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Cold, common

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Upper respiratory infections (URI) as a group are responsible for 50% of all acute illnesses, with the common cold syndrome being most familiar. Colds are caused by over 200 viruses and are characterized by sore throat, congestion and mucus secretion. When exposed to viruses or other infectious agents, only a proportion of people develop clinical illness. Reasons for variability in response are not well understood and the possibility that psychological factors play some role in the aetiology and progression of infectious disease has received increased attention.

It is commonly believed that stressful life events influence the onset of URI by causing negative affective states (e.g. anxiety and depression) which, in turn, exert direct effects on biological processes or behavioural patterns that increase disease risk. The influence of stress on the immune system is considered the primary biological pathway through which stress can influence infectious disease susceptibility. While there is substantial evidence that stress is associated with changes in immune function (Herbert & Cohen, 1993; Segerstrom & Miller, 2004) (see 'Psychoneuroimmunology'), the implications of stress-induced immune changes for susceptibility to disease have not been established. To date, studies of stress and URI susceptibility have focused on establishing a link between stress and disease with little attention to pathways through which such an association might occur. The major findings of these studies are examined below.

There is consistent evidence that persons under stress report greater levels of URI symptoms and that stress results in greater health care utilization for URI (Cohen & Williamson, 1991). For example, Glaser *et al.* (1987) demonstrated that medical students report more infectious (mostly URI) illness during examination periods than at other times. Similarly, Stone *et al.* (1987) found that, for 79 married couples followed over three months, daily life events rated as undesirable increased three to four days prior to onset of self-reported symptoms of URI, close in time to the incubation period of many common cold viruses. The self-reported symptoms of URI measured in these studies may tap underlying pathology; however, it is also possible that they reflect a stress-induced misinterpretation of physical sensations without underlying illness. The latter interpretation is supported by studies in which effects of stress on symptoms, but not verified disease, are observed, and by evidence that stress is associated with increased symptom reporting in general, not only with symptoms directly associated with infectious pathology (Cohen & Williamson, 1991) (see 'Symptom perception').

Other investigators have verified the presence of pathology by physician diagnosis or biological methods. Several of these studies provide evidence that life stressors increase risk for verified upper respiratory disease. For example, Meyer and Haggerty (1962) followed 100 members of 16 families for a 12-month period. Daily life events that disrupted family and personal life were 4 times more likely to precede than to follow new streptococcal and non-streptococcal infections (as diagnosed by throat cultures and blood antibody levels) and associated symptomatology. Similar results were reported in a study of viral URIs in 235 members of 94 families (Graham *et al.*, 1986). Here, high stress, as defined by scores on reported major stressful life events, daily events and psychological stress, was associated with more verified episodes and more symptom days of respiratory illness. Turner Cobb and Steptoe (1996) also found that higher levels of life event stress were associated with increased clinically-verified URI among 107 adults followed for 15 weeks. In sum, studies verifying infectious episodes suggest that stress increases risk for upper respiratory disease. However, community studies, like these, do not control for the possible effects of stressful events on exposure to infectious agents. Moreover, the literature on this topic is not entirely consistent; indeed, several studies have failed to find a relation between stress and upper respiratory disease (for review see Cohen & Williamson, 1991) (see also 'Life events and health').

Several prospective studies have eliminated the possible role of psychological effects on exposure by experimentally inoculating healthy individuals with common cold viruses (viral challenge studies). Here, volunteers are assessed for degree of stress and then experimentally exposed to a cold virus or placebo. They are then kept in quarantine and monitored for the development of infection and illness. Early viral challenge studies were limited by a range of methodological weaknesses (Cohen & Williamson, 1991), including insufficient sample sizes and lack of control for factors known to influence susceptibility to viral infection (including pre-existing antibodies to the infectious agent and age). Furthermore, the possible role of stress-elicited changes in health practices such as smoking and alcohol consumption was not considered. These limitations may account for initial failures to find consistent relations between stress and susceptibility to URI.

In contrast, recent viral challenge studies have included multiple controls for factors known to be independently associated with susceptibility to viral infection (e.g. Cohen *et al.*, 1991, 1993, 1998; Stone *et al.*, 1992). These studies consistently find a positive association between stress and susceptibility to URI. For example, among 394 healthy adults, stressful life events, perceptions of current stress and negative affect were all associated with an increased risk of developing biologically verified URI, with greater stress related linearly to

susceptibility (Cohen *et al.*, 1991, 1993, 1998). Further examination of findings revealed that perceptions of stress and negative affect increased risk for illness through a different pathway than stressful life events. The former measures increased the probability of becoming infected (replicating virus), while the latter increased the probability of infected people developing clinical symptoms (Cohen *et al.*, 1995). More recent research from this group suggests that the longer the duration of stressful life events, the greater risk of becoming infected (Cohen *et al.*, 1998). A large group of control factors including age, sex, allergic status, body weight, season and virus-specific antibody status before challenge, could not explain the increased risk of colds for persons reporting greater stress. Smoking, alcohol consumption, diet, exercise and sleep quality also failed to explain the association between stress and illness.

In a similar study, Stone *et al.* (1992) examined development of symptoms among persons infected with rhinovirus. They found that those with more life events were more likely to develop clinical colds, although perceptions of current stress and negative affect were unrelated to symptom development. In contrast to studies described earlier, this investigation included only infected persons and hence could not assess susceptibility to infection where Cohen and colleagues found perceptions of stress and negative affect were related to susceptibility.

Recent attention has turned to an examination of psychosocial factors that may decrease susceptibility to URI by regulating emotion-sensitive biological systems and/or encouraging health-enhancing behaviours. These factors include dispositional positive affect and social engagement. Initial findings from viral challenge studies suggest that susceptibility to URI decreases in a dose-response manner with increased diversity of the social network and with trait sociability (Cohen *et al.*, 1997; Cohen *et al.*, 2003*a*) (see Social Support and Health). Furthermore, higher positive affect has been associated with decreased incidence of objective symptoms of upper respiratory illness among infected people (Cohen *et al.*, 2003*b*). These relationships appear to be independent of negative emotional styles, baseline immunity, demographics and health practices. Thus, there is some initial evidence that certain psychosocial factors are protective, being related to greater resistance to infection and the expression of fewer objective clinical symptoms.

In sum, well controlled studies support prospective studies of community samples in indicating that psychological stress is associated with increased susceptibility to the common cold. In addition, there is consistent evidence for increased symptom-reporting under stress. Recent studies also suggest that positive emotional styles, sociability and more diverse social networks are associated with greater resistance to developing colds. A number of potential pathways exist through which an association between psychosocial factors and infectious pathology might occur, including behavioral, hormonal and immune mechanisms. Future work is needed to explore these alternatives.

(See also 'Asthma' and 'Chronic obstructive pulmonary disease'.)

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