心理学新进展影印丛书

美国心理学会（APS）组编

Current Directions

健康心理学新进展 in Health Psychology

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出版说明

“心理学新进展影印丛书”由北京师范大学出版社从培生教育出版集团引进出版，包括《普通心理学新进展》《发展心理学新进展》《认知心理学新进展》《社会心理学新进展》《人格心理学新进展》《健康心理学新进展》《变态心理学新进展》共7册英文影印图书，全部由美国心理学会（APS）组编，撰写者均为心理科学各自研究领域的世界著名学者和专家。入选的文章代表了各心理学分支自2000年以来的最新研究成果，同时各册所涉及的主题也全面体现了该分支学科的研究动态。

作为北京师范大学出版社成立26年以来第一批英文原版影印图书，我们真诚希望“心理学新进展影印丛书”的出版，可以为中国广大心理学研究者、教师以及相关专业的研究生，带来国际心理学界近十年的综合发展趋势，从研究思路、概念界定、研究方法与设计、统计技术以及未来的研究方向等方面，国内的学者能够及时把握到国际同行的关注热点，并感受到他们对传统理论的挑战与创新。

在各册图书中，既包括文献综述、对已有研究的质疑，也结合了先进的实验手段、技术和其他学科的综合知识，研究更多地关注和探索心理现象机制层面的复杂原因。每册均有问题思考，以启发学者们深入思索今后的研究热点和可能产生实质性飞越的突破口。

为便于读者阅读，我们特别邀请了北京师范大学心理学院的申继亮教授作为丛书审校专家，各册分别由北京师范大学的青年学者加入了简要的中文进行导读，同时还评价了研究的优缺点。

此套丛书可以作为各高校教师开设心理学新进展课程或专题讲座的教学用书，同时可以作为相关领域的研究人员发表文献综述的内容依据，尤其还适合作为心理学专业英语课程的教材进行学习和讨论。

策划编辑

谢　影

2007年1月18日

Contents

[导读]

第一编　疾病的心理社会和环境成因

健康心理学关注心理学和医学之间的相互联系，该领域的一个重要目的在于考察心理社会和环境因素在多大程度上影响身体疾病（包括头痛、便秘等急发症状，以及癌症、心脏病等慢性疾病）的发展变化，以及在多大程度上实现对人类寿命的预测等。本节概述了影响疾病发生、发展的心理社会和环境因素，这些因素包含多种水平：它们既可以是个体的状态或特质，如压力；也可以是社会环境特点，如家庭结构或工作环境；同时，还可以是更大范围的收入分配等社会因素。

在个体水平上，Cohen 运用了一种独特的研究设计，即将被试暴露于感冒病毒中，并将他们隔离几天。这一创新性方法保证每个被试暴露于同样剂量的感冒病毒中，从而解决了自然环境下高压力者因寻求社会支持而更可能感染病毒的问题。最后，Cohen 的研究发现，个体生活中的压力源以及对压力的感知，会影响他们患普通感冒的概率。接下来，Schulz 等人的研究发现抑郁的个体死亡率更高，尤其是心脏疾病引起的死亡。研究者发现，抑郁使疾病变得更加糟糕，增加了过早死亡的危险性。

尽管健康心理学关注抑郁、敌对等负性心理特征对健康的作用，但我们也不可忽略对积极心理状态的重视，研究积极的心理状态有助于了解健康问题的缓冲因素。Martin 的综述告诉我们，幽默和大笑可以对健康起到积极的保护作用，尽管其作用不像负性心理特征那么显著。Martin 描述了四种促进健康的潜在机制：大笑改变心理状态；幽默、大笑改变情绪状态；幽默、大笑帮助缓解压力并提高个体的社会支持水平。总之，这是一个新兴领域，还需要更多的研究来进一步证实。

在更大范围的社会因素层面，家庭成员和工作环境是社会联系的重要来源。Vitaliano 等人的研究发现，那些照料慢性疾病患者的护理者更容易生病和死亡，他们更需要就医。Spector 则发现，工作压力与生病和高血压关系紧密，并且，感知到的工作控制力在其中发挥重要的作用。

总之，本编主要阐述了心理社会和环境因素对疾病发展的影响，提示我们“心—身”联系更多反映在个体、工作和家庭生活、社会等多层面上。这些研究为我们打开了新的思路，引导我们从生物—心理—社会角度去考察疾病的发生与发展。

Psychosocial and Environmental Antecedents of Disease

Health psychology deals with the interconnections between psychology and medicine. A major goal of this field is to understand the extent to which psychosocial and environmental factors contribute to the development and progression of medical conditions. These conditions can range from acute physical symptoms, such as headaches and constipation, to chronic illnesses such as cancer and heart disease, to predicting the length of time a person lives.

This section provides an overview of the psychosocial and environmental characteristics that have been shown to contribute to the onset and course of medical illnesses. Note that these characteristics range across many levels of organization. They can be states or traits of an individual such as stress; features of the social environment such as family structure or work climate; and larger socital-level factors such as income distribution. At the individual level, Cohen demonstrates through a novel and unique research design that both the stressors in an individual's life, as well as the perception of stress, influence one's likelihood of developing the common cold. His research studies involve actually exposing participants to a cold virus and then keeping them in quarantine for several days. This rigorous experimental methodology allows Cohen to be certain that everyone is exposed to the same dose of a virus, and thus an alternative explanation that people who have higher stress are more likely to get exposed to viruses cannot account for his findings.

The Schulz et al. article reveals that individuals who experience a negative emotional state depression are prone to higher rates of mortality, particularly from diseases of the heart. While a person who develops heart disease is likely to become somewhat depressed, Schulz et al. 's work shows that these feelings can also worsen the disease,

and increase the risk of dying prematurely or having another heart attack. Much of the research in health psychology has focused on the role of negative psychological traits, such as depression and hostility, in health. However, it is also important to study positive psychological states that may help buffer us from detrimental health outcomes. Toward this end, Martin reviews the evidence for humor and laughter as protective factors. Martin describes four potential mechanisms to better health—that laughter may change physiological states, that humor/laughter may change emotional states, that humor/laughter may help make stressful times less stressful, and that humor/laughter may increase one's level of social support. The evidence for laughter as a pathway to health is not as strong as for negative psychological traits; however, this is a recent and emerging field, and more research will soon help to clarify its effects.

Next we move from the level of the individual to a broader perspective on the social factors in an individual's life. One important source of social contact is family members. Vitaliano et al. 's article describes what happens when an individual is placed in the role of caring for a family member with a chronic and debilitating illness. What happens in these circumstances? Researchers such as Vitaliano show that caregivers have changes in stress-sensitive biological systems, are more prone to illness and death, and need to take more medications. Outside of family members, the work environment is another major source of social connection for adults. Spector describes how job stress is associated with more illness symptoms and higher blood pressure. He also emphasizes the importance of perceived control at work, and builds the case for a novel theory of workplace conditions. It holds that high levels of demand can be toxic, but only when a person lacks control over his/her decisions and activities.

Finally, we move to the broadest level—socital factors that influence health. These factors include how people of different ethnic backgrounds are treated by others in society, as well as how the socioeconomic status that a person has within society affects health. Contrada et al. describe the processes by which discrimination occurs, including the perception of often ambiguous and subtle cues in the environment, as well as various ways in which people cope with discrimination. Contrada et al. also describe other types of ethnicity-related stressors, such as being concerned with confirming negative stereotypes about one's ethnic group as well as pressure from one's own ethnic group to behave in certain ways. Contrada presents evidence that these

types of ethnicity-related stressors are related to health and well-being. Lastly, Chen presents evidence that individuals lower in socioeconomic status (SES) have poorer health across a variety of conditions. Interestingly, these effects are not just due to poverty, since each increase in SES is associated with better health across the whole SES spectrum. Moreover, these effects are robust across numerous diseases in both childhood and adulthood.

Together, these articles provide a glimpse of the evidence linking psychosocial and environmental characteristics to the development and progression of medical illness. They show that this "mind-body" connection arises from multiple sources, ranging from characteristics of an individual, to the structure of work and family life, to broader demographic and socioeconomic factors in society.

[导读]

心理压力、免疫力和上呼吸道感染

压力会使个体更容易感染普通感冒、流感和其他传染性疾病，这一观点已得到了人类社会文化的普遍认可，并得到了相关研究的证实。本文通过对已有文献进行综述，探讨了压力对传染性疾病易感性的影响机制。

研究发现，压力降低了人类免疫系统的作用：当个体感知到压力并对自己应对压力的能力作出评估后，一般会出现一些负面的情绪反应，这些情绪反应继而改变我们的神经纤维、行为模式（如抽烟、不规律饮食和睡眠的出现）以及荷尔蒙的分泌，从而影响到人体免疫系统的活力，并导致疾病易感性的提高。

在自然环境下考察压力对个体传染疾病的易感性的作用，是分析二者关系更直接的一种方法。大量的研究发现，心理压力与自我报告和生物检测的上呼吸道疾病——一种最流行的传染性疾病（如普通感冒和流感）有关，但对这些研究结果的解释非常困难。一方面，二者之间的关系受到第三变量的影响，如社会阶层、年龄和种族等；另一方面，压力导致个体暴露于受传染来源的可能性增强（如个体更多地寻求他人的帮助），而并非导致与压力有关的免疫抑制水平得到提高。鉴于此，作者采取了病毒导向的研究策略，在被试完成压力问卷后，为他们注射普通感冒病毒，并在随后的 5 天或更长的时间里监控被试是否感染疾病。这一研究策略可以很好地解决自然环境研究中的第二个问题，同时，基于该策略的很多研究可以有效地验证压力和上呼吸道疾病之间的因果关系。但需要说明的是，这些研究无法解释上呼吸道疾病易感性的提高是否与压力引发的免疫抑制有关。未来的研究应加入基因分析模式，以帮助探讨压力和其他传染性疾病之间的关系。

Psychological Stress, Immunity, and Upper Respiratory Infections

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The belief that when we are under stress we are more susceptible to the common cold, influenza, and other infectious diseases is widely accepted in our culture. It is the topic of numerous contemporary newspaper and magazine articles and has even been addressed in the lyrics of a popular song ("Adelaide's Lament" from *Guys* and *Dolls*). The wide acceptance of this belief is also supported by data collected from participants in my studies. Sixty percent report that they are more likely to catch a cold during stressful than nonstressful periods of their lives. In this article, I review the scientific evidence that addresses this belief. How could psychological stress influence susceptibility to infectious disease? Is such a relation biologically and psychologically plausible? Is there convincing evidence that psychological stress influences susceptibility to upper respiratory infections?

HOW COULD STRESS INFLUENCE SUSCEPTIBILITY TO INFECTIOUS DISEASE?

Although constantly exposed to bacteria, viruses, fungi, and parasites that can cause infectious disease, we only periodically develop infectious illnesses. This is because our immune system protects us from infectious microorganisms. This defensive function is performed by the white blood cells and a number of accessory cells, which are distributed throughout the organs of the body. Stress is thought to influence susceptibility to infectious disease by compromising the effectiveness of the immune system. Persons with suppressed immune function are less able to fight off infectious agents and hence, given exposure to an agent, more likely to develop an infectious disease.

A simplified view of how stressful events in our lives might alter immunity is presented in Figure 1. When our demands are perceived to exceed our ability to cope, we label ourselves as stressed and experience a negative emotional response (Lazarus & Folkman, 1984). In turn, negative emotional responses could alter immune function

through three different pathways (Rabin, Cohen, Ganguli, Lyle, & Cunnick, 1989). Nerve fibers connecting the central nervous system and immune tissue provide one path by which emotional responses may influence immunity. These nerves terminate in immune tissue, where they release chemicals that are thought to suppress the function of immune cells. Stress-induced emotions may also act through their influence on the central nervous system's production and release of hormones such as epinephrine and cortisol. These hormones circulate in the blood and can attach to receptors on immune cells, resulting in the cells' protective functions "turning off." The third mechanism by which stress may affect health derives from the role of behavioral patterns that reflect attempts to cope with negative emotional responses. For example, persons experiencing psychological stress often engage in unhealthy practices such as smoking and not eating or sleeping properly, and such behavioral changes may suppress the activity of the immune system. They may affect immune responses directly or may influence immune function by altering hormonal responses.

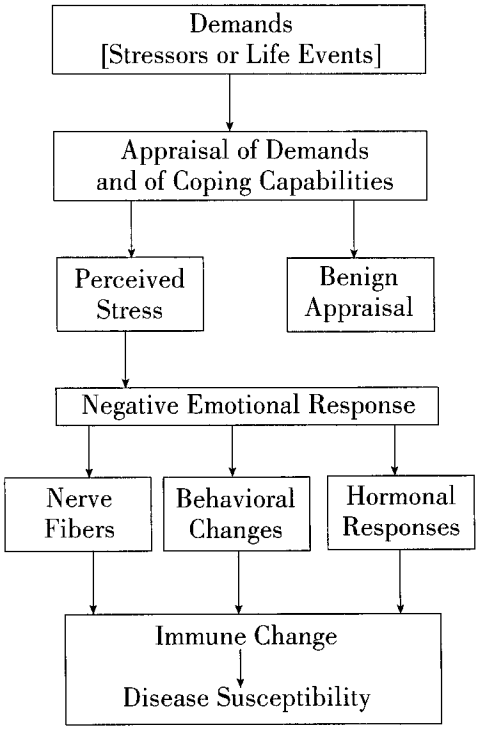

Fig. 1. Pathways through which stressful life events might influence the onset and progression of

infectious disease. For simplicity, arrows are drawn in only one direction, from psychological characteristics to disease. This convention does not imply any assumptions about the existence of alternative paths.

DOES STRESS INFLUENCE IMMUNE FUNCTION?

As just discussed, the key link between psychological stress and susceptibility to infectious agents is thought to be the immune system. There is substantial evidence supporting the role of stress in the regulation of the human immune system. Suppression of immune function has been found among persons taking important examinations (e. g., Kiecolt-Glaser et al., 1984); caring for relatives with chronic diseases (Kiecolt-Glaser, Glaser, et al., 1987); living near the site of a serious nuclear-powerplant accident (McKinnon, Weisse, Reynolds, Bowles, & Baum, 1989); suffering marital conflict (Kiecolt-Glaser, Fisher, et al., 1987); and reporting relatively high levels of unpleasant daily events (Stone et al., 1994), negative moods (Stone, Cox, Valdimarsdottir, Jandorf, & Neale, 1987), or perceived stress (e. g., Jabaaji et al., 1993). Suppression of immune function (called *immunosuppression*) has also been found in response to acute laboratory stressors, including working on challenging cognitive tasks, such as mental arithmetic, and delivering public speeches (e. g., Manuck, Cohen, Rabin, Muldoon, & Bachen, 1991). Clinical depression has also been associated with decreased immune response (Herbert & Cohen, 1993).

DOES STRESS INFLUENCE SUSCEPTIBILITY TO UPPER RESPIRATORY INFECTIONS?

Do studies that demonstrate induced immunosuppression under stressful conditions provide compelling evidence for stress-induced susceptibility to infectious disease? In general, these data are thought to be consistent with, but not definitively supportive of, the hypothesis that stress results in increased susceptibility to disease. The immune response involves a complex cascading series of events. Because studies of stress and immunity are limited to assessing very few markers of immune function in a limited time span, they can provide only a very rough estimate of the body's ability to mount such a defense (Cohen & Williamson, 1991).

Naturalistic Studies of Stress and Upper Respiratory Infection

A more direct approach to addressing the role of psychological stress in susceptibility to infection is examining the correlation between stress and infectious disease in natural settings. Because upper respiratory infections are by far the most prevalent of infectious diseases, the common cold and influenza have been adopted as the primary models for studying how stress might influence susceptibility. A large group of studies has found correlations between psychological stress and self-reported colds and influenza (reviewed in Cohen & Williamson, 1991). This work, however, is generally difficult to interpret. In many cases, third factors such as social class, age, or ethnic background might be responsible directly for increases in both stress and disease. Moreover, because this work is primarily retrospective, being ill may have caused stress rather than vice versa. Another problem is that unverified self-reports of illness are difficult to interpret. Although they may indicate underlying disease pathology, they may also reflect stress-induced biases to view ambiguous physical sensations as symptoms, and to interpret symptoms as indicating the onset of disease (e. g. , Cohen et al. , 1995).

There are a few investigations that have associated psychological stress and biologically verified (as opposed to self-reported) upper respiratory disease (e. g. , Graham, Douglas, & Ryan, 1986; Meyer & Haggerty, 1962). Verification was accomplished by establishing the presence of a responsible bacterium or virus in nasal secretion or of an elevated level of antibody to the infectious agent in blood (serum).[2] In these studies, measures of psychological stress were administered to healthy subjects who were subsequently monitored for up to 12 months for the development of upper respiratory infections. For those reporting infections, nasal secretions or blood samples were used to biologically verify the disease. These studies have found links between psychological stress and the subsequent development of colds and influenza. These results, however, may be attributable to stress-induced increases in exposure to infectious agents, rather than stress-induced immunosuppression. For example, persons under stress often seek out other people, consequently increasing the probability of exposure. The studies also fail to provide evidence about behavioral and biological mechanisms through which stress might influence a person's susceptibility to infection.

Viral-Challenge Studies

In my own work, I have adopted a procedure in which after completing stress questionnaires, volunteers are intentionally exposed to a common cold virus (in nasal drops) and then quarantined and monitored for 5 or more days for the development of disease.[3] Approximately one third of the volunteers exposed to a virus develop a biologically verified clinical cold. The viral-challenge procedure has a number of advantages over naturalistic studies. By experimentally exposing persons to a virus and limiting their contact with other people, I eliminate the possibility that the results are attributable to stress increasing social contact and hence exposure to infectious agents. Moreover, because participants are closely monitored after exposure, it is easier to verify disease onset and to assess the roles of behavioral and biological pathways that might link stress to disease susceptibility. Finally, this methodology allows for a more refined assessment of the body's response to a virus. Specifically, after exposure to a virus, persons can become infected (i.e., their cells replicate the virus) without developing symptoms. In the viral-challenge trials, body fluids used to determine infection are drawn from subjects both with and without upper respiratory symptoms, allowing the identification of subclinical (i.e., with few if any symptoms) as well as clinical infections.

In an attempt to take advantage of the strengths of this methodology, my colleagues and I conducted a viral-challenge study addressing the role of stress in susceptibility to the common cold (Cohen, Tyrrell, & Smith, 1991, 1993). By using a prospective design in which psychological stress is assessed before participants are exposed to a virus, we were able to eliminate the possibility that illness causes stress as an interpretation of our results. Because the primary outcome in viral-challenge studies is categorical (sick or not), large sample sizes are required to maximize study sensitivity. Hence, we accumulated data from 420 healthy volunteers. Collection of these data required more than 40 separate 1 week trials conducted over 4 years. Our main hypothesis was that the higher the level of psychological stress, the higher the risk of developing the upper respiratory illness caused by the virus.

Each participant completed psychological stress questionnaires just prior to being exposed to one of five viruses known to cause common colds. A group of control participants received saline in nasal drops instead of a virus. After 7 days of quarantine, each participant was classified as not infected, infected but not ill, or infected and ill