National Institutes of Health
National Institute of Diabetes and Digestive and Kidney Diseases

Gut Feelings: Interoceptive Contributions to Obesity and Disorders of Gut–Brain Interaction

Hybrid Workshop
Neuroscience Center Building
6001 Executive Boulevard
North Bethesda, MD 20852
April 30–May 1, 2024

EXECUTIVE SUMMARY

Background and Overview

Interoception is the process through which the nervous system senses, interprets, and integrates signals from within the body regarding internal physiological states that are used to motivate behavior to meet physical needs. Interoceptive processes are mechanistically integral to the pathogenesis of both obesity and disorders of gut–brain interaction (DGBI), which are heterogenous disorders associated with changes in gastrointestinal (GI) physiology. Both obesity and DGBI are associated with numerous symptoms, underlying factors, and serious health effects. While exciting progress for treating obesity has emerged in the form of nutrient-stimulated hormone-based pharmacotherapies that work by altering interoception for hunger and satiety, treatments for DGBI have shown limited and variable success. A workshop entitled "Gut Feelings: Interoceptive Contributions to Obesity and Disorders of Gut–Brain Interaction" was sponsored by the National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) to outline the state of the science of research on interoception that is relevant to obesity and DGBI, and to identify important gaps and opportunities for advancing research and fostering new collaborations.

The keynote presentation emphasized that GI interoception concerns the bidirectional interplay between the gut and brain. GI interoception can potentially be defined as the process by which the nervous system senses, interprets, and integrates signals originating from the GI system, including the gut microbiome, to provide moment-by-moment mapping of the gut across conscious and unconscious levels. Interoception involves a body-brain feedback loop, and many brain areas and circuits subserve interoceptive processing, e.g., the brain's gastric network, including the somatosensory cortices, insular cortex, and the midline parietal and occipital regions. Research on interoception requires interdisciplinary familiarity and transdisciplinary collaboration, and researchers must develop better means of identifying whether an individual patient's symptoms are related to sensory transduction problems or perceptual influences that dominate the sensory experiences of their body. Perturbing GI signals is an optimal way to understand how sensory perception occurs in a disorder-relevant fashion. Research in this area requires advancing minimally invasive methods for measuring interoception and translation of these approaches to the clinic.

Scientific Sessions

Meeting speakers presented on focus topics and responded to questions from in-person and virtual attendees. A summary of emerging studies and roadblocks, gaps, and opportunities for each theme is outlined below.

Neural Sensing, Pathways, and Circuits for Food Intake

Emerging Studies

- The GI tract uses two key interoceptive signals to control the process of satiation: (1) the sense of internal stretch, primarily delivered via the vagal and spinal mechanoreceptors that innervate the

stomach and intestine, and (2) detection of nutrients in the small intestine by entero-endocrine cells (EECs), which transform nutrient-related signals into hormones, including cholecystokinin (CCK), glucagon-like peptide-1 (GLP-1), and glucose-dependent insulinotropic peptide (GIP). Vagal and spinal afferent and efferent nerves form the two major pathways that mediate signals between the gut and brain. In addition to these direct neural pathways, circulating gut-related hormones and nutrients are detected in two key brain areas: (1) the nucleus of the solitary tract (NTS) and area postrema (AP) in the dorsal vagal complex (DVC) of the hindbrain; and (2) the arcuate nucleus (ARC) of the hypothalamus. Cells in the hindbrain inhibit food intake and drive satiation, while a key population of neurons in the ARC—agouti-related protein (AgRP) neurons—drive hunger. Key questions related to this theme include how food ingestion is sensed by the brain, how sensory signals are represented in the dynamics of specific neural circuits, how these neural circuits control food intake, what mechanism is used by which individual cells to control each aspect of a meal, and how sensing and behavioral control are altered by weight gain, GLP-1 receptor agonist drugs, and bariatric surgery.

- Signals from the gut to the brain via the caudal NTS control appetite. Using *in vivo* two-photon calcium imaging, NTS activities can be monitored in real time, showing that visceral responses are kinetically diverse. Each organ system detects and interprets various forms of stimuli (e.g., mechanical forces, temperature changes, etc.). However, a particular stimulus can act on different organs (e.g., mechanical stretch in the stomach vs. the bladder), which results in different functional interpretations for the organism. Mechanical and chemical stimuli are detected by distinct peripheral neurons, and representations of different modality signals converge in the NTS, which maps the body via organ identity rather than exact location or stimulus type.
- Glucagon (GCG) and prolactin-releasing hormone (PRLH) neurons are important for different aspects of meal regulation. Neurons expressing pre-proglucagon (GCG neurons) are the primary source of GLP-1 in the brain, receive direct input from the vagal system, and promote non-aversive satiety. Research on PRLH neurons in anesthetized animals suggests that these neurons can be activated by gut stretch; however, studies of neuronal activity during eating in awake, behaving animal models demonstrate that these neurons can also respond to the taste or palatability of food to help modulate meal size and the pace of ingestion. GCG neurons, in contrast, do not respond to sensory signals from the mouth, but respond to GI signals of volume to induce satiety, usually by reducing meal number. These studies illustrate the importance of taste and the ability of neurons to respond to or ignore gut-derived signals depending on behavior and suggest that a hierarchical and contingent relationship exists between meal-related exterosensory and interoceptive cues.
- Although drugs based on GLP-1 are a significant advance in the treatment of obesity, side effects are common, especially nausea and vomiting. Important sites of action for GLP1-based drugs in the central nervous system (CNS) include the NTS and AP of the hindbrain DVC, areas that contain GLP-1 receptor (GLP-1R) expressing neurons that mediate the therapeutic effects of obesity drugs by suppressing food intake. Both AP and NTS GLP-1R neurons are responsive to feeding, with no differences in latency to activation or percent of neurons activated by semaglutide; however, AP neurons appear more responsive to aversive stimuli, such as lithium chloride, while NTS neurons are more responsive to nutritive stimuli. Stimulation of AP GLP-1R neurons significantly reduces hedonic responses and increases aversive responses to a flavor, while NTS GLP-1R neuron activation does not drive aversion. This research shows that hindbrain circuits for satiety and aversion are anatomically and functionally dissociable, so drugs developed to target the NTS GLP-1R neuron population without targeting the AP GLP-1R neurons may lead to more effective obesity pharmacotherapies with fewer side effects.

Roadblocks, Gaps, and Opportunities

- Studies are needed to advance understanding of where different visceral inputs (e.g., vagal, DRG-spinal, chemical, gut-peptides, etc.) converge and are represented in the brain, the regulation of descending control, and how the NTS sorts ascending circuits, including across different states.

- Current models of food intake control do not account for dynamic interaction between exteroceptive signals from the outside world and interoceptive signals from inside the body.
- Understanding how GLP-1R drugs engage various neural circuits to suppress food intake, as well as the mechanisms of their side effects, remains incomplete.
- Further studies are needed on how brain circuitry mediates the effects of GLP-1–based drugs—including differences between mono-, dual-, and tri-agonist strategies, as is information on the conservation of gut–brain interoceptive mechanisms and pathways from animals to humans.

Gut–Brain Signaling and Obesity

Emerging Studies

- Modern basic neuroscience tools, such as *in vivo* calcium imaging, offer powerful ways to interrogate gut-brain dynamics and hormonal effects that subserve incretin drug functions. AgRP neurons, activated by fasting, are necessary and sufficient to drive feeding, and macronutrients infused into the GI tract inhibit AgRP neurons quickly and at a magnitude aligned with the number of calories consumed. CCK, GIP, and GLP-1 appear to inhibit AgRP rapidly and additively or synergistically, but glucose and fats inhibit AgRP neuronal activity through different mechanisms (e.g., lipids act through CCK signaling, and glucose requires GIP signaling). Obesogenic diets blunt AgRP neural responses to interoceptive signals in a macronutrient-dependent way, and the effects of high-fat and high-sugar diets on the gut-brain axis are likely additive.
- The most effective current treatments for obesity, vertical sleeve gastrectomy (VSG) and GLP-1-based pharmacotherapies, target the gut–brain axis and achieve efficacy by altering interoception for hunger and satiety. While VSG and GLP-1R agonists both cause increased activation of neurons in the hindbrain, these treatments appear to be acting on different pathways and are associated with differences in gastric emptying rate, hypoglycemia, and side effects. Moreover, GLP-1R-expressing neurons are not required for the beneficial effects of VSG.
- Alterations in brain serotonin and dopamine systems occur in people with obesity. Lipid-induced dopamine release in the striatum appears reduced in people with obesity compared with lean people, and this effect is not reversed by 10% weight loss. However, there are variations in dopamine responsivity across individuals that may impact food intake.

Roadblocks, Gaps, and Opportunities

- More information is needed on how ingested nutrients alter the gut–brain dynamics that control appetite and how excessive nutrient intake and obesity dysregulate the system.
- Additional work related to incretin-mimetic therapies is needed in the areas of accessibility, lack of efficacy, or efficacy plateau in some patients. Understanding why weight is regained after the cessation of treatment is also important.
- Questions remain regarding the mechanisms that subserve the weight loss effects of bariatric surgery and GLP1-based drugs and the variability in patient response to these treatments.
- Unanswered questions include what drives eating beyond homeostatic need in humans, whether any specific components of diet or eating patterns can be a risk factor for developing disturbed regulation of food intake, and what role brain nutrient sensing plays in the development or maintenance of obesity in humans. More translational work is needed to address these questions.

Environmental and Microbial Variables for Gut–Brain Interoception

Emerging Studies

- The process of food ingestion includes both exteroception and interoception. Environmental and lifestyle factors regulate much of a person's susceptibility to major human diseases. Exercise stimulates many sensory events in the body and is the single best lifestyle intervention available to protect an individual from a wide variety of diseases. The GI microbiome is predictive of exercise performance in mouse studies—a microbiome-dependent dopamine surge in the striatum encourages the mouse to exercise.
- Spatiotemporal EEC activation can be tracked in zebrafish, allowing *in vivo* imaging of the EEC–vagal network. Gut microbiota regulate nutrient-sensing EEC maturation and mitochondrial remodeling and function. Microbes may be sensed by a subtype of EECs (e.g., transient receptor potential cation channel subfamily A member 1 expressing [Trpa1+] EECs), leading to modulation of activity in brain regions such as the hypothalamus.
- Antibiotic-induced depletion of the gut microbiota in mice promotes increased hedonic feeding, but not short-term homeostatic feeding. Palatability, rather than energy acquisition, appears to drive increased feeding behavior. Hedonic feeding following microbiota depletion extends to high-fat diet and other palatable foods—feeding bouts are longer in antibiotic-treated mice and do not appear to be driven by novelty. Microbiota restoration suppresses overconsumption of palatable food. Activity in reward-related brain regions is modulated by select taxa in the gut microbiota, which can alter the incentive salience of a palatable reward. Antibiotic depletion of gut microbiota does not appear to modulate brain responses to homeostatic feeding.

Roadblocks, Gaps, and Opportunities

- Open questions in the environmental control of gut sensory biology include what is being sensed, what are the sensors, how the environment contextualizes the sensing event, and whether this information can be used for interventions.
- More information is needed on the development and plasticity of the EEC–vagal sensory system, environmental regulation of EEC–vagal communication, and how gut microbes modulate brain function and behavior through the EEC–vagal pathway.
- Researchers need to study how the current situation of the organism influences sensory events and affects clinical phenotypes.
- Further research is needed to understand how gut microbes modulate the gut–brain connection in obesity, the effects of stress on gut function, how and what signals from the gut to the brain modulate feeding and whether these cues can be harnessed as therapies, and the effects of diet and gut function on neurodevelopment and neurodegeneration.
- Future directions for research include defining the molecules made by gut bacteria that suppress hedonic feeding, determining whether reward systems in the brain are modulated by specific gut bacteria, identifying circuits accessed by microbes that mediate hedonic feeding, and testing bacterial treatments for obesity-induced desensitization of the reward system associated with hedonic feeding.

Dysfunction of Interoception in DGBI

Emerging Studies

- The most common DGBI, irritable bowel syndrome (IBS), is characterized by interoceptive and exteroceptive sensitivity. Peripheral and central changes are associated with IBS pathophysiology and, once gut–brain interactions are altered, the direction of causality is difficult to determine. Some

IBS cases may be post-infection conditions that often involve genetic predisposition, female sex, and psychological factors at the time of infection, such as anxiety and depression.

- In DGBI, aberrant processing and integration of interoceptive signals with alterations in attentional, perceptual, and affective response to feelings from the GI tract contribute to symptoms, such as abdominal pain. Pain is a modifiable experience resulting from a combination of internal and external input, emotional and cognitive processes, psychosocial context, and biological factors. Patients with DGBI sense not only GI symptoms but also a range of physical and emotional discomfort, and altered homeostatic reflex responses can affect GI motility and secretion.
- DGBI patients exhibit enhanced interoceptive awareness and stress hyper-responsiveness. These disorders are associated with increased ascending input to and activation of brain areas in the interoceptive cortex (e.g., areas of insular and cingulate cortices), which affect how interoceptive input is processed. DGBI patients also show less engagement of descending pain-inhibitory pathways. Intrinsic factors, such as sex and genetics, and external influences, such as stress, increase the risk of impaired homeostasis, resulting in the misinterpretation of internal sensations, which leads to inappropriate autonomic and behavioral responses. DGBI treatments can beneficially modulate interoceptive awareness and accuracy, as well as stress reactivity.
- Normal GI function relies upon this mechanosensation, which is frequently disrupted in DGBI and other disorders (e.g., autism, neurodegenerative diseases). Many first-line DGBI therapies involve manipulating the chemo-physical properties and residual products of diets, but the mechanisms regarding how these diets alleviate DGBI symptoms are largely unknown. Extensive intrinsic and extrinsic vagal, spinal, and pelvic circuits and pathways process and transmit mechanosensory information along the gut–brain axis.
- EECs in the gut epithelia express Piezo2 and transduce “gut touch” information via complex intracellular signaling pathways and in association with release of serotonin. Activation of EECs through Piezo2 and associated intracellular signaling for mechanosensory transduction may be targets for diagnostics and therapeutics for GI-related disorders.
- The sensory vagus provides key interoceptive messages to the central nervous system to keep the body in homeostasis, but vagal dysfunction may underlie some pathologies in mood and other disorders, such as DGBI. High anxiety and depression are associated with many DGBI; in one study, about two-thirds of participants had GI disorders that preceded mood disorders, and one-third had mood disorders that preceded GI disorders. Vagal hyperactivity can explain depression, anxiety, and pain related to functional dyspepsia; the aberrant vagal sensory activity may lead to plasticity in areas of the CNS that modify downstream pathways resulting in concomitant depression, anxiety, pain, and discomfort.

Roadblocks, Gaps, and Opportunities

- More research is required to understand cellular and molecular mechanisms of interoceptive dysfunction in DGBI; the relationship between the degree of interoceptive dysfunction and severity of DGBI symptoms; the relationship between psychological factors and interoceptive accuracy; the interplay between interoceptive dysfunction, exteroceptive dysfunction, and symptom manifestation; the physiological basis for sex differences observed in DGBI prevalence, pathogenesis, and co-morbid conditions, and factors that contribute to individual variability in interoceptive abilities.
- Long-term studies of changes in interoception over time in relation to DGBI symptom progression or remission are needed.
- Research that focuses on altered interoception in DGBI, in consideration of sex differences, and/or discovery of genetic and epigenetic factors that influence interoception could lead to novel therapeutic targets.

- More information is needed on the molecular and cellular details of gut mechanosensing and signal transduction, including targets and wiring diagrams, physiologic and pathophysiologic roles of gut mechanosensation in chemo-physical properties of diets, and the overlap with other diseases.
- Direct evidence is needed of altered interoceptive input to the central nervous system in humans. Novel advances in technologies, signal detection, and machine learning may provide opportunities to monitor sensory activity from both spinal and vagal nerves in clinical disorders of interest. Interventions that specifically modulate interoceptive signaling to the central nervous system in humans also are needed.

Dysregulated Stress: Contributions to Obesity and DGBI

Emerging Studies

- Early-life exposures, such as poverty, adversity, and stress, are linked to nutritionally poor diets for both the mother and developing infant and appear to impact brain circuits and behaviors that control food intake. Adult rodent models that experienced early life stress (e.g., perinatal exposure to a Western diet, maternal separation, or limited bedding and nesting), exhibit higher body weight and suppressed development of GLP1 circuits in the brain. Developmental diet-driven and environmental influences on GLP1 and related eating control circuits may contribute to life-long effects and individual differences in eating and motivation for food.
- Early-life adversity is a significant risk factor for IBS, and chronic stress in adulthood can induce or exacerbate symptoms. In studies with rats, persistent stress for 3 weeks resulted in worse visceral hypersensitivity in females but not males. Behavioral therapies, such as cognitive behavioral therapy (CBT), have been adapted to decrease stress effects and ameliorate IBS symptoms without the side effects that often accompany drug therapies. Environmental enrichment, a CBT analogue for rodents, reverses stress-induced visceral hypersensitivity along the brain–gut axis and associated changes in expression of glucocorticoid receptor in the central nucleus of the amygdala.
- Social discrimination, a type of environmental stress, is associated with a higher risk of obesity. The brain and microbiome communicate cyclically and bidirectionally, and microbes help train the immune system, shape hormone responses, and communicate with the brain and other organs. Patients with food addiction have reduced amounts of gut bacteria that produce the neuroprotective metabolite indole-3 propionate. Discrimination leads to higher cravings for unhealthy foods, increased brain activity in the insular region for sweet foods and in the putamen and orbitofrontal cortex for savory foods, and increased abundance of glutamate metabolites that are linked to inflammation. Resilient individuals may mitigate the development of stress-related health effects through improved emotional regulation, reduced inflammation, and healthy gut barrier integrity. Microbiome modifications may be able to enhance resilience and optimize mental and overall health.

Roadblocks, Gaps, and Opportunities

- More information is needed regarding the development, maturation, and plasticity of neural circuits that control eating as well as how variability in these circuits contributes to individual differences in food intake.
- Sex differences in stress-induced visceral sensitivity—including the specific roles of hormones and the enteric nervous system (ENS)—are not well understood. Research also is needed to investigate physiological mechanisms of behavioral therapies for stress-induced visceral hypersensitivity as well as the utility of these therapies for visceral hypersensitivity that is not induced by stress.
- A holistic understanding of brain–gut–microbiome interactions and related psychosocial and environmental factors—including adverse and protective influences—is needed as interventions may be possible anywhere along the brain-gut-microbiome axis.

Measuring and Modulating Gut–Brain Interactions: Technological Challenges and Opportunities

Emerging Studies

- Studying gut–brain communication is challenging due to the spatial separation, different anatomies, functions, and cell types; thus, developing appropriate tools is difficult. Many translational tools exist to enable studies of the brain using electrophysiology, pharmacology, optogenetics, chemogenetics, and fiber photometry. Emerging wireless, fiber-based neural interfaces created from soft, materials integrated with channels and electronic components like conductors and light-emitting diodes allow for optogenetics, thermometry, electrophysiology, and fluid delivery within miniature and biocompatible architectures that can interface with the gut as well as the brain. These neurotechnologies are suitable for long-term interrogations of the gut and brain simultaneously in awake, behaving animal models.
- Gastric motor functions are coordinated by the ENS, and extrinsic spinal and vagal afferents innervate specific cell types and regions of the stomach. Magnetic resonance imaging (MRI)-based and related tools can be used to noninvasively image and modulate stomach-brain interactions and are easily translatable to humans. Analysis and mapping of GI motor processes from MRI studies have revealed highly coordinated patterns of contractions, peristalsis initiation, and propagation in controls compared to gastroparesis patients but also heterogeneity across individuals.
- DGBI involve altered sensation, stress sensitivity, genetic susceptibility, and multisystem comorbidities, suggesting aberrant autonomic nervous system (ANS) signaling. Percutaneous electrical nerve field stimulation (PENFS) is a non-invasive therapy for neuromodulation of the ANS that has been shown to be effective for DGBI (e.g., IBS and pediatric cyclic vomiting syndrome). Mechanisms of efficacy for PENFS and other neuromodulatory treatments are not well understood, and personalized approaches are needed due to the heterogeneous patient population.

Roadblocks, Gaps, and Opportunities

- Research on the gut is challenged by the lack of transgenic models, difficult surgeries, and gut movement, which impedes electrophysiology and optical imaging. Technological innovation provides opportunities to use approaches such as optogenetics, electrophysiology, thermal sensing, and fluorescent imaging to study GI motility, pressure, nutrient sensing, and the gut microbiome. Wireless microdevices can be leveraged to study the delicate, regionally specific gut tissues and to interrogate the gut and brain simultaneously.
- Translatable tools should be established that are compatible for studying gut-brain interoception in animals and humans, and quantitative tests of stomach-brain interactions are needed that account for individual variation and disease heterogeneity.
- Future opportunities for studying DGBI and ANS dysregulation should include a more holistic view that incorporates investigation of the underlying neural pathways along with classification by GI symptoms and function. Cross-specialty collaborations, emerging non-invasive neuromodulation therapies, reliable biomarkers, and personalized approaches to treatment also would advance the field.

Breakout Discussion Highlights

- Studies of interoceptive sensing and signaling related to feeding and body weight regulation over longer time-scales are lacking, as well as a comparative, cross-species approach in the use of animal models.
- The extent to which successful weight reduction via incretin-mimetic therapies occurs through the impact on GI interoception is not known.

- Current work lacks a comprehensive analysis of how GLP-1 drugs and gut–brain signals act upon multiple circuits and cell types across the brain. Contextualization of these processes with gut signals in specific physiological environments also is required. Cross-laboratory collaboration would facilitate these efforts.
- Research is needed to identify mechanisms and brain regions through which exteroceptive cues and interoceptive signals interact to control feeding, energy homeostasis, and body weight.
- Contributions of higher-order circuits beyond the hypothalamus and brainstem, including emotive and cognitive processes, should be investigated for their critical role in human food intake.
- There is a lack of understanding regarding the heterogeneity of obesity phenotypes or the variation in response to treatments with GLP-1 drugs and bariatric surgery.
- There are many unanswered questions about how the gut microbiome participates in interoceptive processes and the individual's perception of the intestinal milieu.
- Animal models are limited in their translation to humans. Generation of humanized model species through immunological, genetic, or fecal microbiota transplant approaches may lead to more accurately prediction interoceptive responses in humans.
- Translational research on interoception will be accelerated with cutting-edge neurotechnologies that are scalable from animal research to clinical studies, including advanced imaging tools, and wireless, multi-functional, chronically implanted sensors and effectors.
- Multimodal luminal sensors for 3-D intestine measurements across the gut wall, and ways to measure luminal and sensory information at the mucosal level are needed.
- Leveraging the ability to alter or combine approved medical devices, MRI, endoscopy, GI capsules would facilitate research on gut-brain interoception in human subjects and may bring human research capabilities closer to the current state of animal research. Implants and wearables may be future solutions for studying the gut–brain axis.
- Large databases of clinical information and biological data (e.g., brain imaging, heart rate, genetic data) can be leveraged to identify and assess risk factors for DGBI.
- Longitudinal studies with repeated measures are important for assessing changes in symptom perception versus symptom severity in DGBI.
- Peripheral and/or central biomarkers based on GI interoception may help to uncover the evolution of disease, stratify patients, and determine the best treatment options for DGBI.

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