CHAPTER 3

Specificity and Stress Research

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Introduction

There are a number of different ways in which the psychological and physiological aspects of behavior have been interrelated. The connection between the two domains is fundamental. It is the distinction between them that is artificial. Yet there is some administrative virtue in partitioning the unitary responses of a human being or an animal into components that follow disciplinary lines. Each discipline—physiology, endocrinology, psychology, anthropology, and so forth—tends to focus on its aspect of the integrated biobehavioral response with its own set of questions and investigative concepts and techniques. This chapter discusses one of the most common and important of the biobehavioral responses, namely, stress. The perspective will be psychological, and the question to be examined will be the source of individual differences in the diverse factors believed to enter into the stress system.

The term stress has evolved, over the past several decades, to encompass a large variety of phenomena and is used in a number of different ways. In general, however, research on stress falls into one of two broad categories. The first of these categories is essentially physiologically defined. It is the original notion of stress formulated by Selye (1936) in his paper in Nature. Stress here is defined as the reaction of the organism to some sort of outside threat. The organism is reactive and little cognition is involved in the model. In later formulations, Selye attempted to build
in a broader concept of stress, making it applicable to a wider range of human situations, but at heart it remained a reactive model. Research done with this conceptualization was of the sort that followed the medical tradition of animal models, using physical or physiological stressors, and measuring physiological and endocrinological changes as indications of stress.

In contradistinction, a second tradition of stress research can be considered to be transactional; that is, stress is defined as the outcome of interactions between the organism and the environment. The general approaches taken by Lazarus (1966) and Leventhal (1970) are examples of this type of work. In the transactional model, an event in the environment is considered to be a stressor only if the organism’s appraisals of it, and of its own resources, suggest that it is threatening or disturbing. Research done in this tradition tends to be human oriented and uses psychological measures, both for how the subject evaluates the stress and in terms of the subject’s reactions to it. Although the model is said explicitly to apply to physiological and physical stimuli as well as to psychological ones, most of the work done within this framework has been on psychological or nonphysical environmental stimuli.

Pathogen Reaction Model

Selye (1956, 1976) outlined the nature of the pathogen reaction model in a number of different publications. His description of the General Adaptation Syndrome, with three stages of alarm, resistance, and exhaustion, is now well known. The emphasis throughout his explication of the system is that stress consists of the nonspecific consequences of any stressor. In this context (although Selye goes to great pains to make clear what he means), the term nonspecific is slightly confusing. Selye means that every stressor produces certain reactions specific to that stressor as well as nonspecific changes that result from all stressors. In the pathogen reaction model, if an organism or an animal were to break a leg, the consequence of the leg breaking would be specific and local—the fracture of the bone, the tissue damage in that area, the disruption of the blood supply, the edema, and all other consequences brought about precisely by the breaking of the leg would be the specific reaction. The nonspecific reaction to the leg breaking would be the increased output of the adrenal cortical steroids and a variety of other endocrine changes that precede and react to the increased adrenal output (Mason, 1968, 1975; Selye, 1956, 1976). Although it is possible to be precise about these nonspecific changes, they are called nonspecific because the same steroid output occurs in response to each stressor or pathogen. If the same