season and increases again later in the year in temperate climates; in the tropics, asthma occurs more frequently in the wet season (LAIA, 1993; *Lancet*, 1985). In many asthmatic individuals, aspects of weather can exacerbate bronchial hyperresponsiveness. For example, the passage of a cold front followed by strong high pressure was found to be associated with unusually high number of asthma admission days in two U.S. cities (Goldstein, 1980). Sandstorms in Kansas (USA) and the Sudan have been accompanied by increases in bronchitis and asthma (Ayres, 1990).

It is well established that exposure to air pollutants, individually and in combinations, has serious public health consequences. For example, exposure to ozone has been shown to exacerbate asthma and impair lung function in children and the elderly (Beckett, 1991; Schwartz, 1994), and both chronic and acute exposures to fine particles are a cause of excess deaths (Dockery *et al.*, 1993; Pope *et al.*, 1995; Schwartz, 1994) even at exposures below prevailing air-quality standards. Since the combustion of fossil fuels is a major source of both carbon dioxide (a major greenhouse gas) and various air pollutants, climate change can be expected to entail more frequent occasions that combine very hot weather with increases in air-pollutant concentrations. In urban environments, the weather conditions that characterize oppressive air masses (see Section 18.2.1) also enhance the concentrations of air pollutants (Seinfeld, 1986); conditions of low wind speed and high humidity occur periodically in which neither heat nor air pollutants are rapidly dispersed. Further, increases in temperature or in ultraviolet irradiation of the lower atmosphere enhance the chemical reactions that produce secondary photochemical oxidant pollutants such as tropospheric ozone (Akimoto *et al.*, 1993; de Leeuw and Leyssius, 1991; Chamiedes *et al.*, 1994).

In many urban settings, studies have shown that daily mortality from cardiovascular and respiratory diseases is a combined function of temperature and air pollutant concentrations. This combination of exposures is also likely to have interactive impacts on health. Indeed, some epidemiological evidence indicates a synergy (a positive interaction) between stressful weather and various air pollutants, especially particulates, upon mortality (e.g., Shumway *et al.*, 1988; Katsouyanni *et al.*, 1993; Shumway and Azari, 1992). The net effect on morbidity/mortality therefore would be greater than anticipated from prior estimates of the separate effects of weather and pollutants.

18.4. Stratospheric Ozone Depletion and Ultraviolet Radiation: Impacts on Health

Stratospheric ozone depletion is a quite distinct process from accumulation of greenhouse gases in the lower atmosphere (troposphere). Depletion of stratospheric ozone has recently occurred in both hemispheres, from polar regions to mid-latitudes (Kerr and McElroy, 1993; see also IPCC Working Group I volume). The major cause of this ongoing depletion is human-made gases, especially the halocarbons (UNEP, 1994).

The problem can be considered alongside climate change for three reasons: (1) several of the greenhouse gases (especially the chlorofluorocarbons) also damage stratospheric ozone; (2) altered temperature in the troposphere may influence stratospheric temperature and chemistry (Rind and Lacis, 1993); and (3) absorption of solar radiation by stratospheric ozone influences the heat budget in the lower atmosphere (see also IPCC Working Group I volume).

Stratospheric ozone absorbs part of the sun's incoming ultraviolet radiation (UVR), including much of the UV-B and all of the highest-energy UV-C. Sustained exposure to UV-B radiation is harmful to humans and many other organisms (UNEP, 1994). It can damage the genetic (DNA) material of living cells and can induce skin cancers in experimental animals. UV-B is implicated in the causation of human skin cancer and lesions of the conjunctiva, cornea, and lens; it may also impair the body's immune system (Jeevan and Kripke, 1993; Armstrong, 1994; UNEP, 1994).

18.4.1. Skin Cancers

Solar radiation has been consistently implicated in the causation of nonmelanocytic and melanocytic skin cancers in fair-skinned humans (IARC, 1992; WHO, 1994b).

Nonmelanocytic skin cancers (NMSCs) comprise basal cell carcinoma (BCC) and squamous cell carcinoma (SCC). The incidence rates, especially of squamous cell carcinoma, correlate with cumulative lifetime exposure to solar radiation (IARC, 1992; Kricker *et al.*, 1995). Studies of the action spectrum (i.e., the relative biological effect of different wavelengths) for skin carcinogenesis in mice indicate that the UV-B band is primarily responsible for NMSC (Tyrrell, 1994). Malignant melanoma arises from the pigment-producing cells (melanocytes) of the skin. Although solar radiation is substantially involved in melanoma causation (IARC, 1992; Armstrong and Kricker, 1993), the relationship is less straightforward than for NMSC; exposure in early life appears to be a major source of increased risk. The marked increases in incidence of melanoma in Western populations over the past two decades (Coleman *et al.*, 1993) probably reflect increases in personal exposure to solar radiation due to changes in patterns of recreation, clothing, and occupation (Armstrong and Kricker, 1994).

The UN Environment Programme predicts that an average 10% loss of ozone (such as occurred at middle-to-high latitudes over the past decade), if sustained globally over several decades, would cause approximately 250,000 additional cases of NMSC worldwide each year (UNEP, 1994). This prediction assumes that a 1% depletion of stratospheric ozone results in a 2.0% (±0.5%) increase in NMSC incidence (80% of which are BCC). Another estimation of this "amplification factor" gives a figure of 2.25% (Slaper *et al.*, 1992; den Elzen, 1994). At higher geographic resolution, Madronich and de Gruijl (1993) predict that persistence of the ozone losses of the 1979–92