Bay Area Air Quality Management District
375 Beale Street, Suite 600
San Francisco CA 94105

VIA EMAIL
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Victor Douglas

May 8, 2017

Re: Health impacts and implications should be included in the No Project and alternative scenarios and the environmental and regulatory settings sections of the EIR for BAAQMD Rule 12-16

We are writing to encourage the Air District to include a comprehensive health and safety assessment in the final EIR of Rule 12-16, as detailed in the following submission. In particular, by providing a preliminary assessment of potential mortality impacts in the absence of Rule 12-16's preventive measures, this submission demonstrates the feasibility and importance of including a health assessment in the EIR. It is important that such an assessment account for:

- the preventive nature of Rule 12-16
- the influx of heavier crude oil feedstock that is projected in the absence of emissions caps
- resulting exposures and impacts on vulnerable populations, including people who live in proximity to the refineries, have low socio economic standing and / or disadvantaged racial identity, are infants, young children or the elderly, live in already polluted settings, and/or have underlying health conditions

Respectfully
Signatures, listed alphabetically on the following page,

David Bezanson PhD	Clinical psychologist, retired
Claire V Broome MD	Adjunct Professor, Rollins School of Public Health Emory University Assistant Surgeon General, US Public Health Service (retired)
Wendel Brunner MD, PhD, MPH	Former Director of Public Health, Contra Costa Health Services
Robert M. Gould, MD	President, Physicians for Social Responsibility, San Francisco Bay Area Chapter Associate Adjunct Professor, Program on Reproductive Health and the Environment, Dept. of Obstetrics, Gynecology & Reproductive Sciences UCSF School of Medicine (for identification purposes only)
Jonathan Heller PhD	Co-Director and Co-Founder, Human Impact Partners Oakland CA
Richard J Jackson MD MPH	Former California State Public Health Officer Director, CDC National Center for Environmental Health (retired)
Janice L Kirsch MD MPH	Medical oncologist and hematologist
Chaz Langelier MD, PhD	Postdoctoral Scholar Center for AIDS Research University of California, San Francisco (for identification purposes only)
Raymond Neutra MD DrPH	Chief Division of Environmental and Occupational Disease Control, California Department of Public Health (retired)
Thomas B Newman MD MPH	Professor Emeritus of Epidemiology & Biostatistics and Pediatrics, University of California, San Francisco (for identification purposes only)
Bart Ostro PHD	Former Chief of Air Pollution Epidemiology Section, California EPA, currently Research Faculty, Air Quality Research Center, UC Davis
Linda Rudolph MD MPH	Director, Center for Climate Change and Health, Public Health Institute Oakland, CA
Seth BC Shonkoff PhD, MPH	Executive Director | PSE Healthy Energy Visiting Scholar | Dept. Environmental Science, Policy, & Management, UCB Affiliate | Energy Technologies Area, Lawrence Berkeley National Lab
Patrice Sutton, MPH	Research Scientist, Program on Reproductive Health and the Environment, University of California, San Francisco (for identification purposes only)

Coordinated by

Heather Kuiper DrPH MPH	Public Health Consultant, Oakland CA

May 8, 2012

To the Bay Area Air Quality Management District Board:

This submission alerts the Air District that the Rule 12-16 draft EIR does not adequately analyze or discuss the health impacts that were identified in a letter submitted December 2, 2016 during the Notice of Preparation and Initial Study for the Rule 12-16 DEIR. In particular, the draft EIR does not adequately recognize the preventive nature of Rule 12-16, thus omitting health implications from the "No Project" alternative.

Preventing increases in harmful exposures is a well-established health protection measure. (Curie 2011, Pope 2009, Goodman 2002, Hedley 2002, Dominici 2006). A preventive approach to air quality is important, due to an otherwise anticipated increase in Bay Area refineries' use of heavier, dirtier oil feedstock, [1] (BAAQMD 2012a) which will lead to higher exposures to fine particulate matter (PM2.5). PM2.5 is definitively established as a cause of adverse health impacts, including mortality. Given the dense population of the Bay Area, increased PM2.5 will have large population impacts, presenting a major public health threat. Rule 12-16 is an important public health tool as it caps refinery emissions at current levels, thereby preventing increases in exposure to PM2.5.

Omission of the No Project Alternative (not implementing Rule 12-16) and its health impact

Because Rule 12-16 is a preventive measure, the Air District can anticipate that the "No Project" scenario will increase mortality in the Bay Area population, especially among the disadvantaged. The assessment,[2] detailed in Appendix A, measures the impact of long-term exposure to increased PM2.5 resulting from transitions to heavier oil feedstock. Adjusting for other exposures, it finds that:

- **Rule 12-16 could cumulatively prevent 800 to 3000 deaths of Bay Area residents given a refinery facility lifetime of 40 years following conversion to heavier crude**
- **The additional mortality burden for the Bay Area's disadvantaged residents could be 8 – 12 times that of the Bay Area's general population**
- **Annual monetary valuation of these deaths alone could reach up to $123.2 million, or cumulatively, up to $4.84 billion dollars. (CAP, 2017 p C/7)**

This assessment is conservative in its parameters and many of the model parameters are drawn from BAAQMD's own work. For example, it does not consider indoor air exposures, which may be higher, (Brody, 2009), impacts of ultrafine particulates (Ostro, 2015), or increased combustion, production, and handling of pet coke (US EPA). The submitted analysis is also conservative in scope: It does not include PM2.5-related morbidity, neurological, cognitive, and developmental impairment, (especially of children), hospitalizations, lost productivity, reduced activity, and health-related socio-economic impacts. Significantly, the analysis does not include health impacts associated with flares and other acute PM2.5 exposures, including mortality, cardiac events, hospitalizations, and increased susceptibility to adverse health conditions from the underlying stressors of living in proximity to pollution sources (DeFur 2007, Cutchin 2008, Luginaah 202). It also does not include the significant local climate-related

[1] This assessment is predicated on a finding that, without 12-16, Bay Area refineries will likely undergo large-scale capital conversions for refining heavier crude oils and natural bitumen (including and especially tar sands crude), resulting in increased PM2.5 emissions and toxicity, and increased greenhouse gas emissions. (BAAQMD 2012a, Karras, 2016)

[2] This assessment draws from calculations of emissions increases attributable to heavier crude oil feedstock produced by Greg Karras of Communities for a Better Environment (Karras, 2016) It was conducted in collaboration with CBE.

health hazards and impacts that will be attributable to the Bay Area's increased refining of heavier crude feedstock.

Even so, this analysis demonstrates that is reasonable and feasible for the District to develop and consider health impact projections in its final EIR. The signatories request that the Air District include the attached assessment (Appendix A) in its final EIR and also supplement it with estimates of additional health impacts attributable to increased PM2.5 and greenhouse gas emissions, especially for vulnerable populations. See also Appendices B, and C for information that can support such additional analysis.

Modify the draft EIR's assessment of alternatives

Emission intensity caps (Rule 13-1) and mass emission caps (Rule 12-16) are complementary measures and their combination could protect health better than Rule 12-16 alone. This alternative is not considered in the draft EIR although Rule 13-1 is discussed in combination with Rule 11-18. CEQA requires an alternative to accomplish the main objectives of the project at hand, yet Rules 13-1 and 11-18 do not provide health protection equivalent to 12-16. Rule 11-18 targets various toxic air contaminants but not greenhouse gases and particulate matter and is fundamentally different in terms of health protection strategy and outcome. Rule 13-1, *as currently drafted*, omits direct control of PM2.5 and could allow facility-wide refinery emissions to increase; it is does not provide protections comparable to Rule 12-16. Regardless, it is premature to consider Rule 13-1 in the Rule 12-16 EIR.

Expand the existing environmental and regulatory settings assessments

The following considerations should be included in the environmental settings assessment:

- Cities in the San Francisco Bay Area are among the most polluted in the U.S. (ALA, 2017) High baseline air pollution augments susceptibility to adverse health threats. Due to this baseline condition, Bay Area residents will likely experience augmented health risk and burden from increased emissions. Further, the Air District, Cal EPA, the US EPA and the World Health Organization, all find that, "people exposed to PM at levels below the current EPA standards may still experience negative health effects." (BAAQMD, 2012 p 17). There are no safe levels of particulate matter, and given high baseline pollution, every PM2.5 exposure increment will contribute to increased risk of mortality, morbidity, and lost productivity for Bay Area residents.

- This high baseline pollution is not uniformly or fairly distributed, "PM concentrations – and population exposure to PM – can vary significantly at the local scale… People who live or work near major roadways, ports, distribution centers, or other major emission sources… may be disproportionately exposed to certain types of PM (e.g. ultrafine particles)…" (BAAQMD, 2012, p 14) There is growing evidence that proximity to oil refineries places residents at disproportionate risk for adverse health outcomes. Appendix C provides a partial list of this evidence base. There is also documentation that residents in proximity to refineries are disproportionately vulnerable by virtue of race, economic standing, and higher prevalence of underlying health conditions (Cushing 2016, Pastor 2010). The final EIR should recognize as part of the current landscape that failure to prevent increased refinery emissions will have environmental justice repercussions since they will predominantly occur in communities where residents are low income and/or are people of color and already disproportionately burdened by poor underlying health and multiple-source pollution exposures.

- The draft EIR should recognize that state and local policy specifically precludes placing disproportionate burden on impacted, disadvantaged populations. Senate Bill 32 and Assembly

Bill 197 recognize and protect these populations by requiring consideration of equity and social costs in reducing greenhouse gases and equitable resolution of them, prioritizing direct emissions reductions at large stationary sources. CEQA and the District's own mission also affirm a health mandate. Protecting public health and eliminating health disparities are stated goals of the 2017 Clean Air Plan. Rule 12-16 should be understood in light of this state-level policy framework for environmental health protection and the District's own mission.

- Current conditions with regards to Bay Area emissions are not static. Instead, the setting for Rule 12-16 is trending toward increases in the processing of heavier, higher-emitting, lower quality crude oils, expansion of projects to do so, and expanding fossil fuel export. (BAAQMD, 2013) Switching to heavier crudes will inherently increase emissions of PM2.5 and greenhouse gases, making it imperative that measures be put in place to prevent these future increases in emissions, *in addition to* measures decreasing current emissions. Without the preventive caps offered by Rule 12-16, other District measures will be limited by a context of rising emissions.

- The corresponding increase in fossil fuel exports will lead to an increase in exogenous air pollution in the Bay Area since a portion of the byproducts of combustion of fossil fuels exported from the Bay Area will return to us from Asia through transpacific atmospheric transport. This exogenous air pollution will directly threaten health and also impede progress toward the targets and goals of the Clean Air Plan, 2017. Exogenous / overseas sources of pollution are of increasing concern as they have been directly implicated in deaths in local populations and documented as a greater proportion of exposure than locally-sourced pollution in some settings. (Annenberg 2014, Christensen 2015, Zhang 2007, 2008, 2009).

Lastly, the health comments submitted to the District in December 2016 were omitted from Appendix A of the draft EIR and we ask that they be included.

The signatories believe these adjustments are necessary for the EIR to be complete and accurate and respectfully request they be made in time for Rule 12-16's potential adoption in September.

APPENDIX A:

Impact of Rule 12-16 on mortality associated with exposure to PM2.5 from processing heavier oil in Bay Area refineries

Table 1 Potential health impact of 12-16: Averted all-cause deaths attributable to chronic exposures to oil refinery PM2.5 (see Appendix for calculations)

	Regional Population (9 Bay Area Counties)			Impacted Population* (<=2.5 miles from refinery)		
	Low	Med	High	Low	Med	High
PARAMETERS						
Risk						
a. Risk of all-cause death for adults (>30 yrs) per 1µg/m^3 PM2.5 increase in long-term exposure	1.008	1.01	1.012	1.008	1.01	1.012
b. Incremental Risk: risk of all-cause death for adults attributable to increment in long-term PM2.5 exposure (risk/ per 1µg/m^3 PM2.5 increase)	*0.008*	*0.01*	*0.012*	*0.008*	*0.01*	*0.012*
Exposure						
c. Baseline anthropogenic** exposure (µg/m^3 $PM_{2.5}$)		5.7			5.1	
d. Proportion of baseline anthropogenic exposure attributable to baseline refinery activity		.05			0.5	
e. Percent change from baseline anthropogenic emissions due to higher emitting oil emissions	40%	70%	100%	40%	70%	100%
f. Conversion factor (change in PM2.5 exposure per change in PM2.5 emissions)		0.5		0.4	0.5	0.6
g. Averted exposure: the annual increased PM2.5 concentration attributed to heavier oil that is averted by Rule 12-16 (µg/m^3 $PM_{2.5}$)	*0.057*	*0.10*	*0.143*	*0.408*	*0.893*	*1.53*
Population and Mortality						
h. Adult Population (>25)		5,144,345			81,666	
i. Base all-cause adult death rate / person / year		*0.00*83403			*0.0091899*	
IMPACT						
j. Prevented adult all-cause deaths due to 12-16 averting increases in heavier oil PM2.5 emissions* **	**20**	**43**	**73**	**2**	**7**	**14**
k. Rate of prevented adult all-cause death due to 12-16 averting increases in heavier oil PM2.5 emissions /100,000 population /yr	**0.38**	**0.83**	**1.43**	**3.00**	**8.21**	**16.88**
l. Cumulative prevented deaths due to 12-16 (40 yrs)	**800**	**1700**	**2900**	**98**	**270**	**550**

* The distance of 2.5 miles was selected to correspond with findings from Brody (2009) and Pastor (2010). Those living < 2.5 miles of refineries (Table 5) can roughly be interpreted as a proxy for impacted, vulnerable, and/or Environmental Justice populations. The Air District's CARE program prioritizes communities and populations most impacted by air pollution, i.e., those with higher air pollution levels and worse health outcomes for diseases affected by air pollutions. Vulnerable populations also include those with heightened vulnerability to PM due to age (<5, elderly), low SES, minority race/ethnic status, and underlying health conditions. This proxy is conservative because *disparate impacts on vulnerable populations may occur beyond 2.5 miles.*

** Anthropogenic exposure is the ambient PM2.5 concentration above background levels (e.g., from sea salt).

*** Annual and cumulative deaths are presented as whole numbers. The resulting rounding error explains any discrepancy between presented deaths and rate.

Notes for Table 1

a. For every 1µg/m3 PM2.5 increase in exposure there is x% increased risk of all-cause mortality, e.g., a 1% increased risk of all-cause death per 1µg/m3 PM2.5 exposure increase. Risk estimates are from BAAQMD's literature review, of for example Pope et. al (2002), Krewsk et. al, (2000), and others. Risk may be underestimated as it does not account for 1) greater energy intensity and toxicity of PM2.5 associated with heavy oil and natural refining, 2) ultrafine PM, and 3) greater vulnerability of impacted populations.

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b. Calculated as (all cause death risk in exposed) – (all cause death risk in unexposed), i.e, (risk per increase of 1µg/m^3 PM2.5) – (no increase in exposure) = 1.01 – 1 = .01. For every exposure change of 1µg/m^3 PM2.5 there is a corresponding 1% change in all-cause mortality attributable to PM2.5

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c. *Regional*: CAP 2017 p C/7

Impacted Population (<2/5 miles from refinery): From Brody et. al.(2009) baseline PM2.5 exposure was directly measured in Richmond at distances approximately 2.5 miles from the dominant $PM_{2.5}$ source in the refinery. To isolate exposure above background, control site measures in Bolinas were subtracted from Richmond measures, yielding µg/m3 PM2.5. The PM2.5 was chemically fingerprinted to the refinery, finding, for example, high levels, of vanadium and nickel, which in this setting are isolated to refinery emissions (versus traffic). Validating this measure, CARB "ADAM" data for 2013 subtracts annual mean PM2.5 measures at Pt. Reyes from measures at the monitoring station nearest to the refinery, yielding 5.04 µg/m3 PM2.5. A baseline exposure of 4.5 µg/m3 PM2.5 likely underestimates annual exposure because 1) the Brody study was conducted during the summer when PM2.5 concentrations are lowest and 2) Due to wind patterns, and refinery distribution, populations near the other refineries may experience a concentrating of PM2.5. For these reasons, a conservative adjustment was made to factor in higher wintertime concentrations. The annual median concentration was divided by the median concentration Apr–Sep for three years of monitoring at the three closes sites (San Pablo, Vallejo, Concord). The mean of the resulting ratios was multiplied by the Brody measure (2009) such that 4.5 x 1.13 = 5.1 µg/m3 PM2.5 anthropogenic [ ].

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d. Portion of the baseline anthropogenic exposure that is attributable to baseline refinery activity

Regional: CAP, 2017 p 2/20

Impacted Population: We set the portion at .5 since Brody et. al. (2009) used chemical fingerprinting to find that heavy oil combustion (refineries being the predominant source in the study area) is the most important contributor, more important than traffic, to elevated anthropogenic PM2.5 concentrations in the study area (<2.5 miles from refinery). We consider this measure reasonable in light of 1) BAAQMD grid modeling that ranged from .2 - .6, 2) an independent assessment of the Districts aerial emissions intensity data (2015) found that, on a mass/$mile^2$ basis, within 2.5 miles of the refineries, the areal source strength is more than twice (0.7) the regional average for all sources (CBE, 2015), and 3) accommodation of some lofting of emissions from hot stacks (2017 Staff Report). These parameters nevertheless likely underestimate, since downwind refinery communities could experience consolidation of PM2.5 from multiple refineries. Further, statewide analyses link high exposure to refinery proximity (<2.5 miles) (Pastor et. al. 2010).

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e. Karras (2016) estimated a range of annual tons of PM2.5 emissions that Rule 12-16 would avert, such that, meaning that annually, Rule 12-16 would prevent increases of 364, 728, or 1090 short tons PM2.5 / yr of heavier oil-associated emission, or 40%, 70%, and 100% from current refinery emission rates could be averted through Rule 12-16. Medium Case (0.7) is the midpoint of the 0.4 - 1.0 range

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f. The conversion factor translates emissions into exposure. It is derived from the regional weighted average change in $PM_{2.5}$ exposure for a given change in direct emissions of $PM_{2.5}$. Verified by measurements and assuming a 24 hour "backyard exposure," BAAQMD modeled PM2.5 exposure change on a region-wide 4x4km grid relative to a 20% reduction in all-source PM2.5 emissions finding a range from .2 - .6. (CAP, 2017 D/13),
Regional: We applied .5 as the central measure to recognize that the location of population, emission sources, and meteorological conditions coincide. BAAQMD also applied approximately .5 for their regional average conversion. The conversion factor may underestimate impacted population exposures since refineries are strong PM2.5 emission sources near densely populated communities.
Impacted Population: For the <2.5 miles group, given population density and proximity to refineries, which are strong emitters, we used .4 for the lower bound. The upper bound, .6, may underestimate exposure for this group, given monitoring station locations.

g. The increased concentration of PM2.5 (exposure) attributed to heavier oil refining that is averted by Rule 12-16 ($\mu g/m^3$ $PM_{2.5}$). Calculated as (baseline total anthropogenic exposure) x (portion of baseline anthropogenic exposure attributable to baseline refinery emissions) x (Portion change from baseline anthropogenic emissions due to higher emitting oil emissions that is averted by 12-16) x (conversion factor). For the Medium regional case: 5.7 µg/m3 PM2.5 x .05 x .7 x .5 = *0.10* µg/m3 PM2.5. The attributable exposure may be underestimated because it does not account for: 1). NOx and SO2 PM-precursor emissions, and 2) the greater concentration of toxics associated with refining of heavy crude feedstock.

h. See Tables 2 and 3

i. Calculated as (annual deaths / total population) / yr. May overestimate or underestimate death rate over time should risk factors systematically improve or worsen.

j. Prevented deaths calculated as Attributable Risk x Attributable Exposure x all-cause per cap death rate x population. For middle regional scenario: .01 x .1 x .00589 x 7,447,686 = 44 deaths prevented by Rule 12-16.

k. Calculated as (deaths prevented / population) x 100,000 population / year.

l. Cumulative Impact calculated as deaths prevented x 40 years, since capital projects to accommodate heavier crude feedstock generally operate for 30 - 50 years. This number underestimates cumulative impact if population increases, as is anticipated.

Table 2. Bay Area communities ≤ 2.5 miles from refineries; local-scale population data [a]

Census	Refinery [b]	Tract distance to fence line (miles)		Fraction [c]	Population	
Tract	≤ 2.5 miles	closest	furthest	≤ 2.5 miles	Total	≤ 2.5 miles
3650.02	Chevron	0.5	2.5	1.00	5,462	5,462
3660.02	Chevron	2.3	3.3	0.20	6,093	1,219
3680.01	Chevron	1.5	2.5	1.00	5,327	5,327
3680.02	Chevron	2.0	2.7	0.71	3,404	2,431
3720	Chevron	1.8	3.1	0.54	7,353	3,959
3740	Chevron	2.0	2.8	0.63	4,506	2,816
3750	Chevron	1.3	1.8	1.00	4,389	4,389
3760	Chevron	0.4	1.5	1.00	5,962	5,962
3770	Chevron	0.4	2.4	1.00	6,962	6,962
3780	Chevron	0.0	3.1	0.81	3,435	2,770
3790	Chevron	1.1	3.1	0.70	6,117	4,282
2506.04	Phillips 66	2.1	3.7	0.25	3,842	961
3560.01	Phillips 66	0.0	3.5	0.71	3,759	2,685
3570	Phillips 66	1.0	5.5	0.33	3,018	1,006
3580	Phillips 66	0.0	2.0	1.00	5,298	5,298
3591.04	Phillips 66	2.0	3.0	0.50	1,932	966
3591.05	Phillips 66	2.0	3.0	0.50	4,542	2,271
3592.03	Phillips 66	1.0	3.3	0.65	6,726	4,387
3923	Phillips 66	1.0	2.0	1.00	3,102	3,102
3150	Shell &/or Tesoro	0.0	7.0	0.36	3,281	1,172
3160	Shell &/or Tesoro	0.5	2.0	1.00	1,483	1,483
3170	Shell &/or Tesoro	0.1	1.0	1.00	2,144	2,144
3180	Shell &/or Tesoro	0.7	4.7	0.45	3,267	1,470
3190	Shell &/or Tesoro	0.2	2.0	1.00	7,412	7,412
3200.01	Shell &/or Tesoro	0.0	2.0	1.00	3,615	3,615
3200.03	Shell &/or Tesoro	0.7	1.6	1.00	2,805	2,805
3200.04	Shell &/or Tesoro	0.2	2.0	1.00	6,216	6,216
3211.01	Shell &/or Tesoro	1.4	2.5	1.00	6,549	6,549
3270	Shell &/or Tesoro	2.0	6.0	0.13	6,695	837
3290	Shell &/or Tesoro	2.0	3.6	0.31	6,309	1,972
2520	Valero	1.8	3.5	0.41	4,157	1,712
2521.02	Valero	0.0	6.0	0.42	3,874	1,614
2521.04	Valero	0.0	4.0	0.63	5,536	3,460
2521.05	Valero	1.7	3.0	0.62	3,256	2,004
2521.06	Valero	0.5	2.0	1.00	4,132	4,132
2521.07	Valero	0.0	1.5	1.00	3,592	3,592
2521.08	Valero	1.0	2.0	1.00	3,165	3,165
		Sum of these tract data:			**168,717**	**121,608**

a) 2010 Census: https://factfinder.census.gov/faces/tableservices/jsf/pages/productview.xhtml?fpt=table

b) Plant or plants within 2.5 miles of part or all of the census tract, identified by current owner/operator.

c)) Estimation of population for tracts partly within a 2.5-mile radius: Tract fraction ≤ 2.5 miles = (2.5 - distance of bisection with radius in miles) ÷ (furthest distance – bisection distance in miles). Results are used to estimate the fraction of the total tract population ≤ 2.5 miles from a refinery. This method's simplifying assumption that population is distributed evenly within each tract despite geography and distance from refineries may result in overestimates or underestimates of local-scale population for those tracts that are partly within 2.5 miles of a refinery.

Table 3. Demographic and Vital Statistics for Bay Area Counties, 2013

	Age Group (years)											
Counties	<1	1-4	5-14	15-24	25-34	35-44	45-54	55-64	65-74	75-84	85+	TOTAL
Alameda												
Deaths	88	10	21	117	160	260	647	1,270	1,604	2,041	3,376	9,597
Population	19,493	76,842	190,900	203,954	232,027	231,327	222,525	191,268	111,600	55,333	28,101	1,563,370
Death Rate*	451.4	13.0	11.0	57.4	69.0	112.4	290.8	664.0	1437.3	3688.6	12013.8	613.9
Contra Costa												
Deaths	50	8	9	77	110	162	439	835	1,235	1,647	2,576	7,148
Population	12,240	49,755	146,153	145,402	129,256	143,616	163,677	140,700	86,747	42,739	21,577	1,081,862
Death Rate	408.5	16.1	6.2	53.0	85.1	112.8	268.2	593.5	1423.7	3853.6	11938.6	660.7
Marin												
Deaths	13	3	3	15	16	32	96	169	269	422	849	1,887
Population	2,334	9,858	30,334	26,078	23,766	32,876	41,089	40,325	28,899	13,245	7,460	256,264
Death Rate	557.0	30.4	9.9	57.5	67.3	97.3	233.6	419.1	930.8	3186.1	11380.7	736.4
Napa												
Deaths	6	1	1	9	10	23	51	125	188	269	511	1,194
Population	1,412	6,196	17,164	19,139	17,225	17,305	19,546	18,767	12,674	6,715	3,688	139,831
Death Rate	424.9	16.1	5.8	47.0	58.1	132.9	260.9	666.1	1483.4	4006.0	13855.7	853.9
San Francisco												
Deaths	30	4	6	40	91	172	351	749	809	1,268	2,134	5,655
Population	9,034	32,463	58,301	78,811	172,506	144,989	112,817	102,892	63,511	38,509	19,994	833,827
Death Rate	332.1	12.3	10.3	50.8	52.8	118.6	311.1	727.9	1273.8	3292.7	10673.2	678.2
San Mateo												
Deaths	19	2	5	35	52	94	257	477	673	1,102	1,920	4,636
Population	9,031	36,415	90,434	83,106	96,589	107,539	110,625	97,585	60,491	32,391	17,651	741,857
Death Rate	210.4	5.5	5.5	42.1	53.8	87.4	232.3	488.8	1112.6	3402.2	10877.6	624.9
Santa Clara												
Deaths	83	12	16	99	117	232	571	1,041	1,388	2,314	3,584	9,457
Population	24,112	95,493	245,789	228,340	264,949	282,446	270,707	211,136	126,347	68,609	32,667	1,850,595
Death Rate	344.2	12.6	6.5	43.4	44.2	82.1	210.9	493.0	1098.6	3372.7	10971.3	511.0
Solano												
Deaths	29	5	7	48	68	93	187	442	520	722	851	2,972
Population	5,127	20,641	55,419	59,872	56,830	53,419	61,449	56,360	32,286	15,914	6,731	424,048
Death Rate	565.6	24.2	12.6	80.2	119.7	174.1	304.3	784.2	1610.6	4536.9	12643.0	700.9
Sonoma												
Deaths	17	5	7	30	47	67	215	519	626	893	1,606	4,032
Population	5,070	21,413	58,627	65,627	64,121	59,350	69,251	71,808	45,050	20,879	11,874	493,070
Death Rate	335.3	23.4	11.9	45.7	73.3	112.9	310.5	722.8	1389.6	4277.0	13525.3	817.7
Bay Area												
Deaths	335	50	75	470	671	1135	2814	5627	7312	10678	17407	46578
Population	87853	349076	893121	910329	1057269	1072867	1071686	930841	567605	294334	149743	7384724
Death Rate	381.3	14.3	8.4	51.6	63.5	105.8	262.6	604.5	1288.2	3627.9	11624.6	630.7
<2.5 miles from refinery**												
Deaths	6	1	1	10	14	21	51	103	142	191	277	817
Population	1,402	5,685	16,278	16,577	15,027	15,911	18,180	15,913	9,612	4,736	2,286	121,608
Death Rate	454.9	18.5	7.9	60.9	95.7	129.4	278.1	648.0	1474.4	4039.0	12106.1	672.0

	Regional			<2.5miles		
	Death	Pop	Rt.	Death	Pop	Rt.
Adults >25 yr***	42905	5,144,345	834.03	751	81,666	918.992

*Death rates are age-specific expressed per 100,000 population. Age-adjusted rates are calculated using the 2000 U.S. Standard Population.

** Deaths in the Impacted Population (<2.5 miles from refinery) were derived using a death rate that divided Contra Costa and Solano Counties' combined deaths by their combined populations and applying this rate to the population living within 2.5 miles of a refinery for one year (from Table 2) (9,521 ÷ 1,518,002) x 121,608 = 763. This estimate may underestimate refinery effects on impacted populations because baseline death rates in communities near refineries may be greater than county-wide average rates. The age specific populations and deaths for the <2.5 miles group were arrived at by multiplying the total population by the age-specific death and population distribution of the combined Contra Costa and Solano Counties .

***The total adult deaths were adjusted to remove suicides and accidents by multiplying the unadjusted total by 6%, which represented the average and most frequent percent of deaths by suicide/accident for each county.

Population ≤ 2.5 miles from refinery fence lines estimated from census tract data. See Table 2

Source: State of California, Department of Public Health, Death Records. State of California, Department of Finance, Race/Ethnic Population with Age and Sex Detail, 2010-2060. Sacramento, CA, December 2014

State of California, Department of Finance, Race/Ethnic Population with Age and Sex Detail, 2010-2060. Sacramento, CA, December 2014.

APPENDIX B

Summary of pollutant – health outcome pairs to inform fuller health assessment of the No-Project Alternative

Table 1 Pollutant–health outcome pairs for which HRAPIE project recommends concentration–response functions (modified from WHO 2013b)

Pollutant metric	Health outcome	Group	RR (95 % CI) per 10 μg/m^3
$PM_{2.5}$, annual mean	Mortality, all-cause (natural), age 30+ years	A*	1.062 (1.040–1.083)
$PM_{2.5}$, annual mean	Mortality, cerebrovascular disease (includes stroke), ischaemic heart disease, COPD and trachea, bronchus and lung cancer, age 30+ years	A	GBD 2010 study (IHME 2013)[a]
PM_{10}, annual mean	Postneonatal (age 1–12 months) infant mortality, all-cause	B*	1.04 (1.02, 1.07)
PM_{10}, annual mean	Prevalence of bronchitis in children, age 6–12 (or 6–18) years	B*	1.08 (0.98–1.19)
PM_{10}, annual mean	Incidence of chronic bronchitis in adults (age 18+ years)	B*	1.117 (1.040–1.189)
$PM_{2.5}$, daily mean	Mortality, all-cause, all ages	A	1.0123 (1.0045–1.0201)
$PM_{2.5}$, daily mean	Hospital admissions, CVDs (including stroke), all ages	A*	1.0091 (1.0017–1.0166)
$PM_{2.5}$, daily mean	Hospital admissions, respiratory diseases, all ages	A*	1.0190 (0.9982–1.0402)
$PM_{2.5}$, 2-week average, converted to $PM_{2.5}$, annual average	RADs, all ages	B**	1.047 (1.042–1.053)
$PM_{2.5}$, 2-week average, converted to $PM_{2.5}$, annual average	Work days lost, working-age population (age 20–65 years)	B*	1.046 (1.039–1.053)
PM_{10}, daily mean	Incidence of asthma symptoms in asthmatic children aged 5–19 years	B*	1.028 (1.006–1.051)
O_3, summer months (April–September), average of daily maximum 8-h mean over 35 ppb	Mortality, respiratory diseases, age 30+ years	B	1.014 (1.005–1.024)
O_3, daily maximum 8-h mean over 35 ppb	Mortality, all (natural) causes, all ages	A*	1.0029 (1.0014–1.0043)
O_3, daily maximum 8-h mean over 10 ppb	Mortality, all (natural) causes, all ages	A	1.0029 (1.0014–1.0043)
O_3, daily maximum 8-h mean over 35 ppb	Mortality, CVDs and respiratory diseases, all ages	A	CVD: 1.0049 (1.0013–1.0085); respiratory: 1.0029 (0.9989–1.0070)
O_3, daily maximum 8-h mean over 10 ppb	Mortality, CVDs and respiratory diseases, all ages	A	CVD: 1.0049 (1.0013–1.0085); respiratory: 1.0029 (0.9989–1.0070)
O_3, daily maximum 8-h mean over 35 ppb	Hospital admissions, CVDs (excluding stroke) and respiratory diseases, age 65+ years	A*	CVD: 1.0089 (1.0050–1.0127); respiratory: 1.0044 (1.0007–1.0083)
O_3, daily maximum 8-h mean over 10 ppb	Hospital admissions, CVDs (excluding stroke) and respiratory diseases, age 65+ years	A	CVD: 1.0089 (1.0050–1.0127); respiratory: 1.0044 (1.0007–1.0083)
O_3, daily maximum 8-h mean over 35 ppb	MRADs, all ages	B*	1.0154 (1.0060–1.0249)
O_3, daily maximum 8-h mean over 10 ppb	MRADs, all ages	B	1.0154 (1.0060–1.0249)
NO_2, annual mean over 20 μg/m^3	Mortality, all (natural) causes, age 30+ years	B*	1.055 (1.031–1.080)
NO_2, annual mean	Prevalence of bronchitic symptoms in asthmatic children aged 5–14 years	B*	1.021 (0.990–1.060) per 1 μg/m^3 change in annual mean NO_2
NO_2, daily maximum 1-h mean	Mortality, all (natural) causes, all ages	A*	1.0027 (1.0016–1.0038)
NO_2, daily maximum 1-h mean	Hospital admissions, respiratory diseases, all ages	A	1.0015 (0.9992–1.0038)

APPENDIX C:

Partial listing of evidence establishing association between residential proximity to refineries and adverse health outcomes

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