United States Court of Appeals

FOR THE DISTRICT OF COLUMBIA CIRCUIT

Argued April 6, 2010 Decided May 14, 2010

No. 09-1011

COALITION OF BATTERY RECYCLERS ASSOCIATION,
PETITIONER

v.

ENVIRONMENTAL PROTECTION AGENCY,
RESPONDENT

Consolidated with No. 09-1012

On Petitions for Review of Final Rule
of the United States Environmental Protection Agency

Michael B. Wigmore argued the cause for petitioner Coalition of Battery Recyclers Association in No. 09-1011. *Dennis Lane* argued the cause for petitioner The Doe Run Resources Corporation in No. 09-1012. With them on the briefs were *Robert N. Steinwurtzel* and *Sandra P. Franco*.

Eric G. Hostetler, Attorney, U.S. Department of Justice, argued the cause and filed the brief for respondent.

Before: SENTELLE, *Chief Judge*, ROGERS and GARLAND, *Circuit Judges*.

Opinion for the Court by *Circuit Judge* ROGERS.

ROGERS, *Circuit Judge*: Upon review of the air quality criteria and national ambient air quality standards ("NAAQS"), the Environmental Protection Agency revised the primary and secondary NAAQS for lead. *See National Ambient Air Quality Standards for Lead*, 73 Fed. Reg. 66,964 (Nov. 12, 2008) ("Final Rule"). The revision was designed to provide, in light of recent science, the requisite protection of public health and welfare by revising the permissible level of lead in ambient air and revising the averaging time over which the level must be met. The final rule also revised data handling procedures and emissions inventory reporting requirements and provided guidance on implementation. The Coalition of Battery Recyclers Association and the Doe Run Resources Corporation (together "petitioners") contend EPA action was arbitrary and capricious in multiple ways. Upon review of the rulemaking record, we are unpersuaded and we deny the petitions for review.

I.

Pursuant to sections 108 and 109 of the Clean Air Act, 42 U.S.C. §§ 7408–09, EPA regulates air pollutants, "emissions of which, in [the Administrator's] judgment, cause or contribute to air pollution which may reasonably be anticipated to endanger public health or welfare," *id.* § 7408(a)(1)(A). Lead ("Pb") emitted into the air can be inhaled or ingested and then absorbed into the bloodstream, potentially leading to a broad range of

adverse health effects including adverse neurological effects in children. *See* Final Rule, 73 Fed. Reg. at 66,972–73, 66,975–76. In 1978 EPA established primary and secondary NAAQS for lead of 1.5 micrograms of lead per cubic meter of air (µg/m^3) averaged over a calendar quarter. *See National Primary and Secondary Ambient Air Quality Standards for Lead*, 43 Fed. Reg. 46,246 (Oct. 5, 1978) ("1978 Lead NAAQS"). At this time, adverse neurocognitive effects in children had not been shown for blood lead levels below 50 micrograms of lead per deciliter of blood (µg/dL), and the 1978 NAAQS aimed to prevent most children from exceeding a blood lead level of 30 µg/dL. *See id.* at 46,246, 46,252. However, later studies showed adverse neurocognitive effects in children with blood lead levels below 10 µg/dL. *See* Final Rule, 73 Fed. Reg. at 66,975–76, 66,984.

EPA began reviewing the NAAQS for lead in 2004, considering some 6,000 studies and concluding "there is now no recognized safe level of Pb in children's blood." *Id.* at 66,984. As part of its review, EPA produced a "Criteria Document" assessing the latest scientific information regarding health effects associated with lead in the ambient air. *See id.* at 66,966–67. EPA's review "shift[ed] focus from identifying an appropriate target population mean blood lead level and instead focuse[d] on the magnitude of effects of air-related Pb on neurocognitive functions." *Id.* at 66,984. EPA developed an "evidence-based framework" that examined published studies addressing the relationship between IQ loss in children and air lead levels. *Id.* EPA relied to a lesser extent on risk estimates derived from risk assessment models. *Id.* at 67,006. Through its review, EPA sought to identify an air lead level "that would prevent air-related IQ loss (and related effects) of a magnitude judged by the Administrator to be of concern in populations of children exposed to the level of the standard." *Id.* at 66,997.

To relate IQ loss to air lead levels, EPA used its evidence-based framework to determine relationships between air lead levels and blood lead levels (the "air-to-blood ratio") and between blood lead levels and IQ loss (the "concentration-response" relationship). EPA concluded that for each μg/m^3 increase of lead in air, children's blood lead levels increase by 5–10 μg/dL, i.e., the air-to-blood ratio ranged from 1:5 to 1:10. *Id.* at 67,002. EPA selected an air-to-blood ratio of 1:7 "as a generally central value within this range." *Id.* at 67,004. EPA also concluded that the concentration-response relationship is nonlinear, with greater incremental IQ loss occurring at lower blood lead levels, and thus that analyses of children with blood lead levels closest to those of children in the United States today were most relevant. *Id.* at 67,002. EPA determined that the most recently measured mean blood lead level of U.S. children five years old and younger was 1.8 μg/dL, *id.*, and selected four study groups involving children with mean blood lead levels between 2.9 and 3.8 μg/dL rather than groups with higher mean blood lead levels, *id.* at 67,003. To "avoid[] focus on a single estimate that may be unduly influenced by one single analysis," each of the four selected groups was from a different study. *Id.* Using each group's reported mean IQ point decrease per μg/dL increase in blood lead levels, i.e., using the slope of the concentration-response relationship for each group, EPA calculated the median concentration-response slope to be -1.75 μg/dL. *Id.*

After considering public comments and the recommendations of its independent scientific review committee, the Clear Air Scientific Advisory Committee ("CASAC"), *see* 42 U.S.C. § 7409(d)(2), as well as "the uncertainties in the health effects evidence and related information" and the role of IQ loss in its evidence-based framework, EPA concluded that an allowable airborne lead–related loss of two IQ points should be used to set the

NAAQS standard. Final Rule, 73 Fed. Reg. at 67,005. CASAC had stated that "*a population loss of 1–2 IQ points is highly significant from a public health perspective*" and that such loss should be prevented, recommending an air lead level standard of 0.20 μg/m^3 or less. *Id.* at 66,999–67,000. Combining the blood-to-air ratio of 1:7, the concentration-response slope of -1.75 μg/dL, and the allowable air-related IQ loss of 2 points, EPA concluded that an air lead level standard of 0.15 μg/m^3 "would be sufficient to protect public health with an adequate margin of safety" and "is neither more nor less stringent than necessary for this purpose." *Id.* at 67,007.

EPA also concluded that the appropriate averaging time for the air lead level standard is a rolling three-month period with a maximum (not-to-be-exceeded) form evaluated over a period of three years. *Id.* at 66,996. EPA had initially proposed an averaging time of either a calendar quarter or a calendar month, *National Ambient Air Quality Standards for Lead*, 73 Fed. Reg. 29,184 (May 20, 2008) ("*NPRM*"), with CASAC recommending a monthly averaging period, *see* Final Rule, 73 Fed. Reg. at 66,993. EPA based its conclusion on scientific evidence indicating that blood lead levels increase quickly in response to increased lead exposure, that blood lead levels measured at the same time as an IQ test ("concurrent blood lead levels") are most strongly associated with IQ response, and that these concurrent blood lead levels reflect lead exposure over the past one to three months. *Id.* at 66,992–93. On November 12, 2008, EPA published the final rule, revising the primary and secondary NAAQS for lead to 0.15 μg/m^3 averaged over a rolling three-month period. *Id.* at 66,964.

II.

Petitioners assert that the revised primary lead NAAQS is overprotective, contending that (A) EPA did not provide

sufficient record support for basing the standard on preventing a decrease of more than two IQ points, (B) reliance on particular studies relating blood lead levels and IQ was arbitrary and capricious, and (C) selection of a lead standard of 0.15 $\mu g/m^3$ was arbitrary and capricious when measured as an average over a rolling three-month period. Consistent with our standard of review, *see Lead Indus. Ass'n v. EPA*, 647 F.2d 1130, 1145–48 (D.C. Cir. 1980); 42 U.S.C. § 7607(d)(9), we conclude these contentions lack merit because there is substantial record evidence to support EPA's conclusions that the population of children exposed to air lead levels above the revised NAAQS could suffer, and should be prevented from suffering, average losses of more than two IQ points, that greater incremental IQ loss occurs at lower relative blood levels and the more relevant IQ analyses are those of children with blood levels closest to today's population of children, and that a standard of 0.15 $\mu g/m^3$ measured as a three-month rolling average is required to protect public health with an adequate margin of safety.

A.

Sensitive populations; focus on IQ decrements. Petitioners' assertion that the revised lead NAAQS is overprotective because it is more stringent than necessary to protect the entire population of young U.S. children ignores that the Clean Air Act allows protection of sensitive subpopulations. Primary NAAQS are those "which in the judgment of the Administrator, . . . allowing an adequate margin of safety, are requisite to protect the public health." 42 U.S.C. § 7409(b)(1). In *Whitman v. American Trucking Ass'ns*, 531 U.S. 457, 475–76 (2001), the Supreme Court interpreted "requisite to protect" as "not lower or higher than is necessary . . . to protect the public health with an adequate margin of safety." This court, en banc, cited the Senate Report accompanying the Clean Air Act explaining that EPA should set standards providing "a *reasonable* degree of protection . . . against hazards which research has not yet

identified." *Natural Res. Def. Council v. EPA*, 824 F.2d 1146, 1152 (D.C. Cir. 1987) (en banc) (quoting S. REP. NO. 91-1196, at 10 (1970)) (emphasis and omission in *Natural Res. Def. Council*). And so this court has held that "NAAQS must protect not only average healthy individuals, but also 'sensitive citizens'" such as children, and "[i]f a pollutant adversely affects the health of these sensitive individuals, EPA must strengthen the entire national standard." *Am. Lung Ass'n v. EPA*, 134 F.3d 388, 389 (D.C. Cir. 1998) (quoting S. REP. NO. 91-1196 at 10); *see also Lead Indus. Ass'n*, 647 F.2d at 1152–53.

In the Final Rule EPA explained that the scientific evidence showing the impact of lead exposure in young children in the United States led it "to give greater prominence to children as the sensitive subpopulation in this review," Final Rule, 73 Fed. Reg. at 66,975, and to focus its revision of the lead NAAQS on "the sensitive subpopulation that is the group of children living near [lead emission] sources and more likely to be exposed at the level of the standard," *id.* at 67,000. Given the recent scientific evidence on which it relied, EPA's decision to base the revised lead NAAQS on protecting the subset of children likely to be exposed to airborne lead at the level of the standard was not arbitrary or capricious.

Petitioners' suggestion that EPA failed to explain adequately its shift in focus, from blood lead levels in the original 1978 lead NAAQS to IQ decrements in children in the revised NAAQS, is without merit. In the rulemaking EPA explained that although the 1978 NAAQS was based on determining a maximum safe blood lead level for children, current scientific evidence no longer recognized a safe blood lead level, and EPA consequently adopted a different focus when revising the lead NAAQS. *See NPRM*, 73 Fed. Reg. at 29,229; Final Rule, 73 Fed. Reg. at 66,984. EPA further explained that epidemiological studies of cognitive effects and

lead exposure commonly use IQ scores and that the scientific literature supports the conclusion that lead exposure causes IQ loss in children. *See* Final Rule, 73 Fed. Reg. at 66,976. As EPA noted in its brief, EPA does not view blood lead levels as adverse health effects under the Clean Air Act, and both the 1978 and 2008 lead NAAQS focused on preventing adverse health effects such as neurocognitive effects. *See id.* at 66,983–84; 1978 Lead NAAQS, 43 Fed. Reg. at 46,252–53.

Petitioners further contend EPA's use of IQ decrements to revise the lead NAAQS was arbitrary and capricious because IQ measurements are more uncertain than blood lead level measurements. They assert that confounding factors such as environmental factors affect IQ scores and that the population significance of IQ loss is imprecise. However, EPA explained that a large number of high quality studies support the inference that lead exposure causes population IQ loss, and that animal studies in which confounding factors are not present show that low levels of lead cause neurobehavioral effects. *See* Final Rule, 73 Fed. Reg. at 66,984 n.56. Petitioners claim that EPA has acknowledged the standard error of measurement for IQ is between three and four IQ points, and so assert that an IQ decrement of two points therefore "cannot be detected at the level of an individual." Pet'rs Br. 32. This assertion confuses the "'critical' distinction between population and individual risk," wherein a small change in IQ at the level of an individual is a substantial change at the level of a population, as noted by EPA when citing the discussion of the differences between individual-level and population-level data in the Criteria Document. *See* Final Rule, 73 Fed. Reg. at 66,976. Thus any suggestion by petitioners that EPA did not adequately respond to comments on the IQ test error rates, assuming this issue was not forfeited by their failure to resubmit the comments as directed in the notice of proposed rulemaking, *see NPRM*, 73 Fed. Reg. at 29,190, is without merit.

Petitioners also protest, while conceding that CASAC concluded a population loss of two IQ points "is [a] highly significant" public health problem, Final Rule, 73 Fed. Reg. at 67,000, that EPA did not provide adequate record support for its decision to protect against a population loss of more than two IQ points. In the Final Rule and the Criteria Document EPA explained that a mean population loss of two IQ points would cause both a substantial decrease in the percentage of the population achieving very high IQ scores and a substantial increase in the percentage achieving very low scores. *See id.* at 66,976. In addition to citing CASAC's conclusion, EPA cited the comments of the American Academy of Pediatrics and state health agencies that such a loss should be prevented. *See id.* at 67,000. These explanations sufficiently support EPA's decision to prevent a population loss of more than two IQ points. Regardless of whether EPA sufficiently explained its decision not to follow the recommendation of CASAC and others to further reduce IQ loss, a deficiency there would not lend support to petitioners' contention that the revised lead NAAQS is overprotective.

Additionally, while petitioners suggest EPA ignored record evidence that population IQ has not increased commensurate with decreasing blood lead levels over the past several decades, EPA's response to comments on the proposed rule stated there is evidence that IQ scores have increased by an average of three points per decade in the United States over the last several decades and that IQ tests are routinely renormalized to adjust for these increases. *See* EPA Responses to Significant Comments on the 2008 Proposed Rule on National Ambient Air Quality Standards for Lead, at 30–31 (Oct. 2008) ("Response to Comments"). EPA's response also explained these increases may be due to improvements in nutrition and to other societal and environmental factors as well as to reduced exposure to lead, and concluded from an analysis of the scientific evidence

that the decline in blood lead levels over the past several decades contributed to the increase in IQ over that time period.

For these reasons we conclude that EPA adequately justified its decision to prevent a loss of more than two IQ points in the population of children exposed to the level of the revised NAAQS. Petitioners' reliance on *In re Permian Basin Area Rate Cases*, 390 U.S. 747 (1968), to support their contention that the revised lead NAAQS is overprotective, is misplaced. The record shows EPA met its "oblig[ation] at each step of its regulatory process to assess the requirements of the broad public interests entrusted to its protection by Congress," *id.* at 791, by considering what lead NAAQS "is requisite to protect public health, including the health of sensitive groups, with an adequate margin of safety," Final Rule, 73 Fed. Reg. at 67,006.

B.

Selection of scientific studies. Petitioners contend that the record does not support EPA's decision to exclude from its determination of the concentration-response slope three additional study groups involving children with blood lead levels greater than those of current U.S. children but below 10 µg/dL. The four study groups EPA used to determine the concentration-response slope were (1) a group from the "Lanphear" study analyzing blood lead levels below 7.5 µg/dL, (2) a group from the "Téllez-Rojo" study analyzing blood lead levels below 5 µg/dL, (3) the "Canfield" study analyzing blood lead levels below 10 µg/dL, and (4) the "Bellinger and

Needleman" study analyzing blood lead levels below 10 µg/dL.[1] *See* Final Rule, 73 Fed. Reg. at 67,003 (Table 3). Like the Canfield and the Bellinger and Needleman studies on which EPA relied, the three study groups petitioners wanted EPA to use, namely an additional group from the Lanphear study and two additional groups from the Téllez-Rojo study, involved blood lead levels below 10 µg/dL. Petitioners contend that by excluding the three study groups EPA did not rely on the latest scientific knowledge, that the studies' concentration-response slopes do not continuously become steeper as blood lead levels decrease, that there is a larger difference in the steepness of such slopes above and below 10 µg/dL than above and below 5 µg/dL, and thus that EPA should have included the three additional study groups because they involved blood lead levels below 10 µg/dL.

However, EPA explained that the scientific evidence, as well as CASAC's recommendations, supported its conclusion that the concentration-response relationship is nonlinear, in the sense that a given increase in blood lead levels would cause a greater IQ loss in a population with low blood lead levels than a population with higher blood lead levels. *See id.* at 67,002. EPA reasonably concluded from this evidence that studies involving children with blood lead levels closer to the mean

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[1] *See* B.P. Lanphear et al., Low Level Environmental Lead Exposure and Children's Intellectual Function: An International Pooled Analysis," 113 Envtl. Health Perspectives 894 (2005); M.M. Téllez-Rojo et al., Longitudinal Associations between Blood Lead Concentrations < 10 µg/dL and Neurobehavioral Development in Environmentally-Exposed Children in Mexico City, 118 Pediatrics e323 (2006); R.L. Canfield et al., Intellectual Impairment in Children with Blood Lead Concentrations below 10 µg per Deciliter, 348 New Engl. J. Med. 1517 (2003); D.C. Bellinger & H.L. Needleman et al., Intellectual Impairment and Blood Lead Levels [letter], 349 New Eng. J. Med. 500 (2003).

blood lead level of today's population of U.S. children would provide a more representative estimate of the concentration-response slope. *Id.* EPA's decision not to rely on the three studies identified by petitioners is practically self-evident from the table included in the rulemaking, see *id.* at 66,978–79 (Table 1), and EPA's explanation for not relying on these three studies was reasonable. EPA explained that it had included from the Lanphear and Téllez-Rojo studies only the study group with "a mean blood Pb level closest to today's mean for U.S. children." *Id.* at 67,003. EPA further explained that it had "identified four different studies" to use in determining the concentration-response slope and thus "avoid[ed] focus on a single estimate that may be unduly influenced by one single analysis." *Id.*

No less, contrary to petitioners' view, did EPA reasonably explain why it relied more on the evidence-based framework than on the risk assessment model results, results that petitioners assert contradict EPA's conclusion that a given increase in blood lead levels would cause a greater IQ loss at lower blood lead levels than at higher blood lead levels. EPA explained it considered its risk assessment model results but gave them less weight than the results in the published studies considered in its evidence-based framework, because the risk assessment models were associated with "important uncertainties and limitations" related to modeling air lead dispersion, lead exposure pathways, and other factors. *Id.* at 66,981, 67,006. In addition, EPA noted that the results obtained from the risk assessment models were "roughly consistent with and generally supportive of" the air-related IQ loss estimates EPA determined using its evidence-based framework. *Id.* at 67,006 & n.84. To the extent petitioners rely on the location-specific urban case studies from the risk assessment models, EPA noted that the population exposures modeled in the general urban and primary lead smelter sub-area case studies "relate more closely to the air-related IQ loss evidence-based framework" than the location-

specific urban case studies. *Id*. at 67,006 n.84. To the extent petitioners rely on the lower ends of the ranges of IQ losses estimated by the general urban case study, EPA observed both that these lower ends "do[] not fully represent the risk associated with all air-related pathways," *id.* at 66,981, and that at a level of 0.2 $\mu g/m^3$ the range of IQ loss estimates provided by the general urban and primary lead smelter sub-area case studies "are inclusive of the range of estimates" derived from the evidence-based framework for that level, *id.* at 67,006 n.84.

Petitioners fare no better in contending EPA unreasonably assumed the relationship between blood lead levels and IQ was linear by determining a single number (-1.75 µg/dL) for the concentration-response slope. EPA concluded from "the weight of the current evidence" that the concentration-response relationship is "nonlinear, with steeper slopes at lower blood Pb levels," but "recognize[d] uncertainty in the quantitative characterization of the nonlinearity in the blood Pb–IQ loss relationship." *Id.* at 67,003. Petitioners' reliance on *Natural Resources Defense Council*, 824 F.2d at 1165, where the court stated that "there is no particular reason" to think straight-line extrapolation from known to unknown harm levels provides an accurate prediction of harm, undercuts rather than supports their contention. Even if EPA were using the value of -1.75 µg/dL to extrapolate an IQ at a lower blood level rather than to estimate an IQ loss at a particular air lead level, the court there concluded from the limitations of extrapolation not that EPA could not extrapolate, but that "by its nature the finding of risk is uncertain and the Administrator must use his [or her] discretion to meet the statutory mandate" of the particular Clean Air Act provisions involved. *Id.*

EPA thus adequately justified its decision to rely on analyses of IQ in children with blood lead levels closest to those of today's population of children when revising the lead

NAAQS. To the extent petitioners contend the connection between IQ loss and lead exposure is quantitatively uncertain, EPA has fulfilled its obligations to "engage in reasoned decision-making," *Am. Lung Ass'n*, 134 F.3d at 392, and to "err on the side of caution by setting primary NAAQS that 'allow[] an adequate margin of safety,'" *Am. Trucking Ass'ns v. EPA*, 283 F.3d 355, 369 (D.C. Cir. 2002) (quoting 42 U.S.C. § 7409(b)(1)) (alteration in *Am. Trucking*).

C.

Non-conversion with three-month rolling average. Petitioners contend EPA was arbitrary and capricious not in selecting a rolling three-month averaging period, but in failing to convert the 0.15 $\mu g/m^3$ air lead level from an annual basis to a three-month basis. This contention lacks merit. EPA based its selection of a rolling three-month average on different studies than its selection of the 0.15 $\mu g/m^3$ standard. *Compare* Final Rule, 73 Fed. Reg. at 66,991–96 *with id.* at 66,996–67,007. From the studies considered in determining the averaging period, EPA concluded that the scientific evidence "does not specify the duration of a sustained air concentration associated with a particular blood Pb contribution" but does "support[] the importance of time periods on the order of three months or less." *Id.* at 66,994. Petitioners' characterization of the 0.15 $\mu g/m^3$ standard as the product of a unit conversion error is thus inapt.

EPA did rely on at least two published studies referring to annual or yearly averages: a World Health Organization study providing an air lead guideline of 0.5 $\mu g/m^3$ averaged over an annual period, and a study by S.R. Hilts reporting results of

annual blood lead level tests of children living near a smelter.[2] EPA relied on both studies in deriving an air-to-blood ratio rather than in determining the relationship between changes in blood lead levels and IQ. *See* Final Rule, 73 Fed. Reg. at 66,973–74. Petitioners fail to demonstrate either study determined the length of exposure to a particular average ambient air lead level that resulted in a particular blood lead level response, *see id.* at 66,994, and so fail to show that EPA's conclusions regarding the lead NAAQS level are valid only for exposures to lead averaged over a period of one year. Furthermore, petitioners do not dispute that EPA explained that the studies on which it based its selection of the averaging period indicated that adverse health effects may result from lead exposure over a period of one to three months. *See id.* at 66,993.

Petitioners erroneously conclude that because EPA's risk assessment models could accept inputs only of annual average ambient air lead concentrations, *see id.* at 66,980 & n.45, EPA calculated the 0.15 $\mu g/m^3$ level based on annual average air lead exposure rather than three-month exposure. As detailed in EPA's Criteria Document, the annual average values were used in the risk assessment models to estimate blood lead levels rather than IQ responses to blood lead levels. However, as EPA explained, the use of annual average ambient air lead concentrations in the risk assessment modeling was an artifact of the models themselves, and EPA adjusted the input annual concentrations to correspond to monthly or quarterly averaging times. *See id.*; *see also id.* at 66,995; Response to Comments at 9.

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[2] *See* World Health Organization, Air Quality Guidelines for Europe 33 (2d ed. 2000); S.R. Hilts, Effect of Smelter Emission Reductions on Children's Blood Lead Levels, 303 Sci. Total Env't 51, 52 (2003).

The rulemaking thus demonstrates that EPA adequately explained that it did not determine the 0.15 μg/m^3 air lead level by assuming exposure to that level over a period of one year, and that EPA reasonably concluded and adequately explained that a lead NAAQS of 0.15 μg/m^3 measured as a three-month rolling average is requisite to protect public health with an adequate margin of safety.

III.

The Lanphear study investigated the concentration-response relationship between blood lead levels and IQ changes, and provided what EPA described as "the most compelling evidence" for effects of lead on IQ at blood lead levels below 10 μg/dL and for the nonlinearity of these effects. Final Rule, 73 Fed. Reg. at 66,977. Petitioners contend the Lanphear study contained such errors that EPA acted arbitrarily and capriciously in relying on results from the study without first obtaining and making public the underlying data for the study. However, in *American Trucking*, 283 F.3d 355, this court rejected the notion that EPA had improperly failed to obtain and make public data underlying studies on which it had relied during a NAAQS rulemaking, holding that "[t]he Clean Air Act imposes no such obligation" and that "requiring agencies to obtain and publicize the data underlying all studies on which they rely would be impractical and unnecessary." *Id.* at 372 (quotation marks omitted).

Petitioners attempt to distinguish their request on the ground that in *American Trucking* the court was addressing requests for data underlying several studies, while they request only that EPA obtain and make public the data underlying the Lanphear study. This distinction finds no support in the reasoning of *American Trucking*. Rather than distinguishing between an agency's burden in obtaining data from one versus

many studies, the court distinguished EPA's reliance on a study's results from its reliance on the raw data underlying such results, noting that raw data often is unavailable due to proprietary interests of a study's scientific investigators or confidentiality agreements with study participants. *See id.* Petitioners do not contend EPA possessed the underlying data but failed to include it in the rulemaking record. *Cf. Am. Radio Relay League, Inc. v. FCC*, 524 F.3d 227, 237–38 (D.C. Cir. 2008).

Petitioners' reliance on EPA's regulation concerning requests for data under the Freedom of Information Act, 5 U.S.C. § 552 ("FOIA"), is forfeited. This regulation provides that in response to a FOIA request, EPA shall request and make public "research data relating to published research findings produced under an award" of an EPA grant, subject to privacy and other considerations. 40 C.F.R. § 30.36(d). Petitioners cited this regulation for the first time during rebuttal oral argument, affording EPA no opportunity to respond. *See Ark Las Vegas Rest. Corp. v. NLRB*, 334 F.3d 99, 108 n.4 (D.C. Cir. 2003). Nor did petitioners preserve this argument by mentioning their separate FOIA request for the Lanphear study data in a footnote of their brief, without citing 40 C.F.R. § 30.36(d). *See, e.g.*, *Nat'l Mining Ass'n v. Mine Safety & Health Admin.*, 599 F.3d 662, 671 (D.C. Cir. 2010); *NSTAR Elec. & Gas Corp. v. FERC*, 481 F.3d 794, 800 (D.C. Cir. 2007). In any event, the issue raised by petitioners in the instant case involves whether EPA has a general duty to obtain and make public underlying data as part of a rulemaking, rather than any specific duties related to FOIA releases; more generally the regulation concerns EPA's reservation of rights in intangible property related to its grant awards, and the court has no occasion to note more than that the regulation does not apply here.

Similarly unavailing is petitioners' contention that EPA did not respond adequately to comments concerning errors in the Lanphear study. EPA responded to petitioners' comments by noting that errors in Table 4 and two typographical errors in the Lanphear study had been corrected and explaining that these corrected errors did not affect the portions of the study on which EPA had relied. *See* Response to Comments at 72. EPA also explained in response to petitioners' questioning of Figure 3 of the Lanphear study how petitioners had misinterpreted the statistical methods involved in the figure and that EPA's conclusions from the Lanphear study did not depend on the figure. *See id.* at 24–25. EPA further observed that its confidence in the Lanphear study had been reaffirmed by the "Rothenberg" study, which had re-analyzed the Lanphear data and confirmed the nonlinear relationship between IQ and blood lead levels shown in Figure 3 of the Lanphear study.[3] *See id.* at 25.

Consequently, petitioners have failed to identify errors in the Lanphear study that would make EPA's reliance on it arbitrary and capricious, and EPA thus appropriately considered the Lanphear study and was not required to obtain and make public the data underlying the Lanphear study.

IV.

Finally Doe Run Resources, Inc., separately contends that EPA erroneously concluded it lacked statutory authority under the Clean Air Act to consider the bioavailability of lead sulfides when determining compliance with the lead NAAQS. Doe Run asserts that lead sulfides are less bioavailable than other forms

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[3] *See* S.J. Rothenberg & J.C. Rothenberg, Testing the Dose-Response Specification in Epidemiology: Public Health and Policy Consequences for Lead, 113 Envtl. Health Persp. 1190 (2005).

of lead and thus less likely to contribute to health risks, and that EPA's NAAQS compliance evaluations consequently should treat low-bioavailable lead sulfides as "policy relevant background" with "in effect, a waiver from the general NAAQS rule." Pet'rs Br. 55–56. EPA declined to determine compliance with the lead NAAQS based on the bioavailability of lead sulfides, explaining that it "must determine compliance with the standard" pursuant to Clean Air Act § 109(b), 42 U.S.C. § 7409(b). *See* Response to Comments at 65.

The term "policy-relevant background" does not appear in the Clean Air Act, and in the context of the lead NAAQS, EPA defines "policy-relevant background" as natural emissions of lead into the air from non-anthropogenic sources as well as lead from non-air sources. *See NPRM*, 73 Fed. Reg. at 29,192. In responding to comments EPA stated that evidence indicates the bioavailability of lead sulfides increases over time and that EPA considered this variable bioavailability in revising the lead NAAQS. *See* Response to Comments at 10. In its brief on appeal, EPA noted that concentrations of ambient lead near lead mines or smelters are not naturally occurring background concentrations. Resp't Br. 57 n.27. Under the circumstances, EPA reasonably concluded Doe Run's request to treat the bioavailability of lead sulfides differently was effectively a request for a waiver from the lead NAAQS.

Applying the familiar two-step analysis under *Chevron, U.S.A., Inc. v. Natural Resources Defense Council, Inc.*, 467 U.S. 837 (1984), *see Natural Res. Def. Council v. EPA*, 489 F.3d 1364, 1371 (D.C. Cir. 2007), we conclude Doe Run's contention fails at both steps. First, Congress provided in the Clean Air Act that primary NAAQS "shall be ambient air quality standards the attainment and maintenance of which in the judgment of the Administrator . . . are requisite to protect the public health." 42 U.S.C. § 7409(b)(1). EPA is required to designate "any area that

does not meet (or that contributes to ambient air quality in a nearby area that does not meet) the national primary or secondary ambient air quality standard for the pollutant" as "nonattainment." *Id.* § 7407(d)(1). Further, EPA "may not promulgate a redesignation of a nonattainment area (or portion thereof) to attainment unless" it "determines that the area has attained the national ambient air quality standard." *Id.* § 7407(d)(3)(E)(i). Additionally, Congress addressed the circumstances under which attainment could be waived, e.g., as with certain particulate matter sources, *see* 42 U.S.C. § 7513(f), and with emissions emanating from outside the United States, *see* 42 U.S.C. § 7509a, but provided no authorization for EPA to waive NAAQS attainment requirements in the manner requested by Doe Run. Even assuming the Clean Air Act was ambiguous with regard to whether EPA was empowered to grant other waivers, EPA's interpretation of its authority under the statutory scheme is permissible under *Chevron* step two, 467 U.S. at 843, and entitled to deference by the court.

Accordingly, we deny the petitions.