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ORIGINAL ARTICLE

Climate change, global warming and the upper respiratory airways-a review

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The progressive rise in air pollution has been linked to increased respiratory morbidity and mortality. Recent decades have seen increased demands for medical treatment accompanied by an increased risk of hospitalization and rise in premature deaths. Climate change contributes to air pollution by affecting the dispersal of primary pollutants, principally particulate matter $PM_{2.5}$ and by increasing the formation of secondary pollutants, mainly surface ozone close to ground level. Climate change is inherently associated with an increase in the prevalence of rhinitis, and its associated deterioration in quality of life.

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Climate change and air pollution involve a complex web of inter-relationships with various elements affecting human respiratory health. This paper reviews the relationship between the different principal elements associated with climate change and their effects on respiratory epithelium. The main contributing factors to nasal and pulmonary pathology are discussed and include carbon dioxide, ozone and particulates. The pathological mechanisms acting on nasal and bronchial mucosa are also described.

CLIMATE CHANGE

Global warming and the increase in emissions causing air pollution have caused a progressive deterioration in air quality. Since airborne allergens and air pollutants are frequently increased contemporaneously in the atmosphere, an enhanced IgE-mediated response to aeroallergens and enhanced airway inflammation could account for the increasing frequency of rhinitis and asthma in atopic subjects in the last 5 decades.[1]

Increasing carbon dioxide concentration in the earth's atmosphere is the main contributor to the greenhouse effect with increased mean temperatures. Higher surface temperatures lead to evaporation of water from soil so the land loses the ability to cool down with further trapping of heat. Extreme weather events such as lightning and heat waves have been predicted to become more common.[2]

Climate extremes are encouraged by increased water vapour concentrations in the air due to rising surface sea temperatures.[3]

Increased heat extends the pollination season of grasses and their geographical range resulting in more widespread and prolonged dispersal of pollens.[4] Pollens react with diesel particulates and ozone to increase their immunogenicity.[5,6]

Climate change is likely to influence the seasonal period and therefore grass growth, reproductive cycles and intensity of allergenic pollen load. In addition, weed species are expected to proliferate. These changes may vary from one region to another, due to the variation in amounts of UV radiation and rainfall.

The annual number of weeks in the year with high air pollen and mould spore concentrations has been progressively rising over time and has been shown to be positively correlated with temperature rise.[7]

Due to environmental pollutants which also act as irritants to skin and mucous membranes, pollen grains can be altered in the atmosphere leading to the release of lipid mediators (so-called pollen-associated lipid mediators) having pro-inflammatory effects.[8] Exposing ragweed plants to doubled CO_2 atmospheric concentration experimentally increased pollen production by 61%. In addition, ragweed pollen collected along roads with high traffic showed more allergenicity then the same pollen in rural areas.[9]

There is increasing evidence that climate change with events such as flooding may increase the dampness of buildings and therefore the growth of indoor moulds.[10] Increased fungal spore counts have been associated with an increase in asthma symptoms, increase in asthma medication and hospital admissions.[11,12]

Our forecasts of the future impacts of climate change depend on weather forecasts and may vary according to the weather models used which assume different scenarios of greenhouse gas concentrations.[13]

EXTREME WEATHER CONDITIONS

An increasing body of evidence correlates the occurrence of severe asthma epidemics with thunderstorms in the pollen season. Several epidemics of asthma have been reported following thunderstorms in various geographical zones, prevalently in Europe and Australia.[13]

In the first 20-30 minutes of a thunderstorm, there is evidence of increasing concentrations of air-borne allergens. This is due to dry updrafts that waft whole pollens into the cloud base where humidity is high. Here pollens may rupture and cold downdrafts carry the fragments to ground level where outflows distribute them. Whole pollen grains are too large to penetrate the deeper airways. However fragments may manage to do so thus enhancing bronchial hyperreactivity.

Due to strong electric fields that develop during thunderstorms, positive ions are released from the ground and could attach to pollen particles enhancing pollen rupture.

CARBON DIOXIDE

Carbon dioxide is the commonest greenhouse gas which is released by the burning of fossil fuels. Its concentration in the atmosphere is presently approximately 400 parts per million (ppm). Compared

to pre-industrial levels of 280 ppm, this is a rise of 43%. The trapping of heat by greenhouse gases has caused a rise in average global temperature of approximately 1°C. If atmospheric carbon dioxide continues to increase, projections calculate a further global average rise of between 2 and 6°C. [14]

Burning of fossil fuels contributed 75% of anthropogenic (man-created) CO_2 emissions to the earth's atmosphere.[15]

Tropical forests have acted as a carbon sink by taking up carbon dioxide from the atmosphere. Deforestation and clearing with burning of tropical forests contribute almost 25% of CO_2 emissions-the largest proportion of anthropogenic CO_2 emissions after fossil fuel combustion.[16]

OZONE

Ground-level ozone is a secondary pollutant formed by the interaction of the ultraviolet component of sunlight[17] with precursors which include nitrogen oxide emissions from traffic pollution, volatile organic compounds coming from plant metabolism and methane from bovine sources.[18,19]

Ground-level ozone is recognized as one of the worst urban pollutants. In experimental human and animal models, exposure to ozone impairs pulmonary function, increases airway responsiveness, and induces inflammation in the lower airways. At the cellular level, ozone can trigger epithelial cellular membranes to discharge cytokines and arachidonic acid metabolites such as cyclooxygenase and lipoxygenase derivatives. In addition, ozone can indirectly decrease mucociliary clearance and free radical production.[20]

The other main urban pollutant besides traffic related air pollutants (TRAP) is Particulate Matter (PM) of various dimensions.

PARTICULATE MATTER (PM)

Particulate matter is defined as coarse or fine. Coarse particles between 2.5 to 10 microns in diameter are deposited in upper airways while fine particles less than 2.5 microns in diameter are deposited into the lung. Particles can directly exacerbate existing respiratory disease such as asthma.[21] They can also further promote climate change by affecting ambient temperatures.[19] $PM_{2.5}$ levels are associated with anthropogenic sources like soot particles which absorb heat and increase local temperature.[22]

Particles may originate from anthropogenic sources such as construction activity, burning fossil fuels and forest fires, but also from natural sources such as volcanic eruptions and sand in dust storms.

Pollutant emission and dispersal or deposition are influenced by meteorological variables.[23] Reduced air quality affect humans directly but also affect humans indirectly by impacting ecosystems.

In the future, air quality is expected to worsen in cities.[24] Southern Europe is projected to be more affected than Northern regions.[25]

DIESEL EXHAUST PARTICLES (DEP)

DEP has a solid aggregate of elemental carbon and metals, in addition to a gaseous phase composed mainly of non-toxic inorganic gases such as oxygen and nitrogen. Organic components of DEP such as benzene, pyrenes, and others, are collectively termed poly-aromatic hydrocarbons, or PAH.

Based on epidemiological data, WHO and the International Agency for Research on Cancer have classified DEP as highly carcinogenic to humans.[26,27] through possible pulmonary genetic damaging effects and inflammatory toxicity.

Furthermore, climate chamber studies involving both ragweed and house dust mite (HDM) allergic patients also suggest a synergistic effect of DEP on atopic inflammatory mediators following respective allergen challenge and exposure[20] However consistent experimental simulation of real-life exposure conditions in animal studies is difficult to achieve due to the high complexity of DEP composition.

TRAFFIC RELATED AIR POLLUTANTS

Urban traffic related air pollutants (TRAP) have a complex structure composed of solid and gaseous phases, namely black carbon from diesel exhaust with gases like nitrous oxides and carbon monoxide, originating from general traffic and petrol exhaust. Other constituents include metals like zinc and copper originating from car brakes and tyres, respectively.[28]

Nitrogen dioxide contributes to ground level ozone formation which causes an inflammatory effect on the respiratory tract.

EFFECT ON THE RESPIRATORY SYSTEM

In a large epidemiological study from Brazil children living in polluted areas reported a 7% incidence of rhinitis compared to those living in non-polluted areas where the incidence was only 4%.[29]

Following the reunification of Germany, declines in air pollutant levels in Eastern Germany were correlated with a decrease in questionnaire-based respiratory tract symptoms.[30]

A 1°C rise in temperature is associated with increased mortality from respiratory causes, particularly in the elderly.[31]

Due to increased urbanization of populations and increased time spent working remotely means more exposure to indoor air pollutants such as House Dust mite and animal dander (eg cat) and possibly tobacco smoke. Tobacco smoking in the home has been identified as a factor increasing particulate matter (PM) and toxic chemical agents.[32] Remote working increased recently due to the SARS 2 pandemic.

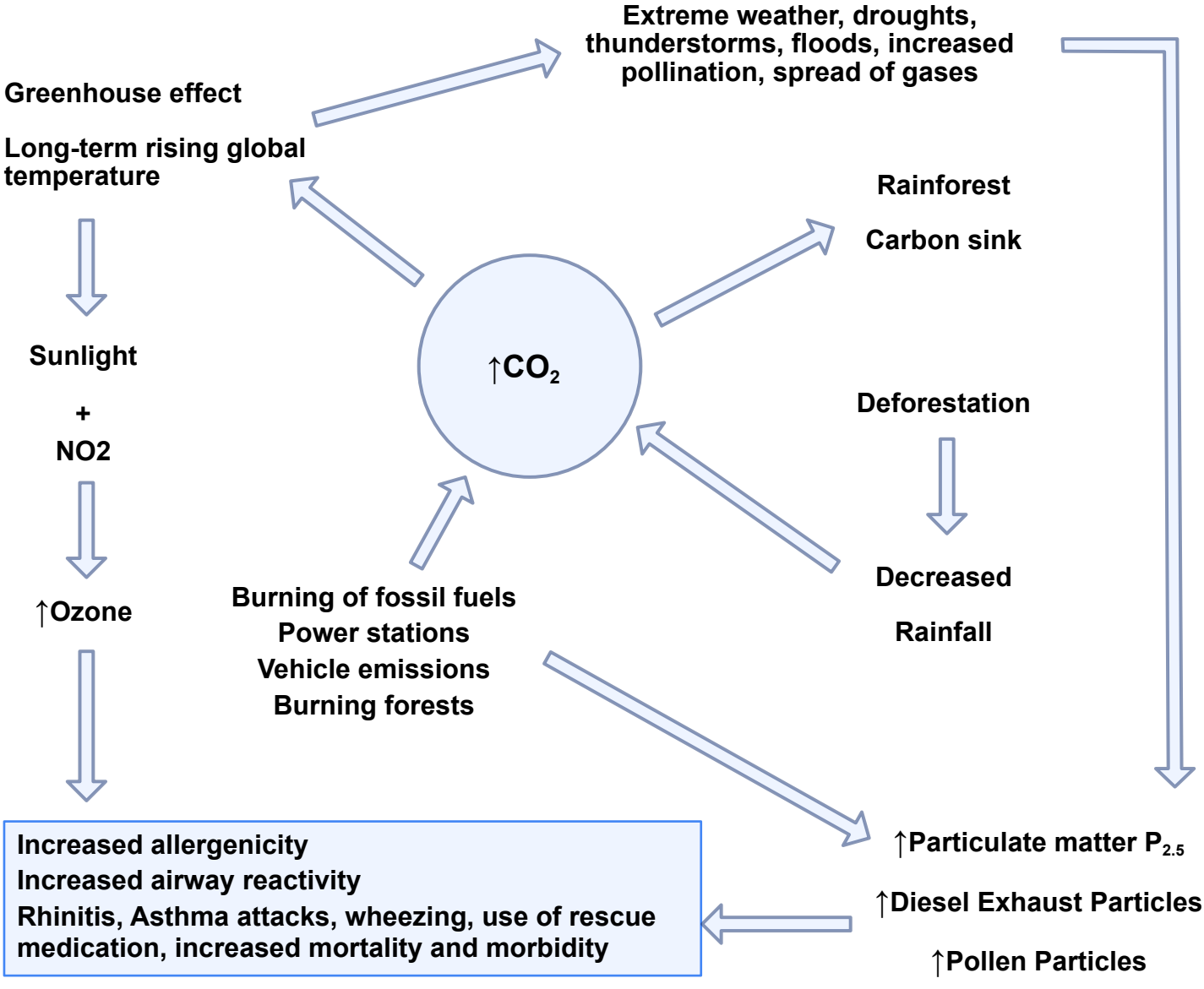
There is also an overlap between indoor and outdoor pollutants due to air exchange between outdoor and indoor environments. Cooking or heating using solid fuels in some cultures (coal, wood) can produce fine and coarse indoor particulate matter, nitrogen dioxide, carbon monoxide and sulphur dioxide.

The immuno-modulatory changes which air pollutants exert on respiratory diseases include recruitment of neutrophils and eosinophils in airway mucosa. Nonspecific airway reactivity with increased IL-33 expression and secretion of molecules from dying cells (damage associated molecular patterns, DAMP) activates and boosts the response of the innate immune system leading to increased IL-1b and decreased IL-10 production, and enhanced response to -inhaled allergens.[20,33,34,35]

DISCUSSION

Figure 1 summarises the principal connections between various elements involved in climate change, air pollution and rhinitis.

Table 5 *Interrelationships in Climate Change*



Emerging clinical data may be used to encourage governments into further action in order to mitigate the effects of climate change. Climate mitigation is any action taken to permanently eliminate or reduce the long-term risk and hazards of climate change to human life and property. Climate adaptation refers to the ability of a system to adjust to climate change (including climate variability and extremes) in order to reduce potential damage.

CONCLUSION

In conclusion clinicians are to expect an increase in incidence and morbidity due to rhinitis and asthma as a result of climate change. Allowances should be made in terms of health budgets and time dedicated to the burden of this condition in the coming decades.

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