Childhood asthma and anthropogenic CO$_2$ emissions

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Introduction
Trends in the incidence of childhood asthma worldwide have paralleled the sharp increase in carbon dioxide (CO$_2$) emissions, over at least the last two decades. The prevalence of asthma in the United States has quadrupled over the last 20 years in part due to climate-related factors. In a report released by Harvard Medical School and the Center for Health and the Global Environment, it was noted that there was an increase in asthma incidence of 160% from 1980–1994 among preschool children. This observation was linked to the global rise in CO$_2$ emissions, which in turn affects respiratory exposure to a variety of atmospheric pollens, mold, and fungi. While asthma is associated with genetic predisposition, the changing environment and air pollution are major contributory factors in the pathogenesis of the disease, and may help explain the rapid change in the incidence of asthma over the last few decades.

Even though the actual amount of CO$_2$ in the atmosphere is minute, greenhouse gases are very effective in forming a blanket that prevents heat from escaping the earth’s atmosphere. Dr Charles Keeling’s research laboratory at Mauna Loa on the Big Island of Hawaii records the amount of CO$_2$ in the atmosphere, adjusted annually for seasonal variations. Analysis of ancient air bubbles trapped by glaciers reveals that the amount of CO$_2$ consistently varied between 200 to 300 parts per million (ppm) for over 80,000 years.

Since 1960, for the first time in the known history of the earth, CO$_2$ emissions exceeded 300 ppm. In 1980, the levels approached 350 ppm, and have been increasing relatively rapidly ever since, according to the Keeling curve. In turn, global temperatures fluctuate in a pattern that is closely associated with the amount of CO$_2$ in the atmosphere.

Fossil fuels and deforestation: the major anthropogenic sources of CO$_2$
Carbon dioxide emissions derive primarily from burning fossil fuel. Approximately 75% of all CO$_2$ emissions during the last 20 years resulted from the burning of fossil fuels and the rest from deforestation. CO$_2$ is described as the most important greenhouse gas by some authors and has high inertia, and long residence in the atmosphere. Two of the largest sources of CO$_2$ emissions have been China and Brazil.
of respiratory toxins, including CO₂, which may provoke bronchospasm.²,³

**Childhood asthma incidence**

The parallel trends in the global incidence of asthma and the rise in CO₂ emissions are remarkable. As atmospheric CO₂ levels have risen and global temperature fluctuations have increased, so has the incidence of childhood asthma. According to one CDC-based survey, the number of children under 17 years of age with asthma increased from almost 40 to 60 per 1000 from 1980 to 1993.⁴ Globally, data collected using the international study of allergy and asthma in childhood (ISAAC) questionnaire showed that the recent incidence of childhood asthma in China had increased in 2008–2009 (n = 24,290), in three selected cities in China (Beijing, Chongqing, and Guangzhou) compared to prior reports from the same cities in the 1990s.⁷ The study also noted that the incidence of allergic rhinitis was increasing in those areas. In Phase 2 of the ISAAC study, at Spanish-speaking study sites, the prevalence of current wheezing in Brazil (25.6%) was the highest.⁵,⁹ There are regional variations of asthma prevalence in Latin America, which may be related to use of fossil fuels. The World Health Organization (WHO) estimates that 300 million people worldwide currently have asthma, including 6.2 million children in the US. While air pollution and CO₂ emissions alone cannot account for the increase in asthma observed globally, factors cited by proponents of the hygiene hypothesis, such as cleaner indoor environments, reduction in family size, early use of antibiotics and fewer infections during infancy, also cannot fully account for the atopy and asthma incidence trends. Regional variation in climate change is impacted by rainfall, urbanization, and transportation patterns and the association between climate change and health is therefore complex.¹⁰–¹³

**Pollen exposure and CO₂ levels**

Higher CO₂ levels hasten blooming of certain plants. In a 2002 study of 365 British plants, it was reported that the average first bloom had advanced by 4.5 days. One-sixth of the plants studied demonstrated advanced growth by an average of 15 days.¹⁴ This observation was also made by studies of the European olive trees in Spain. Based on projected estimates, pollen release is expected to occur earlier over the next century. As CO₂ levels hypothetically double, the pollen season for oaks will start earlier and concentrations will be 50% higher.¹⁵ Similar findings have been reported for other allergens such as mugwort and ragweed.¹⁶,¹⁷ The observations and predictions are not uniformly applicable though to all plants, as indicated by a study of Japanese cedar tree pollen, in which the authors investigated the levels of airborne pollen at eleven sites in Japan from 1987 to 1998 using a gravity sampler.¹⁸ They did not observe a trend of increasing pollen levels nor earlier pollen seasons. There are several studies that support the cause and effect relationship between increased atmospheric CO₂, and the increase in biomass and pollen release. Regional atmospheric conditions can be related to the amount reaching the airway. Airborne pollen concentrations depend on the degree of urbanization, air temperature, and wind conditions that spread pollen. In one study, urban sites demonstrated a 7-fold increase in ragweed growth, an average of 2°C increase in temperatures, and 30% higher CO₂ levels, compared to other sites. Based on this study, urban residents may be more affected by higher pollen concentrations.¹⁹,²⁰

In a study by Wayne and colleagues, ragweed was grown under regulated conditions in a greenhouse. The authors reported that stand level pollen production was 61% higher in elevated vs ambient CO₂ environments (P < 0.005), but that the size of the pollen grains was not altered. Most studies support the concept of longer pollen exposure and an earlier start to the pollen season.²¹ This has been confirmed in studies from diverse geographic regions.¹⁴,¹⁹,²⁰ One study from Switzerland, however, described a shorter pollen season in Basel, and an earlier onset of high pollen levels.²² Higher latitudes are warming at a faster rate than mid-latitudes, and the pollen season length has lengthened in proportion to the rate of warming.²³ Spore counts of molds have also mirrored this trend and the spore counts for Alternaria have increased since 1992.²⁴ The expansion of grass growth in northern areas (e.g., Denmark) is also related to CO₂-enriched environmental conditions.²⁵

**Seasonality and global temperature trends**

The Fourth Assessment Report by the United Nations commission panel on climate change stated unequivocally that climate warming is an established trend.²⁶ Global temperatures have been rising for the last 40 years. These
climate changes have altered the pollen season, increased pollen mass and led to forest fires which generate large amounts of air pollutants, compounding the adverse effect on the respiratory system.27,28

As the earth “breathes”, summer levels of CO₂ decrease slightly with the growth of CO₂-absorbing plants. In the winter, this effect is lost and levels of CO₂ emissions again increase.² Seasonal variation in the number of childhood asthma exacerbations has a wide variety of causes, including outbreaks of respiratory viruses during the winter season. It is intriguing to speculate that the rise in CO₂ emissions during the winter season may contribute to the increase in childhood winter asthma exacerbations.

Conclusion
Asthma is a complex disease and its pathogenesis has multiple causes and contributing factors. Among the non-atopic factors, the level of CO₂ emissions and its respiratory health effects are among the most important. The global health impact of a potential decline in anthropogenic CO₂ emissions, as new energy policies are enacted, may provide more evidence of the link between disease pathogenesis and CO₂ emissions.

Disclosure
The author declares no conflicts of interest in this work.

References