



Carbon dioxide overload, detected in human blood, suggests a potentially toxic atmosphere within 50 years

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Abstract

Anthropogenic activities are increasing the amount of carbon dioxide (CO₂) in the atmosphere. There is mounting experimental evidence that lifetime exposure to these increasing atmospheric CO₂ levels can negatively impact the normal physiology of organisms. However, directly assessing this in humans is very difficult. We analysed serum bicarbonate (HCO₃⁻), calcium (Ca) and phosphorus (P) levels from the U.S. National Health and Nutrition Examination Survey (NHANES) from 1999 to 2020 as indirect proxies for atmospheric CO₂ exposure. Over this period, average bicarbonate levels in this population show an increasing trend which parallels rising atmospheric CO₂ concentrations. Both Ca and P have decreased steadily over the same period. If these trends continue, blood bicarbonate values could be at the limit of the accepted healthy range in half a century, and Ca and P will be at the limit of their healthy ranges by the end of this century. Studies indicate that, after this time, elevated atmospheric carbon dioxide, leading to CO₂ accumulation in the body, has the potential to cause a range of adverse health effects. These findings highlight the urgent need for significant reductions in anthropogenic CO₂ emissions to safeguard public health.

Keywords Carbon dioxide · Serum bicarbonate · Blood chemistry · Health

Introduction

In aerobic organisms, including humans, CO₂ is produced as a by-product of cellular respiration and needs to be removed from the body as a waste product via expiration (Raven et al., 2007). CO₂ crosses the cell plasma membrane, enters the blood and almost all of it (90–95%) diffuses into the red blood cells, where it is rapidly hydrated to hydrogen (H⁺) and bicarbonate (HCO₃⁻) by the enzyme carbonic anhydrase (CA) (Arlot-Bonnemains et al., 1985;

Lan et al., 2025). HCO₃⁻ in blood is the most important means of transport for CO₂ throughout the body (Sherwood, 2013) and when that blood reaches the lungs, the reaction is reversed. HCO₃⁻ in the lungs combines with H⁺ produced by the oxygenation of deoxyhemoglobin to produce water and CO₂, which is exhaled as a waste product (Lan et al., 2025). Carbonic anhydrase thus allows a large pool of otherwise slowly reacting plasma HCO₃⁻ to be utilized in CO₂ excretion (Arlot-Bonnemains et al., 1985). A quantitative relationship exists between plasma HCO₃⁻ and levels of CO₂ in the blood whereby when CO₂ increases, so does HCO₃⁻ and vice versa. This is evident in multiple clinical situations (Martinu et al., 2003; Ueda et al., 2009). Importantly, abnormal CO₂ retention, or impaired CO₂ elimination, which is seen in diseases (Mendez et al., 2019; Palmer & Clegg, 2023), impaired ventilation, or excessive CO₂ inhalation (Robertson, 2006), can result in reduced blood pH. The body then attempts to buffer this ‘acidosis’ by various mechanisms, including increased ventilation (if possible), increased renal excretion of acid and conservation of filtered HCO₃⁻ (Sherwood, 2013), retention of calcium, phosphates and other substances (Gray et al., 1973) and nervous system stimulation to counteract the direct effects of

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pH changes on heart contractility and vasodilation (Burton, 1978; Eckenhoff & Longnecker, 1995). Calcium and phosphate (PO_4^{3-}) play important supporting roles in maintaining blood acid-base balance, working alongside the HCO_3^- buffer system. Phosphate can accept or donate hydrogen ions to help stabilize pH, and when blood becomes acidic, calcium and phosphate can be released from bone to help neutralize excess acid (Salcedo-Betancourt & Moe, 2024).

This leads to the question as to whether increases in atmospheric CO_2 pose a threat to human health. There are now many studies that review health effects in the range 600–5,000 ppm CO_2 (Azuma et al., 2018; Bierwirth, 2025; Carr et al., 2025; Jacobson et al., 2019) although there is still a paucity of studies investigating the effects of long-term exposure at relevant levels of CO_2 . As such, the U.S. National Health and Nutrition Examination Survey (NHANES) study, which recorded blood chemistry parameters, such as HCO_3^- , calcium (Ca), and phosphorus (P), from about 7,000 people every two years between 1999 and 2020, provides a unique opportunity to assess potential secular trends in human blood biochemistry that may be a result of a changing air composition.

Atmospheric carbon dioxide throughout human evolution

The ancestor of modern humans is thought to have evolved between 5 and 8 million years ago, with the first *Homo sapiens* appearing in the fossil record around 150,000 years ago (Wood, 1996). Although not precise, it appears that throughout most, if not all, of the ensuing period of human evolution, levels of CO_2 in the atmosphere remained relatively stable around 300 parts per million (ppm). These data were derived from a combination of studies of relict features including air trapped in ice cores (Barnola et al., 1987), the composition of fossil plankton (Zachos et al.,

2001) and Carbon-13 (^{13}C) content in fossil plant material (Cui et al., 2020). However, since the advent of widespread industrialisation, atmospheric CO_2 levels have exponentially increased (Fig. 1). In just the last ~50 years it has risen from <340 ppm (in 1980), to >420 ppm in 2025 (Lan et al., 2025). Atmospheric CO_2 is currently increasing at more than 2 ppm each year, largely due to humanity's activities, such as the burning of fossil fuels (Eggleton, 2012).

Temporal trend in human serum bicarbonate, calcium and phosphorus levels

As levels of atmospheric CO_2 increase, it is obvious that humans will have no option but to inhale more CO_2 . It is also known that increased blood loading with CO_2 (hypercapnia) is physiologically correlated with HCO_3^- levels (Malte & Wang, 2024), due to the hydration of CO_2 to form carbonic acid, which dissociates into H^+ and HCO_3^- . To compensate, renal mechanisms increase HCO_3^- reabsorption and generation to buffer H^+ and partially restore pH homeostasis (Alka & Casey, 2014). This potentially represents a health risk if HCO_3^- levels increase above the normal healthy range and/or the duration of increased HCO_3^- is excessive. In healthy adult humans, arterial HCO_3^- is typically between 22 and 26 mEq/L (Larkin & Zimmanck, 2015), although a recent re-evaluation suggests that 22.1–28.3 mEq/L is more appropriate for arterial blood, and up to 30 mEq/L is appropriate for venous blood (Kraut & Madias, 2018).

Although temporal population biochemical data relating to HCO_3^- are rare, a previous study (Zheutlin et al., 2014) examined data from the U.S. National Health and Nutrition Examination Survey (NHANES) from 1999 to 2012 (7 cycles) looking at the population average HCO_3^- levels in blood samples from a total of 33,546 adults (~5,000 per cycle). Between 1999 and 2012, there was an upward trend in serum HCO_3^- levels in the study population, with it increasing approximately 5%, from ~23.8 mEq/L in 1999–2000 to ~25.0 mEq/L in 2011–2012. This increase paralleled atmospheric CO_2 levels which increased by a similar proportion over the same period (from ~369 ppm to ~393 ppm) (Lan et al., 2025). The authors question whether “the increasing trend in serum bicarbonate found in our study is related to elevated ambient CO_2 and climate change”.

Methods

The overarching NHANES protocol is ethically approved by the Ethics Review Committee of the National Center for Health Statistics, which involved obtaining informed consent from all participants. The NHANES project assesses health and nutrition in a representative sample of adults and

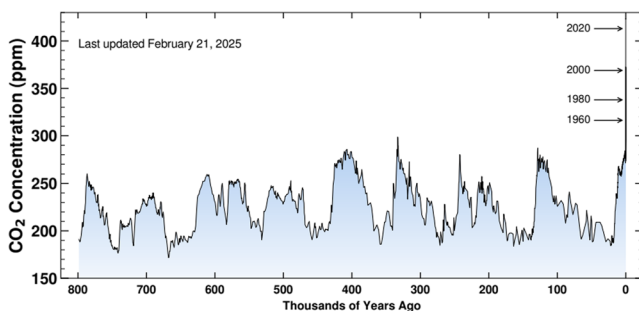


Fig. 1 Atmospheric carbon dioxide concentrations (in ppm) over the last 800,000 years, based on measurements of air trapped in Antarctic ice (Lüthi et al., 2008; Rubino et al., 2019), and direct measurements made at the Mauna Loa Observatory (1958 to present) (Keeling et al., 2005)

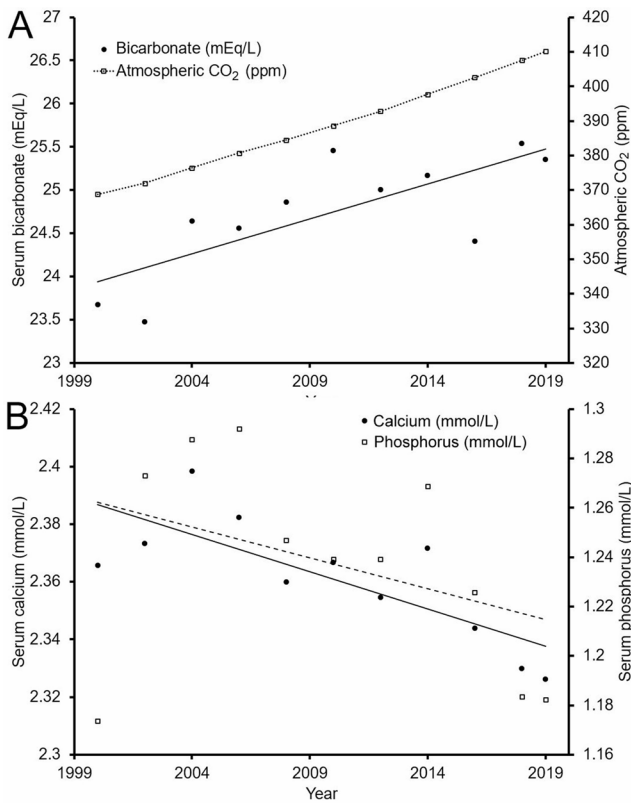


Fig. 2 (A) Comparison between temporal trend in population serum bicarbonate (filled circles, left y-axis) in U.S. adults from the NHANES biochemistry database and measured atmospheric concentration CO₂ (open boxes, dotted line, right y-axis) at Mauna Loa, Hawaii (Lan et al., 2025). The black line is the trendline for HCO₃⁻ with the formula $y = 0.081x - 138.15$. (B) Temporal trends in population serum calcium (filled triangles, solid line left y-axis) and phosphorus (open diamonds, dotted line, right y-axis) in U.S. adults from the NHANES biochemistry database over the same period of time

children in the United States via interviews, physical examinations and laboratory tests.

In the present study, we extend the 7-cycle trend analysis of Zheutlin et al. (2014) to include more recent and comprehensive NHANES data. Our analyses encompass an additional 4-cycles (up to 2019) and include all records from the NHANES database (i.e. from birth to 80+ years of age; ~7,000 records per cycle). From the database, we extracted data on levels of bicarbonate (mEq/L), calcium (mmol/L) and phosphorus (mmol/L) in participant serum. These data were averaged across all participants for each cycle, giving mean values at each time point. Atmospheric carbon dioxide levels at Mauna Loa, Hawaii over the same time period were obtained the Global Monitoring Laboratory dataset (Lan et al., 2025).

Results

Our key finding was that the increase identified by Zheutlin et al. (2014) has continued (Fig. 2). The most recent serum HCO₃⁻ levels measured in the NHANES population (which uses venous blood) was 25.3 mEq/L (in 2019–2020), representing a ~7% increase from 1999. Similarly, this has paralleled atmospheric CO₂ over the same period. As stated above, the upper healthy limit for HCO₃⁻ in venous blood can be taken as 30 mEq/L (Kraut & Madias, 2018), although this value requires further scrutiny particularly given that the high HCO₃⁻ condition would likely be perpetual in the future as atmospheric CO₂ continues to increase. Assuming a linear relationship and a ~0.34% increase rate per year, the calculated trendline in Fig. 2A predicts that the healthy maximum HCO₃⁻ level of 30 mEq/L will be reached in the year 2076. Due to the large sample sizes, population level variation in serum HCO₃⁻ levels is low (SE based on unweighted data < 0.032 for all years).

Similarly, Fig. 2B illustrates changes in serum calcium (Ca) and phosphorus (P) levels in the same population between 1999 and 2020. Mean serum Ca levels have decreased ~2% over this period, while P levels have decreased by ~7% (if the potentially spurious measurement in NHANES 1999–2000 is excluded). Total serum Ca levels for healthy adults are generally accepted to be between 2.1 and 2.6 mmol/L, with levels below this termed hypocalcemia (Bazydlo et al., 2014). For P the healthy range is 0.81 to 1.45 mmol/L (Bazydlo et al., 2014), below which hypophosphatemia occurs. Like serum HCO₃⁻, due to the large sample sizes, population level variation in both Ca and P are low (SE based on unweighted data < 0.0012 for Ca and < 0.0027 for P for all years). If we also assume linear relationships for these parameters, the calculated trendlines in Fig. 2B predict that the lower limits for Ca and P would be reached in the years 2099 and 2085 respectively. These are clearly estimations given the variability in the data. As with bicarbonate, these thresholds could be reached earlier as atmospheric CO₂ levels continue to increase.

Discussion

The trends evident in NHANES data indicate that human physiology has progressively and consistently altered over the last ~25 years. These changes are consistent with effects that might occur due to breathing increasing levels of atmospheric CO₂. Under normal circumstances CO₂ is excreted from the body via breathing with the ventilation rate determined by chemoreceptors measuring blood pH (Eckenhoff

& Longnecker, 1995). This is likely a system that has been tuned to the usually stable level of atmospheric CO₂ at around 280 ppm throughout our evolution (Fig. 1).

In situations of excess CO₂, the bones provide an early compensation mechanism by storing CO₂ as bicarbonate and carbonate (Schaefer et al., 1979a; Stumm, 2023). Additionally bone resorption may occur (Holy et al., 2012), whereby minerals such as Ca and phosphate (PO₄³⁻) are released from the bones into the blood, thereby temporarily increasing blood pH (Arnett, 2010). Studies of long-term exposure to 5,000 ppm CO₂ in guinea pigs show that blood levels of Ca and P are cyclic after CO₂ exposure (Schaefer et al., 1979a). When bone (and kidney) Ca increase, blood Ca decreases. Ca and P levels are mirrored. In chronic acidosis, however, the ability of the kidneys to handle Ca is impaired, such that there may be an increase in Ca excretion and a net loss of Ca from the body in urine (hypercalciuria) over time (Bushinsky et al., 2003). Schaefer (1982) determined that after CO₂ exposure, an initial decline in blood calcium corresponds to a decline in blood pH and marks a period of deposition of CO₂ in bones at 1.5% CO₂ (Schaefer, 1982). At 7,000 ppm CO₂, human blood levels of Ca and P were seen to rise after 20 days (Holy et al., 2012) with loss of these elements from the bones. At 1.5% CO₂, plasma calcium, mirroring the changes in pH, showed a decrease during the first 23 days of exposure and a return to initial levels during the latter part of exposure (Schaefer et al., 1963). The significant decrease in plasma calcium during this period of CO₂ retention suggests that Ca was deposited in the skeleton together with CO₂ (Schaefer et al. 1963). Thus, the effects of breathing elevated CO₂ for extended periods on serum levels of calcium and phosphorus/phosphates is complex. This is further complicated by most studies being conducted at higher CO₂ levels than the current atmosphere and involving acidic (low pH) conditions. However, a recent study provides some insight by demonstrating that, at near neutral pH, excess HCO₃⁻ ions can become incorporated in precipitating amorphous calcium carbonate (Huang et al., 2021). It appears that increasing HCO₃⁻ in the blood, as shown by the NHANES data, is involved in the exchange of H⁺ with Ca and P and the subsequent storage in the bones of CO₂ via calcium carbonate and phosphates. This is a potential reason for declining calcium and phosphate levels in the blood.

Whatever the exact mechanisms, the changes seen in human blood chemistry over the last quarter of a century, as illustrated by the NHANES data, are greatly concerning from a human health perspective. They show we may have already entered a period where there is permanent and developing physiological compensation for CO₂ in our bodies.

Health effects of long-term increases in bicarbonate and carbon dioxide

As already stated, HCO₃⁻ is a critical physiological anion that plays crucial roles in the transport of CO₂ throughout the body and in buffering pH (Sherwood, 2013). The fact that its level in the blood is increasing (at a population scale) in parallel with increasing atmospheric CO₂ is concerning. The question remains, however, whether this is really a problem, or whether human physiology will not be negatively impacted. Indeed, some authors have elegantly argued that blood acid-base compensatory mechanisms will easily be able to handle further increases in atmospheric CO₂ (Malte & Wang, 2024; Saunders & Habgood, 2023). Unfortunately, these arguments ignore the mounting evidence that even small, short-term increases in atmospheric CO₂ do indeed impact physiology in many species ranging from gastropods (Mardones et al., 2022; Navarro et al., 2022), to rodents (Kiray et al., 2014; Larcombe et al., 2021; Martrette et al., 2017; Wyrwoll et al., 2022) and humans (Allen et al., 2016; Lu et al., 2015; Satish et al., 2012; Seppänen et al., 1999). Additionally, they do not consider the impact of longer-term (i.e. life-course) exposure, or exposure during critical windows of development and the potential for such exposures to alter our physiological compensation mechanisms. To the best of our knowledge, currently there are no studies of long-term (approximating lifetime) health effects of exposure to atmospheric CO₂ in the range that is vitally important for the near future (500–800 ppm) (IPCC, 2022). However, two recent publications from our research group explore the effects of “life-time” (*in utero* to early adulthood) exposure to levels just above this (890 ppm) using a mouse model (Larcombe et al., 2021; Wyrwoll et al., 2022). We measured effects on lung structure and function, behaviour and neural expression of genes in CO₂ exposed groups.

The possible effects of increased HCO₃⁻ and CO₂ can also potentially be observed from other studies of short-term exposure to slightly higher CO₂ levels, such as those commonly experienced in indoor environments (e.g. 1,000 to 3,000 ppm CO₂) (reviewed in (Azuma et al., 2017)). For example, an older study (Eliseeva, 1964) reported marked changes in human respiration, circulation, and cerebral electrical activity at 1,000 ppm CO₂, while more recent studies of humans and animals have shown harmful effects of CO₂ exposure at these levels, such as changes in heart rate, kidney calcification, oxidative stress, neural damage and inflammation (Kiray et al., 2014; MacNaughton et al., 2016; Schaefer, 1982; Thom et al., 2017; Vehviläinen et al., 2016), reviewed in (Jacobson et al., 2019). This is important due to

many human populations spending a significant proportion of their time indoors, where CO₂ levels are typically higher. For example, The National Human Activity Pattern Survey (NHAPS) found that Americans spent an average of 87% of their time in enclosed buildings and ~6% of their time in enclosed vehicles in 1992–1994 (Klepeis et al., 2001). This has potentially increased since the COVID-19 pandemic (Young et al., 2024), with many people working-from-home in environments with poor mechanical air-ventilation (Nazarov, 2021). The proportion of time spent in indoor, CO₂ enriched environments could also contribute to the changes in blood chemistry noted in the NHANES data and is an important consideration in interpreting changes over time, and forecasts for the future.

In animals, CO₂ exposure has been found to play a role in oxidative stress caused by reactive oxygen species (ROS) (Ezraty et al., 2011; Kiray et al., 2014). For example, Ezraty et al. (2011) demonstrated that current, and slightly elevated, atmospheric CO₂ levels play a role in exacerbating oxidative stress in a bacterial model (Ezraty et al., 2011). Kiray et al. (2014) concluded that 1,000 ppm CO₂ is associated with oxidative stress and oxidative damage to brain tissue in mice (Kiray et al., 2014). ROS are produced by aerobic metabolism of molecular oxygen and play a major role in various clinical conditions including malignant diseases, diabetes, atherosclerosis, chronic inflammation and neurological disorders such as Parkinson's and Alzheimer's diseases (Waris & Ahsan, 2006). In particular, oxidative damage to cellular DNA can lead to mutations resulting in the initiation and progression of cancer.

As discussed above, increased exposure to CO₂ leads to increased blood acidity, and as part of the body's compensation mechanisms, the kidneys retain bicarbonate helping to normalise blood pH (Schaefer et al., 1979b). This clearly means that bicarbonate levels are normally intimately tied to processes that rely on acid-base balance. However, with long-term high levels of CO₂ in the blood, compensation mechanisms are no longer sufficient, metabolic acidosis occurs and the kidneys do not respond in producing bicarbonate. Under these conditions it appears that Ca²⁺ ions are mobilized to replace H⁺ ions, producing calcification (CaCO₃) of the kidneys and other body tissues such as arteries (Schaefer, 1982). Tissue calcification has been observed in the kidneys of guinea pigs and rats exposed to 1,500 ppm CO₂ for ~6 to ~15 weeks (Schaefer et al., 1979b). The effect may be driven by the over-expression of the carbonic anhydrase (CA) enzyme caused by having more CO₂ to catalyse (Phelan et al., 2021) since high CA activity is associated with calcification (Adeva-Andany et al., 2014). This is a protein malfunction that appears possible at projected future CO₂ levels given lifetime exposure. CA malfunction due to elevated CO₂ and HCO₃⁻ can also

have indirect effects on the development and progression of diseases such as cancer and diabetes (Aspatwar et al., 2021). Similarly, a recent report suggests that exposure to increasing atmospheric CO₂ “is likely to lead to proteome malfunction” – due to “protein misfolding, aggregation, charge distribution, and altered interaction with other molecules” (Duarte et al., 2020). The authors suggest that these changes could help explain the increasing prevalence of certain syndromes including diabetes and neurological disorders. In a similar vein, Kryvenko and Vadász (2021) describe how acute and chronic hypercapnia can impair endoplasmic reticulum (ER) function. The ER is a cellular organelle that serves many roles including protein synthesis and calcium storage (Voeltz et al., 2002). Elevated HCO₃⁻ and CO₂ levels are thought to cause ER stress, which alters ER protein-folding homeostasis potentially leading to tissue and organ malfunction (Kryvenko & Vadász, 2021). More research is required to understand the potential for slightly elevated atmospheric CO₂ levels to lead to these changes.

Another physiological effect of exposure to slightly elevated atmospheric CO₂ is its potential to detrimentally impact learning, cognitive abilities and mental health in humans (Allen et al., 2019; Allen et al., 2016; Satish et al., 2012; Scully et al., 2019). There is now a considerable body of published data showing impacts at levels < 1,000 ppm CO₂, although the effects of exposure remain controversial. For example, one study found no impact of exposure to levels up to 15,000 ppm (Rodeheffer et al., 2018), however the study population was a group of highly trained US Navy submariners. Conversely, studies in young adults (Satish et al., 2012), office workers (Allen et al., 2016) and university staff/students (Snow et al., 2019) showed negative effects at CO₂ levels as low as 950 ppm. Of potential importance, the study by Snow et al. (2019), found that there were no clear physiological drivers underlying the measured impacts on cognitive performance. Such studies are supported by assessment of CO₂-induced changes in human brainwaves, measured by electroencephalography (EEG) combined with cognitive tests (reviewed in (Zhang et al., 2024)). Such studies show that exposure to CO₂ between 1,000 and 2,500 ppm results in heightened brain activity. Although the mechanisms underlying these impacts are not clear, Stumm (2023) postulates that CO₂ induced increase in extracellular calcium ions may restrict the movement of sodium ions thereby reducing neuron excitability (Stumm, 2023). Another explanation may be that CO₂ signalling activates the autonomic nervous system causing stress which affects cognitive performance (Azuma et al., 2018). Brain activity holds paramount importance for human functioning, acting as the central regulator that coordinates various bodily functions and cognitive processes (Zhang et al., 2024). Given that CO₂ exposure at moderate levels appears to prove

detrimental to brain activity, additional research is urgently required to assess whether life-time exposure to CO₂ levels predicted by climate change modelling will have significant impacts on human cognitive function.

Carbon dioxide is also known to cause anxiety and panic attacks in humans (Battaglia, 2017). CO₂ sensitivity is one of the most basic and general alarm/avoidance systems within the realm of biology. While panic and anxiety attacks generally occur at high levels of CO₂, the distribution of liability to CO₂ sensitivity is continuous and normally distributed in humans and animals. This means that there are potentially small anxiety increases even at the current and near-future elevated levels of atmospheric CO₂. To this effect, increased hormones associated with anxiety have been observed in mammals at levels of CO₂ in the range 700–1000 ppm (Kiray et al., 2014; Martrette et al., 2017; Wyrwoll et al., 2022). Even a small permanent increase in global human anxiety could have a dangerous impact on societies being associated with greater fear, mental disturbance, conflicts, etc.

Health effects of long-term decreases in calcium and phosphorus

Both calcium and phosphorus are essential for human health, with insufficient levels (hypocalcemia and hypophosphatemia) being associated with a range of adverse health impacts (Gaasbeek & Meinders, 2005; Pepe et al., 2020).

Calcium is essential to maintaining total body health, with low levels potentially impacting virtually any organ or system (Pepe et al., 2020). For example, calcium is essential in muscle contraction, oocyte activation, building strong bones and teeth, blood clotting, nerve impulse, transmission, regulating heart-beat and fluid balance within cells (Pravina et al., 2013). The requirements are greatest during periods of growth such as childhood. Low blood calcium (hypocalcemia) produces symptoms including muscle cramps, lethargy, numbness and tingling in the fingers, and problems with heart rhythm. Systemic Ca²⁺ is regarded as a hormone itself, as it can modulate the function of the parathyroid gland, the thyroid gland, the kidney, and other organs and cells via the calcium-sensing receptor (Proudfoot, 2019).

Similarly, phosphorus plays many critical roles in the body (Gaasbeek & Meinders, 2005). These include metabolic processes within the cell such as energy metabolism and protein phosphorylation which are key mechanisms that control many cellular functions, including metabolism, growth, and muscle contraction (Shaker & Deftos, 2023). It is a vital component of adenosine triphosphate (ATP), the body's primary energy carrying molecule (Bonora et al., 2012), and 2,3-diphosphoglycerate (2,3-DPG), which

is found in erythrocytes and is crucial in oxygen transport (Macdonald, 1977). Accordingly, abnormally low levels of phosphate can lead to tissue hypoxia and cellular function disruption (Gaasbeek & Meinders, 2005). Phosphorus also plays a role in nucleotide metabolism which is used to build DNA, RNA and it influences phospholipid metabolism that forms a vital part of cell growth and function (Shaker & Deftos, 2023).

Conclusions

The analysis of average blood HCO₃[−] levels in a large human population shows an increase between the years 2000 and 2020, while both calcium and phosphorus show a decrease over the same period. The increase in average blood HCO₃[−] (~0.34% per year) is comparable to the increase in atmospheric CO₂ levels (~0.5% per year). This trend suggests there may be a causal link between ambient CO₂ and systemic bicarbonate levels, a finding that warrants additional attention. Trend analysis suggests that HCO₃[−] levels will be at the currently accepted limit of the healthy range within 50 years, with calcium and phosphorus reaching the currently accepted minimum levels shortly after. Clearly these are estimations, as there are uncertainties due to factors such as variations in the numbers of participants, their environment leading up to blood sampling and differences in measurement procedures during each collection. Also, the relationships may not be linear. However, on the face of it, this raw analysis of biochemical data suggests the distinct possibility that, within a half century from now, HCO₃[−] levels in human blood will reach unhealthy levels. What effects this may have on physiology remain to be elucidated, but urgently need to be considered.

Given that the entire evolution period for humans has seen a stable and relatively low atmospheric CO₂ level (<300 ppm), it's possible that our physiology is finely tuned for a range of CO₂ that will not be much greater than this level. As the atmospheric CO₂ levels rise, already at 420 ppm, the increasing levels of bicarbonate, and decreasing levels of calcium and phosphorus in our blood represent permanent and growing changes in human blood chemistry. These changes can be explained by CO₂ retention and overload in the body. The extra CO₂ being inhaled isn't expelled via increased ventilation, as ventilation rate is controlled by pH which has likely remained stable due to acid-base regulation. CO₂ storage in the body then becomes a major issue presenting a risk to population health and an existential threat for many species especially given that the rise in atmospheric CO₂ may be a phenomenon that has significant momentum. Realistically, to mitigate this approaching

threat, the alarm needs to be raised immediately. Unfortunately, currently there is little awareness or inclination for action on this issue.

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Declarations

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Consent to participate Not applicable.

Consent to publish Not applicable.

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