

This is an Open Access document downloaded from ORCA, Cardiff University's institutional repository:<https://orca.cardiff.ac.uk/id/eprint/154634/>

This is the author's version of a work that was submitted to / accepted for publication.

Citation for final published version:

Bodine, Sue C., Brooks, Heddwen L., Coller, Hilary A., Domingos, Ana I., Frey, Mark R., Goodman, Barbara E., Kleyman, Thomas R., Lindsey, Merry L., Morty, Rory E., Petersen, Ole H. , Ramírez, Jan-Marino, Schaefer, Liliana, Thomsen, Morten B. and Yosten, Gina L. C. 2022. An American Physiological Society cross-journal call for papers on "The physiology of obesity". *American Journal of Physiology - Lung Cellular and Molecular Physiology* 323 (5) , L593-l602. 10.1152/ajplung.00335.2022

Publishers page: <http://dx.doi.org/10.1152/ajplung.00335.2022>

Please note:















Changes made as a result of publishing processes such as copy-editing, formatting and page numbers may not be reflected in this version. For the definitive version of this publication, please refer to the published source. You are advised to consult the publisher's version if you wish to cite this paper.

This version is being made available in accordance with publisher policies. See <http://orca.cf.ac.uk/policies.html> for usage policies. Copyright and moral rights for publications made available in ORCA are retained by the copyright holders.



EDITORIAL

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

 Sue C. Bodine,¹  Heddwen L. Brooks,²  Hilary A. Collier,^{3,4,5}  Ana I. Domingos,⁶  Mark R. Frey,^{7,8,9}  Barbara E. Goodman,¹⁰  Thomas R. Kleyman,^{11,12,13}  Merry L. Lindsey,^{14,15}  Rory E. Morty,^{16,17}  Ole H. Petersen,¹⁸  Jan-Marino Ramirez,^{19,20,21}  Liliana Schaefer,²²  Morten B. Thomsen,²³ and  Gina L. C. Yosten²⁴

¹Division of Endocrinology and Metabolism, Department of Internal Medicine, Carver College of Medicine, University of Iowa, Iowa City, Iowa; ²Department of Physiology, University of Arizona College of Medicine, Tucson, Arizona; ³Department of Molecular, Cell and Developmental Biology, University of California, Los Angeles, California; ⁴Molecular Biology Institute, University of California, Los Angeles, California; ⁵Department of Biological Chemistry at the David Geffen School of Medicine, University of California, Los Angeles, California; ⁶Department of Physiology, Anatomy and Genetics, University of Oxford, Oxford, United Kingdom; ⁷The Saban Research Institute, Children's Hospital Los Angeles, University of Southern California Keck School of Medicine, Los Angeles, California; ⁸Department of Pediatrics, University of Southern California Keck School of Medicine, Los Angeles, California; ⁹Department of Biochemistry and Molecular Medicine, University of Southern California Keck School of Medicine, Los Angeles, California; ¹⁰Division of Basic Biomedical Sciences, Sanford School of Medicine of the University of South Dakota, Vermillion, South Dakota; ¹¹Department of Medicine, University of Pittsburgh, Pittsburgh, Pennsylvania; ¹²Department of Cell Biology, University of Pittsburgh, Pittsburgh, Pennsylvania; ¹³Department of Pharmacology and Chemical Biology, University of Pittsburgh, Pittsburgh, Pennsylvania; ¹⁴School of Graduate Studies and Research, Meharry Medical College, Nashville, Tennessee; ¹⁵Research Service, Nashville VA Medical Center, Nashville, Tennessee; ¹⁶Department of Translational Pulmonology and the Translational Lung Research Center Heidelberg, University Hospital Heidelberg, member of the German Center for Lung Research (DZL), Heidelberg, Germany; ¹⁷Department of Lung Development and Remodelling, Max Planck Institute for Heart and Lung Research, Bad Nauheim, Germany; ¹⁸School of Biosciences, Cardiff University, Cardiff, United Kingdom; ¹⁹Department of Neurological Surgery, University of Washington Medical Center, Seattle, Washington; ²⁰Center on Human Development and Disability, University of Washington, Seattle, Washington; ²¹Center for Integrative Brain Research at the Seattle Children's Research Institute, University of Washington, Seattle, Washington; ²²Institute of Pharmacology and Toxicology, Goethe University Frankfurt am Main, Frankfurt, Germany; ²³Department of Biomedical Sciences, University of Copenhagen, Copenhagen, Denmark; and ²⁴Department of Pharmacology and Physiology, Saint Louis University School of Medicine, St. Louis, Missouri

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 Willendorf, 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

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 $<30 \text{ kg}/\text{m}^2$ are considered overweight, and persons with a BMI $\geq 30 \text{ kg}/\text{m}^2$ 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 ≥ 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 inter-organ 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://ajphheart.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/Ca²⁺-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. Heddwyn 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 adaptive 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. Collier, 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.

ACKNOWLEDGMENTS

The authors thank Colette E. Bean, Chief Publishing Officer of the American Physiological Society, Dr. David D. Gutterman, Chair of the Publications Committee of the American Physiological Society, and Michael Pogachar, Managing Editor of *AJP-Lung*, for reading and editing this Editorial.

GRANTS

H.L.B. is supported by the National Institutes of Health (NIH) through R01 HL131834, R01 NS110749-01 and AZ ABRC, ADHS16-162517. H.A.C. is supported by the NIH through R01 AR070245-01A1 and R01 CA221296-01A1, the Melanoma Research Alliance, The Cancer Research Institute, the Broad Stem Cell Center, and the Jonsson Comprehensive Cancer Center. M.R.F. is supported by NIH Awards R01DK095004 and R01DK119694. B.E.G. is partially supported by the NIH through P20GM103443. T.R.K. is supported by the National Institutes of Health through P30 DK079307. M.L.L. acknowledges funding from the NIH under Award Number HL137319, and from the Biomedical Laboratory Research and Development Service of the Veterans Affairs Office of Research and Development under Award Number 5101BX000505. R.E.M. is supported by the University of Heidelberg, the Max Planck Society (MPI-HLR), the German Center for Lung Research (*Deutsches Zentrum für Lungenforschung*; DZL) (DZL-TLRC); and the German Research Foundation (*Deutsche Forschungsgemeinschaft*; DFG) through EXC2026 [390649896], SFB1213 [268555672], KFO309 [284237345], Mo1789/1-1 [160966624], and Mo1789/4-1 [420759458]. O.H.P. is supported by a grant from Innovate UK (10033786). J.-M.R. is supported by the NIH through R01 HL151389, R01 HL126523, R01 HL144801, and P01 HL090554. L.S. is supported by the DFG through SFB1039 and SFB1177 [259130777] and EXC 2026 [390649896].

DISCLOSURES

S. C. Bodine is the Editor-in-Chief of the *Journal of Applied Physiology*. H. L. Brooks is the Editor-in-Chief of the *American Journal of Physiology-Renal Physiology*. H. A. Collier is the Editor-in-Chief of *Physiological Genomics*. A. I. Domingos is the Editor-in-Chief of the *American Journal of Physiology-Endocrinology and Metabolism*. M. R. Frey is the Editor-in-Chief of the *American Journal of Physiology-Gastrointestinal and Liver Physiology*. B. E. Goodman is the Editor-in-Chief of *Advances in Physiology Education*. T. R. Kleyman is the Editor-in-Chief of *Physiological Reports*. M. L. Lindsey is the Editor-in-Chief of the *American Journal of Physiology-Heart and Circulatory Physiology*. R. E. Morty is the Editor-in-Chief of the *American Journal of Physiology-Cellular and Molecular Physiology*. O. H. Petersen is the Editor-in-Chief of *Function*. J.-M. Ramírez is the Editor-in-Chief of the *Journal of Neurophysiology*. L. Schaefer is the Editor-in-Chief of the *American Journal of Physiology-Cell Physiology*. M. B. Thomsen is the Deputy Editor-in-Chief of *Physiological Reports*. G. L. C. Yosten is the Editor-in-Chief of the *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*. All Editors are compensated by the American Physiological Society for their services. They were not involved and did not have access to information regarding the peer-review process or final disposition of this article. An alternate editor oversaw the peer-review and decision-making process for this article.

AUTHOR CONTRIBUTIONS

S.C.B., H.L.B., H.A.C., A.I.D., M.R.F., B.E.G., T.R.K., M.L.L., R.E.M., O.H.P., J.-M.R., L.S., M.B.T., and G.L.C.Y. drafted manuscript; edited and revised manuscript; and approved final version of manuscript.

REFERENCES

1. **Cotgrave R.** *A Dictionarie of the French and English Tongues*. London: Adam Islip, 1611, p. 10.
2. **Szombathy J.** Die diluvialen Kulturschichten von Willendorf. Vortrag auf der Monatsversammlung am 12. Januar 1910 vor der Anthropologischen Gesellschaft. *Mitteilungen der Anthropologischen Gesellschaft Wien* 40: 8–9, 1910.
3. **Balke H, Nocito A.** Vom Schönheitsideal zur Krankheit—eine Reise durch die Geschichte der Adipositas. *Praxis (Bern 1994)* 102: 77–83, 2013. doi:10.1024/1661-8157/a001169.
4. **Fogel RW.** *The Escape from Hunger and Premature Death, 1700–2100*. Cambridge, UK: Cambridge University Press, 2004.
5. **Allen RC.** Tracking the agricultural revolution in England. *Econ Hist Rev* 52: 209–235, 1999. doi:10.1111/1468-0289.00123.
6. **Pingali PL.** Green revolution: impacts, limits, and the path ahead. *Proc Natl Acad Sci USA* 109: 12302–12308, 2012. doi:10.1073/pnas.0912953109.
7. **Guerrini A.** *Obesity and Depression in the Enlightenment: The Life and Times of George Cheyne*. Norman, Oklahoma: University of Oklahoma Press, 2000.
8. **Craik EM.** *The 'Hippocratic' Corpus: Content and Context*. Abingdon, Oxon: Milton Park, 2015.
9. **Eknoyan G.** A history of obesity, or how what was good became ugly and then bad. *Adv Chronic Kidney Dis* 13: 421–427, 2006. doi:10.1053/j.ackd.2006.07.002.
10. **Ludwig DS, Sørensen TIA.** An integrated model of obesity pathogenesis that revisits causal direction. *Nat Rev Endocrinol* 18: 261–262, 2022. doi:10.1038/s41574-022-00635-0.
11. **Quetelet A.** Recherches sur le poids de l'homme aux different âges. *Nouveaux Memoire de l'Academie Royale des Sciences et Belles-Lettres de Bruxelles* 7: 1–57, 1832.
12. **Eknoyan G.** Adolphe Quetelet (1796–874)—the average man and indices of obesity. *Nephrol Dial Transplant* 23: 47–51, 2008. doi:10.1093/ndt/gfm517.
13. **Keys A, Fidanza F, Karvonen MJ, Kimura N, Taylor HL.** Indices of relative weight and obesity. *J Chronic Dis* 25: 329–343, 1972. doi:10.1016/0021-9681(72)90027-6.
14. **Henschel A, Keys A.** The influence of test meal composition on gastric emptying in man. *Am J Physiol* 149: 107–111, 1947. doi:10.1152/ajplegacy.1947.149.1.107.
15. **Keys A, Henschel A, Taylor HL.** The size and function of the human heart at rest in semi-starvation and in subsequent rehabilitation. *Am J Physiol* 150: 153–169, 1947. doi:10.1152/ajplegacy.1947.150.1.153.
16. **Keys A, Brozek J.** Body fat in adult man. *Physiol Rev* 33: 245–325, 1953. doi:10.1152/physrev.1953.33.3.245.
17. **World Health Organization.** WHO Fact-sheet: Obesity and Overweight World Health Organization. <https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight> [2022 July 26].
18. **World Health Organization.** *Obesity: Preventing and Managing the Global Epidemic* Geneva, Switzerland: World Health Organization, 2000.
19. **Di Angelantonio E, Bhupathiraju Sh N, Wormser D, Gao P, Kaptoge S, Berrington de Gonzalez A, et al.** Body-mass index and all-cause mortality: individual-participant-data meta-analysis of 239 prospective studies in four continents. *Lancet* 388: 776–786, 2016. doi:10.1016/S0140-6736(16)30175-1.
20. **Haslam DW, James WP.** Obesity. *Lancet* 366: 1197–1209, 2005. doi:10.1016/S0140-6736(05)67483-1.
21. **Buchwald H.** A Brief History of Obesity: Truths and Illusions. <https://www.clinicaloncology.com/Current-Practice/Article/07-18/A-Brief-History-of-Obesity-Truths-and-Illusions/51221> [2022 July 26].
22. **Münzberg H, Floyd E, Chang JS.** Sympathetic innervation of white adipose tissue: to beige or not to beige? *Physiology (Bethesda)* 36: 246–255, 2021. doi:10.1152/physiol.00038.2020.
23. **Singh P, Covassin N, Marlatt K, Gadde KM, Heymsfield SB.** Obesity, body composition, and sex hormones: implications for cardiovascular risk. *Compr Physiol* 12: 2949–2993, 2021. doi:10.1002/cphy.c210014.
24. **Ren J, Wu NN, Wang S, Sowers JR, Zhang Y.** Obesity cardiomyopathy: evidence, mechanisms, and therapeutic implications. *Physiol Rev* 101: 1745–1807, 2021. doi:10.1152/physrev.00030.2020.

25. **Samson WK, Yosten GLC, Remme CA.** A primer on obesity-related cardiomyopathy. *Physiol Rev* 102: 1–6, 2022. doi:10.1152/physrev.00023.2021.
26. **Merritt EK.** Why is it so hard to lose fat? Because it has to get out through your nose! An exercise physiology laboratory on oxygen consumption, metabolism, and weight loss. *Adv Physiol Educ* 45: 599–606, 2021. doi:10.1152/advan.00006.2021.
27. **Pérez-Cornejo P, Corral-Fernandez NE, Guzman-Hernandez ML, Gopalan C.** Nutrition education on obesity and diabetes to medical students. *Adv Physiol Educ* 45: 217–223, 2021. doi:10.1152/advan.00193.2020.
28. **Beier UH, Baker DJ, Baur JA.** Thermogenic T cells: a cell therapy for obesity? *Am J Physiol Cell Physiol* 322: C1085–C1094, 2022. doi:10.1152/ajpcell.00034.2022.
29. **Dufau J, Shen JX, Couchet M, De Castro Barbosa T, Mejhert N, Massier L, Griseti E, Mouisel E, Amri EZ, Lauschke VM, Rydén M, Langin D.** In vitro and ex vivo models of adipocytes. *Am J Physiol Cell Physiol* 320: C822–C841, 2021. doi:10.1152/ajpcell.00519.2020.
30. **Kawai T, Autieri MV, Scalia R.** Adipose tissue inflammation and metabolic dysfunction in obesity. *Am J Physiol Cell Physiol* 320: C375–C391, 2021. doi:10.1152/ajpcell.00379.2020.
31. **Kelly AC, Rosario F, Chan J, Cox LA, Powell TL, Jansson T.** Transcriptomic responses are sex-dependent in the skeletal muscle and liver in offspring of obese mice. *Am J Physiol Endocrinol Physiol* 323: E336–E353, 2022. doi:10.1152/ajpendo.00263.2021.
32. **Aydogan Mathyk B, Piccolo BD, Alvarado F, Shankar K, O'Tierney-Ginn P.** Metabolomic signatures of low- and high-adiposity neonates differ based on maternal BMI. *Am J Physiol Endocrinol Physiol* 322: E540–E550, 2022. doi:10.1152/ajpendo.00356.2021.
33. **Hashem AM, Cifuentes L, Calderon G, Ricardo-Silgado ML, Gonzalez-Izundegui D, Campos A, McRae A, Franks S, Hurtado MD, Burton D, Petterson XM, Lanza IR, Camilleri M, Acosta A.** Effect of caloric intake and macronutrient composition on intestinal cholesterol absorption and bile acids in patients with obesity. *Am J Physiol Gastrointest Liver Physiol* 323: G157–G164, 2022. doi:10.1152/ajpgi.00108.2022.
34. **Paris S, Ekeanyanwu R, Jiang Y, Davis D, Spechler SJ, Souza RF.** Obesity and its effects on the esophageal mucosal barrier. *Am J Physiol Gastrointest Liver Physiol* 321: G335–G343, 2021. doi:10.1152/ajpgi.00199.2021.
35. **Onufer EJ, Han YH, Courtney C, Steinberger A, Tecos M, Sutton S, Sescleifer A, Ou J, Sanguinetti Czepielewski R, Randolph GJ, Warner BW.** Liver injury after small bowel resection is prevented in obesity-resistant 129S1/SvMj mice. *Am J Physiol Gastrointest Liver Physiol* 320: G907–G918, 2021. doi:10.1152/ajpgi.00284.2020.
36. **Thompson MD, Kang J, Faerber A, Hinrichs H, Özler O, Cowen J, Xie Y, Tarr PI, Davidson NO.** Maternal obesogenic diet regulates offspring bile acid homeostasis and hepatic lipid metabolism via the gut microbiome in mice. *Am J Physiol Gastrointest Liver Physiol* 322: G295–G309, 2022. doi:10.1152/ajpgi.00247.2021.
37. **Hernandez-Hernandez ME, Zee RYL, Pulido-Perez P, Torres-Rasgado E, Romero JR.** The effects of biological sex and cardiovascular disease on COVID-19 mortality. *Am J Physiol Heart Circ Physiol* 323: H397–H402, 2022. doi:10.1152/ajpheart.00295.2022.
38. **Kiernan R, Persand D, Maddie N, Cai W, Carrillo-Sepulveda MA.** Obesity-related vascular dysfunction persists after weight loss and is associated with decreased vascular glucagon-like peptide receptor in female rats. *Am J Physiol Heart Circ Physiol* 323: H301–H311, 2022. doi:10.1152/ajpheart.00031.2022.
39. **Heather LC, Hafstad AD, Halade GV, Harmancey R, Mellor KM, Mishra PK, Mulvihill EE, Nabben M, Nakamura M, Rider OJ, Ruiz M, Wende AR, Ussher JR.** Guidelines on models of diabetic heart disease. *Am J Physiol Heart Circ Physiol* 323: H176–H200, 2022. doi:10.1152/ajpheart.00058.2022.
40. **Aluganti Narasimulu C, Singla DK.** Mechanisms of COVID-19 pathogenesis in diabetes. *Am J Physiol Heart Circ Physiol* 323: H403–H420, 2022. doi:10.1152/ajpheart.00204.2022.
41. **Fulghum KL, Smith JB, Chariker J, Garrett LF, Brittan KR, Lorkiewicz PK, McNally LA, Uchida S, Jones SP, Hill BG, Collins HE.** Metabolic signatures of pregnancy-induced cardiac growth. *Am J Physiol Heart Circ Physiol* 323: H146–H164, 2022. doi:10.1152/ajpheart.00105.2022.
42. **Caton R.** Case of narcolepsy. *Clin Soc Trans* 22: 133–137, 1889.
43. **Lavie P.** Who was the first to use the term Pickwickian in connection with sleepy patients? History of sleep apnoea syndrome. *Sleep Med Rev* 12: 5–17, 2008. doi:10.1016/j.smrv.2007.07.008.
44. **Burwell CS, Robin ED, Whaley RD, Bickelmann AG.** Extreme obesity associated with alveolar hypoventilation—a Pickwickian Syndrome. 1956. *Obes Res* 2: 390–397, 1994. doi:10.1002/j.1550-8528.1994.tb00084.x.
45. **Comroe JH Jr.** Frankenstein, Pickwick, and Ondine. *Am Rev Respir Dis* 111: 689–692, 1975. doi:10.1164/arrd.1975.111.5.689.
46. **Brock JM, Billeter A, Müller-Stich BP, Herth F.** Obesity and the lung: what we know today. *Respiration* 99: 856–866, 2020. doi:10.1159/000509735.
47. **Peters U, Suratt BT, Bates JHT, Dixon AE.** Beyond BMI: obesity and lung disease. *Chest* 153: 702–709, 2018. doi:10.1016/j.chest.2017.07.010.
48. **Südy R, Peták F, Kiss L, Balogh AL, Fodor GH, Korsos A, Schranc A, Babik B.** Obesity and diabetes: similar respiratory mechanical but different gas exchange defects. *Am J Physiol Lung Cell Mol Physiol* 320: L368–L376, 2021. doi:10.1152/ajplung.00439.2020.
49. **Proskocil BJ, Calco GN, Nie Z.** Insulin acutely increases agonist-induced airway smooth muscle contraction in humans and rats. *Am J Physiol Lung Cell Mol Physiol* 320: L545–L556, 2021. doi:10.1152/ajplung.00232.2020.
50. **Proskocil BJ, Fryer AD, Jacoby DB, Nie Z.** Pioglitazone prevents obesity-related airway hyperreactivity and neuronal M2 receptor dysfunction. *Am J Physiol Lung Cell Mol Physiol* 321: L236–L247, 2021. doi:10.1152/ajplung.00567.2020.
51. **Calco GN, Proskocil BJ, Jacoby DB, Fryer AD, Nie Z.** Metformin prevents airway hyperreactivity in rats with dietary obesity. *Am J Physiol Lung Cell Mol Physiol* 321: L1105–L1118, 2021. doi:10.1152/ajplung.00202.2021.
52. **Iyer AS, Dransfield MT.** The “Obesity Paradox” in chronic obstructive pulmonary disease: can it be resolved? *Ann Am Thorac Soc* 15: 158–159, 2018. doi:10.1513/AnnalsATS.201711-901ED.
53. **Smulders L, van der Aalst A, Neuhaus E, Polman S, Franssen FME, van Vliet M, de Kruijff MD.** Decreased risk of COPD exacerbations in obese patients. *COPD* 17: 485–491, 2020. doi:10.1080/15412555.2020.1799963.
54. **DeLapp DA, Glick C, Furmanek S, Ramirez JA, Cavallazzi R.** Patients with obesity have better long-term outcomes after hospitalization for COPD exacerbation. *COPD* 17: 373–377, 2020. doi:10.1080/15412555.2020.1781805.
55. **Gong MN, Bajwa EK, Thompson BT, Christiani DC.** Body mass index is associated with the development of acute respiratory distress syndrome. *Thorax* 65: 44–50, 2010. doi:10.1136/thx.2009.117572.
56. **Zhi G, Xin W, Ying W, Guohong X, Shuying L.** Obesity “Paradox” in acute respiratory distress syndrome: a systematic review and meta-analysis. *PLoS One* 11: e0163677, 2016. doi:10.1371/journal.pone.0163677.
57. **Dana R, Bannay A, Bourst P, Ziegler C, Lossner MR, Gibot S, Levy B, Audibert G, Ziegler O.** Obesity and mortality in critically ill COVID-19 patients with respiratory failure. *Int J Obes (Lond)* 45: 2028–2037, 2021. doi:10.1038/s41366-021-00872-9.
58. **Ball L, Serpa Neto A, Pelosi P.** Obesity and survival in critically ill patients with acute respiratory distress syndrome: a paradox within the paradox. *Crit Care* 21: 114, 2017. doi:10.1186/s13054-017-1682-5.
59. **Yu Q, Wang D, Wen X, Tang X, Qi D, He J, Zhao Y, Deng W, Zhu T.** Adipose-derived exosomes protect the pulmonary endothelial barrier in ventilator-induced lung injury by inhibiting the TRPV4/Ca²⁺ signaling pathway. *Am J Physiol Lung Cell Mol Physiol* 318: L723–L741, 2020. doi:10.1152/ajplung.00255.2019.
60. **Stefan N, Birkenfeld AL, Schulze MB, Ludwig DS.** Obesity and impaired metabolic health in patients with COVID-19. *Nat Rev Endocrinol* 16: 341–342, 2020. doi:10.1038/s41574-020-0364-6.
61. **Pathangey G, Fadadu PP, Hospodar AR, Abbas AE.** Angiotensin-converting enzyme 2 and COVID-19: patients, comorbidities, and therapies. *Am J Physiol Lung Cell Mol Physiol* 320: L301–L330, 2021. doi:10.1152/ajplung.00259.2020.
62. **Morty RE, Ziebuhr J.** Call for Papers: the pathophysiology of COVID-19 and SARS-CoV-2 Infection. *Am J Physiol Lung Cell Mol Physiol* 318: L1016–L1019, 2020. doi:10.1152/ajplung.00136.2020.
63. **Kovesdy CP, Furth SL, Zoccali C; World Kidney Day Steering Committee.** Obesity and kidney disease: hidden consequences

- of the epidemic. *Kidney Int* 91: 260–262, 2017. doi:10.1016/j.kint.2016.10.019.
64. **Monu SR, Wang H, Potter DL, Liao TD, Ortiz PA.** Decreased tubuloglomerular feedback response in high-fat diet-induced obesity. *Am J Physiol Renal Physiol* 322: F429–F436, 2022. doi:10.1152/ajprenal.00307.2021.
 65. **McPherson KC, Shields CA, Poudel B, Johnson AC, Taylor L, Stubbs C, Nichols A, Cornelius DC, Garrett MR, Williams JM.** Altered renal hemodynamics is associated with glomerular lipid accumulation in obese Dahl salt-sensitive leptin receptor mutant rats. *Am J Physiol Renal Physiol* 318: F911–F921, 2020. doi:10.1152/ajprenal.00438.2019.
 66. **Poudel B, Shields CA, Brown AK, Ekperikpe U, Johnson T, Cornelius DC, Williams JM.** Depletion of macrophages slows the early progression of renal injury in obese Dahl salt-sensitive leptin receptor mutant rats. *Am J Physiol Renal Physiol* 318: F1489–F1499, 2020. doi:10.1152/ajprenal.00100.2020.
 67. **Shi Y, Wang C, Zhou X, Li Y, Ma Y, Zhang R, Li R.** Downregulation of PTEN promotes podocyte endocytosis of lipids aggravating obesity-related glomerulopathy. *Am J Physiol Renal Physiol* 318: F589–F599, 2020. doi:10.1152/ajprenal.00392.2019.
 68. **Lee HS, Suh JY, Kang BC, Lee E.** Lipotoxicity dysregulates the immunoproteasome in podocytes and kidneys in type 2 diabetes. *Am J Physiol Renal Physiol* 320: F548–F558, 2021. doi:10.1152/ajprenal.00509.2020.
 69. **Abler LL, O'Driscoll CA, Colopy SA, Stietz KPK, Wang P, Wang Z, Hartmann F, Crader-Smith SM, Oellete JN, Mehta V, Oakes SR, Grimes MD, Mitchell GS, Baan M, Gallagher SJ, Davis DB, Kimple ME, Bjorling DE, Watters JJ, Vezina CM.** The influence of intermittent hypoxia, obesity, and diabetes on male genitourinary anatomy and voiding physiology. *Am J Physiol Renal Physiol* 321: F82–F92, 2021. doi:10.1152/ajprenal.00112.2021.
 70. **Kim AK, Hamadani C, Zeidel ML, Hill WG.** Urological complications of obesity and diabetes in males and females of three mouse models: temporal manifestations. *Am J Physiol Renal Physiol* 318: F160–F174, 2020. doi:10.1152/ajprenal.00207.2019.
 71. **Chapman CL, Grigoryan T, Vargas NT, Reed EL, Kueck PJ, Pietrafesa LD, Bloomfield AC, Johnson BD, Schlader ZJ.** High-fructose corn syrup-sweetened soft drink consumption increases vascular resistance in the kidneys at rest and during sympathetic activation. *Am J Physiol Renal Physiol* 318: F1053–F1065, 2020. doi:10.1152/ajprenal.00374.2019.
 72. **Everaert I, He J, Hanssens M, Stautemas J, Bakker K, Albrecht T, Zhang S, Van der Stede T, Vanhove K, Hoetker D, Howsam M, Tessier FJ, Yard B, Baba SP, Baelde HJ, Derave W.** Carnosinase-1 overexpression, but not aerobic exercise training, affects the development of diabetic nephropathy in BTBR ob/ob mice. *Am J Physiol Renal Physiol* 318: F1030–F1040, 2020. doi:10.1152/ajprenal.00329.2019.
 73. **Hu G, Xu L, Ma Y, Kohzuki M, Ito O.** Chronic exercise provides renal-protective effects with upregulation of fatty acid oxidation in the kidney of high fructose-fed rats. *Am J Physiol Renal Physiol* 318: F826–F834, 2020. doi:10.1152/ajprenal.00444.2019.
 74. **van Velzen DM, Smits MM, van Bommel EJM, Muskiet MHA, Tonneijck L, Kramer MHH, Joles JA, den Heijer M, Nokoff N, Bjornstad P, van Raalte DH.** Kidney hemodynamic function in men and postmenopausal women with type 2 diabetes and preserved kidney function. *Am J Physiol Renal Physiol* 320: F1152–F1158, 2021. doi:10.1152/ajprenal.00660.2020.
 75. **Hua Y, Herder C, Kalhoff H, Buyken AE, Esche J, Krupp D, Wudy SA, Remer T.** Inflammatory mediators in the adipo-renal axis: leptin, adiponectin, and soluble ICAM-1. *Am J Physiol Renal Physiol* 319: F469–F475, 2020. doi:10.1152/ajprenal.00257.2020.
 76. **Mannon EC, Sartain CL, Wilkes TC, Sun J, Polichnowski AJ, O'Connor PM.** Renal mass reduction increases the response to exogenous insulin independent of acid-base status or plasma insulin levels in rats. *Am J Physiol Renal Physiol* 321: F494–F504, 2021. doi:10.1152/ajprenal.00679.2020.
 77. **Petrick HL, Foley KP, Zlitzni S, Brunetta HS, Paglialunga S, Miotto PM, Politis-Barber V, O'Dwyer C, Philbrick DJ, Fullerton MD, Schertzer JD, Holloway GP.** Adipose tissue inflammation is directly linked to obesity-induced insulin resistance, while gut dysbiosis and mitochondrial dysfunction are not required. *Function (Oxf)* 1: zqaa013, 2020. doi:10.1093/function/zqaa013.
 78. **Goossens GH, Blaak EE.** Unraveling the pathophysiology of obesity-related insulin resistance—a perspective on “Adipose Tissue Inflammation Is Directly Linked to Obesity-Induced Insulin Resistance, while Gut Dysbiosis and Mitochondrial Dysfunction Are Not Required”. *Function (Oxf)* 1: zqaa021, 2020. doi:10.1093/function/zqaa021.
 79. **Juhaszova M, Kobrinsky E, Zorov DB, Nuss HB, Yaniv Y, Fishbein KW, de Cabo R, Montoliu L, Gabelli SB, Aon MA, Cortassa S, Sollott SJ.** ATP synthase K(+)- and H(+)-fluxes Drive ATP synthesis and enable mitochondrial K(+)-“uniporter” function: I. Characterization of ion fluxes. *Function (Oxf)* 3: zqab065, 2022. doi:10.1093/function/zqab065.
 80. **Stancill JS, Hansen PA, Mathison AJ, Schmidt EE, Corbett JA.** Deletion of thioredoxin reductase disrupts redox homeostasis and impairs beta-cell function. *Function (Oxf)* 3: zqac034, 2022. doi:10.1093/function/zqac034.
 81. **Ivanova YM, Blondin DP.** Examining the benefits of cold exposure as a therapeutic strategy for obesity and type 2 diabetes. *J Appl Physiol (1985)* 130: 1448–1459, 2021. doi:10.1152/jappphysiol.00934.2020.
 82. **Kullman EL, Kelly KR, Haus JM, Fealy CE, Scelsi AR, Pagadala MR, Flask CA, McCullough AJ, Kirwan JP.** Short-term aerobic exercise training improves gut peptide regulation in nonalcoholic fatty liver disease. *J Appl Physiol (1985)* 120: 1159–1164, 2016. doi:10.1152/jappphysiol.00693.2015.
 83. **Ratchford SM, Lee JF, Bunsawat K, Alpenglow JK, Zhao J, Ma CL, Ryan JJ, Khor LL, Wray DW.** The impact of obesity on the regulation of muscle blood flow during exercise in patients with heart failure with a preserved ejection fraction. *J Appl Physiol (1985)* 132: 1240–1249, 2022. doi:10.1152/jappphysiol.00833.2021.
 84. **Bates JHT, Peters U, Daphtary N, MacLean ES, Hodgdon K, Kaminsky DA, Bhatawadekar S, Dixon AE.** Altered airway mechanics in the context of obesity and asthma. *J Appl Physiol (1985)* 130: 36–47, 2021. doi:10.1152/jappphysiol.00666.2020.
 85. **Dickson SL, Chown JA.** Neuroscience of obesity. *Neuroscience* 447: 1–2, 2020. doi:10.1016/j.neuroscience.2020.09.047.
 86. **Crouse WL, Das SK, Le T, Keele G, Holl K, Seshie O, Craddock AL, Sharma NK, Comeau ME, Langefeld CD, Hawkins GA, Mott R, Valdar W, Solberg Woods LC.** Transcriptome-wide analyses of adipose tissue in outbred rats reveal genetic regulatory mechanisms relevant for human obesity. *Physiol Genomics* 54: 206–219, 2022. doi:10.1152/physiolgenomics.00172.2021.
 87. **Rowlands DS, Page RA, Sukala WR, Giri M, Ghimbovski SD, Hayat I, Cheema BS, Lys I, Leikis M, Sheard PW, Wakefield SJ, Breier B, Hathout Y, Brown K, Marathi R, Orkunoglu-Suer FE, Devaney JM, Leiken B, Many G, Krebs J, Hopkins WG, Hoffman EP.** Multi-omic integrated networks connect DNA methylation and miRNA with skeletal muscle plasticity to chronic exercise in Type 2 diabetic obesity. *Physiol Genomics* 46: 747–765, 2014. doi:10.1152/physiolgenomics.00024.2014.
 88. **Liu S, Lorenzen ED, Fumagalli M, Li B, Harris K, Xiong Z, Zhou L, Korneliusen TS, Somel M, Babbitt C, Wray G, Li J, He W, Wang Z, Fu W, Xiang X, Morgan CC, Doherty A, O'Connell MJ, McInerney JO, Born EW, Dalén L, Dietz R, Orlando L, Sonne C, Zhang G, Nielsen R, Willerslev E, Wang J.** Population genomics reveal recent speciation and rapid evolutionary adaptation in polar bears. *Cell* 157: 785–794, 2014. doi:10.1016/j.cell.2014.03.054.
 89. **Kazankov K, Bojsen-Møller KN, Møller HJ, Madsbad S, Grønbaek H.** Macrophage activation marker sCD163 is associated with liver injury and hepatic insulin resistance in obese patients before and after Roux-en-Y gastric bypass. *Physiol Rep* 10: e15157, 2022. doi:10.14814/phy2.15157.
 90. **Singh P, Covassin N, Sert-Kuniyoshi FH, Marlatt KL, Romero-Corral A, Davison DE, Singh RJ, Jensen MD, Somers VK.** Overfeeding-induced weight gain elicits decreases in sex hormone-binding globulin in healthy males—implications for body fat distribution. *Physiol Rep* 9: e15127, 2021. doi:10.14814/phy2.15127.
 91. **Kurgan N, Islam H, Matusiak JBL, Baranowski BJ, Stoikos J, Fajardo VA, MacPherson REK, Gurd BJ, Klentrou P.** Subcutaneous adipose tissue sclerostin is reduced and Wnt signaling is enhanced following 4-weeks of sprint interval training in young men with obesity. *Physiol Rep* 10: e15232, 2022. doi:10.14814/phy2.15232.
 92. **Maharjan BR, Martinez-Huenschullan SF, McLennan SV, Twigg SM, Williams PF.** Exercise induces favorable metabolic changes in white

- adipose tissue preventing high-fat diet obesity. *Physiol Rep* 9: e14929, 2021. doi:10.14814/phy2.14929.
93. **Sissala N, Myllymäki E, Mohr F, Halmetoja R, Kuvaja P, Dimova EY, Koivunen P.** Hypoxia ameliorates maternal diet-induced insulin resistance during pregnancy while having a detrimental effect on the placenta. *Physiol Rep* 10: e15302, 2022. doi:10.14814/phy2.15302.
94. **Bel JS, Tai TC, Khaper N, Lees SJ.** Chronic glucocorticoid exposure causes brown adipose tissue whitening, alters whole-body glucose metabolism and increases tissue uncoupling protein-1. *Physiol Rep* 10: e15292, 2022. doi:10.14814/phy2.15292.
95. **Tanimura R, Kobayashi L, Shirai T, Takemasa T.** Effects of exercise intensity on white adipose tissue browning and its regulatory signals in mice. *Physiol Rep* 10: e15205, 2022. doi:10.14814/phy2.15205.