Individual coping strategies and vulnerability to stress pathology

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7. EMOTION AND MOTIVATION

SY038

Stress modulation of disease. *Overmier, J. B. Center for Research in Learning, Perception, and Cognition, University of Minnesota, Minneapolis, USA.*

Psychology, physiology and immunity, long treated as independent disciplines, are now being discovered to be intimately wed and constantly influencing one another, although the mechanisms of influence are still unclear. This has led to a new field of research and training called psychoneuroimmunology with its own core journal, BRAIN *Brain Behaviour and Immunity*. Much of the work grows out of Selye's early contributions showing that "stress" - whatever that is - modulated vulnerability to a range of challenges that had disease states as a consequence. The present symposium explores from several different perspectives how psychological, physiological and immune systems are interdependently involved in the modulation of disease processes. Emphasis is on sampling diverse contemporary European research thrusts, but eschewing the stress X heart psychosomatic disease interaction that is most familiar.

SY038.1

Stress and disease: A psychobiological perspective. ***Dantzer, R.*** *INSERM, Bordeaux, France.* Understanding of the way different systems are organised and their different substances communicate with each other at different levels of organisation opens new perspectives on pathophysiology. This will be illustrated by examples drawn from the rapidly expanding field of psychoneuroimmunology.

SY038.2

Conditioned increase of natural killer (NK) cell activity in humans. ***Buske-Kirschbaum, A.*** *University of Trier, Clin. & Physio. Psychology, Trier, Germany.* In order to study conditioning of the human immune function healthy volunteers were investigated in a classical conditioning protocol. Conditioned subjects received repeated pairings of a sherbet sweet with an injection of epinephrine (s.c.) on four consecutive days. After 20 mins epinephrine injection resulted in a significant increase of natural killer cell activity (NKCA). After re-exposure of the sherbet sweet conditioned subjects showed significant increase of NKCA. No alteration of NKCA could be observed in three control groups. In a second conditioning study using a differential design, subjects were provided with a taste A (CS+) in combination with epinephrine injection or with a taste B (CS-) which remained unreinforced. After repeated presentation of the stimuli re-exposure of the CS+ and the CS- in the first two test trials resulted in no alteration of NKCA. After the second re-exposure of the CS+ on the test trial 3, however, significant increase of NKCA was observed.

SY038.3

Individual coping strategies and vulnerability to stress pathology. ***Koolhaas, J.M.*** *Department of Animal Physiology, University of Groningen, AA Haren, The Netherlands.* Psychosocial factors are implicated in the individual development, course and recovery from disease. Studies with animal models using social interactions show that the magnitude and direction of changes in the immune, neuroendocrine and cardiovascular system are highly correlated with the social position of the animal. The relationship appears to depend on the animal's coping strategy both in terms of behaviour and neuroendocrinology, and hence in immunology as well. It will be argued that disease processes should be considered as a function of the baseline behavioural, neuroendocrine, and immunological state.

SY038.4

The pathological power of the past. ***Murison, R.*** *Department of Biological and Medical Psychology, University of Bergen, Norway.* Much research on stress-disease interactions employs simple single stressor exposure designs. However, the behavioural and physiological consequences of a stress exposure are modulated by variables other than those associated with the final stressor. These include genetic variation, previous experience(s) of stressful situations, prior coping history, and similarities between prior and final stressor situations. Thus the extent of stress-induced pathology may be proactively modulated by the organism's learning history. The ability of, and the mechanisms by which, prior stressors