserves, blunted inflammatory responses and inflammatory preconditioning, endotoxin neutralization by adipose tissue, prevention of muscle wasting and weakness, and increased stability of the circulatory system due to higher baseline function of the renin-angiotensin system may underlie the protective effects of obesity (58), but few of these ideas have been tested in preclinical experimental systems. Some experimental progress has been made and reported in 2020 in *AJP-Lung* (59), where investigators demonstrated that adipose-derived exosomes in obese rats protected against ventilator-induced lung injury, ostensibly through inhibition of a TRPV4/Ca^{2+}-mediated pathway. It is clear that much more exciting physiological research remains to be done, and *AJP-Lung* welcomes the submission of reports addressing any of these topics.

Remaining with a critical care setting, probably the most recent noteworthy observation related to obesity and the respiratory system was the identification of obesity as a key risk factor for COVID-19 (60), which remains an active area of investigation to which reports published in *AJP-Lung* have also contributed (61), and where a separate Call for Papers on "The Pathophysiology of COVID-19 and SARS-CoV-2 Infection" is currently open (62).

If your manuscript addresses any aspect of the impact of obesity on the respiratory system, from the nose to the alveolus, please submit to *AJP-Lung* via the eJPress portal at https://ajplung.msubmit.net. Please address any questions related to this Call for Papers to *AJP-Lung* Editor-in-Chief, Dr. Rory E. Morty, at rory.morty@med.uni-heidelberg.de and *AJP-Lung* Managing Editor, Mr. Michael Pogachar, at mpogachar@physiology.org.

AMERICAN JOURNAL OF PHYSIOLOGY-REGULATORY, INTEGRATIVE, AND COMPARATIVE PHYSIOLOGY

The *American Journal of Physiology-Regulatory, Integrative, and Comparative Physiology* (*AJP-Regu*) has a broad scope that encompasses most organ systems and experimental models, from invertebrates to humans. *AJP-Regu* in particular is focused on how organ systems interact to maintain normal physiologic functioning, and how those interactions are disrupted in disease states. Obesity is a complex disorder that affects all organ systems, and, increasingly, multiple species, as the effects of the obesogenic environment constructed by humans exerts influence on the metabolism of the species that cohabitate with humans. In addition, emerging evidence indicates that the negative impacts of human industrialism on the environment are also negatively influencing the metabolic function of humans and other species as well.

AJP-Regu welcomes manuscripts that seek to delineate how obesity affects interorgan communication and metabolic functioning in humans and in standard and nonstandard animal models. In addition, *AJP-Regu* is particularly interested in manuscripts investigating the impact of environmental insults on metabolic health of humans and nonhuman species. To submit a manuscript to *AJP-Regu* as part of this Call for Papers, please visit https://ajpregu.msubmit.net at the eJPress portal. Please address any questions regarding this Call to *AJP-Regu* Editor-in-Chief, Dr. Gina L. C. Yosten, at gina.yosten@health.slu.edu and *AJP-Regu* Editorial Assistant, Ms. Katie Kornuta, at kkornuta@physiology.org.

AMERICAN JOURNAL OF PHYSIOLOGY-RENAL PHYSIOLOGY

Obesity is a potent risk factor for the development of kidney disease and significantly increases the risk of hypertension, diabetes, and chronic kidney disease. Indeed, obesity and its association with kidney disease was highlighted for World Kidney Day education in 2017 (reviewed in Ref. 63). Obesity causes hyperfiltration at the glomerular level and a recent study in the *American Journal of Physiology-Renal Physiology (AJP-Renal)* determined that tubuloglomerular feedback is reduced in response to high-fat diet-induced obesity (64) and altered renal hemodynamics is associated with glomerular lipid accumulation (65).

Renal inflammation and fibrosis pathways are known to contribute to obesity-induced kidney damage, and manuscripts that examine the modulation of macrophage, T cell, or cytokine profiles that contribute to disease are welcome, such as one recent report that assessed how macrophage depletion impacted the progression of renal injury (66). Interestingly, podocytes can endocytose lipids and processes that aggravate this uptake can accelerate obesity-related glomerulopathy (67) or dysregulate podocyte immunoproteasomes (68). *AJP-Renal* also publishes studies beyond the kidney, that address bladder physiology and function, where obesity is a known modulator of voiding function (69) and urological complications, in both males and females (70).

Given the high incidence of obesity in the western world, the high fructose-fed rat model has been successfully used to demonstrate the role of diet composition in obesity-induced kidney damage. In humans, high-fructose corn syrup-sweetened soft drink consumption increases vascular resistance in the kidneys, at rest and during sympathetic activation [(71), which was *AJP-Renal* "Paper of the Year"]. Importantly, the role of exercise as an intervention in preventing kidney damage is a hot topic, and exercise studies as they relate to protection against obesity-induced kidney damage are welcomed (72, 73). Sex differences are known to occur in the onset of kidney disease and *AJP-Renal* welcomes preclinical and clinical studies that aim to identify how differences in physiological function can contribute to differences in obesity-induced kidney disease onset (74). *AJP-Renal* also welcomes studies that examine the cross-roads between interorgan signaling, for example the role of adipocyte signaling on kidney function (75) or the interplay between kidney mass and insulin signaling (76).

If your manuscript addresses any aspect of the impact of obesity, metabolic syndrome, or type 2 diabetes on kidney function, from a single-cell to the whole organism, please submit your manuscript to *AJP-Renal* via the eJPress portal at https://ajprenal.msubmit.net; and please contact *AJP-Renal* Editor-in-Chief, Dr. Heddwen L. Brooks, at hlbrooks@physiology.org and *AJP-Renal* Associate Managing Editor, Ms. Teki Best, at tbest@physiology.org with any questions that you may have about this Call.

FUNCTION

From organelles to organisms, *Function* seeks papers that contribute to defining the mechanistic basis of living systems in health and disease. *Function* aims to publish major advances that extend physiological understanding of biological function and the changes associated with disease states. In the field of obesity, *Function* is particularly interested in findings that are of general interest, being relevant to different organ systems and illuminating general functional aspects. This includes, for example, the role of inflammation in adipose tissues (77, 78). Fields that are related to obesity, such as mitochondrial function (79) and general aspects of the function of the insulin system (80) continue to be core areas of interest for *Function*. We would also be interested in papers relating to control of food intake by the brain.

Function publishes not only original full research articles, but also short Focus articles that report an important new observation, based on solid evidence, but without the full exploration required for a full paper. All full research articles are accompanied, or followed up, by a perspective article, written by an acknowledged expert in the field. Evidence Review Articles, quoting only original papers, are commissioned from leading experts, but suggestions for such articles are welcome. Please submit your manuscript, through the online portal at https://mc.manuscriptcentral.com/function. Please address any questions related to this Call for Papers to the *Function* Editor-in-Chief, Dr. Ole H Petersen, at functioneditor@physiology.org and *Function* Managing Editor, Dr. Chris England, at cengland@physiology.org.

JOURNAL OF APPLIED PHYSIOLOGY

The *Journal of Applied Physiology* (*JAPPL*) publishes original research on the acute and adaptative responses of multiple organ systems to physiological, environmental, and pathological stressors. The *JAPPL* has a long-standing interest in research related to the effects of obesity on the physiological function of individual tissues and organ systems, as well as its role in the progression and severity of age-related dysfunction and diseases such as diabetes (81), nonalcoholic fatty liver disease (82), cardiovascular disease (83), and respiratory diseases (84). Previous research in the *Journal* has addressed the role of exercise on weight loss, as well as the role of physical inactivity as a risk factor for becoming obese. In response to this Call for Papers, the *JAPPL* is especially interested in original research that examines sexual dimorphism in the physiological response of tissues and organ systems to a high-fat diet and obesity. Furthermore, the *Journal* is interested in research articles that examine sexual dimorphism in the response of both visceral and subcutaneous adipose tissues to both endurance and resistance exercise. The *Journal* continues to be interested in research examining the effect of intracellular adipose tissue on the function of organs/tissues such as skeletal muscle, heart, and liver. Furthermore, research on the identification and function of adipose derived factors (adipokines) on interorgan cross talk and adaptation to exercise is of great interest.

Submit your manuscript to *JAPPL* via eJPress portal at https://jappl.msubmit.net. Please address any questions related to this Call for Papers to the *JAPPL* Editor-in-Chief, Dr. Sue C. Bodine, at sue-bodine@uiowa.edu and *JAPPL* Associate Managing Editor, Ms. Teki Best, at tbest@physiology.org.

JOURNAL OF NEUROPHYSIOLOGY

Obesity and the obesity epidemic is a major health issue worldwide. The neurophysiology of obesity is at the core of many aspects of this epidemic, and we are just beginning to appreciate the complexity of the neurophysiology of obesity or "obesities" (85). Obesity is the result of an imbalance in energy intake and energy expenditure, which can have many causes. Genetic and epigenetic factors, age, sex, diet, and exercise influence basal expenditure that makes up 50%–70% of our total energy expenditure. Energy intake is very dependent upon environment and eating behavior, which have strong neuroscience components. Obesity has also a strong neurodevelopmental component, as prenatal and postnatal influences can have long-term consequences for eating behavior that can even be passed on to the next generation, a topic of great neuroscience-related interest. It is well established that the hypothalamus plays a critical role in regulating the physiological homeostasis. This brain region integrates information about the metabolic state of an individual, which is mediated by hormones, neuromodulators, and nutrients. Neuroscience research has identified peptides that are critical for the communication between the gut and the nervous system, and disturbances in this control can lead to maladaptive feeding behavior. A major focus has become the microbiome itself, which seems to be involved in various problems associated with obesity. Food addiction is another research area with important clinical implications. There is also an important scientific overlap with psychology and psychiatry when it comes to a variety of eating disorders, which includes binge eating disorder. In many cases, eating disorders can cause an energy imbalance with opposite consequences when compared with obesity. These disorders include anorexia or bulimia nervosa.

Although the focus of this call is on obesity, understanding the drivers of energy imbalances in general is of great interest for the *Journal of Neurophysiology* (*JNP*). Submit your manuscript to the *JNP* via the eJPress portal at https://jn.msubmit.net/. Please address any questions related to this Call for Papers to *JNP* Editor-in-Chief, Dr. Jan-Marino Ramirez, at nino1@uw.edu and *JNP* Assistant Managing Editor, Ms. Jami Jones, at jjones@physiology.org.

PHYSIOLOGICAL GENOMICS

Physiological Genomics (*PG*) publishes Research Articles, Review articles, and Rapid Reports in a wide area of research focused on uncovering the links between genes, proteins and pathways, and physiology at all levels of biological organization. *PG* welcomes articles that address the relationship between genes, proteins, metabolites, and pathways, and function in the broad area of obesity. *PG* is enthusiastic about receiving articles in the area of genome-wide association studies investigating the genetic basis of obesity. Applications of "omics" technologies such as transcriptomics, proteomics, metabolomics, and microbiome metagenomics to understand the physiological alterations that result from different diets, that contribute to obesity, or that result from obesity, in any tissue or organ system, would be an excellent fit for *PG*, for example (86). In addition, investigation of the signaling pathway networks including microRNAs, long noncoding RNAs, and protein-protein interaction networks that result from different diets, that contribute to the development of obesity, or that arise as a result of obesity, in any tissue or organ system (for example, see Ref. 87), are also of great interest.

Submit your manuscript to the *PG* via eJPress portal at https://physiolgenomics.msubmit.net. Please address any questions related to this Call for Papers to *PG* Editor-in-Chief, Dr. Hilary A. Coller, at hcoller@ucla.edu and *PG* Assistant Managing Editor, Ms. Jami Jones, at JJones@physiology.org.

PHYSIOLOGICAL REPORTS

Physiological Reports is an open-access journal that accepts manuscripts submitted directly by the authors or manuscripts that have been rejected but referred to us by the other APS journals. If you receive a rejection and referral, you can revise your manuscript based on the reviews you already received from the supporter journal, before transferring the manuscript to *Physiological Reports*. We publish research across all areas of basic and translational physiology. Case Reports and Review articles are acceptable, so long as they have a physiological focus.

We welcome manuscripts from studies covering the effects of obesity or adipose tissue on several organ systems, including cellular studies of the physiology of adipocytes. Weight gain and obesity not only affect the development of many multi-organ diseases, but also impacts our physiology during, for example, physical exercise, cognitive challenges, pregnancy, and childhood growth. Time-restricted feeding, new pharmaceuticals, and exercise associated with weight loss and weight-loss maintenance are hot topics in experimental and clinical physiology. Comparative physiology may give us clues to how, for example, brown and polar bears can survive a diet high in fats and cholesterol with extreme seasonal fluctuations (88).

Some of the key studies published in *Physiological Reports* relating to obesity include a study on how the liver responds to a gastric bypass procedure (89) and how overfeeding changes body fat distribution via a mechanism involving gonadal hormones (90). Exercise impacts the physiology of adipose tissue in overweight men (91), but the type of exercise appears to have little importance, at least in mice (92). Maternal obesity increases the risk of gestational diabetes, which affects both mother and fetus, and reducing ambient oxygen tension can reduce some risks for the mother, but not the offspring (93). Finally, two papers report on the effects of changing adipose tissue from white to brown and vice versa (94, 95).

Physiological Reports welcomes manuscripts detailing studies that are descriptive, with incomplete mechanistic insights, studies that falsify the hypothesis (i.e., "negative" studies) and studies that confirm major conclusions previously published. Please submit your manuscript, through the online portal at https://mc.manuscriptcentral.com/physiologicalreports. For any questions regarding this Call for Papers, please contact *Physiological Reports* Editor-in-Chief, Dr. Thomas Kleyman, at kleyman@pitt.edu, and *Physiological Reports* Deputy Editor-in-Chief, Dr. Morten B. Thomsen, at mbthom@sund.ku.dk.

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AUTHOR CONTRIBUTIONS

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