Health and implications of increased gastrointestinal microbial populations, prolonged stress, and its effects on the gut microbiota have become areas of active research. The field of PsychoNeuroImmunology (PNI) has clearly demonstrated that exposure to psychological stressors can disrupt immune functioning. For the most part, studies have focused on innate and adaptive immune responses, with few studies focusing on more basic defenses, such as the commensal microbiota. The healthy human is colonized by a vast array of microbes that exceed cells of the body by a factor of 10^11 (i.e., 10^14 microbes:10^13 human cells). These microbes have many beneficial effects on the host and comprise a strong natural barrier to invading pathogens; disrupting the microbiota is known to enhance susceptibility to enteric diseases. The factors regulating the types of bacteria that comprise the intestinal microbiota, as well as the levels to which they can grow, are not well defined. But, it is known that certain components of gastrointestinal functioning strongly influence microbial populations. Moreover, these functions are altered during stress response. Thus, it was hypothesized that stressor exposure would affect the intestinal microbiota. Initial studies in rhesus monkeys (Macaca mulatta) supported this hypothesis by demonstrating that exposure to stressors early in the lifespan significantly reduced levels of commensal lactobacilli that are thought to protect the intestines from infection. In more recent murine studies, exposure to a prolonged stressor (i.e., repeated restraint stress) led to a significant overgrowth of Gram-negative bacteria. Moreover, DNA sequencing, using pyrosequencing methodology, indicated that microbial diversity in the intestines was reduced from 1200 to 900 operational taxonomic units after stressor exposure. While the physiological importance of these alterations is currently being studied, the data indicate that prolonged stressors significantly alter intestinal microbial populations, and implicate an additional mechanism through which stress can affect health.

Aerodynamics of the northern flying squirrel (Glaucomys sabrinus)

Most studies on mammalian gliders deduce glide aerodynamics from the location of the launch and landing, using an assumption of steady-state aerodynamics. During steady-state gliding, all forces are balanced, velocity is constant, and motion is passive, powered only by gravity. However, considering the dynamic nature of the wings structure (articulated limbs and compliant membranes with muscles imbedded in the skin) and the complex forest environments where the animals glide, the assumption of steady-state aerodynamics may not be warranted. To test the hypothesis that mammalian gliders use steady-state aerodynamics we used high speed video to record the 3-D trajectories of wild flying squirrels over a variety of glide distances in their natural habitat. From these trajectories, we calculated velocities, accelerations, forces, force coefficients, glide ratios, and lift-to-drag ratios. Our results show that flying squirrels do not use steady-state aerodynamics at any point in glides of any of the distances examined. Instead, the squirrels generate more net aerodynamic force than their bodyweight, allowing significant upward and forward accelerations. Additionally, the squirrels show changes in their force coefficients and lift-to-drag ratios during glides, indicating active and coordinated control of non-steady glide paths. This way, squirrels achieve the same glide ratio as they would using steady-state aerodynamics, but with considerably greater total and horizontal velocity. Because the squirrels are able to change force coefficients, and redirect force upward or forward by adjusting the lift-to-drag ratio, they are able to modulate their lift and thrust generation.

Impact of stressor exposure on intestinal microbiota

The field of PsychoNeuroImmunology (PNI) has clearly demonstrated that exposure to psychological stressors can disrupt immune functioning. For the most part, studies have focused on innate and adaptive immune responses, with few studies focusing on more basic defenses, such as the commensal microbiota. The healthy human is colonized by a vast array of microbes that exceed cells of the body by a factor of 10^11 (i.e., 10^14 microbes:10^13 human cells). These microbes have many beneficial effects on the host and comprise a strong natural barrier to invading pathogens; disrupting the microbiota is known to enhance susceptibility to enteric diseases. The factors regulating the types of bacteria that comprise the intestinal microbiota, as well as the levels to which they can grow, are not well defined. But, it is known that certain components of gastrointestinal functioning strongly influence microbial populations. Moreover, these functions are altered during stress response. Thus, it was hypothesized that stressor exposure would affect the intestinal microbiota. Initial studies in rhesus monkeys (Macaca mulatta) supported this hypothesis by demonstrating that exposure to stressors early in the lifespan significantly reduced levels of commensal lactobacilli that are thought to protect the intestines from infection. In more recent murine studies, exposure to a prolonged stressor (i.e., repeated restraint stress) led to a significant overgrowth of Gram-negative bacteria. Moreover, DNA sequencing, using pyrosequencing methodology, indicated that microbial diversity in the intestines was reduced from 1200 to 900 operational taxonomic units after stressor exposure. While the physiological importance of these alterations is currently being studied, the data indicate that prolonged stressors significantly alter intestinal microbial populations, and implicate an additional mechanism through which stress can affect health.

Seasonal and developmental expression of growth hormone regulatory neuropeptides in Atlantic salmon (Salmo salar)

In Atlantic salmon, Salmo salar, circulating levels of growth hormone (GH) are known to vary with developmental stage and season. GH levels increase dramatically in spring in fish transforming from freshwater-adapted parr to seawater-adapted smolts. The vernal increase in GH is entrained by photoperiod: early exposure to a long-day photoperiod advances the increase in GH. We investigated the role of the neurohormones pituitary adenylate cyclase-activating polypeptide (PACAP, a GH secretagogue) and somatostatin I and II (inhibitors of GH secretion) in mediating these changes in GH levels by measuring the respective mRNA transcript levels throughout the spring. In one study, brains were collected from parr and presumptive smolts throughout the spring months. In a second study, brains were collected from fish exposed to an advanced photoperiod in early spring. For both studies, RNA was extracted separately from the hypothalamus and telencephalon, and mRNA transcripts were measured by quantitative PCR. Hypothalamic somatostatin mRNA levels declined from March to May in both parr and smolts. Hypothalamic somatostatin mRNA levels also declined in fish exposed to an advanced long-day photoperiod, compared to natural photoperiod controls. No significant differences in levels of PACAP mRNA were detected in the hypothalamus. Analysis of the mRNA levels of these neuropeptides in the telencephalon is in progress and will also be presented.