BEFORE THE PUBLIC UTILTIES COMMISSION
OF THE STATE OF SOUTH DAKOTA

IN THE MATTER OF THE APPLICATION OF SCS CARBON TRANSPORT LLC FOR A PERMIT TO CONSTRUCT A CARBON DIOXIDE PIPELINE.	HP22-001

SURREBUTTAL TESTIMONY OF

MICHAEL LUMPKIN

ON BEHALF OF

SCS CARBON TRANSPORT LLC

SCS EXHIBIT #___

August 31, 2023

I. INTRODUCTION AND QUALIFICATIONS

Q. Please state your name, present position and business address.

A. My name is Michael Harrison Lumpkin, PhD, DABT. I am a Senior Toxicologist with CTEH, LLC ("CTEH"). My business address is 350 Indiana Street, Suite 700, Golden, CO 80401.

Q. On whose behalf are you providing testimony in this docket?

A. SCS Carbon Transport LLC ("SCS").

Q. How are you associated with SCS?

A. I have been engaged to provide consulting services and expertise to SCS regarding toxicology and human health risks related to acute carbon dioxide exposures that could potentially occur in the unlikely event of a carbon dioxide pipeline release.

Q. Please describe your educational and professional background.

A. I earned a Bachelor of Science degree in biochemistry from the University of Georgia in 1994, and a Doctor of Philosophy degree in toxicology from the University of Georgia in 2002. I am board-certified in general toxicology by the American Board of Toxicology. I have practiced toxicology for the past 20 years as a consultant and federal government contractor. I have extensive experience in dose reconstruction, chemical dose-response assessment, physiologically-based pharmacokinetic ("PBPK") modeling, chemical emergency response, and product stewardship and safety assessments. I have developed, critiqued and applied PBPK models for multiple government agencies, including the U.S. Environmental Protection Agency ("USEPA"), U.S. Centers for Disease Control and Prevention ("CDC") and U.S. Department of Defense ("DOD") and have co-

authored numerous peer-reviewed hazard assessments for USEPA and the Agency for Toxic Substances and Disease Registry ("ATSDR").

Since 2014, I have been employed by CTEH, where I have practiced consulting toxicology, human risk assessment and risk communication. I regularly provide health risk communication and training to emergency response managers at the federal, state, and local levels, first responders, national guard responders, and local communities concerned with chemical releases or facility fence-line exposures. I also lead CTEH chemical emergency response teams to collect environmental data and recommend health protective measures to first responders, incident commanders, response workers, and the public during and following chemical incidents. I have served as a responding toxicologist to numerous pipeline releases of various substances. I was the lead toxicologist at CTEH responding to the CO_2 pipeline release in 2020 near Satartia, Mississippi. A copy of my resume is included as Exhibit 1.

Q. Have you previously provided testimony in this proceeding?

A. No, I have not.

Q. What is the purpose of your surrebuttal testimony?

A. The purpose of my testimony is to respond to issues raised in testimony, including the direct testimonies of Landowner Intervenor witnesses Dr. Ted Schettler, Ms. Sheri Deal-Tyne, Ms. Carolyn Raffensperger, Mr. DeEmerris "Debrae" Burns, and Mr. Dan Zegart by providing information regarding the toxicological effects of acute (meaning short-term) CO_2 exposures at varying concentrations higher than typically found in ambient air. My surrebuttal provides information regarding the airborne concentrations to which first responders and the general public may be exposed over a period of minutes to a few hours

without likelihood of adverse health effects that would result in inability to egress from the area or the development of transient or permanent toxic injury.

II. HUMAN HEALTH EFFECTS AND CO_2 EXPOSURE

Q. Numerous witnesses raise concern regarding adverse health effects related to potential CO_2 exposure from a release on the proposed pipeline. Can you provide some context for how CO_2, a gas we all exhale every day, has the potential for adverse health effects?

A. While normal inhalation and exhalation of CO_2 is part of regular bodily function, at very high levels, CO_2 can be toxic to humans. Toxicity is the ability of a chemical to cause harm to biological tissues, organ systems, or individual organisms. It is widely accepted by toxicologists that there exists some dose or exposure level at which a substance changes from harmless to harmful, to an organism. This harm may range from relatively mild and reversible to lethal. This is true for substances ranging from water (a requisite for all living organisms) to botulinum toxin (one of the most potent known neurotoxins).

Q. How does the human body respond to inhalation of a gas such as oxygen or CO_2?

A. Changes in the human body's physiological or biochemical conditions due to inhalation of oxygen, CO_2, or other gases include a well-ordered sequence of biochemical adjustments. These adjustments are intended to establish or re-establish optimum use of energy sources so that bodily functions required for survival are maintained. In the case of normal CO_2 production within the body or inhalation of increasing CO_2 concentrations from ambient air, the body responds with neurological messaging that prompts shifts in breathing rates, blood pressure, and proportional blood flow to critical organ systems in an attempt to re-balance the delivery of oxygen and the removal of waste CO_2 from cells. This re-balancing

process, called physiological compensation, occurs for many processes in the body in addition to oxygen and CO_2 transport and use. However, there are limitations to the body's ability to compensate, after which further perturbations of oxygen or CO_2 inhalation and CO_2 elimination create conditions in blood and tissues that may not be conducive to normal function.

Q. Can you explain the relationship between ambient oxygen concentration, high concentration acute carbon dioxide exposure, and human health effects?

A. Yes. Normal ambient oxygen concentration is 20.9% of air, with the balance consisting primarily of nitrogen, water vapor, trace gases, and other gases and particulates present due to local geography or pollution conditions. Oxygen concentration needed for normal body function is at least 19.5% of inhaled air. As oxygen levels fall below 19.5%, physiological compensation results in higher breathing rates and higher cardiac output through increased pulse rate. However, as oxygen levels drop further, decreased physical coordination and impaired mental acuity increase. At oxygen levels of 6% to 10%, nausea, vomiting, and increasing lethargy increases markedly to the point of unconsciousness. Oxygen levels of less than 6% will result in cessation of breathing, convulsions, cardiac arrest, and death.

An increase of CO_2 in ambient air does not result in a 1-to-1 reduction of oxygen. CO_2 is heavier than oxygen and will physically displace oxygen depending on CO_2 concentration. However, CO_2 also displaces the other gases in ambient air to a greater or lesser extent than it displaces oxygen. For example, assuming minimal water vapor and trace gas content in air, 100,000 ppm CO_2 (a 10% CO_2 atmosphere) would result in an approximate oxygen level of 18.8% under standard conditions of temperature and pressure. A CO_2 atmosphere

of 300,000 ppm (30%) would result in an approximate oxygen level of 14.6% under standard conditions.

In the body, CO_2 is a natural byproduct (along with water) formed by the complete metabolism of glucose, the energy source of human cells. Due to differences in partial pressures of oxygen and CO_2 in the cells and blood, and again in the blood and alveolar sacs of the lungs, waste CO_2 is exchanged for inhaled oxygen in the lungs' alveoli, where it is then exhaled. The presence of CO_2 in blood can result in lowering of blood pH from its typical neutral value of 7.4 to more acidic levels (i.e., less than 7.0). The blood has an active buffering system which functions to maintain a neutral blood pH. However, excessively high levels of an acidic compound in blood can result in a continuous acidic condition called acidosis. Blood acidosis triggers neurological centers in the brain to increase the ability of the lungs to eliminate CO_2 via exhalation. Results of these changes in respiratory properties include changes in blood pressure, increased respiratory rate, and increased heart rate. All these changes act together in an effort to transport more CO_2 from the cells, through the circulatory system, and to the lungs where it may leave the lung blood to the lung air spaces, resulting in a net reduction of blood CO_2 and a return toward neutral blood pH.

Q. Are there published scientific studies regarding the potential health effects of high concentration acute CO_2 exposure?

A. Yes, numerous case reports and studies of volunteers and lab animals contain data on the effect of high concentration acute (up to a few hours) CO_2 exposures. Many of these published studies were conducted in the early to mid-1900's. Several of these studies are limited by details regarding the actual CO_2 and oxygen composition of the test atmospheres

(in the case of experimental studies) or presence of other unmeasured toxic gases (in the case of occupational case reports of injuries and fatalities). Further, these studies reported rather inconsistent results, with some studies suggesting loss of coordination, dizziness and headaches following exposures of 5,000 to 10,000 ppm while others reported no ill effects from acute and longer duration exposures up to 30,000 ppm. Similarly, some reported exposures at or more than 100,000 ppm indicated lethargy and transient loss of consciousness, while others reported death at these same concentrations.

While some study authors have suggested that possible blood acidosis may have led to disruption of electrolyte balance (particularly potassium levels in blood), the details in reporting are lacking to verify this impact. Many authors have opined that the observed effects described in their respective studies are due to "CO_2 toxicity"; however, it is difficult to determine if the observed effects were a result of oxygen deficiency, actual overwhelming of the subjects' blood buffering capabilities, or neurological stimulation of cardiopulmonary changes resulting in secondary effects on mental acuity, coordination, or headaches.

These older studies inform the current National Institute for Occupational Safety and Health ("NIOSH") Immediately Dangerous to Life and Health ("IDLH") limit of 40,000 ppm for healthy people. A NIOSH IDLH limit is an air concentration at or below which healthy workers may be exposed for 60 minutes without risk of permanent harm to health or ability to escape.

Q. Are there more recent studies of the potential health effects of high concentration acute CO_2 exposure?

A. Yes. The most detailed and recent study on the subject is by van der Schrier, et al. (2022) (the "van der Schrier Study"). The study authors reported CO_2 exposures from 60,000 to 120,000 ppm (or 6% to 12% CO_2 atmosphere) to healthy male volunteers. The volunteers were exposed for up to one hour. The inspired oxygen levels ranged from 19.7% down to 18.4% as CO_2 exposure levels increased. In the same study publication, rats were exposed from 100,000 to 500,000 ppm CO_2 for up to one hour. At the conclusion of the rat exposures or at the time of death, rats were necropsied for examination of organs, including the lungs. The blood pH values from the human and rat data were used together to develop a mathematical model that could translate (or predict) human and rat blood pH changes over time given various CO_2 inhalation exposures. This is the first time such an intraspecies model of CO_2-induced blood pH changes has been reported.

The human and rat data were complimentary, moving from sub-100,000 ppm exposures to up to 500,000 ppm exposures. The human subjects showed high tolerability of 60-minute exposures to up to 75,000 ppm and 10-minutes at 90,000 ppm. Blood pH levels decreased over time at all exposure levels but reached an equilibrium of no less than 7.2 at exposure levels up to 90,000 ppm (blood pH has a typical neutral value of 7.4). Transient changes to cardiac parameters and mental acuity were reported, indicating physiological compensation to increase blood elimination of CO_2. Exposures to 100,000 and 120,000 ppm were stopped early due to subject irritability, anxiety, or loss of consciousness. However, all subjects completely recovered in all aspects within minutes of exposure cessation.

Q. What conclusions do you draw from the van der Schrier study?

A. Prior to the publication of the van der Schrier Study, it was difficult to use data from the earlier studies and case reports in humans to distinguish the effects caused by non-toxic physiological compensation to blood CO_2 burdens or actual toxic harm. The detailed effects reported for humans and animals in the van der Schrier Study indicate a lack of toxic effect and the ability to make escape-related decisions for exposures between 75,000 and 90,000 ppm in typical, healthy individuals.

The database for inhaled CO_2 effects at levels from 2,000 ppm to less than 100,000 ppm are often missing sufficient detail to tease out toxic versus compensatory effects in humans. However, the van der Schrier Study provides new details into acute CO_2 tolerability and toxicity for both species. The van der Schrier Study is a solid study on which to base the classification of acutely inhaled CO_2 exposures of less than 100,000 ppm as non-toxic and suggests revisiting the present NIOSH limit of 40,000 ppm for healthy people.

Q Dr. Schettler states, "people may be unaware of excessive exposures until they are experiencing troubling, dangerous symptoms." (Schletter Direct, Ex. 2.) Do you agree with this statement?

A. No. This statement does not consider the lack of evidence for danger at 40,000 ppm described in the 2022 van der Schrier Study. While Schettler's Attachment 2 contains primarily general statements, the document does not reflect that latest scientific research on this issue.

Q. Attachment 2 of Dr. Schettler's direct testimony includes a table labeled "CO_2 toxicity" (Schettler Direct, Ex. 1). Do you agree with the data presented in that table?

A. No. Consistent with the flaws in the remainder of his testimony, this table presents conclusions that do not reflect the best available scientific data. Moreover, the ranges

presented in the table are incredibly broad, and it is not credible to present summaries of potential impacts that remain consistent throughout the ranges he presents.

Q. Do you have real-world experience responding to exposures of CO_2 from CO_2 pipeline releases?

A. Yes. I was the lead toxicologist at CTEH responding to the carbon dioxide pipeline release in 2020 near Satartia, Mississippi. I arrived in Satartia on January 23, 2020, to support the incident command during a community meeting held at the First Baptist Church of Satartia. During the meeting, I briefed the community on the CTEH air monitoring data collection to date and explained the basic toxicology of acute carbon dioxide and hydrogen sulfide exposure. I answered community members' questions during and immediately following the meeting. Over the next several days, I was available to meet and answer toxicology questions of community members who came to the Satartia City Hall. Several community members, including first responders, gave account of their whereabouts and health effect experiences in the evening of January 22, 2020. Health effects described to me ranged from no noticeable effects to transient lightheadedness, dizziness, and short-term loss of consciousness.

Q. How do the health effects reported to you by persons in Satartia compare to the scientific literature on the subject?

A. The experiences conveyed to me by community members and first responders who were in Satartia on the evening of January 22, 2020, are consistent with transient effects reported in the scientific literature for acute carbon dioxide exposure. Subjective effects reported to me such as momentary lightheadedness and subsequent headache are consistent with known physiological compensation of the circulatory system to adjust cerebral blood flow

for increased elimination of carbon dioxide from the body. These same subjective effects, in my experience as a responding toxicologist, are also common in populations that are reacting to and/or evacuating from a reported chemical release incident, reflecting an individual's physiological response to a real or perceived hazardous situation.

Q. Several Landowner Intervenor witnesses provide accounts of long-term health impacts following the exposure to CO_2 in Satartia, MS (e.g., Burns, Zepata). For example, Mr. Burns reports that he continues to experience health effects because of his exposure in the Satartia release (Burns Direct, 1-2). Do you have a response to these reports of long-term health effects from CO_2 exposure?

A. The effects that Mr. Burns describes, which he attributes to his exposure to the release in Satartia, simply do not comport with the data for the effect of acute CO_2 exposure on the human body. The allegation of long-term respiratory difficulty is inconsistent with reports of short-term CO_2 exposures in humans, including at 100,000 ppm. The allegations of ongoing, subjective neurological deficits are also inconsistent with Mr. Burns' account of short-term exposure to released CO_2 and residual hydrogen sulfide (which would be less than 42 ppm in air based on laboratory analysis of the pipeline gas feed) present and smelled per Mr. Burns' testimony.

Q. Do you definitively rule out Mr. Burns's claims?

A. No. Without access to Mr. Burns's medical records, including those from the emergency room visit he made immediately following the incident, Mr. Burns's statements cannot be completely evaluated. A more detailed assessment of the association of Mr. Burns's alleged recurrent health effects and his testimony of exposure during the release incident may be made upon review of his medical records that may include diagnostic results,

physical exams, and clinical impressions of treating physicians made on the night of the incident who subsequently released Mr. Burns from care at the emergency department.

Q. In an OP-ED letter published in the Los Angeles Times, Ms. Deal-Tyne and Ms. Raffensperger assert that CO_2 pipeline rupture could result in a release of CO_2 with the potential to "kill everything in its path" (Raffensperger, Attachment No. 2 and Deal-Tyne, Attachment No. 2). Do you agree with that statement?

A. No. Ms. Raffensperger and Ms. Deal-Tyne appear to be policy-focused and against CO_2 pipelines, but there is no scientific basis for such a hyperbolic statement.

Q. In your professional opinion, is there undue risk to human health from CO_2 exposure when considering the probability of a pipeline release occurring?

A. In my professional opinion as a toxicologist and human health risk assessor, the risk of actual toxicological harm or a reduced capacity to escape is small. Based on best available science on the physiological and toxicological effects of acute carbon dioxide exposure, inhalation of at least 75,000 ppm carbon dioxide by healthy individuals is unlikely to result in harm or reduction in decision making capacity. My review of risk modeling of pipeline incidence performed for the SCS-proposed pipeline routes, which integrates air dispersion modeling for 40,000 ppm and 80,000 ppm concentration buffers, informs my opinion that there is no undue risk of adverse effects to human health from the operation of the proposed SCS pipeline system.

Q. Does this conclude your surrebuttal testimony?

A. Yes.

Dated this 31st day of August, 2023.

/s/ Michael Harrison Lumpkin

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Michael Harrison Lumpkin