

UNIVERSITY OF PITTSBURGH
DEPARTMENT OF PSYCHOLOGY

Stress, Glucocorticoids, and Inflammation:
Resolving the Paradox

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3:00 PM

**Martin Colloquium Center
4127 Sennott Square**

Scientists and physicians have long known that chronic psychological stressors can accelerate the development and progression of various medical illnesses. Historically, much of the blame for stress-related diseases has been assigned to the hypothalamic-pituitary- adrenocortical axis, which releases cortisol into circulation following many psychologically demanding experiences. Cortisol has a wide variety of physiological consequences including mobilization of glucose, regulation of vascular tone and fluid volume, and modulation of immune function. This chain of events seems unlikely to contribute to many stress-related conditions, however, because one of cortisol's major actions is to suppress inflammation, which plays a key role in coronary, autoimmune, infectious, and allergic diseases. To resolve this paradox and identify underlying mechanisms, we have advanced the hypothesis that chronic stress fosters resistance to glucocorticoids.

This view suggests that chronic stress triggers persistent secretion of cortisol, which leads to compensatory downregulation of glucocorticoid receptor expression and functioning. Such dynamics could enable inflammation to flourish, and also diminish the efficacy of therapeutics that work through GR. In this lecture I will discuss a recent series of studies with chronically stressed populations that substantiate this view, and provide insights into some of the psychobiological pathways involved.

Reception to follow in Room 4125 Sennott Square