January 31, 2018

Jennifer Francis, Ph.D.
Vice Provost for Academic Affairs
Duke University
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Re: Research Misconduct Complaint Against Duke University Professor Junfeng Zhang

Dear Vice Provost Francis,

This is a formal research misconduct complaint against Duke Professor Junfeng Zhang (Zhang) of the Nicholas School of the Environment (https://nicholas.duke.edu/people/faculty/zhang), the Duke Global Health Institute (https://globalhealth.duke.edu/people/faculty/zhang-jim), and the Duke Kunshan University (https://dukekunshan.edu.cn/en/faculty). I have overwhelming evidence that Zhang falsely claimed a positive relationship between fine particulate matter ($PM_{2.5}$) and total mortality in the United States in his attached December 26, 2017 JAMA Editorial "Low-Level Air Pollution Associated With Death: Policy and Clinical Implications" JAMA 2017;318(24):2431-2432 doi:10.1001/jama.2017.18948 (https://jamanetwork.com/journals/jama/article-abstract/2667043).

Zhang completely ignored the null evidence that I have published since 2005 and most recently summarized in my attached October 12, 2017 NEJM Letter "Air Pollution and Mortality in the Medicare Population" N Engl J Med 2017;377:1497-1499 DOI:10.1056/NEJMc1709849 (http://www.nejm.org/doi/full/10.1056/NEJMc1709849). In addition, on November 27, 2017, virtually the same date that JAMA accepted the Zhang Editorial, JAMA rejected my November 22, 2017 manuscript with new evidence of NO $PM_{2.5}$ premature deaths in the ACS CPS II cohort. Although my null findings directly contradict the positive findings in a seminal 2002 JAMA article, JAMA refused to assess my findings with internal or external peer review. A summary of my null findings, as well as the JAMA rejection letter are attached.

I allege that Zhang's Editorial satisfies the definition of "falsification" as stated in Public Health Service Policies on Research Misconduct: "omitting or changing data such that the Research is not accurately represented in the Research Record." Although there is falsification throughout his Editorial, this initial complaint is limited to Zhang's falsification in two concluding sentences: "In 2015, 107 million and 23 million people lived in US counties where air quality did not meet the standards for ozone and $PM_{2.5}$, respectively.[9] While efforts are needed to bring these nonabatement counties into compliance with the current NAAQS, regulators should continue to consider emerging scientific evidence such as that reported by Di et al[2] and should further lower the standards to minimize health risks."

Zhang's sentences deliberately misrepresent the Research Record. Based on Zhang's Curriculum Vitae, particularly his publications and his research funding, he must be very familiar with intense controversy that has existed for 25 years regarding the claim that $PM_{2.5}$ causes premature deaths in the United States. He even teaches the relevant Duke University course "GLHLTH 634: Air Quality: Human Exposure and Health Effects" (https://scholars.duke.edu/display/courseGLHLTH634). However, his Editorial does not mention the intense controversy or the evidence that invalidates his above claims.

Zhang's falsification is illustrated by the *italicized* words in this sentence: "While efforts are *needed* to bring these nonabatement counties into compliance with the current NAAQS, *regulators* should continue to consider emerging scientific evidence such as that reported by Di et al[2] and *should further lower* the standards to minimize health risks." Based on his Reference 9, 19.6 million (85%) of the 23 million people in US counties that do not meet the $PM_{2.5}$ NAAQS live in California, where Enstrom 2017 (cited in my October 12, 2017 NEJM letter) presents overwhelming evidence published during 2000-2017 that there are NO $PM_{2.5}$ premature deaths and NO public health justification for the $PM_{2.5}$ NAAQS. If regulators consider ALL the existing and emerging evidence, such as, the evidence cited in my October 12, 2017 NEJM letter, they will find that there is NO conclusive evidence that further lowering of the NAAQS will minimize health risks. Zhang must know about the null $PM_{2.5}$ evidence in California, as well as the aggressive CARB and SCAQMD $PM_{2.5}$ regulations in California, because he was a USC Professor of Medicine during 2010-2013, when I was repeatedly presenting this null evidence in California. The overwhelming evidence that there are NO $PM_{2.5}$-related premature deaths in California or the US must be examined and cited.

Zhang did not acknowledge that the Di et al evidence on $PM_{2.5}$ premature deaths in the Medicare population (as published in the June 29, 2017 NEJM and December 26, 2017 JAMA articles by Di et al) is severely flawed, as explained by Steve Milloy, JD, in his requests for retraction dated July 5, 2017 to the NEJM Editor (https://junkscience.com/2017/07/retraction-request-made-for-nejm-air-pollution-kills-study/) and dated January 4, 2018 to the JAMA Editor (https://junkscience.com/2018/01/junkscience-com-requests-jama-retract-new-harvard-pm2-5-study-on-basis-of-scientific-misconduct/). In addition, research misconduct complaints against Di et al have been filed with the US Office of Research Integrity by Mr. Milloy on September 5, 2017 (https://junkscience.com/2017/09/junkscience-requests-federal-research-misconduct-investigation-for-air-pollution-study/) and by John D. Dunn, MD, JD, on January 30, 2018.

Furthermore, Zhang knows there is ***no etiologically plausible or proven mechanism*** by which inhaling the current average US level of about 40 micrograms of $PM_{2.5}$ per day (about 1 gram per lifetime) can cause premature death. In addition, he knows that the weak observational epidemiologic evidence in Di et al ***does not justify*** the multi-billion-dollar EPA-CARB-SCAQMD $PM_{2.5}$ regulations that have adversely impacted millions of American businessmen during the past 20 years and have driven millions of blue collar jobs from the US to China. Indeed, Zhang wrote a January 2015 article with USC Professor Jonathan M. Samet on this very subject: "Chinese haze versus Western smog: lessons learned," J Thorac Dis. 2015 Jan;7(1):3-13. doi: 10.3978/j.issn.2072-1439.2014.12.06 (http://jtd.amegroups.com/article/view/3573/html).

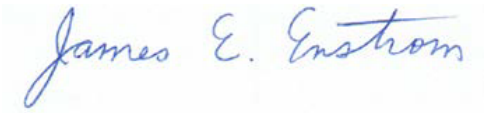
The Abstract of Zhang's article is attached and it includes these sentences "Air pollution in many Chinese cities has been so severe in recent years that a special terminology, the 'Chinese haze', was created to describe China's air quality problem. . . . Hence it is important to provide a global and historical perspective to help China combat the current air pollution problems." Key sentences in this article include: "One of the consequences of the increased stringency of pollution control regulations in these [Western] countries is the outsourcing of more polluting manufacturing jobs into the low- and middle-income countries by multi-national corporations. In fact, China has been recognized as 'the world's factory' in the last few decades. . . . Daily $PM_{2.5}$ concentrations [in China] exceeded 100 $\mu g/m^3$ for more than half of the days and reached as high as 744 $\mu g/m^3$, more than 20 times the US Environmental Protection Agency's (EPA) 24-hour standard for $PM_{2.5}$ of 35 $\mu g/m^3$."

Both Duke University and the Harvard TH Chan School of Public Health have close ties with China and Chinese scientists like Zhang, Di, and numerous others. These scientists exaggerate and falsify the health and mortality effects of air pollution in the US and promote unjustified regulations in the US. However, they know full well that air pollution is much worse in China and that stricter regulations need to be enforced in China, not in the US. Zhang must be held accountable for falsifying the Research Record in this Editorial.

Please acknowledge receipt of my complaint against Zhang and issue a formal assessment.

Thank you very much.

Sincerely yours,



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EDITORIAL

Low-Level Air Pollution Associated With Death
Policy and Clinical Implications

Junfeng Zhang, PhD

Globally, an estimated 3.3 million annual premature deaths (5.86% of global mortality) are attributable to outdoor air pollution,[1] although ambient air pollution has been regulated under national laws in many countries. In the United States under the Clean Air Act, the primary National Ambient Air Quality Standards (NAAQS) are intended to protect human health, with an adequate margin of safety, including sensitive populations such as children, older adults, and individuals with respiratory diseases. Under the Clean Air Act, the standards are reviewed every 5 years to account for new scientific evidence regarding their appropriateness and adequacy for protecting public health.

Related article page 2446

Historically, this science-based review process has resulted in continued evolution of the NAAQS. For example, an annual and 24-hour standard for fine particulate matter ($PM_{2.5}$) and an 8-hour standard for ozone were added in 1997. The 24-hour $PM_{2.5}$ standard was lowered from 65 μg/m^3 in 1997 to 50 μg/m^3 in 2006. The 8-hour ozone standard was lowered from 0.08 parts per million (ppm) in 1997 to 0.075 ppm in 2008 and then to 0.070 ppm in 2015. At the next review of NAAQS for $PM_{2.5}$ and ozone, new scientific evidence will be evaluated in recommending whether the current standards should be revised.

In this issue of *JAMA*, Di et al[2] report findings that day-to-day changes in $PM_{2.5}$ and ozone ambient concentrations were significantly associated with higher risk of all-cause mortality at levels well below the current daily NAAQS. Using a case-crossover design and conditional logistic regression analysis in a data set involving 22 million deaths among US Medicare participants during 2000-2012, the authors estimated that a 10-μg/m^3 increase in $PM_{2.5}$ and a 10-parts-per-billion increase in warm-season (ie, between April 1 and September 30) ozone in the 2 days prior to death were, respectively, associated with a 1.05% (95% CI, 0.95%-1.15%) and 0.51% (95% CI, 0.41%-0.61%) increase in daily mortality rate. The authors also identified susceptible subgroups, reporting that nonwhite individuals, Medicaid-eligible individuals, women, and adults 85 years and older had significantly higher mortality risk associated with increased $PM_{2.5}$ levels and that individuals aged from 75 to 84 years and 85 years and older had higher mortality risk associated with increased ozone levels. Importantly, the authors did not find evidence of a threshold in the exposure-response relationship for either pollutant, suggesting that there is no "absolute" safe level of exposure to $PM_{2.5}$ or ozone.

The Medicare cohort used in this study includes individuals residing in rural areas without nearby air pollution monitors, but the authors were able to estimate exposure to $PM_{2.5}$ and ozone using predictive models of data from remote air monitors, satellite-based measurements, and other data sets.[2] Pollutant concentrations in rural areas are generally lower than in urban areas. The findings from this study add unique evidence, applicable to both rural residents and more vulnerable groups, to raise public awareness concerning health risks associated with low-level $PM_{2.5}$ and ozone pollution. The findings suggest that the current NAAQS for these pollutants should be reevaluated.

The findings from this epidemiological investigation by Di et al[2] are supported by mechanistic insights from recent studies of pathophysiological responses to $PM_{2.5}$ and ozone exposure. It is now well accepted that short-term exposure to $PM_{2.5}$ has cardiorespiratory effects through increased pulmonary and systemic inflammation, increased oxidative stress, enhanced thrombogenesis, and autonomic dysfunction.[3] At relatively high concentrations, ozone impairs lung function and increases the incidence of asthma attacks. As a highly reactive oxidant, ozone has long been considered to mainly affect the respiratory system. However, a recent study showed that at levels below those capable of causing lung function changes, ozone is associated with increases in pulmonary inflammation, blood pressure, and platelet activation (a risk factor for thrombosis).[4] Rodent studies show that ozone compromises immune function against bacterial infection.[5] Not only do these mechanistic studies support the biological plausibility of exposure-mortality associations, such as those found by Di et al,[2] but they also provide insights for potential "therapeutic" interventions. For instance, a limited number of studies suggested that antioxidant supplementation may reduce the effects of $PM_{2.5}$ or ozone.[6] More intervention trials should be conducted to examine the efficacy of using dietary supplementation, medications, or personal protective equipment in alleviating the adverse health effects of air pollution in the general population and particularly in more susceptible populations.

The findings of Di et al[2] may have implications for forecasting and personal monitoring of exposure to $PM_{2.5}$ and ozone, which could allow individuals at increased risk to reduce or mitigate their exposure. The study showed that when $PM_{2.5}$ or ozone concentration was higher on a particular day, more deaths occurred 2 days later. Predictions of pollutant concentrations for the next few days, such as weather forecasting, can be made readily available to the public. (For example, this has already

been done in China.) Individuals can be advised to minimize their outdoor activities when outdoor pollutant levels are projected to be higher. However, staying indoors may be more helpful in avoiding exposure to ozone than to $PM_{2.5}$ because less than 30% of ambient ozone penetrates indoor spaces when windows and doors are closed, whereas more than 80% of $PM_{2.5}$ enters the indoor space in the absence of an air cleaning device such as central or room filtration.

In the study by Di et al,[2] several subgroups of Medicare recipients, including nonwhite individuals, women, Medicaid-eligible individuals, and older adults (>70 years) were found to have increased susceptibility to $PM_{2.5}$ and ozone. These susceptibility factors should be considered in developing personalized protection strategies, such as staying indoors on heavy pollution days and during exacerbations of underlying respiratory conditions, and wearing personal protective equipment, such as N95 face masks and respirators when outdoors.[7] Individuals at increased risk may also wish to avoid places such as heavily polluted city streets.[8] Furthermore, with rapid technological advancements, it becomes increasingly feasible to use low-cost, light-weight pollutant monitors in residences and workplaces or to be worn by individuals. Such exposure data can be integrated into a mobile health platform as part of an overall health management plan to achieve maximal risk reductions.

Such individual-level protections, however, are only a complement to the ultimate solution of emission controls. In 2015, 107 million and 23 million people lived in US counties where air quality did not meet the standards for ozone and $PM_{2.5}$, respectively.[9] While efforts are needed to bring these nonabatement counties into compliance with the current NAAQS, regulators should continue to consider emerging scientific evidence such as that reported by Di et al[2] and should further lower the standards to minimize health risks. Some may argue that it would be too costly to make further improvements in air quality when pollution levels are relatively low. However, pollution controls required by the Clean Air Act have been associated with preventing an estimated hundreds of thousands of premature deaths and with estimated economic benefits exceeding the costs.[10] It can be assumed that even greater health benefits could result from further emission reductions, which can be achieved through cleaner energy production (eg, by renewable, nonpolluting sources such as wind and solar power) and a cleaner transportation fleet (eg, with electric and hybrid vehicles and low-emission mass transportation).

ARTICLE INFORMATION

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Conflict of Interest Disclosures: The author has completed and submitted the ICMJE Form for Disclosure of Potential Conflicts of Interest. Dr Zhang reported receiving funding from Underwriters Laboratories Inc in the form of a research contract to Duke University to support a study of the health impact of using air purifiers in the bedrooms of children with asthma in Shanghai, China. From 2012 to 2017, he served as a member of the Oxides of Nitrogen Primary NAAQS Review Panel for the US Environmental Protection Agency. He has also received an honorarium for attending a global advisory board meeting on air pollution from the RB Company in London, England.

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No potential conflict of interest relevant to this letter was reported.

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DOI: 10.1056/NEJMc1709849

TO THE EDITOR: The article by Di et al. contains weak noncausal evidence that $PM_{2.5}$ is related to total mortality in the Medicare population. It does not cite the previous evidence reported by Zeger et al.[1] of a large, unexplained geographic variation in the risk of death associated with $PM_{2.5}$ and of no risk of death associated with $PM_{2.5}$ if the risk is based on a local regression coefficient that indicates the association between location-specific trends in pollution and mortality, as described in the detailed statistical analysis reported by Greven et al.[2]

The article by Di and colleagues also does not cite recent data showing no risk of death associated with $PM_{2.5}$ in the National Institutes of Health–American Association of Retired Persons Diet and Health Study cohort[3] and the Cancer Prevention Study cohort.[4] We think that before the findings of the federally funded study by Di et al. are accepted as valid, the underlying Medicare data should be analyzed independently in accordance with the HONEST (Honest and Open New EPA Science Treatment) Act.[5]

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No potential conflict of interest relevant to this letter was reported.

1. Zeger SL, Dominici F, McDermott A, Samet JM. Mortality in the Medicare population and chronic exposure to fine particulate air pollution in urban centers (2000-2005). Environ Health Perspect 2008;116:1614-9.
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5. H.R. 1430, Honest and Open New EPA Science Treatment (HONEST) Act of 2017 (http://www.govtrack.us/congress/bills/115/hr1430/text).

DOI: 10.1056/NEJMc1709849

THE AUTHORS REPLY: In response to Majeed and Majeed: our definition of warm-season ozone is consistent with that in the study by Jerrett et al., the results of which were also published in the *Journal*.[1] Although ozone levels peak over the summer, in recent decades, summer ozone levels have decreased, but spring and autumn ozone levels have increased. Using a statistical approach for causal inference, we have previously shown that exposure to high levels of ozone in the spring, summer, and fall is associated with an increased risk of death.[2]

In response to Raymond: given that there is no threshold for the relationship between $PM_{2.5}$ and mortality, any reduction in air pollution is beneficial. Establishing a restriction on diesel idling would reduce air pollution without cost.

Enstrom points to some studies with null findings that we did not cite. Our conclusions would not have changed on the basis of which of the hundreds of studies of air pollution we might have cited. This is because our study is not a meta-analysis. It is an analysis of new nationwide data and an assessment of exposure with high spatial resolution (i.e., daily $PM_{2.5}$ and ozone concentrations for nationwide grids that were 1 km by 1 km), and we reported strong, not weak associations. Sensitivity analyses showed that smoking and socioeconomic status are unlikely to confound the association, and we controlled for spatial variation (see the Supplementary Appendix, available with the full text of our article at NEJM.org). Moreover, meta-analyses of all published cohort studies show strong, robust associations of $PM_{2.5}$ with mortality,[3] and two recent studies have shown similar associations with the use of causal modeling techniques.[4,5] The Medicare beneficiary denominator file from the Centers for Medicare and Medicaid Services is a publicly available data source, and therefore this study can be independently replicated.

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Since publication of their article, the authors report no further potential conflict of interest.

Air Pollution and Total Mortality in Cancer Prevention Study Cohort Reanalysis

James E. Enstrom, Ph.D., M.P.H., FACE

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November 22, 2017

Text Word Count = 2,950

Key Points

Question: Is fine particulate matter ($PM_{2.5}$) related to total mortality in the 1982 American Cancer Society Cancer Prevention Study (CPS II) Cohort?

Findings: Independent reanalysis found that $PM_{2.5}$ and SO_4^{2-} had no significant relationship with total mortality during 1982-1988 in the CPS II cohort when the best available $PM_{2.5}$ and SO_4^{2-} data were used. Furthermore, this reanalysis found several other null findings that challenge the validity of the positive findings in a seminal 2002 *JAMA* article.

Meaning: There is urgent need for complete reanalysis of the CPS II data used to justify establishment of the 1997 EPA National Ambient Air Quality Standard for $PM_{2.5}$.

From: robert.golub@jamanetwork.org [mailto:robert.golub@jamanetwork.org]
Sent: Tuesday, November 28, 2017 4:10 AM
To: jenstrom@ucla.edu
Cc: Dawn.Cortez@jamanetwork.org; jim_ens@hotmail.com
Subject: JAMA17-9910 Decision Letter

November 27, 2017

Dr James E Enstrom
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RE: **Air Pollution and Total Mortality in Cancer Prevention Study Cohort Reanalysis**

Dear Dr Enstrom:

Thank you for submitting your manuscript to JAMA. Each manuscript is thoroughly evaluated by the JAMA editorial staff, who assess the manuscript's quality and its priority for publication. Those manuscripts judged unlikely to succeed through stringent external review or whose subject matter does not meet our current editorial priorities are rejected at that point.

More than half of the approximately 7000 manuscripts submitted to us annually are rejected after such in-house review, and less than 9% of manuscripts are eventually accepted for publication in JAMA. Based on our evaluation, I regret to inform you that we will not pursue the manuscript you have submitted for publication, and we will not be referring it to JAMA Internal Medicine.

While we realize that you may be disappointed with our decision, we hope that providing you with this information promptly will allow you to submit your manuscript to another journal without the delay entailed by the external review process.

Thank you for the privilege of reviewing your work.

Sincerely yours,

Robert M Golub, MD
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Review Article

Chinese haze versus Western smog: lessons learned

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Abstract: Air pollution in many Chinese cities has been so severe in recent years that a special terminology, the "Chinese haze", was created to describe China's air quality problem. Historically, the problem of Chinese haze has developed several decades after Western high-income countries have significantly improved their air quality from the smog-laden days in the early- and mid-20th century. Hence it is important to provide a global and historical perspective to help China combat the current air pollution problems. In this regard, this article addresses the followings specific questions: (I) What is the Chinese haze in comparison with the sulfurous (London-type) smog and the photochemical (Los Angeles-type) smog? (II) How does Chinese haze fit into the current trend of global air pollution transition? (III) What are the major mitigation measures that have improved air quality in Western countries? and (IV) What specific recommendations for China can be derived from lessons and experiences from Western countries?

Keywords: Air pollution; emissions; health effects; clean air legislation

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History of air pollution and its health consequences

While air pollution in cities has only recently become an alarming concern in China, it has long been recognized as a threat to public health through both its acute and long-term adverse effects. Outdoor or ambient air has always been contaminated with pollutants from natural sources, including, for example, pollens, smoke from fires and volcanoes, and emissions of organic compounds from plants. The current problems and patterns of air pollution date to industrialization and the rise of cities. Together, fossil fuel combustion for heating and cooking and eventually for electric power generation and emissions from factories led to worsening pollution during the Industrial Revolution. The 20th century added mobile sources, including cars, trucks, and other vehicles, as major contributors to urban air pollution.

Urban air pollution was recognized centuries ago as a threat to health. Well-chronicled disasters during the 20th century motivated the actions that have led to marked improvements in air quality in North America and Western Europe. Most notably, the London Fog or the great London smog of 1952, an extreme air pollution event during a week-long episode of atmospheric stagnation, resulted in 10,000 or more excess deaths before the weekly mortality rate returned to the baseline (*Figure 1*) (1). It followed the 1930 episode in the Meuse Valley of Germany (2) and the 1948 episode in Donora, Pennsylvania, in the United States (3); both of these episodes were also accompanied by readily detected excess deaths and were well-documented in the scientific literature. Levels of air pollution during the great London smog were extreme by contemporary standards; "black smoke", a surrogate for airborne particulate matter (PM), reached approximately 4 mg/m^3, orders of magnitude above typical levels in high-income countries of the West. The clear and dramatic loss of lives that was caused by the London smog