**UNITED STATES ENVIRONMENTAL PROTECTION AGENCY**

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| Proposed Rule, National Emission Standards for Hazardous Air Pollutants: Supplemental Notice of Proposed Rulemaking, 89 Fed. Reg. 9088 (February 9, 2023) | Docket No. EPA-HQ-OAR-2017-0015 |

# COMMENTS OF CALIFORNIA COMMUNITIES AGAINST TOXICS, SIERRA CLUB, AND EARTHJUSTICE

*Submitted via regulations.gov and e-mail on March 11, 2024 by Earthjustice*

## BACKGROUND

On July 24, 2020, EPA promulgated a final air toxics rule for Lime Manufacturing Plants ("Lime Kilns") that the agency knew to be flatly unlawful. Although the Clean Air Act unambiguously requires § 112-compliant limits for each hazardous air pollutant that a source category emits and EPA knew that lime kilns emit hazardous air pollutants for which its existing § 112 standards provide no emission limits, EPA refused to promulgate the statutorily-required limits. EPA sought to justify its action by arguing it "does not read CAA section 112(d)(6) as directing the Agency, as part of or in conjunction with the mandatory 8-year technology review, to develop new emission standards to address HAP or emission points for which standards were not previously promulgated." 85 Fed. Reg. 44,960, 44969 (July 24, 2020).

Approximately four months before EPA published that rule, the D.C. Circuit had already rejected the interpretation of § 112(d)(6) on which EPA purported to rely. In *Louisiana Environmental Action Network v. EPA*, 955 F.3d 1088 (D.C. Cir. 2020), the Court held "[t]he section 112(d)(6) requirement that EPA, when it undertakes its eight-year review, revise emission standards 'as necessary' means

that EPA must conform them to the basic requisites of 'emission standards' under section 112, including by setting controls on previously unaddressed hazardous air pollutants." *Id.* at 1098. Nonetheless, fully aware that its interpretation of the Clean Air Act was unlawful, EPA nonetheless chose to rely on it and, indeed, reiterate it in refusing emission limits for the uncontrolled hazardous air pollutants that Lime Kilns emit. 85 Fed. Reg. at 44969. EPA's action demonstrated contempt for the people the Clean Air Act was enacted to protect, for the Court that had clearly rejected the unlawful statutory interpretation EPA advanced, and for Congress, which enacted the statutory requirements that the agency steadfastly refused to obey for more than 20 years.

Sierra Club challenged EPA's unlawful rule in the D.C. Circuit, Sierra Club v. EPA, D.C. Cir. No. 20-1381 (filed September 22, 2020). To give EPA a chance to correct its violation of the law voluntarily, Sierra Club also filed a petition for reconsideration under Clean Air Act § 307(d)(7)(B). EPA-HQ-OAR-2017-0015-0065.

Notably, EPA promulgated the illegal 2020 air toxics rule for Lime Kilns years after the Clean Air Act required the agency to issue lawful standards, and the agency acted only after a court ordered it to do so. *See Blue Ridge Environmental Defense League v. Pruitt*, 261 F. Supp. 3d 53 (D.D.C. 2017) ("*BREDL*"). In 2021, EPA returned to the district court and admitted that its 2020 rule did not satisfy the Clean Air Act or even its obligations under the district court's order to promulgate the overdue rule in *BREDL.* Blue Ridge Environmental Defense League v. Regan, D.D.C. No. 16-364 (CRC), Joint Motion to Extend Deadlines under Court Order, April 13, 2021, Ex. A. hereto. EPA sought and obtained consent of plaintiffs and the court in *BREDL* for an extension of the deadline to give the agency a chance to bring its rule into compliance with the statute. *Id.* The deadline for EPA to issue a rule that complied with the Clean Air Act and satisfied the district court's order was extended to February 23, 2023.

Shortly, before the final rule was due, EPA sought and obtained a second extension of the deadline, to August 1, 2023, claiming its "analysis here took

longer than expected." Blue Ridge Environmental Defense League v. Regan, D.D.C. No. 16-364 (CRC), EPA's Unopposed Motion to Extend Deadline, January 23, 2023, Ex. B.

In July of the same year, EPA sought yet another extension. Requesting an entire year of further delay, EPA claimed

> Some comments challenged underlying assumptions that EPA had made when it decided, under the Regulatory Flexibility Act, that the proposal would not have "a significant economic impact on a substantial number of small entities." 5 U.S.C. § 605(b). Because EPA can no longer certify that the proposal would not have such effects, the agency now plans to convene a review panel under 5 U.S.C. § 609(b), invite public comments on an initial regulatory flexibility analysis, and issue a final regulatory flexibility analysis to accompany the final rule. *See id.* §§ 603-04.

Blue Ridge Environmental Defense League v. Regan, D.D.C. No. 16-364 (CRC), EPA's Unopposed Motion to Extend Deadline, July 20, 2023, Ex. C (emphasis added).

Despite its representations to plaintiffs and the court, EPA used the extension not merely to conduct the SBREFA analysis described in its motion but to pursue its apparent policy goal of weakening the Lime Kilns rule to benefit industry at the expense of people who are exposed to Lime Kilns' toxic emissions. EPA did not inform the court or plaintiffs or the court that it was going to propose such amendments when it obtained consent for the extension or, indeed, that it was going to use the time to anything other than conduct SBREFA analysis. Nor did EPA mention that it was engaged in proposing such amendments at any subsequent point before publishing the weakening amendments in the Federal Register.

## COMMENTS

## I. EPA WOULD VIOLATE THE CLEAN AIR ACT AND ACT ARBITRARILY BY SETTING A "HEALTH-BASED EMISSION LIMIT" FOR HYDROGEN CHLORIDE.

EPA seeks comment on a request by the Small Business Advocacy Review Panel to set a "health-based emission limit" (HBEL) for hydrogen chloride (HCl) that is even weaker than the HCl limit EPA proposed. 89 Fed. Reg. 9088, 9092 (February 9, 2024). It is worth providing some context for EPA's consideration of this request. "The mission of EPA is to protect human health and the environment." https://www.epa.gov/aboutepa/our-mission-and-what-we-do. Given this mission, one might expect EPA would be an advocate for "human health and the environment" and to represent ordinary Americans' interests in being protected from toxic pollution at least as zealously as it represents business owners' economic interests. As is plainly evidenced by EPA's biased and one-sided arguments in favor of the HBEL and EPA's complete failure to consider or even mention the reasons against issuing one that the agency articulated and successfully defended in its air toxics rule for industrial boilers, *see U.S. Sugar v. EPA*, 830 F.3d 579, 624 (D.C. Cir. 2016), the agency has abandoned or lost track of this mission.

Clean Air Act § 112(d)(4) provides that, "[w]ith respect to pollutants for which a health threshold has been established, the Administrator may consider such threshold level, with an ample margin of safety, when establishing emission standards under this subsection." 42 U.S.C. § 7412(d)(4). Nothing in the text of this provision authorizes EPA to set limits that are less stringent than those required by § 112(d)(2)-(3). To the contrary, the text of the statute indicates that Congress intended EPA to consider health thresholds, where they have been established, as a factor in making the limits that EPA promulgates under § 112(d)(2)-(3) more stringent. Indeed, by providing that thresholds be "consider[ed]" "when establishing emission standards under this subsection," *Id.* (emphasis added), the statutory text indicates that Congress wanted (d)(4) to provide for more stringent standards. Specifically, § 112(d)(2) provides that

standards "promulgated under this subsection" must meet the stringency requirements in § 112(d)(2) and (d)(3). 42 U.S.C. § 7412(d)(2). Because § 112(d)(2) requires the "maximum" reductions that are achievable "including a prohibition on emissions where achievable," reading § 112(d)(4) as authorizing more stringent standards is consistent with § 112(d)(2). Reading § 112(d)(4) as allowing standards that are less stringent than § 112(d)(2)-(3) – i.e., that do not comply with these provisions – is inconsistent with § 112(d)(4)'s directive to consider health thresholds "when establishing emission standards." *Id.*

In the past, EPA has invoked a floor statement by then Senator Durenberger as authority for reading § 112(d)(4) as allowing it to set limits that are less stringent than those required by § 112(d)(2)-(3). In its 1998 rule for pulp and paper mills, for example, EPA stated

> With respect to the pollutants for which a safe threshold can be set, the authority to set a standard less stringent than maximum achievable control technology is contained in subsection (d)(4). With respect to carcinogens and other non-threshold pollutants, no such authority exists in subsection (d) or in any other provision of the Act.

63 Fed. Reg. 18,754, 18,765 (April 15, 1998) (quoting Environment and Public Works, A Legislative History of the Clean Air Act Amendments of 1990, Vol. 1 at 876, statement of Senator Durenberger during Senate Debate of October 27, 1990).

A floor statement does not trump the statutory text. Because the text of § 112(d)(4) does not authorize EPA to set limits less stringent than those required by § 112(d)(2)-(3) – and strongly indicates that Congress intended only that EPA consider more stringent limits under these provisions – EPA cannot rely on Senator Durenberger's floor statement for authority to set limits that are less stringent than § 112(d)(2)-(3) require.

Assuming *arguendo* that § 112(d)(4) does authorize EPA to issue limits that less stringent than § 112(d)(2)-(3) require in some circumstances, doing so for HCl would still be unlawful. As EPA itself explains:

> The EPA presumptively applies section 112(d)(4) only to HAP's that are not carcinogens because Congress clearly intended that carcinogens be considered nonthreshold pollutants. … "With respect to the pollutants for which a safe threshold can be set, the authority to set a standard less stringent than maximum achievable control technology is contained in subsection (d)(4). With respect to carcinogens and other non-threshold pollutants, no such authority exists in subsection (d) or in any other provision of the Act."

63 Fed. Reg. at 18,765 (quoting Staff of the Senate Committee on Environment and Public Works, A Legislative History of the Clean Air Act Amendments of 1990, Vol. 1 at 876, statement of Senator Durenberger during Senate Debate of October 27, 1990) (emphasis added). EPA has never altered its interpretation of what "Congress clearly intended." To the contrary, EPA has reiterated it consistently over the last two decades. *See, e.g.*, 75 Fed. Reg. 32,006, 32,031 (June 4, 2010); Sierra Club v. EPA, D.C. Cir. No. 15-1487 (filed April 28, 2017) ("EPA Brick Kilns Brief), Ex. D hereto, at 23 (citing 79 Fed. Reg. 75,622, 79,639 (December 18, 2014).

Notably, the D.C. Circuit has upheld EPA's refusal to set a HBEL for HCl because, *inter alia*, it did not "consider the potential acute or carcinogenic effects that might be caused by HCl exposure." *U.S. Sugar v. EPA*, 830 F.3d 579, 624 (D.C. Cir. 2016) (citing 75 Fed. Reg. at 32,031). Conversely, where EPA sought to set a HBEL for HCl, the D.C. Circuit found EPA's action unlawful because the agency failed to carry its burden of showing with "substantial evidence" that HCl is not carcinogenic. *Sierra Club v. EPA*, 895 F.3d 1, 10-11 (D.C. Cir. 2018).

In the supplemental proposal, EPA does not provide substantial evidence that HCl is not carcinogenic or, indeed, even attempt to provide any showing at all. For this reason alone, issuing a HBEL for HCl would be unlawful and arbitrary.

Evidently seeking to evade the holding in *Sierra Club* and its own consistent interpretation of the Clean Air Act, EPA claims to "now recognize[]" that carcinogens can be "threshold pollutants." 89 Fed. Reg. at 9092 (citing EPA, Guidelines for Carcinogen Risk Assessment (2005)). Thus, EPA seeks to bypass the fact that it lacks substantial evidence to show that HCl is not a carcinogen by

now claiming it is irrelevant whether HCl is a carcinogen and the agency does not have to make any such showing.

EPA's claim to have newly "recognize[d]" that carcinogens can be threshold pollutants – based on an EPA guidance document from 2005 – is irrelevant under the Clean Air Act. Because "Congress clearly intended that carcinogens be considered nonthreshold pollutants," 63 Fed. Reg. at 18,765 (emphasis added), EPA's views on whether carcinogens can be considered threshold pollutants do not matter. The meaning of the Clean Air Act does not change just because EPA's views on whether carcinogens are threshold pollutants change. Where Congress has clearly expressed its intent, EPA's job is to effectuate that intent, not to subvert it, regardless of whether EPA's views have come to differ from the clearly expressed intent of Congress.

Further, if it existed at all, *but see supra*, EPA's purported authority to set limits under § 112(d)(4) that are less stringent than those required by § 112(d)(2)-(3) comes not from the statutory text but Senator Durenberger's floor statement. 63 Fed. Reg. at 18,765. That is the only authority EPA has ever cited for this reading of § 112(d)(4). And if that floor statement truly expressed Congress's intent that EPA can set less stringent limits under § 112(d)(4), it would equally express Congress's "clear[] inten[t] that carcinogens be considered nonthreshold pollutants." *Id.* Thus, if EPA wants to issue a HBEL for HCl it must establish with substantial evidence that HCl is not a carcinogen – as the D.C. Circuit has already made plain to EPA. *Sierra Club*, 895 F.3d at 10-11.

Remarkably, the supplemental proposal does not even mention EPA's interpretation of the statute. EPA does not acknowledge that its purported "recogni[tion]" that carcinogens can be threshold pollutants conflicts directly with its own repeated findings that Congress clearly intended that carcinogens cannot be treated as threshold pollutants. EPA does not say why it believes itself entitled to treat carcinogens as threshold pollutants, despite its understanding that Congress "clearly" did not intend such treatment of them. Nor does EPA purport to alter its statutory interpretation, let alone provide a reasoned basis for doing so. For this reason as well, EPA's action is unlawful. *See, e.g.*, *Encino Motorcars v. Navarro*, 579 U.S. 211, 221-222 (2011).

Likewise, EPA makes no attempt to reconcile its apparent current position that the lack of research into whether HCl is carcinogenic is irrelevant, 89 Fed. Reg. at 9,092, with its previous finding that the lack of conclusive research is a reason not to issue a HBEL for HCl, 75 Fed. Reg. at 32,031. *See U.S. Sugar v. EPA*, 830 F.3d at 624. Indeed, EPA does not even see fit to acknowledge the position it took in the boilers rule and the *U.S. Sugar* case, let alone provide a rational basis for departing from it.

Even if EPA were free to ignore the text of the statute, *Sierra Club*, and the agency's own long-held interpretation of the Clean Air Act and issue a HBEL for HCl without showing that HCl is not carcinogenic, setting a HBEL for HCl based on the supplemental proposal would be unlawful and arbitrary. Once again, just as in *Sierra Club*, EPA assumes it can equate absence of evidence with evidence of absence. *See Sierra Club*, 895 F.3d at 10-11. The only difference here is that EPA deploys that assumption to claim it can treat HCl as a threshold pollutant rather than to claim it can treat HCl as a non-carcinogen. *Compare* 89 Fed. Reg. 9092 *with Sierra Club*, 895 F.3d at 10-11. *See also* EPA Brick Kilns Brief at 23-25.

Having disavowed any obligation to show that HCl is not carcinogenic, EPA next claims "[a]n important consideration when determining if a carcinogen has a threshold is whether it is mutagenic." 89 Fed. Reg. at 9,092. Even assuming *arguendo* that EPA can simply bypass the need to show that a pollutant is not carcinogenic, EPA does not and cannot claim that mutagenicity is the sole test for whether a pollutant has a threshold, either for cancer of any other health harm. EPA does not discuss all the other indicators of whether a pollutant has a threshold, let alone why it has chosen not to consider them.

Further, far from providing "substantial evidence" that HCl is not mutagenic, EPA makes clear that the current evidence regarding whether HCl is mutagenic is outdated and, at best, equivocal. All EPA claims is that "[i]n the case of HCl, the available evidence does not indicate that HCl has a mutagenic effect." *Id.* That is precisely the same lack of evidence sought to rely on in *Sierra Club*, and falls far short of the required demonstration with substantial evidence that HCl is not mutagenic. *See* 895 F.3d at 10-11.

Indeed, apart from a 1992 study on "bacteria," the only studies EPA can identify indicate that HCl is mutagenic in mammals. *Id.* EPA seeks to discount these studies by claiming – without providing any citation whatsoever – that some unidentified researchers have found in unidentified research that these results were caused by exceptionally high doses of HCl. 89 Fed. Reg. at 9092. EPA's citation-free and unsubstantiated critique of the only mammalian evidence it has – evidence showing that HCl is mutagenic – scarcely amounts to a demonstration with substantial evidence that HCl is not carcinogenic. *See Sierra Club*, 895 F.3d at 10-11.

EPA's attempt to rely on "[a]nother important consideration," the mechanisms that can cause cancer, is similarly flawed. 89 Fed. Reg. at 9092. EPA states that increased cell production and tissue enlargement, known as hyperplasia, can lead to cancer. *Id.* EPA further states that an animal study has shown that HCl exposure causes hyperplasia in rats. *Id.* Then, however – just as with mutagenicity – EPA seeks to discount its own evidence by arguing that the same study did not show that HCl caused the rats to get cancer. *Id.* But it is EPA's argument that hyperplasia, as a purported "mechanism" of cancer, is relevant to whether HCl is a pollutant for which a cancer threshold exist. EPA's only evidence is that HCl does cause hyperplasia. Therefore, EPA's hyperplasia argument scarcely demonstrates with substantial evidence that HCl is not a threshold pollutant. As for EPA's claim that the study does not demonstrate HCl causes cancer, it is irrelevant. It is EPA's burden to demonstrate with substantial evidence that HCl does not cause cancer. *Sierra Club*. 895 F.3d at 10-11. EPA does not claim that its 1995 rat study provides such evidence, nor could it. *Id.*

EPA next claims to have "similarly recognized the existence of a threshold of exposure for hyperplasia and resulting cancer outcomes from exposure to chloroform. 89 Fed. Reg. at 9092-9093 The agency claims "[c]hloroform was labeled as likely to be carcinogenic to humans under high-exposure conditions that cause hyperplasia. However, the EPA concluded that chloroform is not likely to be carcinogenic to humans under exposure conditions that do not cause hyperplasia." *Id.* That argument merely underscores the extent to which EPA is defying what the agency itself describes as Congress's clear intent that carcinogens not be treated as

threshold pollutants. Chloroform is, by EPA's own finding, a probable human carcinogen. https://iris.epa.gov/ChemicalLanding/&substance_nmbr=25. EPA has never claimed chloroform is a threshold pollutant, let alone identified a threshold below which it does not cause cancer. EPA's new suggestion that it can treat chloroform as a threshold pollutant is a good example of why OMB cautioned EPA that its new approach was "precedential" and urged the agency to seek "external scientific review before considering finalizing a threshold proposal." OMB Review Draft at 20 (emphasis added).

Although EPA did not find that chloroform is a threshold pollutant, as the agency now inaccurately suggests, it did find the chloroform is a probable carcinogen based on evidence that it causes hyperplasia. https://iris.epa.gov/ChemicalLanding/&substance_nmbr=25. EPA also has evidence that HCl causes hyperplasia. 89 Fed. Reg. at 9092. Thus, EPA's new arguments about HCl are not "similar" to those underlying the chloroform finding, *id.* To the contrary, EPA discounts the finding that HCl caused hyperplasia because the study did not also show that HCl caused cancer. Nowhere does EPA explain why, if it believes hyperplasia is a mechanism for carcinogenicity sufficient to demonstrate that chloroform causes cancer even without an independent direct finding that chloroform causes cancer, it does not reach the same conclusion for HCl.

Further, EPA states only that HCl causes hyperplasia in rats. 89 Fed. Reg. at 9092. It does not claim to have identified any level below which HCl does not cause hyperplasia in rats, let alone people. Thus, even if EPA identified a threshold below which chloroform did not cause hyperplasia – which it has not – that situation would not be "similar[]," *id.*

Remarkably, the record shows EPA so eager to substitute its new views for what the agency itself has described as Congress's clear intent that it ignored cautionary comments from the Office of Management and Budget. In its review of EPA's proposal under EO12866, OMB commented

> Has EPA's approach here for HCl and under 112(d)(4) been peer reviewed by the SAB, since EPA's action here appears to be

precedential? Recommend seeking comment on whether EPA should seek such external scientific review before considering finalizing a threshold proposal.

EPA-HQ-OAR-2017-0015-0201 ("OMB Review Draft") at 20 (emphasis added). Then, after EPA apparently refused to alter its approach, seek peer review by SAB, or even seek comment on the need for "such external scientific" review, OMB requested in a second round of comments that EPA at least take comment on whether "any additional scientific evidence of this health-based threshold proposal for hyperplasia is available that would help inform EPA in their risk assessment and subsequent decision-making … and "[w]hether additional external scientific review is needed before finalizing a threshold-based proposal, since EPA's supplemental proposed action under 112(d)(4) appears, if true, to be precedent." *Id.*

Evidently, OMB's comments were futile. EPA's new approach has not been peer reviewed by SAB, and EPA made no effort to seek comment on seeking SAB review, despite OMB's identification of its new approach to the evaluation of cancer risk as "precedential." Apparently, EPA did not seek "any" additional external scientific review or evidence. In any event, EPA does not mention OMB's concerns in its proposal, let alone respond to them or identify any external review or evidence that address those concerns.

## II. EPA'S APPROACH TO CANCER RISK IS ARBITRARY AND INCONSISTENT WITH ITS OWN GUIDELINES.

As outlined in the *2005 Carcinogenic Risk Assessment Guidelines* ("Guidelines"), it has long been EPA's policy and practice to presume that genotoxic carcinogens do not have a known threshold of exposure and thus, no dose is safe.[1] A second practice is that of employing linear extrapolation for chemicals that undergo carcinogenic risk assessment and are found to act via a mutagenic mode of action, unless it can be demonstrated unequivocally that a chemical acts via a non-mutagenic mode of action.

[1] EPA, *Guidelines for Carcinogen Risk Assessment* ("Guidelines") at 1-11 (2005), https://www.epa.gov/sites/default/files/2013-09/documents/cancer_guidelines_final_3-25-05.pdf.

Indeed, the Guidelines state that "[w]hen the weight of evidence evaluation of all available data are insufficient to establish the mode of action for a tumor site and when scientifically plausible based on the available data, linear extrapolation is used as a default approach (emphasis added), because linear extrapolation generally is considered to be a health-protective approach. Nonlinear approaches generally should not be used in cases where the mode of action has not been ascertained."[2] For clarity on what is meant by 'nonlinear', footnote 3 on page 16 states "[t]he term "nonlinear" is used here in a narrower sense than its usual meaning in the field of mathematical modeling. In these cancer guidelines, the term "nonlinear" refers to threshold models (which show no response over a range of low doses that include zero) and some nonthreshold models (e.g., a quadractic model, which shows some response at all doses above zero)."[3] The Guidelines make clear that nonlinear or threshold approaches should not be employed in the absence of an established mode of action.

EPA has not met its own standard in its current analysis, in the absence of a toxicological risk assessment that evaluated HCl for its carcinogenic potential.[4] Moreover, EPA's proposal to set a health-based exposure level based on the presumption of an existing threshold is not in accordance with other federal guidelines, including the FDA *Guidance for Industry S2B Genotoxicity: A Standard Battery for Genotoxicity Testing of Pharmaceuticals*, states, "Compounds which are positive in tests that detect such kinds of damage [damage to DNA] have the potential to be human carcinogens and/or mutagens, i.e., may induce cancer

[2] Guidelines at 3-21.
[3] Guidelines at 1-11.
[4] *See* Guidelines at 3-21: "When the weight of evidence evaluation of all available data are insufficient to establish the mode of action for a tumor site and when scientifically plausible based on the available data, linear extrapolation is used as a default approach, because linear extrapolation generally is considered to be a health-protective approach. Nonlinear approaches generally should not be used in cases where the mode of action has not been ascertained." Footnote at 3 section 1-11states "The term "nonlinear" is used here in a narrower sense than its usual meaning in the field of mathematical modeling. In these cancer guidelines, the term "nonlinear" refers to threshold models (which show no response over a range of low doses that include zero) and some nonthreshold models (e.g., a quadractic model, which shows some response at all doses above zero)." Importantly, the Guidelines make clear that nonlinear or threshold approaches should not be employed in the absence of an established mode of action.

and/or heritable defects."[5] Further supporting this approach, a 2006 scientific review article by EPA staff person, Michael Cimino, reported that, "The default regulatory assumption is that chemicals that are genotoxic in standard tests can cause mutations in humans (in somatic and/or germ cells), and can contribute to adverse health outcomes, such as cancer, via a genotoxic or mutagenic mode of (toxic) action (MOA)."[6]

Notably, EPA's staff co-published a peer reviewed article describing that different carcinogens act via multiple mechanisms or MOA's and it can be challenging to determine which is the main MOA, especially when considering differences in exposure at different lifestages and at differential doses.[7] This notion is reinforced by comments submitted by Joseph Landolph, an expert and reviewer of EPA's draft Framework for Determining a Mutagenic Mode of Action for Carcinogenicity ("Framework"). While the Framework was never finalized, in response to a charge question referring to the clarity and completeness of the Framework, Landolph "strongly recommend[ed] adding that if a mutagenic chemical carcinogen does not have a conclusive mutagenic MOA, but no other evidence exists to conclusively document a non-mutagenic MOA, then this chemical is treated as acting by a mutagenic MOA for risk assessment and regulatory purposes. For risk assessment purposes, this chemical is presumed to follow a linear, no threshold dose-response curve for cancer induction."[8] Landolph is supported by subject matter expert's understanding that even when the MOA is

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[5] FDA, *Guidance for Industry S2B Genotoxicity: A Standard Battery for Genotoxicity Testing of Pharmaceuticals* at page 1 (July 1997). Available at: https://www.fda.gov/media/71971/download.

[6] Cimino MC. at 384, Comparative overview of current international strategies and guidelines for genetic toxicology testing for regulatory purposes (2006). Environ Molec Mutagen 47:362-390. Available at: https://onlinelibrary.wiley.com/doi/epdf/10.1002/em.20216.

[7] Smith, M. T., Guyton, K. Z., Gibbons, C. F., Fritz, J. M., Portier, C. J., Rusyn, I., DeMarini, D. M., Caldwell, J. C., Kavlock, R. J., Lambert, P. F., Hecht, S. S., Bucher, J. R., Stewart, B. W., Baan, R. A., Cogliano, V. J., & Straif, K. (2016). Key Characteristics of Carcinogens as a Basis for Organizing Data on Mechanisms of Carcinogenesis. *Environmental health perspectives*, *124*(6), 713–721. https://doi.org/10.1289/ehp.1509912.

[8] EPA, Framework for Determining a Mutagenic Mode of Action for Carcinogenicity (External Review Draft) at C-34 (April 4, 2008). Available at: https://archive.epa.gov/osa/mmoaframework/web/pdf/mmoa_report_final508_6-2-08.pdf.

not known (and there is not sufficient evidence to suggest otherwise, especially since it is understood that chemicals can act by non-mutagenic MOA's and still operate with no threshold.[9] To conclude, EPA's current proposal does not provide sufficient and unequivocal evidence to support its claim that HCl's does not act via a mutagenic mode of action.

## III. IN THE ABSENCE OF A CARCINOGENIC RISK EVALUATION, EPA HAS NOT PROVIDED SUBSTANTIAL EVIDENCE THAT HCL CAN BE TREATED AS A THRESHOLD POLLUTANT.

EPA's Information Risk Information System ("IRIS") database for toxicological assessments for HCl states that quantitative estimates of either carcinogenic risk from oral and/or inhalation exposure were "not assessed under the IRIS Program".[10] Indeed, while the proposed rule makes reference to the

[9] *See* Exhibits E and F, TSCA evaluation comments submitted by UCSF PRHE as well as Earthjustice comments published on docket EPA-HQ-OPPT-2023-0265-0005, which reference the National Resource Council's Science and Decisions: Advancing Risk Assessment (2009) and state: "The absence of a known mutagenic MOA is not sufficient evidence to support these statements, as carcinogens acting by other MOAs can operate with no threshold. Further, the NASEM states that human variability, exposure to other chemicals, and background disease processes alone can result in linear dose-response relationships at low doses, regardless of whether mutagenic MOAs are known: Background exposures and underlying disease processes contribute to population background risk and can lead to linearity at the population doses of concern. The current EPA practice of determining "nonlinear" MOAs does not account for mechanistic factors that can create linearity at low dose. The dose-response relationship can be linear at a low dose when an exposure contributes to an existing disease process…Effects of exposures that add to background processes and background endogenous and exogenous exposures can lack a threshold if a baseline level of dysfunction occurs without the toxicant and the toxicant adds to or augments the background process. Thus, even small doses may have a relevant biologic effect. That may be difficult to measure because of background noise in the system but may be addressed through dose-response modeling procedures. Human variability with respect to the individual thresholds for a nongenotoxic cancer mechanism can result in linear dose response relationships in the population…In the laboratory, nonlinear dose-response processes—for example, cytotoxicity, impaired immune function and tumor surveillance, DNA methylation, endocrine disruption, and modulation of cell cycles—may be found to cause cancer in test animals. However, given the high prevalence of those background processes, given cancer as an end point, and given the multitude of chemical exposures and high variability in human susceptibility, the results may still be manifested as low-dose linear dose-response relationships in the human population."

[10] EPA, Hydrogen Chloride Cancer Assessment (n.d). Available at: https://iris.epa.gov/ChemicalLanding/&substance_nmbr=396.

assertion that "EPA now recognizes that carcinogens can be either non-threshold or threshold pollutants"[11], Commenters have shown such a statement to be out-of-step with its own practices and policy. EPA has not evaluated HCl for its carcinogenic potential. To further demonstrate its lack of substantial evidence, EPA's proposal references a single animal study that was used to derive a noncancer reference concentration after finding hyperplasia of the nasal mucosa larynx and trachea in rats.

The aforementioned study was used to derive a point of departure for the reference concentration for HCl and utilized the lowest observed adverse effect level ("LOAEL"), which is less preferential when compared to the more significant no observed adverse effect level ("NOAEL") or even a benchmark dose.[12] By EPA's definition, the NOAEL is "an experimentally determined dose at which there was no statistically or biologically significant indication of the toxic effect of concern" and only in instances where a NOAEL "has not been demonstrated experimentally, the term LOAEL is used."[13] While EPA did include an uncertainty factor for having to extrapolate from a LOAEL to NOAEL, the critical study used to derive the reference concentration failed to identify a level at which no effect occurred and as such, the single study for which EPA is relying on failed to provide definitive evidence that a no effect level (or a threshold of exposure) exists for HCl. This recognition is significant given that EPA argues HCl should be treated as a threshold pollutant even in the absence of adequate carcinogenicity studies.

Genotoxicity studies, which are designed to "detect compounds that induce genetic damage by various mechanisms"[14] are not widely available for HCl. Very few studies have explored either genotoxicity or carcinogenicity, and yet the

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[11] 89 Fed. Reg. 9092.

[12] EPA, Reference Dose (RfD): Description and Use in Health Risk Assessments Background Document (March 15, 1993). Available at: https://www.epa.gov/iris/reference-dose-rfd-description-and-use-health-risk-assessments.

[13] *Id.*

[14]Science Direct, Genotoxicity, Genetic Toxicity Studies (from Toxicology, 2021). Available at: https://www.sciencedirect.com/topics/pharmacology-toxicology-and-pharmaceutical-science/genotoxicity.

absence of such data does not support the conclusion of an existing threshold. In reality, from the few identified studies evaluating genotoxicity, at least two identified concentrations at which HCl was found to induce a response: in a 1981 study, the Escherichia coli DNA-repair assay was positive at a concentration of 25 ug/well as well as a separate study finding chromosomal nondisjunction induced at a concentration of 100 ppm in Drosophila melanogaster in 2008.[15] Chromosomal nondisjunction is a phenomena where the chromosomes fail to separate, producing daughter cells with an abnormal number of chromosomes. This is also considered a molecular initiating event, which is defined as "the initial interaction between a molecule and a biomolecule or biosystem that can be causally linked to an outcome via a pathway".[16] Molecular initiating events are a sequence of events that go along with the adverse outcome pathway framework and serve as the basis for determining genotoxicity and in turn, the potential for mutagenicity. The assessment of HCl's ability to induce a positive response in at least two separate studies provides evidence that exposure to HCl is mechanistically linked to potential genotoxic and potentially mutagenic outcomes (which cannot be ruled out).

Finally, Commenters draw attention to a fatal flaw associated with EPA's noncancer risk value for HCl. According to CalEPA's Office of Environmental Health Hazard Assessment ("OEHHA"), EPA itself rated its HCl database as "low-confidence" given "(1) the use of only one dose; (2) limited toxicity evaluation; (3) the lack of reproductive toxicity data; and (4) the lack of chronic exposure

[15] National Research Council (US), Committee on Emergency and Continuous Exposure Guidance Levels for Selected Submarine Contaminants. Emergency and Continuous Exposure Guidance Levels for Selected Submarine Contaminants: Volume 3. Washington (DC): National Academies Press (US); 2009. 3, Hydrogen Chloride. Available from: https://www.ncbi.nlm.nih.gov/books/NBK219917/.

[16] Timothy, EH et al. Defining Molecular Initiating Events in the Adverse Outcome Pathway Framework for Risk Assessment (2014). *Chemical Research in Toxicology* 2014 *27* (12), 2100-2112 Available at: https://pubs.acs.org/doi/10.1021/tx500345j#:~:text=A%20molecular%20initiating%20event%20(MIE,an%20outcome%E2%80%94adverse%20or%20otherwise.

studies."[17] Given the extremely limited data on the health effects associated with HCl exposure, EPA must include an evaluation of these studies in its final rule.

## IV. EPA'S PROPOSED *RISK APPROACH TO ASSESS A HEALTH-BASED EMISSION LIMITATION FOR HYDROCHLORIC ACID FOR THE LIME MANUFACTURING SOURCE CATEGORY (*2023) MEMORANDUM IS FLAWED AND UNDERESTIMATES THE NONCANCER RISK ASSOCIATED WITH HCL EXPOSURE.

EPA has requested comment on whether its proposed risk analysis and subsequent extrapolation of an emission limit of 300 tons per year ("tpy") would provide an ample margin of safety. In reviewing the Memorandum entitled, *Risk Approach to Assess a Health-Based Emission Limitation for Hydrochloric Acid for the Lime Manufacturing Source Category* ("2023 Memo"), Commenters have identified several flaws, including EPA's unjustified use of a weaker noncancer risk value, its continued underestimation of noncancer risks, and its unsubstantiated proposed risk approach.

**By failing to use the California OEHHA Reference Exposure Level (REL) to evaluate acute and chronic risk of HCl exposure EPA underestimates risks.**

To ensure it follows the best available science, EPA should use the latest OEHHA risk exposure level ("REL") for HCl. EPA has not provided adequate rationale for choosing to ignore OEHHA's REL, which is an order of magnitude stronger than EPA's RfC.[18]

Refusing to follow the OEHHA noncancer risk value conflicts with the Act's direction to employ the best available science, and with EPA's own scientific risk assessment guidelines. As EPA admits, EPA has long recognized that OEHHA's "process for developing [dose-response assessments] is similar to that used by EPA

[17] OEHHA, Appendix D.3 Chronic RELs and toxicity summaries using the previous version of the Hot Spots Risk Assessment guidelines (OEHHA 1999) at 309-312. Available at: https://oehha.ca.gov/media/downloads/crnr/appendixd3final.pdf.

[18] *Id.*

to develop IRIS values and incorporates significant external peer review," as well as recommendations of EPA and the National Academies of Sciences.[19]

It is EPA's longstanding policy to prioritize use of this value as the only non-federal source of dose-response value listed in EPA's hierarchy of scientific values.[20] EPA has made this determination "and prioritized according to (1) conceptual consistency with EPA risk assessment guidelines and (2) level of peer review received," with the goal of "incorporating into our assessments the best available science with respect to dose response information."[21]

In this proposed rule, however, EPA does not use the HCl REL to assess acute or chronic risks. Instead, it uses values that are orders of magnitude less protective and underestimates the acute risk by a factor of 100 (*see* 2023 Memo*)*. EPA's proposal thus relies on a significant underestimate of the acute health hazards of the regulated facilities' emissions. It is both unlawful and arbitrary, as it is misaligned with EPA's longstanding practice, an unsupported change with that practice, and a failure to apply the best available science.

In addition to underestimating acute non-cancer health hazards, EPA is willingly and knowingly exposing communities of color and low-income communities to increased and unnecessary harm from short-term HCl exposure. The ERPG-1 is a guideline level calculated by the U.S. Department of Energy that recognizes exposure to a chemical, like HCl, at this limit will lead to certain health effects.[22] Conversely, the REL developed by OEHHA represents the reference

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[19] EPA, Residual Risk Assessment for the Lime Manufacturing Source Category in Support of the 2019 Risk and Technology Review Proposed Rule (May 2019) at 27. Available at: EPA-HQ-OAR-2017-0015-0033.

[20] *Id.*

[21] *Id.* at 25.

[22] EPA, Dose-Response Assessment for Assessing Health Risks Associated With Exposure to Hazardous Air Pollutants (last updated June 27, 2018), https://www.epa.gov/fera/dose-response-assessment-assessing-health-risks-associated-exposure-hazardous-air-pollutants#tables; EPA, Table 2. Acute Dose-Response Values for Screening Risk Assessments (last updated June 18, 2018) https://www.epa.gov/sites/production/files/2018-06/acutefinaloutput_6_18_2018_7-49-37_am_2.xlsx.

level of exposure under which no adverse effects will occur. EPA provides no explanation for its decision to deviate from years of practice and allow acute health harm to affected communities from exposure to these chemicals.

The AEGL values and Emergency Response Planning Guidelines ("ERPG") values were created for emergency exposure scenarios. Levels defined for "once-in-a-lifetime, short-term exposures" and "emergency planning" for "single exposures" to chemical releases or accidents, are not appropriate tools to use to measure the acceptability of acute risks over a lifetime from one or more potential exposures due to an industrial source's emissions.[23]124 The Science Advisory Board ("SAB") has approved use of the RELs but *not* the EPRGs. As the SAB has explained:

> The incorporation of the available California Reference Exposure Levels (RELs) for the assessment of acute effects is a conservative and *acceptable approach* to characterize acute risks. The Panel *has some concern with the use of* the Acute Exposure Guidelines Limits (AEGLs) and *Emergency Response Planning Guidelines (ERPGs*) AEGL-2 and ERPG-2 values *should never be used in residual risk assessments because they represent levels that if exceeded could cause serious or irreversible health effects.*[24]

[23] 84 Fed. Reg. at 69,192.

[24] EPA Science Advisory Board, Review of EPA's draft entitled, "Risk and Technology Review (RTR) Risk Assessment Methodologies: For Review by the EPA's Science Advisory Board with Case Studies – MACT I Petroleum Refining Sources and Portland Cement Manufacturing" at 6-7 (emphasis added). SAB goes on to state that "an overarching concern with the Agency's chronic inhalation exposure estimates is that children's exposures do not appear to have been adequately addressed"); *see also id.* at 34 n.13 ("In particular is the question of whether the interindividual variability factor for non-carcinogens and the standard cancer unit risk derivation adequately covers children. If it does not, it is a potentially significant uncertainty given the greater intake rate of children via inhalation and sensitivity to carcinogens and other toxicants."). Available at: https://www.epa.gov/sites/default/files/2021-02/documents/epa-sab-10-007-unsigned.pdf.

The AEGL and ERPG numbers would be expected to underestimate risk. Using these numbers is likely to discount or cloak the level of risk to the maximum exposed individual.

As stated, EPA has long recognized CalEPA RELs as authoritative and regularly uses them in risk assessments. It gives no reasoned scientific explanation for refusing to use this value as the best available reference dose here. It must use the most up-to-date and best available scientific information on the risks and health effects of the HCl REL in its risk assessment. Doing so will lead to a recognition of increased acute risk from lime kiln facilities' emissions – especially if EPA is proposing to allow a staggeringly high 300 tpy of HCl emissions based on its approach outlined in the 2023 Memo.

**For chronic noncancer risk and where child-specific reference values are unavailable, EPA must consult science on early exposure impacts and use an additional default or uncertainty factor.**

The increased susceptibility of children, while known to exist, has not been quantified for many toxic chemicals. Until EPA has child-specific or child-based reference values available for a given pollutant, EPA should apply a default or uncertainty factor of at least 10 to account for increased risk from early-life exposures for non-cancer risk in this rulemaking and other risk assessments.

This would be consistent with the NAS recommendation on the need for EPA to use default factors to account for greater risk, with the science developed and considered by OEHHA, and with the 10X factor enacted by Congress in the Food Quality Protection Act.[25] Specifically, as the SAB report explained:

California EPA/OEHHA has determined that inhalation dosimetry for children is sufficiently different from adults to warrant a full 10-fold intra-individual pharmacokinetic uncertainty factor (i.e., an extra 3-fold PK uncertainty for children relative to the IRIS method) as a default approach. In setting non-cancer reference exposure levels (RELs), Cal EPA/OEHHA also considers that

[25] NAS, Science and Decisions: Advancing Risk Assessment (2009) at 190-93, 203. Available at: http://www.nap.edu/catalog.php?record_id=12209.

children may be outliers in terms of chemical susceptibility and on a case-specific basis adds a children's pharmacodynamic factor of 3-fold, making the inhalation risk for children as much as 10 times greater than adults.[26]

In addition, Congress has recognized this science in its unanimous vote on toxics legislation passed in 1996—the Food Quality Protection Act ("FQPA")—in which Congress found the need to use, and enacted, a tenfold Margin of Safety, or "10X factor." Specifically, the Act provides that "an additional tenfold margin of safety for the pesticide chemical residue and other sources of exposure shall be applied for infants and children to take into account potential pre- and post-natal toxicity and completeness of the data with respect to exposure and toxicity to infants and children."[27] Congress's recognition of the need to use this default factor provides a model that EPA should consider and incorporate into its residual risk assessment.

It would be appropriate and within EPA's authority under § 7412(f)(2) to determine that EPA must similarly use a children's tenfold margin of safety factor here, to fulfill the Clean Air Act's "margin of safety" requirement. 42 U.S.C. § 7412(f)(2). In doing so, EPA may rely directly on the science itself, and also on the unanimous guidance from Congress, provided in the FQPA, that the existing evidence of increased harm requires significant action to protect children from toxic exposure.

Further, the child-specific reference doses that OEHHA has created for some pollutants provide support for the use of an additional tenfold margin of safety factor.[28] EPA's current reference values for many pollutants are generally one order of magnitude less protective (i.e., larger) than the values that California has

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[26] OEHHA, Technical Support Document for Cancer Potency Factors: Methodologies for derivation, listing of available values, and adjustments to allow for early life stage exposures (May 2009) at 3-4, 50-51. Available at: https://oehha.ca.gov/media/downloads/crnr/tsdcancerpotency.pdf.

[27] 154 21 U.S.C. § 346a(b)(2)(C) (requiring that, in establishing, modifying, leaving in effect, or revoking a tolerance or exemption for a pesticide chemical residue, "for purposes of clause (ii)(I) an additional tenfold margin of safety for the pesticide chemical residue and other sources of exposure shall be applied" to protect infants and children).

[28] OEHHA, Table of all Child-Specific Reference Doses (chRDs) Finalized to Date (Jun. 22, 2010) Available at: https://oehha.ca.gov/risk-assessment/chrd/table-all-chrds.

recognized as needed to protect children, based on the currently available science and a specific assessment of research relevant to early life exposures.

EPA can have no valid basis for ignoring science showing that pollutants other than carcinogens also can cause substantial harm even at low doses if exposure occurs *in utero* and during the early windows of vulnerability.

**EPA inappropriately proposes to utilize a "risk-approach" for setting a Health Based Emission Limit.**

In the 'Results and Risk Characterization' section of the 2023 Memo, EPA states, "The maximum chronic non-cancer hazard from HCl emissions for this source category resulted in an HQ equal to 0.2, which is 5 times below the (RfC) concentration or a Hazard Quotient (HQ) of 1. Therefore, adverse chronic health effects are not likely to occur." Commenters are not aware of any instance where a hazard quotient (which is not even added against a hazard index) has been used as a mechanism to derive a quantitative equivalent to emissions levels. In fact, while EPA has in multiple instances (*see HON and MON rule)*[29] found noncancer risks to be at or well above 1 and thus, presumably unacceptable[30], it has <u>never</u> utilized the proposed approach to calculate what level of emissions would likely result in chronic and/or acute risks falling below 1 (in fact, chronic and acute risks identified in the MON rule were35x and 30x (respectively) higher than the risks found for the proposed lime kilns rulemaking).[31]

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[29] EPA, Residual Risk Assessment for the Synthetic Organic Chemical Manufacturing Industry (SOCMI) Source Category in Support of the 2023 Risk and Technology Review Proposed Rule (March 2023) at 6. Available at EPA-HQ-OAR-2022-0730-0085; Residual Risk Assessment for the Miscellaneous Organic Chemical Manufacturing Source Category in Support of the 2020 Risk and Technology Review Final Rule (April 2020) at 6. Available at: EPA-HQ-OAR-2018-0746-0189.

[30] *See* OMB response to comments located on page 23 of EPA-HQ-OAR-2017-0015-0211. In reference to the proposed 300 tpy emissions limit (based on the recommendations of the 2023 Memo) the 'Author' states, "This section does not make clear what the "established" "health threshold" EPA is using to consider setting a standard would be. It is also unclear how this could protect against multiple source exposure, or broader impacts." In response, EPA states, "EPA's statutory authority for regulatory decision making under the CAA section 112 is limited to considering source category risks. **EPA has long treated noncancer source category risks at or below a HQ or HI of 1 as being within the ample margin of safety** (emphasis added)."

[31] *Id* at 29.

Furthermore, Commenters find it inappropriate for EPA to simply solicit comments on its approach without going through a validated and transparent review process. Commenters strongly urge EPA to consult its Science Advisory Board with the appropriate charge and charge questions on this significant and highly consequential proposal, which includes novel and unsubstantiated approaches/methodologies employed in its risk approach analysis.