
**BEFORE THE PUBLIC UTILITIES COMMISSION
OF THE STATE OF SOUTH DAKOTA**

**IN THE MATTER OF THE APPLICATION
OF NAVIGATOR HEARTLAND
GREENWAY, LLC FOR A PERMIT UNDER
THE SOUTH DAKOTA ENERGY
CONVERSION AND TRANSMISSION
FACILITIES ACT TO CONSTRUCT THE
HEARTLAND GREENWAY PIPELINE IN
SOUTH DAKOTA**

HP22-002

**DR. TED SCHESSLER
INITIAL PRE-FILED TESTIMONY
IN SUPPORT OF LANDOWNER
INTERVENORS**

Q: Please state your name and purpose for providing testimony in these proceedings.

A: My name is Dr. Ted Schettler. The purpose of my testimony is to provide the PUC information helpful when considering the current proposed carbon capture pipeline of applicant Navigator. Specifically, my research and opinions relate to the “technology” proposed CO2 pipeline companies claim to possess and their claim that approval is necessary to reduce carbon emission and such a reduction is therefore a public benefit.

Q: What experience, education, training, or background qualify you to provide opinions and your concerns as you have herein?

A: Please see a summary of my education and experience in **Attachment No. 1**.

Q: Does Attachment No. 2 to your testimony include research you have done related to CO2 and your opinions and concerns that you want the PUC to be aware of relative to the proposed CO2 pipeline in question?

A: Yes, it does. It is a Fact Sheet I prepared regarding hazards and risks of CO2 exposure. I stand by my positions and opinions discussed and am competent to testify about them as necessary.

I urge the PUC to carefully consider this testimony during the Hearing in this matter and in your deliberations. I further reserve the right to amend or modify

these opinions upon presentation of any additional information that may justify such a change.

/s/ Dr. Ted Schettler, MD, MPH

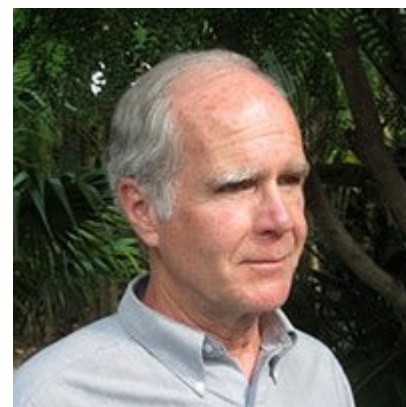
Dr. Ted Schettler, MD, MPH

Ted Schettler, MD, MPH

Science Director, Science and Environmental Health Network

Ted, SEHN's science director, received his MD from Case-Western Reserve University and a master's degree in public health from the Harvard School of Public Health. He practiced medicine for many years in New England.

Ted has worked extensively with community groups and non-governmental organizations throughout the US and internationally, addressing many aspects of human health and the environment. He has served on advisory committees of the US EPA and National Academy of Sciences.



Ted is co-author of *Generations at Risk: Reproductive Health and the Environment*, which examines reproductive and developmental health effects of exposure to a variety of environmental toxicants. He is also co-author of *In Harm's Way: Toxic Threats to Child Development*, which discusses the impact of environmental exposures on neurological development in children, and *Environmental Threats to Healthy Aging: With a Closer Look at Alzheimer's and Parkinson's Diseases*. Ted has published numerous articles in the medical literature, and is frequently quoted in the popular press.

Among many others, Ted's current projects include serving as science director for the Collaborative on Health and Environment (CHE) and active participation in the Health Care Without Harm coalition, contributing to its international campaign to improve the environmental performance of hospitals and other healthcare institutions. Ted works with colleagues from other organizations and maintains an intensive public speaking schedule, giving frequent talks on environmental health, ecological health, and the precautionary principle.



SCIENCE
& ENVIRONMENTAL
HEALTH
NETWORK



CARBON DIOXIDE (CO₂)

NORMAL PHYSIOLOGY & HAZARDS AND RISKS

by Ted Schettler, MD, MPH, Science Director, Science and Environmental Network (SEHN)

CO₂ IN NORMAL PHYSIOLOGY

In humans, carbon dioxide (CO₂) is produced in cells as a byproduct of metabolism.¹ Then it circulates through the bloodstream, mostly within red blood cells, to be exhaled from the lungs as CO₂ and excreted from kidneys as bicarbonate (HCO₃⁻).

CO₂ levels:²

- play a major role in regulating blood and tissue pH (acid-base balance)
- largely determine respiratory drive (rate and depth of breathing)
- influence oxygen attachment to hemoglobin

Internal physiologic processes tightly regulate CO₂ levels in the blood; abnormally high concentrations cause a variety of signs and symptoms. (See CO₂ toxicity below.)

Maintaining blood pH within a narrow range is essential for normal functioning of virtually all organs and physiologic systems. CO₂ plays a central role via these reactions:



where H₂O is water, H₂CO₃ is carbonic acid; HCO₃⁻ is bicarbonate; H⁺ is hydrogen ion.

Humans and virtually every living organism (plants and animals) have evolved to produce an enzyme, carbonic anhydrase³, which dramatically increases the speed of the first part of the above reaction, (CO₂ + H₂O ↔ H₂CO₃), making this an ideal system for very rapidly fine-tuning the regulation of CO₂ levels and blood pH through changes in the rate and depth of respiration.

CO₂ also reacts with water to form carbonic acid in a CO₂ pipeline, even in the absence of carbonic anhydrase, albeit much more slowly. Carbonic acid is highly corrosive to carbon steel.

The main drivers of respiratory rate and depth are normally bloodstream CO₂ levels and associated pH changes. These are maintained within healthy limits by chemoreceptors located in the medulla at the base of the brain (central chemoreceptors) and in the aorta and carotid arteries (peripheral chemoreceptors). Brain receptors detect changes in the CO₂ and pH, aorta receptors detect changes in CO₂ and oxygen, and carotid receptors detect changes in all three. Respiratory rate and depth increase primarily in response to an increase in CO₂/decrease in pH to reduce CO₂ and maintain pH within normal limits.

CO₂: AN ASPHYXIANT AND TOXICANT

CO₂ is colorless, odorless, non-flammable, and heavier than air. As a gas in air, concentrations will be higher near the floor or ground. It can be compressed at high pressures into a liquid or frozen at very low temperatures into a solid (dry ice).

CO₂ is classified as a hazardous substance by the Occupational Safety and Health Administration (OSHA) and National Institute for Occupational Safety and Health (NIOSH)⁴

Workplace exposure limits:

OSHA: Airborne permissible exposure limit (PEL) is 5000 ppm (0.5%) averaged over 8 hours.

NIOSH: Recommended airborne exposure limit (REL) is 5000 ppm averaged over 10 hours and not to exceed 30,000 ppm (3%) over any 15 minute period.

Ambient air contains about 0.04% (400 ppm) CO₂, 20% O₂, 79% nitrogen, and small amounts of other gases.

This air level of CO₂ has no direct adverse health effects in humans. As a potent greenhouse gas, however, this steadily-increasing atmospheric concentration is a major driver of climate change, which has wide-ranging direct and indirect health impacts globally.

Inhalation exposures to concentrations of CO₂ higher than OSHA and NIOSH exposure limits are hazardous in two ways, both of which contribute to signs and symptoms (see also table next page):

- Gaseous CO₂ is heavier than air and when released in concentrated amounts it flows downhill, collects in low-lying areas—indoors or outdoors—or confined, poorly-ventilated spaces such as basements, storage tanks, dry-ice refrigeration systems, fermentation areas, or mines. It displaces ambient air so that oxygen levels can fall to dangerously low levels. For that reason, CO₂ is well-known as an asphyxiant.

- CO₂ also has toxic properties in addition to causing oxygen-deprivation. Inhalation of high concentrations of CO₂ can sharply lower the pH of blood and tissues (acidosis) causing acute effects on the respiratory, cardiovascular, and central nervous systems.

The response to excessive CO₂ inhalation varies even among healthy individuals⁵ so that the timing of onset of signs and symptoms of toxicity can vary from one person to another. This may be due to underlying health status, age, or variability in chemoreceptor sensitivity and respiratory response to acute CO₂ exposures.

Symptoms depend in large part on the concentration of inhaled CO₂ and the length of time a person is exposed. Since CO₂ is odorless and does not cause irritation, unlike some other gases, it gives no warning and people may be unaware of excessive exposures until they experience troubling, dangerous symptoms.

CO₂ TOXICITY⁶

CO ₂ Concentration	Health Effect	Timing
2% (20,000 ppm)	Respiratory center stimulated causing increases in breathing (tidal) volume	Rapid
4% (40,000 ppm)	Increase in breathing rate becomes distressing; development of respiratory acidosis	Immediately dangerous to life and health (IDLH) [NIOSH] *
5-10%	Dimmed sight, sweating, tremor, increased heart rate and blood pressure; can → unconsciousness**	Within a few minutes
More than 10%	Can cause convulsions; coma (less than one minute)	Death within 10 minutes
20-30%	Loss of consciousness; death	Within one minute

* NIOSH considers this level to be immediately dangerous to life and health because it can cause confusion and impair ability to respond and get to safety. Signs and symptoms resulting from low to moderate exposures are generally reversible when a person is removed from a high CO₂ environment.

** Symptoms are due to a combination of lower oxygen levels and CO₂ toxicity.⁷

REFERENCES

1. *Krebs Cycle*. Redwood City, Calif.: Course Hero, Inc., undated; <https://bit.ly/3NfXhzh>
2. G.J. Arthurs and M. Sudhakar, "Carbon dioxide transport," *Continuing Education in Anaesthesia Critical Care & Pain* Vol. 5, No. 6 (Dec. 2005), pgs. 207-210; <https://bit.ly/37V9qto>
3. At least seven families of carbonic anhydrase enzymes have been identified, coded by distinct gene families, and at least one family is present in virtually every living organism. See Rossana Occhipinti and Walter F. Boron, "Role of Carbonic Anhydrases and Inhibitors in Acid-Base Physiology: Insights from Mathematical Modeling," *International Journal of Molecular Sciences* Vol. 20 (2019), 30 pgs. <http://bit.ly/3JEk2us>
4. "Carbon Dioxide," in *NIOSH Pocket Guide to Chemical Hazards* (Atlanta, Georgia: Centers for Disease Control and Prevention, Oct. 30, 2019); <https://bit.ly/3tyCGON>
5. Matthew Gill and others, "Effects of elevated oxygen and carbon dioxide partial pressures on respiratory function and cognitive performance," *Journal of Applied Physiology* Vol. 117, No. 4 (Aug. 15, 2014), pgs. 406-412; <https://bit.ly/3qw4HEL>
6. Wouter ter Burg and Peter M.J. Bos, *Evaluation of the acute toxicity of CO2*. (Bilthoven, The Netherlands: National Institute for Public Health and the Environment [RIVM], July, 2009); <https://bit.ly/3txCj76>. And: Kris Permentier and others, "Carbon dioxide poisoning: a literature review of an often forgotten cause of intoxication in the emergency department," *International Journal of Emergency Medicine* Vol. 10, No. 14 (2017), 4 pgs.; <https://bit.ly/3umxqNv>. And: Food Safety and Inspection Service Environmental, Safety and Health Group, *Carbon Dioxide Health Hazard Information Sheet [ESGH-Health-02.00]* (Washington, D.C.: U.S. Department of Agriculture, no date; retrieved Mar. 23, 2022.); <https://bit.ly/2VCrjqC> And: "Carbon Dioxide," in *NIOSH Pocket Guide to Chemical Hazards* (Atlanta, Georgia: Centers for Disease Control and Prevention, Oct. 30, 2019); <https://bit.ly/3tyCGON>
7. Richard E. Fairfax, "Clarification of OSHA's requirement for breathing air to have at least 19.5 percent oxygen content" in correspondence to William Costello April 2, 2007. Washington, D.C.: Occupational Safety and Health Administration, United States Department of Labor, April 2, 2007; <https://bit.ly/3lsWoQo>