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

1	I.	INTRODUCTION AND QUALIFICATIONS
2	Q.	Please state your name, present position and business address.
3	A.	My name is Michael Harrison Lumpkin, PhD, DABT. I am a Senior Toxicologist with
4		CTEH, LLC ("CTEH"). My business address is 350 Indiana Street, Suite 700, Golden,
5		CO 80401.
6	Q.	On whose behalf are you providing testimony in this docket?
7	A.	SCS Carbon Transport LLC ("SCS").
8	Q.	How are you associated with SCS?
9	А.	I have been engaged to provide consulting services and expertise to SCS regarding
10		toxicology and human health risks related to acute carbon dioxide exposures that could
11		potentially occur in the unlikely event of a carbon dioxide pipeline release.
12	Q.	Please describe your educational and professional background.
13	A.	I earned a Bachelor of Science degree in biochemistry from the University of Georgia in
14		1994, and a Doctor of Philosophy degree in toxicology from the University of Georgia in
15		2002. I am board-certified in general toxicology by the American Board of Toxicology. I
16		have practiced toxicology for the past 20 years as a consultant and federal government
17		contractor. I have extensive experience in dose reconstruction, chemical dose-response
18		assessment, physiologically-based pharmacokinetic ("PBPK") modeling, chemical
19		emergency response, and product stewardship and safety assessments. I have
20		developed, critiqued and applied PBPK models for multiple government agencies,
21		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-22

EXHIBIT A-43

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

3 Since 2014, I have been employed by CTEH, where I have practiced consulting toxicology, 4 human risk assessment and risk communication. I regularly provide health risk 5 communication and training to emergency response managers at the federal, state, and local 6 levels, first responders, national guard responders, and local communities concerned with 7 chemical releases or facility fence-line exposures. I also lead CTEH chemical emergency 8 response teams to collect environmental data and recommend health protective measures 9 to first responders, incident commanders, response workers, and the public during and 10 following chemical incidents. I have served as a responding toxicologist to numerous 11 pipeline releases of various substances. I was the lead toxicologist at CTEH responding to 12 the CO₂ pipeline release in 2020 near Satartia, Mississippi. A copy of my resume is 13 included as Exhibit 1.

14 Q. Have you previously provided testimony in this proceeding?

15 A. No, I have not.

16 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 DealTyne, Ms. Carolyn Raffensperger, Mr. DeEmerris "Debrae" Burns, and Mr. Dan Zegart by
providing information regarding the toxicological effects of acute (meaning short-term)
CO₂ 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

- 1 without likelihood of adverse health effects that would result in inability to egress from the 2 area or the development of transient or permanent toxic injury. 3 II. HUMAN HEALTH EFFECTS AND CO₂ EXPOSURE 4 Q. Numerous witnesses raise concern regarding adverse health effects related to 5 potential CO₂ exposure from a release on the proposed pipeline. Can you provide 6 some context for how CO₂, a gas we all exhale every day, has the potential for adverse 7 health effects? 8 A. While normal inhalation and exhalation of CO₂ is part of regular bodily function, at very 9 high levels, CO₂ can be toxic to humans. Toxicity is the ability of a chemical to cause harm 10 to biological tissues, organ systems, or individual organisms. It is widely accepted by 11 toxicologists that there exists some dose or exposure level at which a substance changes 12 from harmless to harmful, to an organism. This harm may range from relatively mild and 13 reversible to lethal. This is true for substances ranging from water (a requisite for all living 14 organisms) to botulinum toxin (one of the most potent known neurotoxins). 15 How does the human body respond to inhalation of a gas such as oxygen or CO₂? Q. 16 Changes in the human body's physiological or biochemical conditions due to inhalation of A. 17 oxygen, CO₂, or other gases include a well-ordered sequence of biochemical adjustments. 18 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 CO₂
 production within the body or inhalation of increasing CO₂ 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 CO₂ from cells. This re-balancing

process, called physiological compensation, occurs for many processes in the body in
addition to oxygen and CO₂ transport and use. However, there are limitations to the body's
ability to compensate, after which further perturbations of oxygen or CO₂ inhalation and
CO₂ elimination create conditions in blood and tissues that may not be conducive to normal
function.

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

8 A. Yes. Normal ambient oxygen concentration is 20.9% of air, with the balance consisting 9 primarily of nitrogen, water vapor, trace gases, and other gases and particulates present due 10 to local geography or pollution conditions. Oxygen concentration needed for normal body 11 function is at least 19.5% of inhaled air. As oxygen levels fall below 19.5%, physiological 12 compensation results in higher breathing rates and higher cardiac output through increased 13 pulse rate. However, as oxygen levels drop further, decreased physical coordination and 14 impaired mental acuity increase. At oxygen levels of 6% to 10%, nausea, vomiting, and 15 increasing lethargy increases markedly to the point of unconsciousness. Oxygen levels of 16 less than 6% will result in cessation of breathing, convulsions, cardiac arrest, and death.

An increase of CO₂ in ambient air does not result in a 1-to-1 reduction of oxygen. CO₂ is heavier than oxygen and will physically displace oxygen depending on CO₂ concentration. However, CO₂ 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 CO₂ (a 10% CO₂ atmosphere) would result in an approximate oxygen level of 18.8% under standard conditions of temperature and pressure. A CO₂ atmosphere

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

3 In the body, CO_2 is a natural byproduct (along with water) formed by the complete 4 metabolism of glucose, the energy source of human cells. Due to differences in partial 5 pressures of oxygen and CO₂ in the cells and blood, and again in the blood and alveolar 6 sacs of the lungs, waste CO₂ is exchanged for inhaled oxygen in the lungs' alveoli, where 7 it is then exhaled. The presence of CO₂ in blood can result in lowering of blood pH from 8 its typical neutral value of 7.4 to more acidic levels (i.e., less than 7.0). The blood has an 9 active buffering system which functions to maintain a neutral blood pH. However, 10 excessively high levels of an acidic compound in blood can result in a continuous acidic 11 condition called acidosis. Blood acidosis triggers neurological centers in the brain to 12 increase the ability of the lungs to eliminate CO₂ via exhalation. Results of these changes 13 in respiratory properties include changes in blood pressure, increased respiratory rate, and 14 increased heart rate. All these changes act together in an effort to transport more CO₂ from 15 the cells, through the circulatory system, and to the lungs where it may leave the lung blood 16 to the lung air spaces, resulting in a net reduction of blood CO₂ and a return toward neutral 17 blood pH.

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Q.

Are there published scientific studies regarding the potential health effects of high concentration acute CO₂ 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) CO₂ 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 CO₂ 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.

8 While some study authors have suggested that possible blood acidosis may have led to 9 disruption of electrolyte balance (particularly potassium levels in blood), the details in 10 reporting are lacking to verify this impact. Many authors have opined that the observed 11 effects described in their respective studies are due to "CO2 toxicity"; however, it is 12 difficult to determine if the observed effects were a result of oxygen deficiency, actual 13 overwhelming of the subjects' blood buffering capabilities, or neurological stimulation of 14 cardiopulmonary changes resulting in secondary effects on mental acuity, coordination, or 15 headaches.

16 These older studies inform the current National Institute for Occupational Safety and 17 Health ("NIOSH") Immediately Dangerous to Life and Health ("IDLH") limit of 40,000 18 ppm for healthy people. A NIOSH IDLH limit is an air concentration at or below which 19 healthy workers may be exposed for 60 minutes without risk of permanent harm to health 20 or ability to escape.

Q. Are there more recent studies of the potential health effects of high concentration
acute CO₂ exposure?

1	A.	Yes. The most detailed and recent study on the subject is by van der Schrier, et al. (2022)
2		(the "van der Schrier Study"). The study authors reported CO ₂ exposures from 60,000 to
3		120,000 ppm (or 6% to 12% CO ₂ atmosphere) to healthy male volunteers. The volunteers
4		were exposed for up to one hour. The inspired oxygen levels ranged from 19.7% down to
5		18.4% as CO ₂ exposure levels increased. In the same study publication, rats were exposed
6		from 100,000 to 500,000 ppm CO_2 for up to one hour. At the conclusion of the rat
7		exposures or at the time of death, rats were necropsied for examination of organs, including
8		the lungs. The blood pH values from the human and rat data were used together to develop
9		a mathematical model that could translate (or predict) human and rat blood pH changes
10		over time given various CO ₂ inhalation exposures. This is the first time such an intraspecies
11		model of CO ₂ -induced blood pH changes has been reported.
12		The human and rat data were complimentary, moving from sub-100,000 ppm exposures to
13		up to 500,000 ppm exposures. The human subjects showed high tolerability of 60-minute
14		exposures to up to 75,000 ppm and 10-minutes at 90,000 ppm. Blood pH levels decreased
15		over time at all exposure levels but reached an equilibrium of no less than 7.2 at exposure
16		levels up to 90,000 ppm (blood pH has a typical neutral value of 7.4). Transient changes
17		to cardiac parameters and mental acuity were reported, indicating physiological
18		compensation to increase blood elimination of CO ₂ . Exposures to 100,000 and 120,000
19		ppm were stopped early due to subject irritability, anxiety, or loss of consciousness.
20		However, all subjects completely recovered in all aspects within minutes of exposure
21		cessation.

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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 CO₂ 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.

7 The database for inhaled CO₂ effects at levels from 2,000 ppm to less than 100,000 ppm 8 are often missing sufficient detail to tease out toxic versus compensatory effects in humans. 9 However, the van der Schrier Study provides new details into acute CO₂ tolerability and 10 toxicity for both species. The van der Schrier Study is a solid study on which to base the 11 classification of acutely inhaled CO₂ exposures of less than 100,000 ppm as non-toxic and 12 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 "CO₂ 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 CO2 from CO2 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
	А. Q.

22 me such as momentary lightheadedness and subsequent headache are consistent with

in the scientific literature for acute carbon dioxide exposure. Subjective effects reported to

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23 known physiological compensation of the circulatory system to adjust cerebral blood flow

1		for increased elimination of carbon dioxide from the body. These same subjective effects,
2		in my experience as a responding toxicologist, are also common in populations that are
3		reacting to and/or evacuating from a reported chemical release incident, reflecting an
4		individual's physiological response to a real or perceived hazardous situation.
5	Q.	Several Landowner Intervenor witnesses provide accounts of long-term health
6		impacts following the exposure to CO ₂ in Satartia, MS (e.g., Burns, Zepata). For
7		example, Mr. Burns reports that he continues to experience health effects because of
8		his exposure in the Satartia release (Burns Direct, 1-2). Do you have a response to
9		these reports of long-term health effects from CO ₂ exposure?
10	A.	The effects that Mr. Burns describes, which he attributes to his exposure to the release in
11		Satartia, simply do not comport with the data for the effect of acute CO ₂ exposure on the
12		human body. The allegation of long-term respiratory difficulty is inconsistent with reports
13		of short-term CO ₂ exposures in humans, including at 100,000 ppm. The allegations of

14 ongoing, subjective neurological deficits are also inconsistent with Mr. Burns' account of 15 short-term exposure to released CO₂ and residual hydrogen sulfide (which would be less 16 than 42 ppm in air based on laboratory analysis of the pipeline gas feed) present and 17 smelled per Mr. Burns' testimony.

18 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.

- 1 In an OP-ED letter published in the Los Angeles Times, Ms. Deal-Tyne and Ms. **Q**. 2 Raffensperger assert that CO₂ pipeline rupture could result in a release of CO₂ with 3 the potential to "kill everything in its path" (Raffensperger, Attachment No. 2 and 4 Deal-Tyne, Attachment No. 2). Do you agree with that statement? 5 A. No. Ms. Raffensperger and Ms. Deal-Tyne appear to be policy-focused and against CO₂ 6 pipelines, but there is no scientific basis for such a hyperbolic statement. 7 **Q**. In your professional opinion, is there undue risk to human health from CO₂ exposure 8 when considering the probability of a pipeline release occurring? 9 In my professional opinion as a toxicologist and human health risk assessor, the risk of A. 10 actual toxicological harm or a reduced capacity to escape is small. Based on best available 11 science on the physiological and toxicological effects of acute carbon dioxide exposure, 12 inhalation of at least 75,000 ppm carbon dioxide by healthy individuals is unlikely to result 13 in harm or reduction in decision making capacity. My review of risk modeling of pipeline 14 incidence performed for the SCS-proposed pipeline routes, which integrates air dispersion 15 modeling for 40,000 ppm and 80,000 ppm concentration buffers, informs my opinion that 16 there is no undue risk of adverse effects to human health from the operation of the proposed 17 SCS pipeline system. 18
- 19 Q. Does this conclude your surrebuttal testimony?
- 20 A. Yes.
- 21

- 1 Dated this 31st day of August, 2023.
- 2
- 3 /s/ Michael Harrison Lumpkin
- 4
- 5 Michael Harrison Lumpkin