



September 23, 2010

Ms. Joan Denton, Ph.D.
Director
Office of Environmental Health Hazard Assessment
Cal/EPA
1001 I Street
Sacramento, CA 95814

Subject: Comments on OEHHA's report entitled "Cumulative Impacts: Building a Scientific Foundation"

Dear Dr. Denton,

The organizations represented above respectfully submit the attached technical comments on the Office of Environmental Health Hazard Assessment's (OEHHA) report entitled "Cumulative Impacts: Building a Scientific Foundation" that was prepared at our request by Dr. John Bukowski, DVM, MPH.

Although the report is described by OEHHA as building the scientific foundation for a cumulative impacts screening methodology, we find that the OEHHA report generally lacks a science-based approach and is written from an advocacy perspective. The following points highlight our concerns.

We are very concerned that the literature review that is the basis for many of the OEHHA report's findings and conclusions contains extensive inconsistent and contradictory findings and assertions.

We believe that the screening methodology lacks scientific rigor in a number of ways:

- the indicators selected seem to be based on political criteria as opposed to scientific criteria;
- the indicators selected lack a full assessment of all the variables and dimensions that encompass the extent and breadth of the indicator;
- the rationale for assigning the weights to the different variables is weak and subjective;
- the rationale for combining the components is counterintuitive - why is living near an underground storage tank site with no exposure given the same value as another indicator with direct exposure?

We urge that the screening methodology incorporate a science-based process that better assesses, defines and estimates the cumulative impacts and the range of potential impacts. Current environmental regulation is based on exposure that could cause health effects. When a chemical is used safely it does not mean it will automatically cause a detrimental health effect. A potential indicator for cumulative impacts in the proposed screening methodology is use of the U.S. EPA Toxic Release Inventory (TRI) for exposure determination. This is a misleading use of data since the TRI reports all chemicals at a site. Having lead at a site and having lead released unsafely into the environment should not be considered the same.

We do not believe that the screening methodology will identify the priority drivers of cumulative impact necessary to address community issues or risk management decisions that will lead to a positive difference. Cumulative impact is proposed to be measured by five components, each of which is made up of two or more contributing indicators. However, there is no objective, scientific rationale for how the components or indicators were selected and weighted. There is no clear, scientific indication that the final value derived for each community would clearly distinguish level of cumulative impact. Therefore, the uses and goals of the screening method stated in Chapter 3 will not be achieved using the proposed components and scoring values.

The report acknowledges that more discussion is needed to determine what role, if any, cumulative impacts should play in the permitting process. It also states that guidelines must be developed to determine how the screening method should be used. The screening method needs to be streamlined and include a more qualitative approach that ranks communities on specific, predetermined, data-based components instead of relying on a single, vague summary value. This would incorporate objective criteria into the screening method that would result in a transparent process allowing risk management decisions to be tailored to specific community concerns before any permitting discussion or guidelines developed. Otherwise, this proposed screening method will not enhance decision making and instead cause confusion and open the door for extensive litigation.

While risk assessment cannot take into consideration economic impact, risk management can and should. Uneven implementation of this screening tool could lead to adverse economic impacts which would result in further harm to the communities it is striving to protect.

While we appreciate the work that has gone into this report thus far, the business community believes that far more discussion and greater input from a wider breadth of the scientific community is needed before it is ready to be adopted.

Thank you for your consideration of our concerns and of the attached technical comments.

Sincerely,

Curt Augustine



Director of Policy and Government Affairs
Alliance of Automobile Manufacturers

Tim Shestek



Senior Director, State Affairs
American Chemistry Council

Mark Smith



Legislative Advocate
American Council of Engineering Companies, California

Justin Morton



Manager, Government Affairs
American Forest & Paper Association

Matthew Hargrove



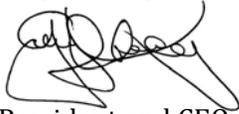
Sr. Vice President, Government Relations
California Business Properties Association

Robert Callahan



Policy Advocate
California Chamber of Commerce

Gary Hambly



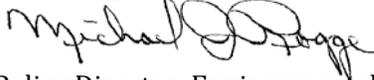
President and CEO
California Construction and Industrial Materials Association

Cynthia L. Cory



Director, Environmental Affairs
California Farm Bureau Federation

Mike Rogge



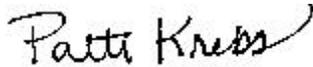
Policy Director, Environmental Quality
California Manufacturers & Technology Association

Laurie Nelson



Advocate
Consumer Specialty Products Association

Patti Krebs



Executive Director
Industrial Environmental Association

Steve Cook



Vice President
National Aerosol Association

Craig S.J. Johns



Project Manager
The Partnership for Sound Science in Environmental Policy

Bryan Bloom



President
Priority Moving, Inc.

William E. Davis



Executive Vice President
Southern California Contractors Association

Gail Delihant



Director, CA Government Affairs
Western Growers

Renee Pinel



President/CEO
Western Plant Health Association

Kevin Buchanan



Senior Coordinator, Bay Area and State Water Issues
Western States Petroleum Association

Ted LaDoux



Executive Director
Western Wood Preservers Institute

ATTACHMENT

Review of the CalEPA report entitled Cumulative Impacts: Building a Scientific Foundation, Public Review Draft, August 19, 2010

John Bukowski, DVM, MPH, PhD

WordsWorld Consulting

I. EXECUTIVE SUMMARY

The authors of the CalEPA report on cumulative impacts repeatedly refer to science and science-based approaches throughout the document. Even the title is focused on “building a scientific foundation.” Unfortunately, the document itself is not based on objective science, but is rather is a political text designed to advocate for the approach supported by the Agency.

The CalEPA authors rely heavily on the literature review performed by Zuk and Morello-Frosch, which is written from an advocacy rather than scientific perspective. Zuk and Morello-Frosch present evidence in support of EJ assertions, rather than an unbiased review of the literature. The strongest assertions in this document are that health and exposure disparities exist by race and socioeconomic status (SES). The weakest assertion is that minority status and low SES increase sensitivity to the health effects associated with pollution. This latter assertion is largely theoretical, based on vague mechanistic arguments such as “weathering” and “allostatic load.”

Both the CalEPA and Zuk documents provide only a superficial view of the literature, without critical assessment of study strengths and weaknesses. Contrary/inconsistent evidence is either ignored or downplayed, so that the resulting summary presents a one-sided assessment of the literature, apparently intended to persuade rather than inform.

Studies cited in support for EJ assertions contain inconsistent/contradictory findings that cast doubt on the results. A major limitation of these studies is the potential for residual bias from SES and other risk factors. Inconsistent contrary findings may also stem from modeling deficiencies, in that covariates are often highly correlated and study authors generally make no mention of model diagnostics.

The screening methodology set forth by CalEPA does not appear to be based on scientific or objective criteria. The screening scale is made up of five components: SES, sensitivity, exposure, environmental effects, and public health effects. Neither the components of the scale

nor the indicator variables making up these components were selected and weighted via an objective, science-based process. Many indicators seem to have been chosen based on ease of data access, political expediency, guesswork, or some other non-scientific rationale.

No attempt is made to identify and capture all the important dimensions within a component. For example, the SES component consists only of race and two highly correlated estimates of income/wealth (income and poverty), with other important dimensions of SES (eg, education, occupation, and social class) ignored. The rationale for assigning ranges (ie, weights) to the variables and then combining components is similarly weak, subjective, and in some cases counterintuitive. For example, components with less certainty are assigned narrower ranges than those with more certainty, which is contrary to accepted views regarding the impact of data uncertainty.

The screening tool could be improved by adopting a simpler, more qualitative approach. Rather than relying on a single, opaque, summary value, it would seem most appropriate to qualitatively rank each community on specific, predetermined, data-based components. This would be a more transparent process, allowing concerned parties to see how communities ranked on various aspects of sensitivity or impact, and allowing risk management decisions to be tailored to specific community concerns.

Before cumulative impacts are used for decision making, the process needs to be refined to reflect a science-based process that generates objective and transparent estimates of impact/sensitivity. This process should begin with an unbiased (non-advocacy) technical review of the scientific literature. CalEPA should also consider the economic impacts associated with implementing cumulative impact assessment (eg, regulation, permitting), as well as the potential for uneven protection across racial groups.

II. ASSESSMENT OF THE CALEPA REPORT

The authors of the CalEPA report on cumulative impacts (hereafter referred to as the CI report) state repeatedly their intention to use scientific evidence to lay out a “science-based” approach to cumulative impacts. Indeed, the title of the report includes the phrase “building a scientific foundation.” However, even a casual reading of the CI report suggests a lack of scientific rigor, with the focus of the document concerned with advocacy and political motives.

The advocacy/political tone of the document is established in the introduction. The rationale for the assessment is based largely on addressing residential “worry” and showing that regulators share the concerns of the “more vulnerable” communities burdened with “a toxic legacy.” The introduction goes on to highlight a need to “move beyond analysis” and encourage “public participation and public contribution to the science.” While goals such as empathy and public participation may be worthy political efforts, they hardly represent appropriate contributions to the science. Finally, the avowed purpose of the report is to present a screening methodology that is described as being “neither comprehensive nor detailed.” In short, one could argue the scientific basis for using empathy and public input as a means of moving away from analysis via application of a screening method that lacks comprehension and detail.

Chapter 1: Scientific Evidence

The political/advocacy tone of the CI report continues within the presentation of scientific evidence for disproportionate impacts. Study results are presented superficially, without critical evaluation of strengths, weaknesses, potential for bias, consistency, or plausibility. In most cases, findings in support of the political/advocacy goal of the report are presented qualitatively, without even listing measures of association and their corresponding confidence intervals. Negative or contrary evidence is generally not presented. Risk factors such as genetics, lifestyle, and SES are mentioned only as an aside, despite the fact that these non-pollution causes represent the major risk factors for disease.

The superficial nature of the presentation in chapter 1 can be explained somewhat by reliance on a third-party summary of the evidence. The authors indicate that the literature on CIs has been “organized in a report prepared for OEHHA by researchers at the University of California Berkeley,” citing Zuk and Morello-Frosch (2009). The authors of the CI report rely heavily on Zuk and Morello-Frosch and generally repeat the material presented by those authors.

Unfortunately, as will be described later, the report by Zuk and Morello-Frosch takes a non-critical, advocacy approach to the evidence. Therefore, relying on an advocacy document for scientific evidence has defeated the scientific purpose of the CI report even before it was written.

Deficiencies in presenting the CI literature are discussed extensively in the later section on **Review of the Tech-Support Document by Zuk and Morello-Frosch** (see below), and will not be repeated here. However, a few examples are presented to highlight how lack of critical evaluation, selective reporting, and one-sided discussion can skew interpretation of the evidence.

- (1) Page 16 of the CI report cites Krewski et al. (2000) as evidence of increased sensitivity driven by non-intrinsic factors, noting that these authors found a higher pollution-mortality risk among those with less than a high school education. However, the authors of the CI report fail to point out that all of the increased risk is among those with less than a grade 12 education, with no pollution risk for total or cardiopulmonary mortality among those with greater than high school education. Neither the authors of the CI report nor Krewski et al. provide a plausible biological mechanism for why the cardiac and lung tissues of educated persons would be unaffected by pollution. Similarly, neither of these sets of authors discusses the possibility that the risk associated with pollution may actually represent residual confounding by low SES, which is removed by restricting analyses to those with higher education.
- (2) On page 19, the authors of the CI report cite Lee and McConville (2007) as evidence of racial difference in mortality, pointing out “higher death rates for African Americans than whites in California ... regardless of socioeconomic position, as measured by educational attainment.” However, they fail to note that education is only one measure of SES and is unlikely to fully account for all SES-related differences. The authors of the CI report go on to state that “mortality among Hispanic and Asian populations is slightly lower than among whites,” without providing an explanation for decreased risk among two minority populations, a finding that runs contrary to EJ theory.
- (3) On page 18, the authors of the CI report cite a 2006 study by Braveman on the impact of social disadvantage. However, they ignore an earlier study in which Braveman et al. (2001) point out that there are a large number of measures of SES and that these are often only moderate correlated among each other, suggesting that they represent somewhat independent constructs. Therefore, one cannot fully capture the influence of SES by

adjusting for one or even a few variables, and there is bound to be a certain amount of residual confounding in any multivariable model. Braveman et al. (2001) conclude that “it is difficult to interpret results of studies that attempt to adjust for socioeconomic influences on health while examining the influence of other factors, such as race.”

- (4) On page 17 of the CI report, Clougherty et al. (2007) is cited as evidence that family stress increases the association between pollution and asthma. However, the authors of the CI report ignore the fact that there is no association whatsoever between pollution exposure and asthma among children when violence is absent, and that violence itself is not an independent risk factor, making it highly unlikely that it would so strongly modify the impact of pollution so as to create a strong association where none existed. Furthermore, other potentially important a priori variables, such as maternal smoking, education, and preexisting asthma are not strongly or significantly associated with childhood asthma, calling into question the findings on pollution and violence.
- (5) On page 20, the authors of the CI report cite various studies that reported racial/SES differences in hospitalizations for asthma, without discussing the impact of SES on the dynamics and treatment of this disease. It has been fairly well established that poor/minority families use hospital emergency rooms for primary care, thereby increasing the possibility of being admitted to the hospital. The authors of the CI report also fail to note that Boudreaux et al. (2003) reported hospitalization rates that were almost identical among races for the initial/index visit, and that most racial differences in acute effects disappeared after adjustment for SES.

Chapter 2: Definition of Terms

The advocacy tone of the rest of the document is continued to a lesser degree in chapter 2. Rather than confining this discussion to definition of terms, as suggested by the title, the authors include one-sided discussion of pollution-related effects that are enhanced by low SES or minority status. As with the first chapter, supportive results are presented without critical evaluation. For example, on pages 29-30 of the CI report, a study by Bell et al (2007) is cited as evidence of race-based increased sensitivity to the impact of particulate pollution on birth weight. However, Bell et al. (2007) reported a significant racial impact for PM_{2.5} exposure, but no racial difference regarding PM₁₀ exposure (average decrease of 9 grams for whites and 8

grams for blacks), even though PM_{10} includes the $PM_{2.5}$ fraction. This suggests that fine PM exposure greater than 2.5 microns (ie, $PM_{2.5-10}$) must have a protective effect for African-American women compared to white women. Such inconsistencies cast serious doubt on the findings by race. Furthermore, Bell et al. (2007) cite the mechanism behind cigarette smoking as an explanation for their findings, but report effects across all trimesters of exposure, whereas smoking exerts its effect primarily during the third trimester (Bukowski 2004).

Chapter 3: Screening Methodology

Chapter three is entitled “A scientific screening methodology for analyzing cumulative impacts in communities.” Although the proposed method appears to assign ranks in an objective manner, it is neither inherently objective nor “scientific.” That is to say, this tool has not been developed in a scientifically defensible manner so as to lead to scientifically defensible outcomes.

The scale is derived based on five components, each of which is made up of two or more contributing indicators. However, the authors of the CI report give no indication that either the components or their indicators were selected and weighted via an objective, science-based process. Rather, many indicators seem to have been chosen based on ease of access, political expediency, guesswork, or some other non-scientific rationale.

SES:

The authors provide no scientific rationale for why the indicators of minority status, income level, and poverty were selected to represent SES. As stated elsewhere (see review of tech-support document), SES is an extremely complex variable with multiple independent constructs, including education, social class, occupation, and wealth, to name a few. These constructs can be represented by multiple variables, including employment status, type of occupation, house value, net worth, years of education, and neighborhood characteristics. There is no indication that the authors used a scientific process to select those constructs and variables that best captured the various dimensions of SES and best encompassed the wide range of health-related factors (eg, diet, health behaviors, obesity, etc.) subsumed under the broad rubric of SES. In fact, all three components would seem to cover similar constructs, with at least two of them (income and poverty) being highly correlated.

Sensitive populations:

This component uses the proportion of elderly and children as a measure of sensitive populations. Although this choice seems reasonable, a scientific discussion would point out that there is considerable variation in sensitivity among these groups, and that children and seniors are not inherently defective in their ability to handle chemical exposures. In fact, for most drugs used in human medicine, children and seniors receive similar doses as do young or middle-aged adults, with only moderate (eg, 50%) reduction in the event of clinically significant organ dysfunction (Bukowski and Lewis 2004).

Exposures:

As with SES, no scientific rationale is given for selection of the particular exposures. The selected exposures seem to be heavily weighted toward exposure to traffic-related emissions, in that levels of PM_{2.5} and ozone are driven by mobile sources, a measure of which is also included in the component.

Environmental effects:

Again, no scientific rationale is included for choosing proximity to hazardous waste sites and leaking underground tanks as measures of environmental effects. Furthermore, it is unclear if these components are meant to reflect psychological stress from living in an ugly/unhealthful neighborhood, or additional exposures.

Public health effects:

The public health effects cover a fairly broad range of outcomes, but it is not at all clear why they were chosen or how they relate to the other components. Furthermore, it would seem that this component overlaps SES in that it has been fairly well established that the various risk factors related to low SES (eg, smoking, poor diet, poor health behaviors, lack of preventive care, etc.) lead to increase health effects.

Weighting and assigning scores:

There appears to be no scientific rationale for how components were weighted (eg, the range of values) or combined. Within a component, indicators appear to have been assigned equal weights because of insufficient information to suggest otherwise, but this is in and of itself an unscientific process. Ranges appear to have been assigned based largely on a subjective assessment of the strength of the available data, with a wider range provided when the data were thought to be richer and more certain. This approach seems to be based on a process of guesswork, rather than scientific scrutiny. Furthermore, the approach itself appears flawed, in that a wider range is applied when there is more certainty and a smaller range when there is less, which is contrary to accepted practice in which uncertainty is seen as increasing variability. For example, it is not at all certain that pollution exposure at current levels represents a potentially three times greater risk (10) than does the impact of SES (3). In fact, traditional public health theory suggests that the myriad risk factors associated with SES would outweigh the cancer burden associated with pollution (Doll 1998).

The rationale for why exposure, public health effects, and environmental effects are added, and then multiplied by the sum of SES and sensitivity, also seems vague and somewhat subjective. No references are given for the “evidence from human studies indicating multiplication is appropriate” (p34), especially for the variables selected by the authors of the CI report.

There is no clear, scientific indication that the final value derived for each community would clearly distinguish level of impact. That is to say, is a community with a high cancer rate at similar risk to one with a high level of asthma? How does this compare to areas with a high level of poverty, high traffic load, or numerous waste sites? Is the presence of a retirement center equal to that of a grade school, and how will this influence health effects? Furthermore, the summary nature of the final statistic hides the components that went into generating it, leading to a nontransparent process. One is left only with a summary value meant to indicate a level of “badness,” which has no real public health or scientific utility.

Suggested improvements:

Input variables for estimating the relative impacts of concepts such as health, vulnerability, and cumulative hazard are incompletely understood and challenging to define, let

alone quantify. Attempts to do so often provide a false sense of certainty/accuracy that is not in keeping with the limitations of the underlying data. Complex, poorly-defined concepts such as “vulnerability” and “cumulative hazard” are also difficult to fully grasp, even for those with scientific training, and quantitative approaches add a layer of technical complexity that further obscures the uncertainties and limitations associated with the underlying inputs. There is also a real danger that interested parties might change/adjust model inputs to conform to expected or desired outcomes.

Given the considerable uncertainty associated with the process, it would seem most appropriate to develop a simpler, more qualitative approach to identifying impacted communities. Rather than relying on a single, opaque, summary value, it would seem most appropriate to use objective criteria to choose specific, predetermined components for which communities could be ranked qualitatively. This would be a transparent process by which concerned parties would be able to see how communities ranked on various aspects of sensitivity or impact. Risk management decisions could then be tailored to specific community concerns, and the rationale behind decisions would be clearly visible. Inputs, goals, and weights could be decided beforehand in consultation with the public and other interested parties, so that all communities would be judged based on predetermined criteria in which stakeholders had a vested interest.

Chapter 4. Decision Making

The authors of the CI report foresee cumulative impacts and the aforementioned screening tool potentially applied to a variety of regulatory settings, including permitting, site clean up, enforcement, monitoring, risk assessment, standard setting, financial assistance, and education and outreach. However, before this occurs, it is important to refine the process to address existing deficiencies and the broader environment in which regulatory decisions are placed.

Both the screening tool and agency view of cumulative impacts need to be refined to reflect a scientific process that generates objective estimates of impact/sensitivity that are transparent to concerned stakeholders. This process should begin with an unbiased, nonadvocacy review of the scientific literature on cumulative impacts and the underlying tenets

of the EJ movement. To this end, it is recommended that CalEPA expand their base of experts beyond the narrow clique of favored researchers at UC Berkeley.

The authors of CI report need to consider the economic impacts of additional or more stringent regulation based on cumulative-impact arguments. Indiscriminate application of CI assessment could harm key industries that are important sources of taxes and employment for the state of CA in general and minorities in particular. Furthermore, using CI speculation to drive more stringent regulation could increase the costs of energy, thereby impacting the costs of all products that require energy to produce or transport (eg, food, consumer goods, pharmaceuticals, etc.). A large body of research shows that health is closely linked to wealth/income/poverty, so that economic disruption would probably have a bigger impact on health (including minority health) than the public health and EJ benefits intended under the regulation (Annapolis Center 2006). This is especially a concern given the severe economic problems that currently exist in the state of CA.

The authors of the CI report also need to consider how application of CI-based assessment and regulation would influence their current mandate to protect “all” races and socioeconomic groups. For example, the current screening process assigns precautionary priority to minority and low-income neighborhoods, which could be construed as less concern for the health protection of whites and those with higher SES. Great care needs to be taken so that protection of the disadvantaged does not unfairly lead to real or perceived increased risk among the advantaged.

Chapter 5. Proposed Actions and Next Steps

The authors of the CI report outline next steps, including more detailed guidelines, methodology for more comprehensive analysis, and need to gather more data. However, these steps should be undertaken only after the current report and screening plan are subjected to a more scientific and unbiased evaluation, so as to place the process into a more technically accurate and balanced context.

III. REVIEW OF TECH-SUPPORT DOCUMENT BY ZUK & MORELLO-FROSCH

The authors of the CI report repeatedly indicate that they are seeking scientific evidence and science-based approaches. To this end, CalEPA relies heavily on the Zuk and Morello-Frosch (2009) report entitled, “A synthesis of the scientific evidence for addressing cumulative impacts.” However, the Zuk document is not a critical, scientific review of the evidence. Indeed, Zuk and Morello-Frosch acknowledge that “it is beyond the scope of this report to systematically review the extensive literature.” Instead, they provide supportive evidence for five “assertions” on health disparities, pollution disparities, and enhanced susceptibility of lower SES or minority populations. Such an approach, which seeks to present evidence in support of a position rather than review the pros and cons in the literature, is (by definition) advocacy rather than science.

Zuk and Morello-Frosch provide no critical review of the various cited studies. Instead, supportive results are presented as corroborative evidence, often implying relative certainty by use of phrases such as “it is known,” “researchers have identified,” and it has “become widely accepted.” Contrary findings are mentioned only rarely as an afterthought. Most importantly, none of the studies are critically reviewed so as to put their results in perspective. This leads the reader into believing that the data are more consistent and compelling than is actually the case.

Critical review is especially important given that most of the presented findings come from epidemiologic studies. Epidemiology represents an observational rather than experimental approach, so that some level of bias is unavoidable and can potentially account for some or all of the findings. It should also be remembered that unlike experimentalists who must adhere closely to standardized protocols, epidemiologists have considerably more leeway in deciding how studies will be performed and what results will be reported. Therefore, it is common for epidemiologists to follow lines of inquiry or report/highlight only particular findings that support a preconceived hypothesis. Such problems have been referred to as wish bias, the file-drawer problem, selective reporting, publication bias, or publication bias in situ (Rosenthal 1979, Dickersin 1990, Wynder et al. 1990, Hahn et al 2000, 2002, Phillips 2004).

Zuk and Morello-Frosch exhibit a lack of attention to detail throughout the draft manuscript, which detracts from the evidence presented. Little detail was provided on cited studies, often only saying that they support author assertions without providing information on strength of association, confidence intervals, sample size, participation rates, type of study (eg,

ecological, cross-sectional, etc.), potential for misclassification or selection bias, etc. As stated previously, lack of critical review on the potential for bias is a major limitation of any synthesis of epidemiologic evidence, and detracts heavily from the scientific weight that can be afforded such a synthesis. Furthermore, many citations within the Zuk and Morell-Frosch report are missing from the reference list (eg, Giscombe and Lobel 2005, Mustillio et al. 2004, Zimmerman et al. 2006, and McDaniel et al. 2006), making it difficult for the interested reader to identify and critically review the cited papers.

Inconsistencies in Selected Studies

Given the limitations of the current review, it would be impossible to perform a detailed critique for the large number of studies mentioned by Zuk and Morello-Frosch. Instead, selected biases, inconsistencies, and uncertainties from a few studies will be presented as examples.

Morello-Frosch and Jesdale, 2006

On page 12, Zuk and Morello-Frosch cite Morello-Frosch and Jesdale (2006) as evidence that cumulative exposure and cancer risk have been found to “disparately affect communities of color and low SES.” However, no detailed evaluation of this paper is provided, and inconsistencies within this paper are ignored.

Morello-Frosch and Jesdale report increasing cancer risk with increasing levels of “segregation,” implying that this represents unequal risk driven by environmental injustice. However, upon closer inspection, the results do not support this contention. Risk does indeed increase with increasing level of black or Hispanic segregation, but risk also increases with increasing white segregation. An extremely segregated white community has an adjusted cancer RR of 1.28 (95% CI 1.24-1.33), which is significantly greater than that for white communities that are only highly segregated (RR 1.04, 95% CI 1.01-1.08). In fact, the risk for an extremely segregated white community is considerably higher than that for a highly segregated black or Hispanic community (adjusted RR 1.09). This means that a homogenous white suburb would experience much greater cancer risk than either a mixed or highly segregated minority community. These results run contrary to environmental justice theory that suggests risk is borne predominantly by people of color. Such findings suggest fundamental problems with the modeling approach, in that “segregation” (as defined) tends to increase risk regardless of the group being segregated. Furthermore, the authors provide no insight into why extremely

segregated Hispanics are most affected (adjusted RR 1.74), while black, white, and Asian estimates of risk are similar (adjusted RR 1.38, 1.28, and 1.32 respectively).

Morello-Frosch and Jesdale (2006) also note that racial gradients of risk remains despite adjustment for ecological measures of SES, “suggesting that segregation affects pollutant burdens in a manner independent of area-level poverty.” However, rather than showing two independent effects (ie, segregation and poverty), the data actually show no increased risk with increasing poverty, as demonstrated by the essentially flat curves seen in Figure 3. Such a result runs contrary to environmental justice theory and its supporting data, which suggest greater air pollution exposure/risk among the poor. Such a finding suggests that SES has not been well controlled, and that increasing “segregation” is acting as a surrogate for decreasing SES. This is consistent with the crude, ecological adjustment for SES in this study. Indeed, Morello-Frosch and Jesdale (2006) adjusted for only a single measure of income (based crudely on poverty level) and a “material deprivation” index, without controlling for education, type of occupation, or other SES measures that influence where people live. Therefore, residual bias is to be expected.

Apelberg et al. 2005

This paper is also cited as evidence of disproportionate cancer risk for poor or minority communities. Apelberg et al. reported on a cancer-risk study similar to that performed by Morello-Frosch and Jesdale (2006). They noted an increased risk when more African Americans were in a census tract, but only for the lowest quartile of household income. In the highest quartile of income, risk actually decreased significantly with increasing percentage of African Americans. This is contrary to a racially based effect, in which one would also expect to see increased risk by race at least within middle-income families (and possibly in higher-income ones). Such findings suggest residual bias by SES, rather than differential risk by race. This is consistent with the ecological nature of this study, in which only census-tract-level data on SES were available.

The authors also reported a consistent and statistically significant protective effect associated with the presence of Hispanics in a neighborhood (RR approximately 0.4-0.5). Furthermore, there was no consistent association between income and major emission sources (eg, power plants or waste sites). These findings run contrary to environmental justice theory and suggest possible problems with the modeling approach.

Kington & Smith 1997

This paper is cited to show that “while racial disparities in health are substantially reduced when accounting for individual SES, they are not completely eliminated” (p.3). This cross-sectional study concludes that SES plays a relatively small role in explaining race-based differences in chronic-disease prevalence. However, closer examination of modeling results shows unusual findings that contradict the EJ theory supported by Zuk and Morello-Frosch. In the full model controlling for age, gender, and SES, blacks have significantly higher prevalence of hypertension and diabetes than do whites. However, whites have significantly higher prevalence of arthritis (38%) than either blacks (34%) or Hispanics (28%). Whites also have prevalence of heart disease (13%) that is significantly higher than for Hispanics (7%), and similar to that of black (11%). Such findings suggest heterogeneity in the prevalences of chronic disease, rather than uniform increase among minorities.

Mustillo et al. 2004

This paper is cited as evidence that prenatal stress from discrimination can lead to poor reproductive outcomes. The study authors report moderately strong odds ratios of 2-3 for self-reports of racial discrimination. Zuk and Morello-Frosch do not mention the uncertainty surrounding self-reported data, which are subjective measures that cannot be objectively quantified. More importantly, they ignore unusual findings that cast doubt on the underlying models used to derive these results. For example, race/ethnicity, income, and education are not significant predictors of prematurity, with RR ranging from 0.8-1.1. In fact, having less than a college education is nonsignificantly protective (RR 0.87). Furthermore, depressive symptoms (a relatively strong stress) are not associated with prematurity (RR 1.0), and alcohol intake is significantly protective (RR 0.3). Such findings call into question the underlying models used to generate the data and/or the underlying EJ theory.

Clougherty et al. 2007

This paper is cited as evidence for the synergistic effects between pollution and psychosocial stress, such as exposure to chronic violence. However, Zuk and Morello-Frosch do not point out that participation in this study was less than 50%, thereby increasing the potential

for bias. Furthermore, they do not note that although there is significant increase in asthma among those with high violence scores (RR 1.6-2.4), there is no association whatsoever between pollution exposure and asthma among the low violence group (RR 1.0-0.85). Furthermore, violence itself is not a risk factor (RR 0.9-1.1), making it highly unlikely that it would so strongly modify the impact of pollution so as to create a strong association where none existed. Furthermore, other variables that might be expected to be predictive, such as maternal smoking, education, and preexisting asthma are not strong or significant associations (RR 1.1-0.9). Again, such contradictory results call into question the underlying models on which these results are based.

Assertions

Zuk and Morello-Frosch argue for a series of five EJ assertions. The level of evidence supporting these claims varies, but all are discussed from a one-sided perspective of advocacy rather than scientific detachment.

Assertions 1-2:

These are among the strongest assertions made by the authors, because it has been fairly well documented that both health disparities (assertion 1) and differences in exposure (assertion 2) exist by SES and race. However, even here the authors go well past the stated goal of documenting differences and delve into hypothetical theories behind why and how these differences exist. Pejorative terms such as racism, discrimination, and segregation are put forward as reasons why poor and minority citizens experience psychosocial stress that contributes to health disparities. It should be noted that neither these subjective terms, nor the nebulous stress that they are said to produce, nor the mechanism by which these stresses lead to specific diseases are objectively measurable.

Zuk and Morello-Frosch mention opposing points of view only in passing. Furthermore, contradictory findings that exist in cited articles are generally ignored. For example, they cite the National Vital Statistic Reports (NVSR) as evidence of differences in life expectancy by race and SES, but then cite articles on infant mortality as evidence that this gap is increasing over time. However, returning to the NVSR shows that black-white differences in life expectancy have decreased by 30%-40% between 1975 and 2007, dropping from approximately 7.5 years to

5 years among males, and from 5 years to 3 years among females (Xu et al. 2009). Similarly, Zuk and Morello-Frosch cite the fact that Hispanics experience better health than whites, even after controlling for SES (p. 3), as apparent evidence that racism/segregation play an independent role in health. However, superior health among Hispanic minorities (a group that is supposedly repressed and discriminated against) is evidence against their EJ assertion.

Zuk and Morello-Frosch continually point out that racial differences are not completely explained by SES, yet the cited evidence is often not internally consistent. For example, Kington and Smith (1997) is cited, noting that while SES may “explain some of the differences in functional status of people with chronic diseases such as hypertension and heart disease, it was not significantly related to prevalence” (p7). However, Zuk and Morello-Frosch fail to state that the prevalence of heart disease in the Kington study was greatest among whites. Zuk and Morello-Frosch also indicate that black-white mortality differences are dominated by diseases such as “homicide, hypertension, heart disease, diabetes mellitus, respiratory diseases and several types of cancer” (p4) without noting that these illnesses are heavily linked to non-environmental exposures related to SES. In fact, Zuk and Morello-Frosch demonstrate a level of analytical naivety by suggesting that it is easy or even possible to adequately account for all aspects of SES, so that remaining effects must be due to race rather than residual confounding by SES. This is an important, fundamental flaw in their arguments that will be discussed in detail later.

The authors note that proximity to hazard “does not necessarily result in higher exposures,” (p8) but repeatedly point to presence of landfills, factories, etc. as evidence of cumulative hazardous exposures. Furthermore, Zuk and Morello-Frosch suggest that landfills, waste sites, factories, etc. have been sited among minorities and the poor as part of a discriminatory process. However, they largely ignore information suggesting that hazardous waste facilities are not predominantly sited in minority neighborhoods (Anderton et al. 1994, Davidson and Anderton 2000) and that risk from major-source emitters (eg, power plants and large factories) are higher within predominantly white communities (Apelberg et al. 2005). Furthermore, it is likely in many cases that poor and minority residents migrate to the low-priced real estate surrounding preexisting sites, rather than vice-versa.

Assertion 3:

Assertion 3 argues that health disparities by race/SES may be due to differences in pollution exposure. As with all the assertions, this statement ignores the tremendous difficulties involved with separating health effects due to race/SES from those related to environmental exposures strongly linked to race and SES (see below).

Zuk and Morello-Frosch suggest that environmental exposures combine to produce more-than-additive effects that are not addressed by traditional risk assessment. While this has been demonstrated in selected laboratory and epidemiologic settings, it has not been universally demonstrated. Furthermore, observed multiplicative relationships have generally been based on high-level exposures (eg, maximum-tolerated dose or past occupational exposures) that are much higher than the low-level background exposures realistically experienced today. It should be remembered that it is equally possible for exposures to combine to produce additive effects, sub-additive effects, or even hormetic (ie, beneficial) effects. It should also be noted that the risks predicted by animal bioassays (on which most risk assessment is based) are often orders of magnitude higher than those actually experienced by people (Portier 1988). Furthermore, the attributable risks predicted by epidemiologic studies often add up to several hundred percent, suggesting that cumulative cancer risks are less than the sum of their respective causes (Doll 1998). In addition, risk assessment values are by definition conservative estimates that include uncertainty factors intended to add orders-of-magnitude precaution (ie, uncertainty is always assumed to increase risk). All these issues suggest that cumulative risks do not necessarily interact to generate greater risk than suggested by individual exposures.

Assertions 4 and 5:

Assertion 4 points out the fact that biological factors such as age, sex, genetics, and health status can influence how individuals interact with environmental exposures. Although this is widely accepted, most supporting evidence again comes from high-level occupational or laboratory exposures. Zuk and Morello-Frosch provide little mechanistic evidence that so-called sensitive individuals are unable to cope with the low-level environmental exposures that currently exist today. Furthermore, current risk assessments based on individual exposures already include 10-fold uncertainty factors to account for inter-individual sensitivity, as well as additional factors of 3-10 to account for animal-human extrapolation, database uncertainty, etc.

Assertion 5 purports that poor and minority peoples are especially sensitive to pollution exposures because of psychosocial factors unrelated to biology. This is the weakest assertion made by Zuk and Morello-Frosch, and one that is largely theoretical/rhetorical without much scientific support. Vague terms such as stress, “weathering,” and allostatic load are used to suggest mechanisms behind increased sensitivity, but such nebulous theories do not constitute mechanistic evidence. That is to say, the authors provide no well-established, biologically based pathways by which these psychosocial factors increase absorption, decrease detoxification, or generally increase pollution risk. The use of emotional and value-laden terms such as racial discrimination, racism, social deprivation, and racial segregation also appear to suggest advocacy instead of science.

By and large, the studies cited to support assertion 5 do not constitute reliable evidence. For example, Seeman et al. (2001) is cited as evidence that “allostatic load” from low SES and racial inequality increases mortality risk. However, these authors show only that a summary scale based on existing markers of risk, such as blood pressure, obesity, and levels of stress hormones, proved useful in predicting mortality. Neither Seeman et al. nor Zuk and Morello-Frosch link any of these variables to the psychosocial stressors of interest, nor do they show that such markers increase risk from low-level pollution exposures. Similarly, Clougherty et al. (2007) is cited as evidence of increased pollution risk from allostatic stress (in this case due to violence), without noting the inconsistent findings previously described.

Residual Bias from SES (and Other Risk Factors)

There are several major tenets behind the arguments made by Zuk and Morello-Frosch (as well as CalEPA and other EJ advocates), including: (1) pollution exposures are highly correlated with minority status and lower SES (ie, the classic EJ contention), (2) minority/poor populations experience greater health effects than do more affluent/white populations, (3) minorities are mired in lower SES than are whites. Zuk and Morello-Frosch go farther and acknowledge that low SES and/or minority status are strongly associated with a variety of health-related stresses and behaviors, including exposure to crime/violence, family instability, smoking, drunkenness, lack of preventive care, obesity, decreased access to health care, poor diet and food choice, sedentary life style, etc. From an epidemiologic perspective, such a situation creates a fundamental and perhaps insoluble problem differentiating the effects of pollution exposure from

those due to race/SES, and an even bigger problem differentiating effects due to race from those due to SES.

It is difficult to reliably disentangle effects due to pollution from those due to SES/race. The fact that pollution exposure is highly linked to SES/race, and that SES/race is strongly linked to adverse health outcomes, sets up the classic confounding triangle, whereby effects from race/SES can be misinterpreted as arising from pollution. Even when one attempts to control for SES via statistical modeling, there is bound to be residual confounding given the high level of correlation between exposure and SES/race, as well as the many varied and poorly correlated variables that comprise race and SES.

It must be remembered that SES is a “broad concept that refers to the placement of persons, families, households and census or other aggregates with respect to the capacity to create or consume goods that are valued in our society” (Shavers 2007). In essence, the concept of SES represents an attempt to quantify those intangible aspects of a person or group that affects their health and overall wellbeing. This concept is defined using surrogate measures representing the various dimensions of SES, including income, education, accumulated economic assets, occupation, social class, etc. (Braveman et al. 2001, Shavers 2007). Such measures must themselves be estimated using various surrogate variables that capture different characteristics of the dimensions being addressed (see Table). Some measures refer to aspects of the individual, while others refer to aspects of the contextual environment (Shavers 2007).

Table. Selected examples of surrogate measures for the dimensions involved with SES (Braveman et al 2001, Shavers 2007)

DIMENSION	SURROGATE MEASURE
Income	Individual annual income Annual household income Family income
Wealth	Poverty level Net worth Percentage of household owning a home, car, etc. Average home value in neighborhood Percentage on public assistance
Education	Years of education completed Highest level of education Degrees earned Maternal/Paternal education
Occupation	Current employment status (eg, unemployed, retired, etc.)

	Specific occupation
	Aggregate occupation (eg, blue collar)
Social class	Employment category (eg, professional vs. laborer)
	Neighborhood characteristics (poverty area, blue collar, etc.)

There are often only moderate correlations among these various aspects of SES, suggesting that they represent somewhat independent constructs (Braveman et al. 2001). Therefore, one cannot fully capture the influence of SES by adjusting for one or even a few variables, and there is bound to be a certain amount of residual confounding in any multivariable model. This has led researchers to conclude that “it is difficult to interpret results of studies that attempt to adjust for socioeconomic influences on health while examining the influence of other factors, such as race” (Braveman et al. 2001) and that “the complexity of the interaction between race/ethnicity and SES makes it difficult to disentangle the independent effects of the two variables” (Shavers 2007). It should also be remembered that SES variables are crude (ie, poorly classified) surrogates for the actual constructs of interest, and that misclassification of a confounder leads to residual confounding (Greenland 1980, Marshall and Hastrup 1996). Therefore, one can never be sure that an association with race/ethnicity is not due to residual confounding by SES, or that effects associated with pollution are not due to residual confounding by race/SES.

There is also considerable potential for uncontrolled confounding by other factors, given the broad range of risk factors associated with the diseases that have been evaluated. For example, a substantial portion of the evidence presented by Zuk and Morello-Frosch deals with linking pollution, race, and SES to adverse birth outcomes such as prematurity and low birth weight. These reproductive outcomes are associated with a large number of important risk factors, including maternal age, education, smoking, alcohol consumption, drug abuse, weight gain, marital status, race, prenatal care; birth order; gestational length; child’s sex; small maternal prepregnancy weight; small maternal stature; complications of the current or previous pregnancy (eg, pregnancy-induced hypertension); maternal illness (eg, fever or untreated hypertension); stress; genetics; and job-related exertion (Bukowski 2004, Kramer 1987, 2003, Lang et al. 1996). In general, the cited studies adjusted for only some of these important risk factors, suggesting a strong possibility of residual confounding that could be misinterpreted as independent effects of pollution or race. Furthermore, many of the above variables are correlated amongst themselves and with SES/race, so that inclusion of one or a few would account for a

substantial effect of the others, making SES and related variables seem relatively unimportant in a model that controls for race. It is also important to remember that

“Socioeconomic status, like race/ethnicity and marital status, is closely related to other demographic, behavioral, environmental, and medical factors that may influence pregnancy outcome and that cannot be easily controlled for in epidemiologic studies. Thus, socioeconomic status is presumably a proxy for these other factors rather than an independent determinant of preterm delivery” (Berkowitz and Papiernik 1993).

That is to say, SES is a surrogate for independent risk factors that are related to SES, race, and (presumably) pollution exposure.

Modeling concerns:

Statistical models are analytical tools that have limitations. In the current context, a major limitation is the inability to reliably distinguish between highly correlated covariates. This problem of “multicollinearity” means that it may be difficult or impossible to reliably separate the confounding effects of one variable from the effects of highly correlated covariates. Even models that appear to adequately fit the data can produce biased and inaccurate results because of multicollinearity. It should be noted that,

“the choice of the particular set of independent variables which are to be included in the model is highly important and that in the presence of multicollinearity, the interpretation of regression coefficients ... must be undertaken with caution. The regression coefficients are affected not only by the other intercorrelated variables in the model but also by intercorrelated variables omitted from the model” (Netter et al. 1985).

As a general rule, the reviewed articles did not mention performing tests for multicollinearity, and even such tests provide no guarantee of detecting (let alone fixing) this problem because “it is possible for variables to pass these tests and have the program run but yield output that is clearly nonsense” (Hosmer and Lameshow 1989).

Other potential modeling concerns include the impact of influential observations, violation of model assumptions (such as normality, independence, and homoscedasticity), and issues of “overfitting” (ie, applying complex models to sparse data thereby creating an artificially perfect fit). As a rule, the studies cited by Zuk and Morello-Frosch have not provided evidence that models have been adequately tested via regression diagnostics. An exception is the paper by

Mustillo et al. (2004), which notes that “the Homer-Lemeshow goodness of fit test statistic was not significant.” However, it should be noted that goodness of fit is only one diagnostic test among many, and that Mustillo et al. have misinterpreted failure to reject as “indicating a good model fit.” In point of fact, goodness of fit tests are weak statistics that can only identify poor fit, with nonsignificant results providing no assurance of good model fit (Greenland 1998).

A recent paper by Messer et al. (2010) point out the limitations related to multicollinearity when trying to determine effects of SES and race. These authors found that it is very difficult to control for confounding in models addressing race/SES because of structural confounding. That is to say, to control for confounding covariates, one needs heterogeneity in said covariates within exposure strata (such as neighborhood or census tract). However, given the close correlation between measures of SES and race, there tends to be homogeneity within strata. For example, none of the 53 Durham county census tracts evaluated by Messer et al. had both high black percentage and high SES, and only one county had high white percentage and low SES. For Wake county, these numbers were 0 and 0, respectively. This surprised the investigators, because very few tracts in either county are highly segregated, suggesting that these counties should be good for social justice research.

Messer et al. (2010) call inferences drawn from such models as “‘off support,” meaning that they are not really supported by the underlying data. These authors also point out that there is no easy fix for this problem, because this is an inherent problem with how people are allocated, so that collecting more data doesn’t really help. One can surmise that adding in a third set of variables on pollution, which is highly related to SES and race, would exacerbate the problem, especially when exposure is assigned at the neighborhood level.

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