For the Record

Still No Evidence: A Reply to Khan et al.

In the Spring 2026 issue of *Regulation*, Arif Khan and coauthors argued that the "boxed warning" on antidepressants discouraged providers from prescribing them to youth, thereby increasing suicides (Khan et al. 2026a). We disputed this in the Summer issue, arguing that use of the drugs themselves correlates with higher suicides, and concluding that the boxed warning is appropriate (Stone et al. 2026). In the same issue, Khan and coauthors responded to our arguments (Khan et al. 2026b). We now write in reply to those arguments.

Their response confirms rather than resolves the concerns we raised. It fails to address most of the empirical arguments we presented, relies heavily on a systematic review that is itself methodologically compromised, introduces new claims that are either self-refuting or unsupported by evidence, and misrepresents the basis for our analysis.

Methodology is not a technicality / Khan et al. characterize our critique as focused on "methodological objections" while ignoring "real-world detrimental effects." This framing treats methodology as an obstacle to be set aside when the stakes are sufficiently high. It is not. Methodology is the mechanism by which we distinguish true findings from artifacts of analytical error (Hammad & Rochon 2026). A study that uses invalid methods does not demonstrate real-world effects; it demonstrates what invalid methods produce.

The "real-world detrimental effects" Khan et al. invoke have not been demonstrated independently of the causal assumption that antidepressant prescribing rates determine population-level suicide incidence. Remove that assumption—which the evidence requires—and there are no demonstrated real-world effects. What Khan et al. present as established fact is the unproven conclusion of a causal argument. They are using their conclusion to dismiss the critique that shows the conclusion is unproven. That is circular reasoning, not evidence.

Causal claim remains unsupported / Khan et al.'s principal response to our timing argument is that the warning's effect was delayed: that it took years to propagate through the healthcare system before becoming visible in population-level outcomes. This argument fails on multiple grounds.

For one, their causal claim contradicts the prescribing timeline. Antidepressant prescribing to young people declined by approximately 20 percent between 2005 and 2007 but, they claim, this had nothing to do with the boxed warning. The "suppression" did not take effect until 2008, when prescribing rates began a sustained increase. They provide no basis for their attribution of specific numbers of excess deaths—2,365 children and 3,593 young adults—to the warning. Suicide rates fell during the period of maximum prescribing decline and did not begin rising until 2007–2009 as prescribing was already recovering. The delayed effect argument requires us to believe that the decline in prescribing from 2004 through 2007, whatever the cause, resulted in falling suicide rates while prescribing recovery produced rising ones—the opposite of what their theory predicts. Between 2016 and 2022, antidepressant prescriptions for adolescents aged 12–17 increased by 43 percent and among those aged 18–25 by more than 47 percent (Chua et al. 2024). Current prescribing is at historic highs, yet suicide rates remain elevated. Khan et al. offer no account of this.

Another problem is that their causal claim contradicts the literature they cite. The majority of the studies Khan et al. invoke—including Gibbons et al. 2007, and Lu et al. 2014—describe adverse effects occurring immediately after the warning, in 2005–2007, precisely the period when youth suicide rates were falling. If the delayed effect argument is correct, these papers detected nothing real. If these papers detected real effects, the delayed effect argument fails. Khan et al. invoke both simultaneously without acknowledging the contradiction.

Several of our strongest arguments receive no response at all. The sex-specific divergence in suicide trends—rates among boys peaked around 2017–2018 and have since declined approximately 30 percent while rates among girls have continued to rise—is not mentioned. Since prescribing decisions are not made differently for boys and girls, a warning-driven suppression of prescribing cannot explain this divergence. The misrepresentation of the comparison group is similarly unaddressed: Suicide rates for adults aged 55–64, presented as an unaffected control population, increased by 25–44 percent over the same period, comparable to the increases in adolescents that Khan et al. attribute to the warning. The pharmaceutical marketing explanation for the 2005 prescribing decline—which affected every age group through 54 and coincided with an estimated annual $800 million drop in manufacturer promotional spending—is ignored. The healthcare contact data showing that more than one third of people who die by suicide had no healthcare contact of any kind in the preceding year are not addressed. The argument that moving the warning to Section 5 would not change the clinical or legal considerations facing physicians receives no response.

Barrier claim is asserted without evidence / The central claim of their response—that the boxed warning creates mean-

ingful barriers to care—is presented as self-evident but supported by no real evidence. No quantification is offered of how many patients who needed antidepressants failed to receive them because of the warning's status specifically as a box, as opposed to other sources of concern about the drugs causing suicidal thinking and behavior, and as distinct from the many other well-documented barriers to mental health care including access, insurance, stigma, and prescriber availability. They base this claim solely on unspecified clinical anecdotes of physicians reporting hesitancy; this appears to be no more than their subjective impression. Khan et al. provide no detail about what these anecdotes actually describe—even qualitatively—and do not describe any organized effort to collect clinical opinion. There is no transcript, no protocol, no indication of how the opinions were solicited, and no way to assess whether the questions put to physicians were neutral or leading. Even if such efforts were made, opinions gathered without methodological discipline are easily shaped—wittingly or unwittingly—by the framing of the inquiry, and what is presented as independent clinical testimony may in fact be the elicited echo of the interviewer's premises.

Khan et al.'s own rendering of these reports is itself processed through their priors: Authors who have argued for more than a decade that the warning causes harm are not neutral curators of physician sentiment; they are predisposed to motivated reasoning in the selection, summarization, and emphasis of such material. The reports themselves are also a self-selected sample: Physicians who feel constrained by the warning and want it removed have reason to come forward and be heard, while physicians who prescribe without difficulty have none. A body of testimony filtered in this way cannot establish the prevalence of the phenomenon it describes. What Khan et al. characterize as "real-world effects" may amount to little more than the projection of their own opinions onto a sympathetic and unverifiable sample.

Anecdote sits at the bottom of the hierarchy of clinical evidence, and Khan et al. invoke it here to override administrative prescribing data. They offer no account of how a barrier-creating warning has presided over a 43 percent increase in adolescent prescriptions since 2016. Nor do they show that patients not prescribed antidepressants because of such hesitancy are those at high risk for suicide. Physicians unwilling to prescribe to high-risk patients would also be obliged to refer such patients to physicians willing to treat them properly.

Khan et al. also argue that the practical effect of a labeling change lags months to years behind the regulatory decision because dispensed inventory carries the old label until stock turns over. This claim reflects a fundamental misunderstanding of how physicians access prescribing information. Physicians decide whether to prescribe before the prescription is written and before any package insert reaches the patient. The insert that accompanies dispensed medication is a patient-facing document; it plays no role in the prescribing decision, which has already been made. Prescribers access drug labeling through electronic references, FDA databases, clinical decision support tools, promotional materials, and professional training.

Widely used drugs such as antidepressants do not require years for supplies to turn over; for most drugs and pharmacies, it is a few months. Most antidepressants have a shelf life of about two to three years from the date of manufacture. Drugs present on pharmacy shelves before the warning was issued would have been dispensed or expired well before youth suicide rates began their increase in 2008. This also directly contradicts the source on which Khan et al. primarily rely: Soumerai et al. (2024) describe the effects of the warning as "sudden, simultaneous, and sweeping," an account irreconcilable with a mechanism that operates gradually as old inventory clears.

The logic of the barrier argument also defeats itself. A physician who believes a patient is at high risk for suicide and thinks an antidepressant could reduce that risk has every reason to prescribe despite the warning because withholding treatment only increases the likelihood of the outcomes they fear, including a lawsuit should a suicide occur. The population for whom a physician could be deterred from prescribing by the warning must therefore consist of lower-risk patients, for whom the drug-induced suicidality risk most plausibly exceeds the therapeutic benefit. The barrier Khan et al. invoke, if it exists at all, operates precisely where removing it would do the most harm.

These anecdotal reports also deserve scrutiny before being accepted as evidence of a genuine barrier. Managing emotionally troubled patients—particularly adolescents—is difficult, and many providers feel ill-equipped to do so. The invocation of the boxed warning may just be a convenient rationalization to avoid managing such patients. If so, changing the label would not address the underlying problem. The barriers Khan et al. describe are more plausibly symptoms of inadequate training and systemic gaps in mental health care than consequences of drug labeling, and those underlying problems would not be addressed by any change to the warning.

Self-referential citations / Khan et al. characterize the body of evidence supporting their position as "14 years of strong research," citing Soumerai et al. (2024) as its synthesis. Neither the systematic review nor the broader citation list survives scrutiny.

The Soumerai review is substantially self-referential. Christine Y. Lu appears as author or co-author on at least four of the 11 studies the review includes as primary evidence: Lu et al. 2014, Lu et al. 2018, Lu et al. 2020, and Soumerai et al. 2024 itself. Stephen Soumerai appears on two. Across Khan et al.'s broader citation list of 11 studies, the same two investiga-

tors account for the majority of entries. This is not an independent and converging literature; it is a self-referential citation network in which a small group of researchers repeatedly cites its own prior work as corroboration. That is not independent replication; it is iteration.

The selection criteria of the review favor a predetermined methodology. Of 1,841 studies screened, 11 were selected: overwhelmingly interrupted time series analyses and similar before-and-after comparisons—precisely the methodology Stone (2018a) documented in detail to be invalid for this purpose. Restricting a systematic review to a methodologically invalid study design does not overcome that invalidity.

Several of the 11 included studies use hospital admissions for poisoning (whether self-inflicted or not) by psychotropic agents as a proxy measure for suicide attempts. Stone (2018a) showed that this proxy was explicitly rejected as a useful proxy in the Patrick paper that Lu and Soumerai misrepresent as validating it. In a published point–counterpoint exchange in the journal *Medical Care* (Lu et al. 2018; Stone 2018b), Stone charged that Lu et al. "knowingly and repeatedly misrepresented their proxy as peer-reviewed when the Patrick paper, in fact, rejected its use," and that "whether due to ineptitude or dishonesty, errors of this magnitude ethically require a paper's retraction." The Soumerai et al. (2024) systematic review was written with full knowledge of it and does not address it.

Lu defended the proxy by citing its 99.3 percent specificity. Stone (2018b) showed this statistic is meaningless here: Because suicide attempts are rare relative to all hospital admissions, almost any diagnosis code looks highly specific by this calculation. Appendectomy, which has no connection to suicide, scores 99.1 percent by the same method. The proxy also moves in the wrong direction: between 2004 and 2009, hospital admissions for psychotropic drug misuse were strongly negatively correlated (−0.85) with actual suicide attempt rates in national data.

The proxy measure has additional problems that Lu's response does not address. It does not require that the poisoning be a suicide attempt; many are accidental overdoses. But even if psychotropic drug poisonings were a generally satisfactory proxy for attempted suicide, it makes little sense to use it as a proxy when the concern is that FDA actions discouraged the prescription of antidepressants and other psychiatric care. Antidepressants and antipsychotics are psychotropic drugs. People who present with poisoning from antipsychotics or antidepressants cannot reasonably be considered to have been discouraged by FDA actions from receiving these drugs. According to the Drug Abuse Warning Network, nearly half of the psychotropic drugs involved in suicide attempts were antidepressants (29 percent) or antipsychotics (17 percent) (Stone 2018a).

As our *Regulation* article documents in detail, suicide rates fell for several years after the warning was issued, and prescribing has risen well above pre-warning levels throughout the period the review covers. The claim that consistency of observed harms across 14 years "indicates this is not a coincidence" is circular: Consistency across a literature built on shared methodological errors reflects consistency of method, not convergence of truth.

Khan et al.'s continued citation of Gibbons et al. 2007 is particularly difficult to justify. Our *Regulation* article documented—as Jureidini (2007) had pointed out in correspondence to the journal at the time of publication—that this paper presented two graphs not aligned to the same period: The suicide uptick it presents as evidence of harm occurred in 2004, before the warning existed, while the prescription downtick occurred in 2005, after it. An effect that preceded its alleged cause by a year is not evidence; it is a logical impossibility. The paper was never retracted despite this documented error and continues to be cited as though it had never been identified. Both Khan and Soumerai are well aware of this. For them, it appears, evidence is "strong" when it confirms their priors—logic, methodology, and documented error notwithstanding.

Khan's data transparency charge / Khan et al. state that Stone "obtained data from unknown pharmaceutical companies" and that the data "were confidential," framing this as a transparency concern. Both characterizations are incorrect. Stone et al. (2009) explicitly identify every product included in the analysis, and the sponsors of these products are publicly known: They are marketed pharmaceutical products whose manufacturers are a matter of public record. The data are not merely confidential; they are proprietary clinical trial data submitted to the FDA under legal obligation as part of the regulatory review process. Releasing such data to third parties without the companies' permission would violate federal law governing trade secrets and confidential commercial information. The analysis was examined and reviewed by both senior FDA officials and an advisory committee that had full access to the data but found no reason to doubt its conclusions.

The appropriate vehicle for transparency is peer-reviewed publication, which is precisely what occurred. The *BMJ*, which published Stone et al. 2009, maintains among the most rigorous peer review standards in medicine. Khan et al.'s demand for independent access to the underlying data, while ignoring the published analysis those data produced, is not a principled call for transparency; it is a rhetorical device for casting doubt on findings they have not otherwise attempted to refute.

There is also an irony in their demand. Authors who rest their central barrier claim on undocumented, unsourced, and unverifiable physician anecdote—with no transcript, no protocol, and no accounting of who said what to whom—are not well placed to insist on

Table 1
FDA-Approved Pediatric Indications for Antidepressants

Drug	Indication	Age Range
Fluoxetine	Major Depressive Disorder	8–18 Years
	Obsessive-Compulsive Disorder	7–17 Years
Escitalopram	Major Depressive Disorder	12–17 Years
	Generalized Anxiety Disorder	7–17 Years
Sertraline	Obsessive-Compulsive Disorder	6–17 Years
Fluvoxamine	Obsessive-Compulsive Disorder	8–17 Years
Clomipramine	Obsessive-Compulsive Disorder	10–17 Years
Duloxetine	Generalized Anxiety Disorder	7–17 Years
Imipramine	Childhood Enuresis	6–17 Years

independent access to data already submitted under legal obligation, published in peer-reviewed form, and reviewed by the FDA and its advisory committee. The standard of disclosure they demand of others is one they conspicuously decline to meet themselves.

The irony extends further: Having dismissed methodological critique as a technicality, they nonetheless invoke a methodological argument—the non-significance of individual drug analyses—to cast doubt on the pooled finding. However, all risk ratios shown in their table are greater than one, which means that in each case there were more suicidal events with the active drug than placebo; when combined, the excess was statistically significant. Objecting to the result of the combined analysis because the findings for the drugs considered separately are not significant is a misapplication of statistical reasoning. Non-significance in individual drugs evaluated in two to five trials each is precisely the problem that meta-analysis exists to address. When events are rare and sample sizes are small, individual studies lack the statistical power to detect real associations. Pooling data across trials and drugs is not a methodological convenience; it is the only valid way to evaluate a signal under these conditions. Khan et al.'s argument reflects a misunderstanding of why meta-analysis is conducted in the first place. Researchers who do not grasp this are not in a position to conduct the independent analysis they are requesting.

Self-refuting European evidence / Khan et al. cite Bachmann et al. (2016) in support of their European argument. Bachmann's data do the opposite. From 2005 to 2012, antidepressant (ATD) use increased markedly in both the United States and Europe, with usage rates consistently higher in the United States: "In 2012, ATD prevalence was 1.6% (US), 1.1% (UK), 1.0% (DK), 0.6% (NL) and 0.5% (DE). Increase was greatest in 10–14-year-olds (NL, UK) and 15–19 year olds (DK, DE, US)." If prescribing was rising on both sides of the Atlantic, and was highest in the United States, the boxed warning cannot explain divergent suicide trends. Once again, Khan et al. cite as support evidence that directly contradicts their own argument.

Khan et al. also argue that the European approach preserves clinical discretion while the FDA warning constrains it. This is exactly backwards. The FDA warning does not advise against prescribing; it instructs clinicians to weigh documented risk against clinical need. European labeling, by contrast, states that antidepressants should not be used to treat depression in children and adolescents under 18 except under limited circumstances. That is a direct restriction on prescribing, and one the FDA warning does not impose.

The difference is equally clear in approved indications. In Europe, only one antidepressant—fluoxetine—has a pediatric indication, and that indication is narrow: moderate to severe major depression, only after psychological therapy alone has failed after four to six sessions, and only in combination with ongoing psychotherapy. In the United States, by contrast, seven antidepressants have FDA approval for pediatric use across a range of indications (Table 1). If youth self-harm mortality declined in the European Union under this more restrictive framework, that fact cuts directly against Khan et al.'s claim that the FDA warning uniquely created harmful barriers to care.

Risk side is ignored / A proposal to remove a drug safety warning cannot be taken seriously unless it shows that the benefits of doing so outweigh the risks. Khan et al. do not come close. The confirmed risk of drug-induced suicidal behavior in young patients—an odds ratio of 2.30 in adults under 25, statistically significant across multiple methods, according to the most comprehensive analysis of the question ever conducted (Stone et al. 2009)—receives no substantive response. Khan et al. question the analysis on procedural grounds addressed elsewhere in this reply, but they do not argue the finding is wrong, and they cite no evidence that it has been superseded.

They do not apply the same scrutiny to the evidence for benefit, which is considerably weaker. Clinical trial data show that approximately 85 percent of patients with major depressive disorder derive no drug-specific benefit beyond placebo, with even weaker efficacy in children and adolescents (Stone et al. 2022). Expanding prescribing through warning removal therefore exposes more patients

to the risk of treatment-emergent suicidality without a proportionate increase in therapeutic benefit. For patients with less severe depression or non-depression indications such as generalized anxiety disorder—an increasingly common indication for adolescent prescribing—the risk of drug-induced suicidal thinking or behavior may exceed the risk posed by the disorder itself. A proposal that interrogates the risk evidence while taking the benefit evidence on faith is not a balanced policy argument.

Attribution and disclosure / Khan et al. (2026b) acknowledge that our pre-submission review was quoted without attribution and that the wording incorrectly implied current FDA affiliation. We appreciate the partial acknowledgment, but the problem was not a failure of courtesy; it was a false attribution. Quoting a passage from a private review and linking it to "the FDA" as an institution misrepresents both the source and the standing of the critique.

Khan et al. repeat the meritless claim, originating with Lu and Soumerai (2018), that Stone failed to disclose his FDA affiliation in his publications. Not only did his affiliation appear with the published pieces, the articles were written at the invitation of the journals, and the editors were fully aware of his position at FDA. In direct contradiction of this accusation, Khan et al. also suggest that those publications represented Stone speaking for the FDA. They did not; each explicitly stated that the views expressed were Stone's own and did not represent the FDA's position. As for their claim that they were unaware of the authors' current affiliations, two of the three of us were contacted by Khan et al. directly through our LinkedIn profiles and personal email addresses, not FDA addresses, channels that display institutional affiliations explicitly. Making claims when in possession of information that directly contradicts them is consistent with the pattern their responses exemplify throughout.

Finally, the characterization of the Stone et al. 2009 meta-analysis as "not designed for detecting suicidality" is incorrect. The FDA specifically requested that manufacturers submit trial data for the purpose of examining suicidality risk across the age spectrum. Every event was adjudicated using a validated, blinded assessment methodology designed precisely for that purpose. The trials provided the data; the meta-analysis provided the purpose-built analytical framework. Furthermore, Khan et al. fail to appreciate that any inaccuracy in the identification of suicidality would diminish the possibility of a positive finding, not promote a spurious one; ironically, their objection actually supports the conclusions of the study.

Conclusion / Khan et al. (2026a) began by asserting that the FDA's antidepressant boxed warning caused the deaths of thousands of American children. When examined against the evidence—including evidence they themselves cited—that assertion failed at every link in the causal chain. Their response (2026b) does not repair those failures. It introduces a delayed-effect argument incompatible with the literature they invoke, relies on a systematic review built largely from the review authors' own prior work, asserts barriers to care without evidence, and ignores most of the specific arguments we made. It asks to weaken a prominent drug safety warning without confronting the risk such a change would create for the very patients the warning is meant to protect. What remains, after the rhetoric is stripped away, is not a substantiated case for policy change, but unchecked confirmation bias: questionable evidence that confirms their conviction is accepted uncritically, while unequivocal evidence that contradicts it—the falling suicide rates during the prescribing decline, the mistiming of their own cited studies, the documented and unretracted errors—is dismissed without cogent explanation or simply ignored. The aphorism they deploy against us—"I wouldn't have seen it if I hadn't believed it"—describes their own approach with precision: For these authors, belief does not follow the evidence, it dictates which evidence is permitted to count. In the end, their position is less an argument than an act of denial running in two directions at once: The confirmed risk that the drugs themselves cause suicidality does not register, while the harms they ascribe to the warning—refuted at every step in our original response—remain to them as real as ever. They do not want to believe it, and so they do not see it.

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Defending the Endangerment Finding Rescission

In separate articles in the Summer 2026 issue of *Regulation*, Jonathan Adler and Henry Miller criticize the Environmental Protection Agency's recent rescission (EPA 2026) of its previous finding (EPA 2009) that greenhouse gas (GHG) emissions from vehicles and engines regulated under Section 202(a) of the Clean Air Act (CAA) "may reasonably be anticipated to endanger public health or welfare." Their arguments are deeply problematic.

Adler / In his article, Adler repeatedly refers to GHG emissions from "motor vehicles" rather than emissions from "new" motor vehicles (and engines); "new" in this context means not preowned. Those are what are regulated under Section 202(a). (He clearly understands that distinction in that he quotes accurately from two court decisions that refer to "new motor vehicles.") He then argues that the CAA "merely requires that the EPA administrator 'reasonably anticipate' negative effects on health or welfare, effects that the CAA defines to include effects on climate, 'economic values,' and 'personal comfort and well-being.'"

Nowhere does he ask whether those purported negative effects would satisfy a de minimis threshold, the relevance of which the EPA itself noted in its 2009 finding (p. 66506): "To be sure, any finding of a 'contribution' *requires some threshold* to be met; a truly trivial or de minimis 'contribution' might not count as such" (emphasis added). EPA (2009) then shunted that aside, stating, "The Administrator therefore has ample discretion in exercising her reasonable judgment."

The argument that GHG emissions from section 202(a) vehicles would satisfy a de minimis threshold—that "negative effects on health or welfare" are reasonable to anticipate—is preposterous. Achievement of net zero GHG emissions *by the entire US economy* by 2050 would reduce global temperatures in 2100 by about 0.104–0.137°C, applying the EPA's climate model under assumptions that exaggerate the effects of reduced GHG emissions. That impact would be virtually undetectable because the standard deviation of the surface temperature record is about 0.1°C. EPA (2009) then falsely claims that Section 202(a) vehicles emit 23.5 percent of all US GHG emissions; in fact, that is the percentage produced by *all* US vehicles. Even if we assume the 23.5 percent number, the year 2100 temperature effect of eliminating those GHG emissions would be roughly 0.024–0.032°C, an effect obviously not significant statistically, undetectable, and irrelevant in terms of policy formulation. And the effects of GHG emissions from Section 202(a) vehicles would be vastly smaller.

Adler then argues, correctly, that the 2007 Supreme Court ruling in *Massachusetts v. EPA* is the law of the land, giving the agency the authority to regulate GHG emissions. But if the EPA has authority—as distinct from a requirement—to regulate, then it also has the authority not to regulate. Adler seems to be arguing that the agency may not change its mind:

> It does not matter if the EPA (again) believes the *Massachusetts* decision was wrong and the CAA was never meant to apply to greenhouse gases. The Court has given the act a definitive interpretation to the contrary, and the Court rarely reconsiders its own prior statutory interpretations. If the Court got a statute wrong, that is a mistake for Congress to fix.

This argument conflates two distinct issues: (1) whether GHG emissions are covered by the CAA—whether the EPA has the *authority* to regulate GHG emissions—and (2) whether the EPA is *required* to do so—that is, whether GHG emissions from Section 202(a) vehicles "may reasonably be anticipated to endanger public health or welfare."

Adler then notes:

> Section 111 of the Clean Air Act ... only applies to emissions from sources that "cause or contribute significantly" to the air pollution at issue, suggesting

> the EPA need not—indeed, perhaps, cannot—regulate sources of de minimis contributions under that provision. The absence of any such qualification in Section 202, however, is a mortal blow to the EPA's position.

That argument cannot be correct. In the context of Section 202(a), "cause or contribute significantly" is replaced with "reasonably be anticipated to endanger public health or welfare." Is Adler ignoring this language, or is he actually arguing that GHG emissions yielding temperature effects almost literally equal to zero nonetheless can "reasonably be anticipated to endanger public health or welfare"? "Reasonably anticipate" as a legal matter cannot mean merely to "assert." It must mean that there is some rational basis for a finding. Is Adler arguing that the phrase "reasonably anticipate" imposes no constraint whatever?

He then argues in effect that it is not proper for an agency "to stake a major rulemaking on the Supreme Court's willingness to reject *stare decisis*." Again, he is conflating two distinct issues: (1) whether *Massachusetts v. EPA* was decided correctly, and (2) whether the 2009 EPA endangerment finding is consistent with objective analysis and the evidence both before and since. Even if we assume that *Massachusetts v. EPA* was decided correctly and will not be overturned, that has nothing to do with whether the 2009 endangerment finding itself is sound.

Adler then makes three arguments to the effect that reversing the endangerment finding would be unwise:

- He writes, "Should the *Massachusetts* holding be undone, however, placing greenhouse gases and climate change concerns beyond the scope of the law, there would no longer be any basis to bar [climate lawsuits] from federal court." Apart from the fact that reversing *Massachusetts v. EPA* is not required for a reversal of the endangerment finding, there are several such lawsuits even with *Massachusetts v. EPA*, and one of the central issues is whether they belong in state or federal court.

- He also writes, "Congress may have put an end to CAA waivers for California greenhouse gas emission standards for new motor vehicles through the Congressional Review Act, but if greenhouse gas emissions from motor vehicles are no longer subject to CAA regulation, it is not clear why any such waiver would be required, or why such standards could only be adopted by California—*though other statutes may still preclude state standards that operate as de facto fuel efficiency requirements*" (emphasis added). He here answers his own question: The Energy Policy and Conservation Act of 1975 includes an explicit preemption provision: "When an average fuel economy standard prescribed under this chapter is in effect, a State or a political subdivision of a State may not adopt or enforce a law or regulation related to fuel economy standards or average fuel economy standards for automobiles covered by an average fuel economy standard under this chapter." The only way to control vehicle GHG emissions is with fuel economy standards, whether direct or indirect.

- Adler further writes: "The endangerment finding itself is no obstacle to the relaxation or rescission of existing greenhouse gas emission regulations, including those imposed on motor vehicles. The relevant statutory text provides ample basis for ending regulation of such emissions from stationary sources, and there are strong legal arguments having nothing to do with endangerment that the EPA lacks the statutory authority to push automakers toward the production and promotion of electric cars." He cannot be serious; with the 2009 endangerment finding in force, efforts to end regulation of GHG emissions will confront litigation monstrous in scale and effect. Why else would we now observe massive litigation against the 2026 rescission?

Miller / Miller begins his argument with a reference to the "robust" "scientific consensus on anthropogenic climate change." Apart from the reality that the phrase "scientific consensus" is a contradiction in terms, only someone who knows little about the literature on anthropogenic climate change can believe such balderdash. Merely consider the recent official United Nations Intergovernmental Panel on Climate Change disavowal of the most extreme GHG scenarios (the most important of which is "representative concentration pathway 8.5, or RCP8.5); until now they commonly were defined as "business as usual," but now they "have become implausible." In the Sixth Assessment Report (AR6), every adverse climate effect of increasing GHG concentrations viewed by the IPCC as likely for 2050 or 2100 was driven by RCP8.5 (Table 12.12 of the "Physical Science Basis" chapter), even though the IPCC itself in the AR6 argued that "the likelihood of high emissions scenarios such as RCP8.5 or SSP5-8.5 is considered low."

Note first that the EPA (2009) analysis of future GHG emission scenarios is characterized by some nontrivial confusion: "Atmospheric levels of greenhouse gases will … continue to climb, and thus lead to ever greater rates of climate change." The asserted parameter "rates of climate change" is wholly unclear, but to the extent that it means some sort of acceleration in the observed effects of additional emissions, it is incorrect because the effects of additional emissions are smaller at higher atmospheric concentrations of GHG than at lower ones, and at current concentrations are effectively zero.

EPA (2009) states, "For the year 2030, projections of the six greenhouse gases

show an increase of 25 to 90 percent compared with 2000 emissions." The European Union's Emissions Database for Global Atmospheric Research (EDGAR) shows global GHG emissions (in carbon dioxide–equivalent units) in 2000 at 36,175.2 million metric tons, and 53,206.4 mmt in 2024. That is an increase of 47.1 percent. But, as EPA (2009) recognized, it is atmospheric concentrations that are relevant for anthropogenic climate change. Data from the National Oceanic and Atmospheric Administration's Global Monitoring Laboratory show an increase in atmospheric concentrations of carbon dioxide from 369.7 parts per million in 2000 to 427.4 ppm in 2025, or 15.6 percent. An increase of 25 percent from 2000 to 2030 would require carbon dioxide concentrations that year of about 462.1 ppm, or 34.7 ppm higher than in 2025. That implies an annual average increase over 2025–2030 of over 6.9 ppm. The single largest increase in the NOAA historical data was 3.5 ppm in 2024.

Accordingly, it is obvious that the EPA (2009) GHG scenarios have proven substantially incorrect in the context of the ensuing data, and that an endangerment finding based upon them cannot be supported. In a detailed analysis, Pielke (2025) examines the nine scenarios used in the 2009 endangerment finding. The radiative forcings implied by the nine scenarios range from 4.2°C to 9.2°C. Pielke concludes:

> These nine scenarios are heavily skewed to very high levels of 2100 radiative forcing, with two even more extreme than RCP8.5. Eight of the nine project a central estimate of global average temperature increase to 2100 (above pre-industrial) of greater than 3 degrees Celsius—a value today viewed to be unlikely.

Notwithstanding Miller's assertions, there is no evidence—none—of a climate "crisis." More to the point, Miller fails to examine the projections of climate phenomena used by EPA (2009) to justify its finding that GHG emissions—from Section 202(a) vehicles—can "reasonably be anticipated to endanger public health or welfare." Every one of them is inconsistent with the evidence both in the years leading to 2009 and after.

Ozone ambient levels / According to EPA (2009), "Increases in ambient ozone are expected to occur over broad areas of the country." But the EPA website currently shows a 26 percent *decline* in the ozone national trend for 1980–2009, and ambient ozone levels have declined since then (EPA 2026).

Hurricanes and floods / EPA (2009) states, "The evidence concerning how human-induced climate change may alter extreme weather events also clearly supports a finding of endangerment, given the serious adverse impacts that can result from such events and the increase in risk, even if small, of the occurrence and intensity of events such as hurricanes and floods." Further, "there is the potential for hurricanes to become more intense (and even some evidence that Atlantic hurricanes have already become more intense)."

Projections of future weather events—EPA (2009) uses "may alter"—are not "evidence." With respect to hurricanes, the satellite data show no long-term trends either in numbers or intensities since 1973 (Maue, n.d.). With respect to the intensity of Atlantic hurricanes, the data show an increase since the late 1960s, but that clearly is driven by three outlier years: 2005, 2010, and 2020 (Buchholz 2025). Accordingly, it is difficult to believe that the increase is statistically significant, and EPA (2009) had only the 2005 data available; does Miller believe that one year of observations is sufficient to draw inferences about climate phenomena? And the EPA offered no analysis of the degree to which anthropogenic emissions (or increasing atmospheric concentrations) of GHG were responsible; that would be impossible based upon one year of data. Does Miller believe that there are no natural variations in the intensities of Atlantic hurricanes?

With respect to flooding, the IPCC's AR6 (p. 1568) states: "The SREX (Seneviratne et al., 2012) assessed low confidence for observed changes in the magnitude or frequency of floods at the global scale. This assessment was confirmed by AR5 (Hartmann et al., 2013)." For the United States, flooding over the past century is uncorrelated with increasing GHG concentrations (Hirsch & Ryberg 2011).

Heat waves / EPA (2009, p. 66497) refers to "increases in average temperatures, which increase the likelihood of heat waves." The data show little trend in the number of "hot" days over the period 1895–2017; 11 of the 12 years with the highest number of such days occurred before 1960 (Spencer 2018, Fig. 5). In the EPA heat wave index, there is a sharp increase during the 1930s, but no other trend is visible for the 1895–2021 period (OWID, n.d.).

Standard atmospheric physics theory does not predict that increasing atmospheric concentrations of GHG will yield an increase in average (or record) high temperatures. Instead, we should expect to see a rise in average low temperatures, essentially because the warming effects for the most part are predicted for the coldest, driest air masses in the northern hemisphere—eastern Asia and western North America—in the winter. Koonin (2024) notes that for the United States for 1900–2019, "the average coldest temperature of the year has clearly increased since 1900, while the average warmest temperature has hardly changed over the last sixty years and is about the same today as it was in 1900."

Since 2005, NOAA has maintained the US Climate Reference Network, comprising 114 meticulously maintained temperature stations spaced more or less uniformly across the lower 48 states, 21 stations in Alaska, and two stations in Hawaii. They are placed to avoid heat island effects and other such distortions as much as possible. The reported data show no trend over the

available 2005–2025 period.

Net mortality & morbidity / According to EPA (2009):

> The impact on mortality and morbidity associated with increases in average temperatures, which increase the likelihood of heat waves, also provides support for a public health endangerment finding. There are uncertainties over the net health impacts of a temperature increase due to decreases in cold-related mortality, but some recent evidence suggests that the net impact on mortality is more likely to be adverse, in a context where heat is already the leading cause of weather-related deaths in the United States.

Yet, EPA (2009) failed to say what that "recent evidence" is. *The Lancet* has published three major studies on net mortality caused by heat and cold (Gasparrini et al. 2015; Zhao et al. 2021; Masselot et al. 2023). Each found vastly larger net mortality from cold. Accordingly, any asserted increase in mortality caused by higher temperatures—which to some degree are driven by some combination of anthropogenic and natural phenomena—must be offset by reductions in net mortality from reduced cold. Walkowiak et al. (2025) found that "colder-than-optimal temperatures had a greater effect on mortality than warmer ones." Referring to several studies, Lomborg (2016) notes, "In the U.S. about 9,000 people die from heat each year but 144,000 die from cold."

Wildfires, flooding, drought / EPA (2009) states, "Across the sectors, the potential serious adverse impacts of extreme events, such as wildfires, flooding, drought, and extreme weather conditions provide strong support for" an endangerment finding. With respect to wildfires, the annual number of US wildfires shows no trend since 1985 (NIFC, n.d.). The number of US wildfires has declined sharply since 1926, and acreage burned has declined even more sharply since the preindustrial period 1500–1800, but it has increased since 1983 (NIFC 2001). That latter trend is likely the result of perverse forest management practices, particularly in government forests (CBO 2022). Could Miller, or anyone, explain why increasing atmospheric concentrations of GHG would engender wildfire effects vastly greater in government forests than in private ones?

Global acreage burned declined 24 percent for 1998–2015, as reported by NASA (Ramsayer 2017). On global wildfires, Doerr and Santin (2016) concluded:

> The data available to date, however, do not support a general increase in area burned or in fire severity for many regions of the world. Indeed, there is increasing evidence suggesting that there is overall less fire in the landscape today than there has been centuries ago, although the magnitude of this reduction still needs to be examined in more detail.

With respect to drought, the Palmer Drought Severity index for the contiguous 48 states shows no trend since 1895. Vicente-Serrano et al. (2022) report, "Meteorological droughts do not show any substantial changes at the global scale in at least the last 120 years."

Food production / EPA (2009) claims, "The body of evidence points towards increasing risk of net adverse impacts on U.S. food production and agriculture over time, with the potential for significant disruptions and crop failure in the future." However, FAO (n.d.) reports that global per-capita food production increased by over 40 percent from 1980 to 2024. In the United States, production increased 13.9 percent.

Water quality / According to EPA (2009), "Water resources across large areas of the country are at serious risk from climate change, with effects on water supplies, water quality, and adverse effects from extreme events such as floods and droughts." GAO (n.d.) offers several studies of water quality trends for the United States, and there is no simple answer to the question of whether those trends are positive or negative because there are many variables (salinity, sediment concentrations, contaminants, etc.) to consider. Moreover, many such variables have little or nothing to do with anthropogenic climate change. At the most general level, GAO (n.d.) reports, "Over the past 50 years, the nation's water quality and drinking water have improved, but threats to water quality and safety remain." There is no support in official analyses that "water resources across large areas of the country are at serious risk from climate change."

Sea levels & coastal storms / According to EPA (2009), "Public health is expected to be adversely affected by an increase in the severity of coastal storm events due to rising sea levels." Further, "The evidence concerning adverse impacts in the areas of water resources and sea level rise and coastal areas provides the clearest and strongest support for an endangerment finding, both for current and future generations."

A recent US Department of Energy draft report (subsequently withdrawn for legal reasons) provides the latest evaluation of sea levels along US coasts (Climate Working Group 2025). It offers this summary analysis:

> Since 1900, global average sea level has risen by about 8 inches. Sea level change along U.S. coasts is highly variable, associated with local variations in processes that contribute to sinking and also with ocean circulation patterns. The largest sea level increases along U.S. coasts are Galveston, New Orleans, and the Chesapeake Bay regions—each of these locations is associated with substantial local land sinking (subsidence) unrelated to climate change. Extreme projections of global sea level rise are associated with an implausible extreme emissions scenario and inclusion of poorly understood processes associated

with hypothetical ice sheet instabilities. In evaluating AR6 projections to 2050 (with reference to the baseline period 1995–2014), almost half of the interval has elapsed by 2025, with sea level rising at a lower rate than predicted. U.S. tide gauge measurements reveal no obvious acceleration beyond the historical average rate of sea level rise.

These data do not support the EPA (2009) assertion that "the most serious potential adverse effects are the increased risk of storm surge and flooding in coastal areas from sea level rise and more intense storms."

The "social cost of carbon" is a dishonest parameter used to justify policies with undetectable effects.

Miller complains that the EPA now has redefined "air pollutant," which is an irrelevant argument in that the issue is not whether GHG emissions are "pollutants," but instead whether they can "reasonably be anticipated to endanger public health or welfare." As an aside, a certain minimum atmospheric concentration of carbon dioxide, generally assumed to be about 150 ppm, is necessary for life itself. At what higher concentration does carbon dioxide suddenly become a "pollutant?" Miller does not tell us. Nor does he tell us why water vapor—by far the most important GHG in terms of the radiative properties of the troposphere—is not also a "pollutant." Is it because ocean evaporation is a natural process? So are volcanic eruptions, the emissions from which are "pollutants" by any definition.

Miller's assertion that "Section 202(a) explicitly applies to motor vehicle emissions, as they are a large source of air pollutants," is incorrect: Section 202(a) applies only to "new" vehicles and engines. Somewhat inconsistently, he then asserts that the trivial effect of Section 202(a) GHG emissions—or all vehicle emissions or all US emissions—is irrelevant because "climate change is an inherently cumulative phenomenon." This is irrelevant: The issue is not whether all global GHG emissions can "reasonably be anticipated to endanger public health or welfare." The issue is whether emissions subject to regulation under the CAA satisfy that legal requirement. The obvious answer is "No."

Miller then endorses the "social cost of carbon" parameter (SC-GHG) as "substantial by virtually any serious economic accounting." This is more balderdash. Merely by raising the analytic discount rate to 5 or 7 percent from the absurd 2.5 percent figure used by the Biden administration *while changing no other parameter*, the SC-GHG falls from about $125 per metric ton to about $35 and less than $7, respectively. At $35, the social cost of GHG emissions per gallon of gasoline would be about 31¢; at $7 it would be about 6¢. Are those "social costs" "substantial"?

Even that ignores such benefits of increasing carbon dioxide concentrations as the net decline in mortality from heat and cold, increased agricultural productivity, and planetary greening. For many reasons, the "social cost of carbon" is a fundamentally dishonest parameter (Zycher 2025); it has been used to justify policies that yield undetectable climate effects.

Note also that the SC-GHG as estimated by the Biden administration mischaracterizes the effects on gross domestic product of rising GHG concentrations as projected in the central integrated assessment models; they are much smaller than commonly asserted. In the Dynamic Integrated Climate and Economy (DICE) Model, for which William Nordhaus won the Nobel Prize in Economics in 2018, global GDP in 2100 varies by about 3 percent across policy scenarios, including no climate policies at all—a figure that is both very small and almost certainly not statistically significant given the variations inherent in economic forecasting, the magnitude of annual changes in global economic growth, and the number of years remaining before the end of this century. Per-capita consumption varies only by about 1.3 percent across policy scenarios—also a very small number and almost certain not to be statistically significant.

The IPCC—even in its most alarmist analyses—arrives at a conclusion very close to that reported in the DICE analysis. IPCC (2018, Ch. 3) finds that the damage from anthropogenic climate change unmitigated by policy initiatives will reduce global GDP by 2.6 percent by 2100. In other words, if we assume, conservatively, global GDP growth of, say, 2 percent per year, climate change unmitigated by policy initiatives would shift the global GDP growth path backward by about 15.6 months, an approximate reduction magnitude observed commonly during economic recessions. By 2100, the IPCC projects that individual incomes on average will be at least 400 percent greater than is the case today, so that climate change unmitigated by policy initiatives would make individuals in 2100 "only" 398 percent wealthier than individuals today.

Miller's argument that "Policy Should Be Based on Science, Not Politics" is confused. Science, whatever the disagreements and controversies, can tell us about the workings of the natural world; it tells us nothing about the unavoidable tradeoffs inherent in policymaking. Policymaking is, therefore, political by its very nature. His assertion that "the 2009 endangerment finding did not emerge from ideology" is comedy gold: That politics did not infect decision-making by an Obama EPA led by Lisa Jackson is preposterous. But Miller, without realizing it, has debunked "attribution science," arguing that EPA (2026) "has constructed a legal standard that demands what atmospheric physics cannot deliver: a clean, isolated causal chain from one vehicle category to a measurable global outcome."

Conclusion / The 2026 EPA rescission of the 2009 EPA endangerment finding is fully justified. The 2009 analysis was confused in several dimensions, dishonest, inconsistent with the data on climate phenomena before and after 2009, and inconsistent with the EPA's own climate model. If the phrase "arbitrary and capricious" does not apply to the 2009 endangerment finding, then it is devoid of meaning. The dissenting arguments from Adler and Miller, respectively, do nothing to challenge that conclusion.

—Benjamin Zycher, senior fellow, American Enterprise Institute, and member, REGULATION *Editorial Advisory Board*

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Errata

In "Reconsidering Ivermectin" (Spring 2026), we made two errors of attribution. We wrote:

> The Popp et al. meta-analysis included four of the Knackered Nine clinical studies in Table 1: Buonfrate et al. (2022), López-Medina et al. (2021), Reis et al. (2022), and Vallejos et al. (2021). This shouldn't be surprising when we consider that the Popp et al. authors include some of the authors of the Knackered Nine, such as Stefanie Reis and Stephanie Weibel. Reis was the lead author of the TOGETHER Trial study; Weibel was a coauthor of Buonfrate et al. In fairness to the authors, it's natural to include your own study, but it's also natural to overlook the weaknesses of your own study. It's important to note that the TOGETHER Trial (Reis et al.) was both big and bad, raising serious questions about its final results.

While we correctly cited the Reis et al. article, we incorrectly claimed that Stefanie Reis was one of its authors. In fact, the author was Gilmar Reis, something we should have noticed. That mistake was understandable. Less understandable is our claim that Stephanie Weibel was one of the many co-authors of the Buonfrate et al. study. She was not. These two errors substantially undercut our implicit claim that the authors of the Cochrane study had a conflict of interest.

We thank Pierre Crot Gallaz for pointing out our errors.

—Charles L. Hooper, president, Objective Insights; and David R. Henderson, research fellow, Hoover Institution

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