# 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), 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, Reed & Neale (1987) found that, for 79 married couples followed over three months, daily life events rated as undesirable increased 3 to 4 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).

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 sixteen families for a 12-month period. Daily life events that disrupted family and personal life were four 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, Douglas, Ryan, 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. 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).

Several prospective studies have eliminated the possible role of psychological effects on exposure by inoculating healthy volunteers with common cold viruses in attempts to determine whether psychological factors (measured prior to the viral exposure) influence susceptibility to URI. However, early viral inoculation 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 (e.g. pre-existing antibodies to the infectious agent, gender, 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 failure of initial viral challenge studies to find consistent relations between stress and susceptibility to URI.

In contrast, Cohen, Tyrrell & Smith (1991, 1993) performed a large-scale viral inoculation study, including multiple controls for factors known to be independently associated with susceptibility to viral infection. In this prospective investigation, 420 healthy adults were assessed for degree of stress, and then experimentally exposed to one of five cold viruses or placebo. Increases in stressful life events, perceptions of current stress and negative affect were all associated with an increased risk of developing biologically verified URI. However, the investigators found 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. 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 the study 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.

In sum, recent, 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. A number of potential pathways exist through which an association between stress and infectious pathology might occur, including behavioural, hormonal and immune mechanisms. Future work is needed to explore these alternatives.

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# Colour blindness


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Inherited deficiencies of colour vision are so common in western populations that some have supposed that these variant forms of human vision are maintained by a biological advantage; and certainly it is possible to show in the laboratory that a colour-blind observer can detect small variations in texture that are masked for the normal by uncorrelated variations in colour. Some 2% of Caucasian men are dichromats: to match all colours they require only two primary wavelengths, rather than the three needed by the normal. A further 6% of men are anomalous trichromats: they need three primaries in a colour-matching experiment but for most test lights they mix the primaries in different proportions from the normal. Anomalous trichromacy is commonly, but not invariably, associated with poorer colour discrimination than normal. Both dichromacy and anomalous trichromacy are usually sex-linked conditions: to exhibit a defect, a woman must normally inherit it from both parents. Thus frank colour blindness is seen in less than 0.5% of women, although some 15% of all women are carriers and can often be detected by subtle tests.

Normal colour vision depends on the presence in the retina of three classes of cone cell, with peak sensitivities respectively in the violet (short-wave), green (middle-wave) and yellow-green (long-wave) regions of the spectrum. Embedded in the membranes of the cones are light-sensitive molecules, members of the superfamily of heptahelical molecules (the family also includes the dopaminergic and serotonergic receptors). It is differences in the amino acid sequences of these light-sensitive pigments that lend the cones their different spectral sensitivities. However, an individual cone cannot distinguish the photons of different wavelength that it absorbs, and so the visual system must neurally compare the quantum catches of different types of cone in order to distinguish colour from intensity.

The common forms of inherited colour blindness arise from alterations of either the long-wave or the middle-wave photopigment. The genes that code for these pigments lie in a cluster on the X-chromosome (at locus Xq28) and are thought to have arisen from duplication of a single ancestral gene at an early stage of primate evolution. The juxtaposition and the homology of the long- and middle-wave genes appear to encourage unequal crossing-over, giving rise to a rich variety of genotypes and phenotypes. The cluster may contain more than one copy of the normal genes as well as hybrid genes that draw part of their sequence from the long-wave

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