

REVIEW ARTICLE

The Stress Response: A New Perspective

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A theory of the response to chronic intermittent stress, integrating many diverse studies, is presented. Chronic intermittent stress is presumed to be the type of stress most frequently encountered and most likely to cause physiological changes which predispose an organism to tissue damage. The theory states that all organisms are genetically predisposed to adapt to stress and that the physiological pattern of adaptation is similar across species. This pattern consists of a conditioned endocrine response before the stressor is presented accompanied by a decrease in arousal during the stress. These changes occur because the organism is predisposed to learn cues predictive of stress and to assess the threat potential of the stressor. This pattern is adaptive because it conserves resources and promotes homeostasis. Maladaptation is discussed in terms of failure to learn situational expectancies and appropriate responses. Implications of this theoretical perspective are examined.

Stress is a poorly defined phenomenon which has been extensively studied for the past three decades. Definitions of stress have focused upon stimulus (1), response (2), or interactional elements (3) of the process wherein an organism encounters a situation and reacts to it. Many theories (4-6) have been postulated to explain the organism's physiological response to stress, although none has been totally satisfactory. The limitations of these theories and the resultant confusion in the field have been discussed elsewhere (7-9). As one reviews the literature, it becomes apparent that many authors (10-12) have recognized the need for a more precise definition and theory of stress and have occasionally called for a moratorium on stress research until the confusion is resolved (13). Attempts to

clarify the situation have typically resulted in semantic definitional disputes (14) or in argumentation concerning a specific aspect of a vague, complex theory (15). Therefore, despite advances in our understanding of the physiological correlates of the stress response, the field is still lacking an integrative framework which can explain the majority of research results in a logical, theoretical manner.

At this point it would be helpful to define the terms used in this article, since clear terminology is a prerequisite for the communication of ideas. One of the most commonly accepted definitions of stress is that it is anything which causes an alteration of homeostatic processes (4). The primary problem with this definition is that homeostasis, referring to the maintenance of the body's physiological resting state, is constantly changing in minute ways as a result of any behavior, including sitting or yawning, etc. Each movement activates physiological mechanisms which promote a return toward baseline. Under this definition, any type of postural change is considered a stress. To circumvent this difficulty, stress is defined here

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Received for publication June 11, 1979; revision received October 1, 1979.

as anything which causes an alteration of psychological homeostatic processes. A "stressor" is the specific stimulus in the transaction and the "stress response" is the organism's relatively nonspecific physiological response. Psychological homeostasis is a relatively new concept (16) referring to the maintenance of the normal mood state of an individual at rest. All emotions are alterations from this state. Falick and Britton (17) suggested that what is commonly known as the "grief cycle" (18, 19) is really an adaptive mechanism activated whenever an organism is faced with a change. Thus, the phenomena of denial, anger, guilt, depression, and acceptance enable the organism to integrate the event into consciousness and to promote psychological adaptation. Psychological changes are usually accompanied by alterations of physiological homeostatic responses (20). It is not known whether these systems act in parallel (21) or are interactive (22). By defining stress as an alteration of psychological homeostasis, differentiation is possible between those events which cause alterations of physiological responses and are psychologically stressful and those which are not. Implicit in this definition is that the psychological concomitants of an event make it stressful and that psychological homeostatic responses include both conscious and unconscious states. This definitional interpretation means that perception of an event's occurrence is necessary if it is to be classified as a stressor. Once it has been perceived, it may be either integrated or defended against on a conscious or unconscious level. Under this definition, people classed as "repressors" (23) or "deniers" (24) can be distinguished from those classed as "suppressors" (25) or individuals who do not recognize the stimulus as

stressful (26). One problem common to several research studies (27, 28) has been differentiating those individuals who report a stimulus as nonstressful yet are physiologically aroused, from those individuals who do not respond either psychologically or physiologically. According to the above framework, the repressors/deniers have perceived the event and are defending against its conscious admission. Yet, this unconscious mechanism has altered psychological normality, resulting in a physiological stress response dissociated from subjective awareness. The other group has not perceived the event or has consciously decided to minimize its impact. Thus, no alteration of either psychological or physiological homeostatic mechanisms has occurred. Although it is not possible to test directly the concept of unconscious recognition of an event resulting in an altered psychological state, several methods (29-31) exist for indirect validation of this hypothesis. Another point in question for this definition of stress is the case of a pure physiological stressor. The definition assumes that the majority of physiological stressors (e.g., bodily injury, disease, etc.) have psychological components due to the organism's perception of the event. According to the definition, a pure physiological stimulus—one which occurs unbeknownst to the organism—is not a stressor. It does require physiological adaptation but, because it is not perceived, psychological functioning is not altered.

Data to support the position that the psychological concomitants of physiological stimuli are responsible for the stress response come from Mason's work with monkeys (32) and humans (33). Mason has found that commonly accepted physiological stressors (e.g., heat, exer-

cise, hunger) do not elicit a stress response when they are presented in a way that eliminates their psychological sequelae (e.g., uncertainty, fear). When the organism is presented with these stimuli dissociated from their psychological components, the physiological response is specific to each stimulus and can be predicted on the basis of metabolic change induced by the stimulus. Thus, it appears that psychological alterations are a necessary prerequisite for a transaction to be termed "stressful." It is also apparent that both animals (34-36) and humans (37, 38) respond to purely psychological stimuli and, thus, this definition is applicable to all mammals. No other article has made this distinction between physiological and psychological homeostasis in the definition of stress.

Another important definitional issue concerns differences between acute, chronic, and chronic intermittent stress. Acute stress is any event which occurs within a given (usually short) time period and does not reoccur frequently, if at all. Most investigators do not distinguish between chronic and chronic intermittent stress, subsuming them both under the heading of chronic stress. However, since an organism's reaction to these states is typically different, it becomes important to clarify this relationship. Chronic intermittent stress is a discrete stimulus to which the organism is repeatedly exposed, over a given time period, for a specified amount of time (usually less than 1 hr). On the other hand, chronic stress is a stimulus to which the organism is continuously exposed. It is reasonable to assume that because different factors are involved in chronic and chronic intermittent stress, the reactions to these two states would also differ. It is quite possible that much of the confusion in the

stress field is due to equating these two situations and not differentiating explanatory mechanisms. The purpose of this article is to present a simple theory of the physiological response to chronic intermittent stress based on evolutionary (39, 40) and learning (41) principles.

THEORETICAL FRAMEWORK

In reviewing the literature the following characteristics of the stress response have been found in many studies.

1. When an organism initially encounters a stressor, it responds with sympathetic arousal and adrenocortical secretion.
2. After repeated exposures to this same stressor, the arousal decreases.
3. After repeated exposures to this same stressor, an orienting, anticipatory response is present.
4. If the organism, after experiencing a chronic intermittent stress, is exposed to a novel stressor or injected with ACTH, adrenocortical secretion increases to a level comparable to that incurred by initial exposure to the chronic stress.
5. After repeated exposure to a stressor, the time course of the arousal response is altered such that the maximal response occurs prior to stressor exposure instead of during it.

This pattern of results is evident across all species with adrenocortical systems (42). Given the above pattern of results it is possible to postulate a mechanism for achieving adaptation in cases of chronic intermittent stress. Hypothetically, in order to be maximally fit, an organism must achieve three goals:

- 1) maintenance of homeostasis;
- 2) conservation of resources;
- 3) effective defense against stress.

In the case of acute stress it is easy to achieve all three objectives without compromising any of the others. Arousal of the sympathetic nervous system causes physiological changes necessitated to cope with the stressor, but since the stressor is short-lived, homeostasis is quickly regained and resources conserved. However, in the case of chronic intermittent stress, this response would result in chronic depletion of resources and homeostatic alterations. Thus, a more appropriate response to this type of stress would be one which conserves resources (43) while adequately defending the organism against stress. The response pattern which most readily fulfills these criteria is the one reported above: an overall decrease of arousal during stress accompanied by an anticipatory response. By decreasing the maximal level of the response, resources are conserved. Furthermore, by beginning the response prior to stress onset, resources are mobilized before the time of stress exposure, allowing effective defense with minimal energy expenditure. Yet in order to accomplish these changes, the organism must learn what cues predict stress (i.e., when to begin the anticipatory response) and how serious the stress is (i.e., how much resource mobilization is necessary). These types of cues (predictive, consequential) are easily learned by most organisms and are described by Bolles (41) as fundamental learning principles. Thus, it is hypothesized that learning inhibits the stress response and that conditioned anticipatory responses are part of the mechanism facilitating adaptation to chronic intermittent stress. However, it becomes evident when viewing the human stress response that cognitive processes (learning) are not always adaptive and can be responsible for unelicited

chronic arousal. It is presumed that these situations reflect maladaptation and environmental modification of genetic predispositions.

Thus, the theory predicts that adaptation to chronic intermittent stress consists of an anticipatory response accompanied by a decreased overall physiological response and that these responses are accomplished by learning predictive and consequential cues.

In the following paragraphs, papers representative of different areas of stress research will be reviewed and discussed in terms of the theoretical framework.

Many studies have been done utilizing chronic intermittent stress (48-50). All of these experiments reported a decreased glucocorticoid secretion after exposure to a chronic intermittent stressor and an increased secretion after presentation of a novel acute stressor.

ANIMAL AND HUMAN LITERATURE

Sakellaris et al. (44, 45) hypothesized that adaptation involves an increased responsiveness of the entire pituitary-adrenocortical system. In one series of experiments, Sakellaris and Vernikos-Danellis (44) chronically stressed rats with cold exposure, individual housing, or confinement for varying periods of time. An acute stressor (ether, saline injection, or release from confinement) was then presented and blood samples were obtained at intervals from 0-10 min after stress. Results indicated that chronically stressed rats activated the pituitary-adrenocortical system faster than controls even though there was no difference of baseline values between groups. Thus, chronically stressed rats were apparently

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sensitized to stress and responded to a novel stressor more quickly than did control animals. Selye (46, 47) has observed a similar process during what he terms the exhaustion stage of the stress response. Here organisms respond more quickly to a novel stimulus than to the original stressor. Yet Selye felt that this process represented exhaustion instead of adaptation. The apparent discrepancies between Selye's and Sakellaris et al.'s perspectives are probably due to the type of stress, the length of the experiment, and the coping resources available.

Recent studies in our laboratory (51) have found similar results for the pituitary-adrenocortical system in response to chronic intermittent cold stress. Rats were cold stressed for 10 min a day for a period of either 3 weeks or 3 months. Blood was then collected either immediately after cold exposure or instead of it and assayed for plasma corticosterone. Results indicated that, by 3 months, chronic intermittently stressed rats secreted less corticosterone in response to cold than they did at 3 weeks. Furthermore, baseline blood samples, taken at the time the rats were usually stressed, showed a conditioning effect: plasma corticosterone increased over time such that, by 3 months, equal amounts of the hormone were being secreted before as during the stress. A form of conditioning presumably resulted from pairing hormonal release with time and noise cues. In a recent view of endocrinological research (52), it was found that conditioned endocrine responses occur in a major portion of stress research and that these often reflect a conditioned anticipatory response. Organisms begin their response to a chronic intermittent stress before actual exposure to the situation and tend to decrease this response over time.

Fenz (53) reported on a series of experiments done with novice and experienced parachutists. Physiological measures of an "alerting" response (heart rate, respiratory rate, electrodermal response) were taken at varying intervals on the day a parachutist jumped. It appeared that both novice and experienced, and good and poor parachutists could be differentiated by their alerting response pattern. Novice parachutists displayed high arousal on the morning of the jump and continued to increase their arousal up to the time of the jump. Experienced parachutists showed near basal rates the morning of the jump, reaching a maximum 2 hr before the jump, and returning to baseline at jump time. This shift of the time course of autonomic arousal was accompanied by a decrease of total arousal. Good parachutists had a pattern similar to that described for experienced individuals, whereas poor jumpers more closely resembled novices. This research particularly emphasizes the relationship between adaptivity (e.g., the good parachutists) and an alteration (decreased arousal, anticipatory response) in the stress response.

Kurokawa et al. (54) examined plasma growth hormone secretion in response to stress. Male college students were told that they would be shown a stressful film on two consecutive days. Blood samples were taken both before and during the film. Plasma levels of growth hormone were significantly higher before the film than during it and slightly lower at both times on day 2. These results can be interpreted as reflecting a conditioned endocrine response. Lazarus et al. (55) also found evidence of an anticipatory response to stressful films. They measured autonomic variability (heart rate, electrodermal response) and found that there was an increase in arousal prior to the

showing of the first stressful film. What is interesting about these results is that the conditioning occurred prior to the first exposure to a stressor. Thus, it was achieved by cortical processing and past learned associations to the term "stressful."

Liakos et al. (56) stressed individuals by giving them mild shocks at specified times. Autonomic reactivity increased immediately before the time the shock was to be administered but, over trials, a gradual decrease both immediately before and during the shock was noted. Thus, it appears that on a variety of physiological measures, humans show a pattern of an anticipatory stress response followed by decreased reactivity upon habituation to the event.

When psychological factors are investigated concurrently with physiological reactivity, the adaptiveness of the above pattern is even more apparent. Speisman et al. (57), following their studies of response to stressful films, developed different soundtracks to accompany a film depicting circumcision rites in aborigines. One group of subjects was told to focus on the film in an intellectual way, another group was told that the circumcision was not painful or threatening, and a final group heard a version which emphasized the pain and discomfort of the procedure. The results indicated that subjects who heard the intellectual and denial sound tracks had lower levels of autonomic arousal than did controls (no sound track) or subjects exposed to the "trauma" sound track. Furthermore, in another experiment (58), effectiveness of the defense mechanism was found to covary with occupation: airline pilots had least arousal when presented with a denial sound track, while students were least aroused when an intellectual sound

track was used. Frankenhaeuser (59), measuring urinary catecholamine metabolites, found an increase of epinephrine secretion when subjects gained control (either actual or perceived) over the stressor. Rabavilas et al. (60) found that obsessive-compulsive patients had higher autonomic arousal during anticipation of an event than did controls. Furthermore, arousal level was found to be positively correlated with the amount of obsessive imagery.

Ford (61) examined the men aboard the U.S.S. Pueblo, which was detained by the Chinese for 9 months because of "trespassing" in North Korean seas. During that 9-month detainment, the men did not know what was going to happen to them. They were repeatedly questioned and abused by their captors. Upon return to the United States, they underwent psychological testing and have been prospectively followed since then. After their release, Ford interviewed these men and determined the types of defense mechanisms used while they were imprisoned. He found that the "good copers" used reality testing, rationalization, faith, denial, and humor; while the "poor copers" were preoccupied with obsessive ideation (including fantasy, religious ideations). Forty-six percent of the good copers were diagnosed as mentally healthy whereas 35% of the poor copers were given a diagnosis of passive-dependent personality. Furthermore, 21% of the good copers were diagnosed as schizoid, indicating that apparently one way to cope successfully with stress was to divorce one's self from one's surroundings. A follow-up study 5 years later, unfortunately, did not use the same classification of good and poor copers so conclusions about the long-term effects of different coping styles are not possible. Ploeger

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(62) studied miners who had been trapped in a mine for two weeks. During that entrapment, 6 of the 21 miners suffocated. A 10-year follow-up revealed that all of the survivors, except two, experienced some obsessive ideation, nightmares, and phobias. One man who suffered no "after effects" was the group leader both during and after the disaster. The other man was nineteen years old when the disaster occurred. The entrapment apparently was a positive experience for him. He had been very dependent on his mother up to the time of the accident but, afterwards, was able to free himself from her and improve his relations with others. Moreover, he hallucinated during the entrapment and this reaction may have been an effective defense against the fear. Unfortunately, Ploeger did not examine the men according to good and poor copers so it is not possible to compare his results to those of Ford (61). However, the above studies have many findings in common: poor, maladaptive coping appears correlated with obsessive ruminations, while adaptive coping (low physiological arousal) appears related to ways that decrease the impact/importance of the stressor. These ways include denial, intellectualization, rationalization, perceived control, humor, and anticipation. It must be noted that denial may be an adequate defense against short severe stressors, but it may promote physiological maladaptation in the long run. Janis (63) has suggested that under the rubric of denial there are many denial-like reactions, few of which are maladaptive. Even Speisman et al.'s (57) use of the term is inaccurate, since denial precludes conscious perception. Anyone who is watching a film, but told to ignore the stressful parts, is not denying the existence of the film. Rather he/she is suppressing—a conscious decision to ig-

nore something. Vaillant (64) discussed the difference between denial and suppression and noted that psychologically healthy men frequently used suppression but not denial. His conclusions were derived from a thirty-year prospective study of Harvard sophomores. Many interesting results have accrued from this population (64). The one that is the most relevant to this article concerns differences of coping strategies and defense mechanisms used by psychologically healthy or unhealthy men (65). The healthy men primarily used anticipation, humor, suppression, rationalization, and altruism, while the unhealthy groups used processes such as projection, fantasy, obsessive ideation, and dissociation. These results parallel those of other studies that examined adaptive response to stress. The well-adapted individuals apparently cognitively suppressed and ignored the stressful circumstances surrounding them. This cognitive style presumably reflects a psychological mechanism for maintaining homeostasis. As was seen in the physiological data, the organisms best adapted apparently decreased their arousal in response to the stressor over time. Hamburg and Adams (66) found that humans who engaged in cognitive anticipatory stress responses were better prepared to cope with stress than those who did not. Other studies (67, 68) have found that individuals who are well-adapted to life in terms of health status and productivity utilize coping styles which minimize the impact of the stressor, whereas those who are frequently ill are also chronically anxious.

Thus it appears that the human response to stress is a complex process consisting of interactions between psychological and physiological systems. Yet, beyond this process, the outcome which promotes life (e.g., is most adaptive) is

that which minimizes physiological responsiveness for the duration of the stress. Despite all the methodological differences, the least common denominator in human stress research is that adaptation involves anticipation and minimal physiological arousal. This state is typically accomplished by the use of coping and/or defense mechanisms which maintain psychological homeostasis. This adaptive response was also apparent in the animal literature, although on a more physiological level. The theory presented in this article is able to incorporate the results from a variety of stress research studies into a cohesive unit.

IMPLICATIONS

One of the most apparent implications of the theory is that organisms are genetically predisposed to adapt to stress. If, in fact, this is the case, then "exhaustion" must actually be some other process. Selye (46) felt that if a stressor were chronic, an organism would, at some point, fail to adapt due to exhaustion of adaptation energy. The stage of exhaustion was marked by the illness or death of the organism. In the proposed theory, illness or death would also be consequent to a failure to adapt, but would not result from an exhaustion of resources. Rather it would result from another cause (psychological or physiological) which prevented adaptation. In Selye's experiments, animals were typically exposed to a chronic stressor for 1-6 weeks. Selye found that animals reached exhaustion as early as several hours after stressor onset (46, 69). Exhaustion occurred then, presumably, because Selye often utilized severe, inescapable stressors. The present theory would not predict exhaustion—loss of all resources—within such a short time

period but would, instead, predict that a learned helplessness (70) occurred and that the animals gave up trying to cope with the stressor. Several studies have found that both animals (71) and humans (72, 73) who "give up" succumb to disease and/or death. Yet, alternatively, humans have been placed under severe and uncontrollable stress (e.g., Nazi concentration camps) and have survived. The survivors reported (74, 75) that they often ignored their situation, thought of their future, and, unlike their fellow prisoners who died, refused to give up.

Thus, the research literature on failure to adapt to chronic stress suggests that maladaptation results from psychological not physiological exhaustion as was suggested by Selye (46). Of course, some physiological defects will account for a small amount of exhaustion, but the majority of maladaptive responses will be due to psychological causes, as predicted by the theory. The implication of this prediction is that humans can adapt to almost any stress, given the use of appropriate coping resources. (Presumably, animals will be more affected by the stimulus qualities of the stressor than humans, since animals have less complex cognitive structures.)

A second implication of the theory follows from the above statement. If the theory is correct in predicting that maladaptation is due to the use of inadequate coping mechanisms, it should be possible to both identify adaptive coping mechanisms and to teach them to people who have difficulty dealing with stress and/or are frequently ill. This hypothesis has been realized in the form of stress management training (76) in which individuals are taught effective ways to cope with stress. Although many positive results have occurred from these experi-

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ments (77), they have primarily focused on alleviation of specific deficits and not on change of health status *per se*. According to the theory, maladaptation to stress increases one's susceptibility to illness, while adaptation decreases one's susceptibility. Thus, stress management training should result in a decreased incidence of illness (both physical and mental) if the above relationship is valid. Although much correlational research (64, 68) suggests that it is, no intervention studies have been done with humans to determine the feasibility of this approach.

Finally, if, in fact, an adaptive response to chronic intermittent stress does contain the elements of anticipation and decreased arousal, this pattern could serve as an objective measure of treatment effectiveness or of an individual's proclivity for adaptation.

As of this time, no truly objective measures of psychological treatment effectiveness exist. The measures are subjective to some extent (questionnaire, interview, etc.) and, as such, are subject to invalidation phenomena (78). By identifying an adaptive response pattern, a whole new area of research can be opened up to examine and understand this response.

SUMMARY

An integrative theory of the stress response was presented and shown to be a useful tool for analyzing results of a variety of studies. This theory, based on learning and evolutionary principles, states that organisms are predisposed to adapt to

chronic intermittent stress. Adaptation is achieved by anticipation of the stressor and decreased responsivity. These are expressed physiologically as a conditioned endocrine response to environmental stimuli predictive of the stressor, and a decreasing arousal response after stressor onset. Maladaptation is manifested as an increased or maintained arousal response occasionally continuing to occur despite absence of the stressor. Failure to adapt may be due to any number of variables (stressor, organismic characteristics), but is hypothesized to be due most often to learned cognitive states. It was further hypothesized that, rather than stressor exposure *per se*, the maladaptive response to stress is responsible for the increased illness susceptibility frequently found to follow stress. Support for this hypothesis accrues from research on immunosuppression (79) and work which found that healthy people were better able to ignore and decrease their arousal response than were frequently sick people (64, 67). These learned responses, which modify arousal, may be changed through programs such as stress management training and psychotherapy. In conclusion, by integrating the animal and human literature on stress, the relationship between the psychological and physiological stress response was clarified and redefined.

Supported in part by National Institutes of Health Grant No. AM17844 (S.C.W.) and by Graduate School research funds (to S.R.B.). The author wishes to thank Stephen C. Woods for his editorial advice.

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