Cleanaway's Proposed Processing & Co-generation Plant

Why it shouldn't be allowed

Summary

- **The submission by Cleanaway cites a single cohort study in support of its claim of 'safety' on health grounds**
- **The study itself found adverse outcomes, even within a limited 10 km radius of the incinerator plant studied**
- **The evidence supporting adverse health outcomes from nanoparticles such as the ones that will be emitted by the proposed incinerator is vast and rigorous, and far outweighs the evidence of any lack of association between emissions and negative health outcomes**
- **A large body of evidence shows that nanoparticles are associated with a broad range of health conditions and are a global threat to health – PM2.5 exposure can cause functional and pathological damage to the human body, increase the risk of neurological disorders, neurodegenerative disorders, affect neurodevelopment and childhood outcomes, among other negative effects.**

I wish to lodge my objection to the proposed processing and co-generation plant.

Incineration of waste creates dust particles which are less than 2.5 microns in size that can't be filtered out of the stack gases.

The submission by Cleanaway contains a link to a so-called 'facts' page (Energy-from-waste: Concerns and facts).[1]

Among the other so-called facts, the submission states that, regarding emissions and health:

"Fact: The most recent studies by reputable independent researchers show that modern plants do not cause identifiable changes in health effects surrounding facilities."

First of all, it should be noted that the link is to a single study (not studies as stated), a retrospective cohort study of congenital anomalies from England and Scotland. This study only considered congenital abnormalities in babies born to mothers living within 10 km of a municipal waste incinerator (MWI). Ten kilometres is a very limited locus of exposure, given the modelling around the current proposals for incinerators in Sydney. The study itself noted that even within such a small range, findings showed:

- An increased risk of all congenital abnormalities with proximity to the MWI
- Excess risks for congenital heart defects and genital anomalies,
- An increased excess risk of abnormalities for each kilometre closer to the MWI, including a 7% excess risk for hypospadias (a congenital defect of the penis). This is

[1] Energy-to-waste: Concerns and facts: https://www.cleanaway.com.au/sustainable-future/efw-concerns-facts/

supported by existing research showing that early gestational exposure to ambient air pollution increases the risk of hypospadias among full-term infants.[2]

The authors also noted the dearth of studies of the effects of MWI on their area of focus (congenital anomalies). The study did note, however, five studies finding increased risks of the following conditions:

- facial clefts
- renal and urological defects
- neural tube defects
- spina bifida
- lethal congenital heart defects, and
- deaths due to all congenital anomalies combined.

The study provides weak evidence at best on the absence of a link between exposure from incinerators and a limited number of rare health outcomes. No evidence is provided as to the latency of diseases and population migration, and the residual effects of repeated cumulative exposure.

Thus, Cleanaway's submission relies of claims of safety is reliant on a single study. A maxim of scientific research is that 'absence of evidence is not evidence of absence'.

But why the focus on such as limited area of human health? Congenital abnormalities are rare to begin with and much has been written about the inherent difficulties of establishing cause and effect between environmental exposure and outcomes due to the low statistical power of the bulk of the research, leading to negative findings (that is, no evidence of any association). The majority of these studies are designed to only detect risks where they are large.

Studies of congenital anomalies such as this one diagnosed prenatally or at birth focus on 'survivors' and it is possible that environmental exposures may act on the probability of survival of the malformed fetus, rather than causing abnormal morphological development itself. Direct investigation of such an effect is difficult and it has yet to be established that any environmental exposure acts on differential survival of malformed and normal fetuses.[3]

As the submission by Cleanaway failed to do so, the remainder of this submission will focus on what IS known about the effects of these nanoparticles on human health, a much vaster and more rigorous body of evidence.

It is an incontrovertible fact that exposure to fine particulate matter is a leading cause of premature deaths and illnesses globally. A recent publication in the Lancet medical journal[4] found that:

- Ambient $PM_{2·5}$ was the fifth-ranking mortality risk factor in 2015

[2] Huang et al. Periconceptional exposure to air pollution and congenital hypospadias among full-term infants. Environ Res. 2020 Apr;183:109151. doi: 10.1016/j.envres.2020.109151. Epub 2020 Jan 17.

[3] Dolk H and Vrijheid M. 2003. The impact of environmental pollution on congenital abnormalities. British Medical Bulletin, 2003: vol 68, pp 25-45.

[4] Cohen AJ et al. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution. Lancet 2017, May 13;389(10082):1907-1918. doi: 10.1016/S0140-6736(17)30505-6. Epub 2017 Apr 10.

- Exposure to $PM_{2·5}$ caused approximately 4·2 million deaths and 103 million disability-adjusted life-years (DALYs) in 2015, representing 7·6% of total global deaths and 4·2% of global DALYs
- A substantial increase in $PM_{2·5}$ -related illnesses and deaths over the past 25 years, but
- Potential for substantial health benefits from exposure reduction.

It is established that exposure to nanoparticles is directly associated with increased deaths due to stroke, heart disease, lung cancer and respiratory diseases. Additionally:

- PM2.5 can cause functional and pathological damage to the human body by penetrating the respiratory tract and blood and even entering the brain through the blood–brain barrier[5]
- PM2.5 exposure increases the risk of neurological diseases, including neurodegenerative disorders, stroke, and benign brain tumours[6] [7] [8]
- There is growing concern about the detrimental effects of PM2.5 on neurodevelopment, because the immature brain is more susceptible to PM2.5-induced neurotoxicity than the mature brain is[9] [10]
- Further, a marked association between PM2.5 exposure and reduction in working memory has been found in children aged 7–10 years[11], and early postnatal exposure to PM2.5 induced autism spectrum disorder in children and animals[12] [13], possibly due to neuroinflammation, neurotransmitter disruption, and metabolite alteration[14] [15]
- PM2.5 has been linked to elevated risk of respiratory and cardiovascular disease, neurological disorders, and reproductive function impairments[16] [17] [18]

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[5] Bondy, S. C. (2011). Nanoparticles and colloids as contributing factors in neurodegenerative disease. International Journal of Environmental Research and Public Health, 8(6), 2200–2211. 10.3390/ijerph8062200

[6] Calderón-Garcidueñas, L. , & de la Monte, S. M. (2017). Apolipoprotein E4, gender, body mass index, inflammation, insulin resistance, and air pollution interactions: Recipe for Alzheimer's disease development in Mexico City young females. Journal of Alzheimer's Disease, 58(3), 613–630. 10.3233/JAD-161299

[7] Andersen, Z. J. , Pedersen, M. , Weinmayr, G. , Stafoggia, M. , Galassi, C. , Jørgensen, J. T. , … Raaschou-Nielsen, O. (2018). Long-term exposure to ambient air pollution and incidence of brain tumor: The European Study of Cohorts for Air Pollution Effects (ESCAPE). Neuro-Oncology, 20(3), 420–432. 10.1093/neuonc/nox163

[8] Fu, P. , Guo, X. , Cheung, F. M. H. , & Yung, K. K. L. (2019). The association between PM2.5 exposure and neurological disorders: A systematic review and meta-analysis. Science of the Total Environment, 655, 1240–1248. 10.1016/j.scitotenv.2018.11.218

[9] Calderón-Garcidueñas, L. , González-Maciel, A. , Reynoso-Robles, R. , Kulesza, R. J. , Mukherjee, P. S. , Torres-Jardón, R. , … Doty, R. L. (2018). Alzheimer's disease and alpha-synuclein pathology in the olfactory bulbs of infants, children, teens and adults ≤ 40 years in Metropolitan Mexico City. APOE4 carriers at higher risk of suicide accelerate their olfactory bulb pathology. Environmental Research, 166, 348–362. 10.1016/j.envres.2018.06.027

[10] Ning, X. , Li, B. , Ku, T. , Guo, L. , Li, G. , & Sang, N. (2018). Comprehensive hippocampal metabolite responses to PM2.5 in young mice. Ecotoxicology and Environmental Safety, 165, 36–43. 10.1016/j.ecoenv.2018.08.080

[11] Alvarez-Pedrerol, M. , Rivas, I. , López-Vicente, M. , Suades-González, E. , Donaire-Gonzalez, D. , Cirach, M. , … Sunyer, J. (2017). Impact of commuting exposure to traffic-related air pollution on cognitive development in children walking to school. Environmental Pollution, 231, 837–844. 10.1016/j.envpol.2017.08.075

[12] Li, K. , Li, L. , Cui, B. , Gai, Z. , Li, Q. , Wang, S. , … Xi, Z. (2018). Early postnatal exposure to airborne fine particulate matter induces autism-like phenotypes in male rats. Toxicological Sciences, 162(1), 189–199. 10.1093/toxsci/kfx240

[13] Talbott, E. O. , Arena, V. C. , Rager, J. R. , Clougherty, J. E. , Michanowicz, D. R. , Sharma, R. K. , & Stacy, S. L. (2015). Fine particulate matter and the risk of autism spectrum disorder. Environmental Research, 140, 414–420. 10.1016/j.envres.2015.04.021

[14] Allen, J. L. , Liu, X. , Pelkowski, S. , Palmer, B. , Conrad, K. , Oberdörster, G. , … Cory-Slechta, D. A. (2014). Early postnatal exposure to ultrafine particulate matter air pollution: Persistent ventriculomegaly, neurochemical disruption, and glial activation preferentially in male mice. Environmental Health Perspectives, 122(9), 939–945. 10.1289/ehp.1307984

[15] Ning, X. , Li, B. , Ku, T. , Guo, L. , Li, G. , & Sang, N. (2018). Comprehensive hippocampal metabolite responses to PM2.5 in young mice. Ecotoxicology and Environmental Safety, 165, 36–43. 10.1016/j.ecoenv.2018.08.080

[16] Calderón-Garcidueñas, L. , Leray, E. , Heydarpour, P. , Torres-Jardón, R. , & Reis, J. (2016). Air pollution, a rising environmental risk factor for cognition, neuroinflammation and neurodegeneration: The clinical impact on children and beyond. Revue Neurologique, 172(1), 69–80. 10.1016/j.neurol.2015.10.008

[17] Sinharay, R. , Gong, J. , Barratt, B. , Ohman-Strickland, P. , Ernst, S. , Kelly, F. J. , … Chung, K. F. (2018). Respiratory and cardiovascular responses to walking down a traffic-polluted road compared with walking in a traffic-free area in participants aged 60 years and older with chronic lung or heart disease and age-matched healthy controls: A randomised, crossover study. The Lancet, 391(10118), 339–349. 10.1016/S0140-6736(17)32643-0

[18] Xue, T. , & Zhu, T. (2018). Increment of ambient exposure to fine particles and the reduced human fertility rate in China, 2000–2010. Science of the Total Environment, 642, 497–504. 10.1016/j.scitotenv.2018.06.075

- Further, PM2.5 exposure during early life leads to developmental disorders. A meta-analysis revealed low birth weight was positively associated with maternal exposure to PM2.5 pollution[19]
- Exposure to environmental toxicants during early periods of development can exert permanently deleterious effects on neurobiological and behavioural outcomes[20]
- Trends between PM2.5 exposure and its neurotoxic effects are dose-dependent.[21]

A cursory review of the scientific and medical literature shows that research from 2020 alone has concluded exposure to these nanoparticles:

- Increases hyper-inflammation of the immune system in diseases such as multiple sclerosis[22]
- Causes exacerbations of several acute and chronic lung diseases such as asthma and chronic obstructive pulmonary disease[23]
- Increases the risk of mortality form COVID-19.[24]

These studies support the substantial existing research showing that long-term exposure to nanoparticles is significantly associated with increases to overall mortality and deaths from cardiovascular disease.[25]

The potential direct and indirect costs to adverse health outcomes from increased exposure to nanoparticles will more than offset any savings to energy generation. Potential costs from increased cases of all the health outcomes noted in this submission would be an additional substantial burden to an already stressed health system, not to mention negative effects on psychological, physical and emotional wellbeing and reduced quality of life for all residents of Sydney.

Although the possible physical health effects arising from waste management processes have been studied, there has been little research into socio-economic impacts of waste-management options. On behalf of the residents of Sydney, I reject this proposal.

Kind regards,
Dr Daniela Solomon

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[19] Dadvand, P. , Parker, J. , Bell, M. L. , Bonzini, M. , Brauer, M. , Darrow, L. A. , … Woodruff, T. J. (2013). Maternal exposure to particulate air pollution and term birth weight: A multi-country evaluation of effect and heterogeneity. Environmental Health Perspectives, 121(3), 267–373. 10.1289/ehp.1205575

[20] Heyer, D. B. , & Meredith, R. M. (2017). Environmental toxicology: Sensitive periods of development and neurodevelopmental disorders. Neurotoxicology, 58, 23–41. 10.1016/j.neuro.2016.10.017

[21] Liu J, Yang C, Yang J, et al. Effects of early postnatal exposure to fine particulate matter on emotional and cognitive development and structural synaptic plasticity in immature and mature rats. *Brain Behav*. 2019;9(12):e01453. doi:10.1002/brb3.1453

[22] Cortese A, Lova L, Comoli P, Volpe E, Villa S, Mallucci G, La Salvia S, Romani A, Franciotta D, Bollati V, Basso S, Guido I, Quartuccio G, Battistini L, Cereda C, Bergamaschi R. Air pollution as a contributor to the inflammatory activity of multiple sclerosis. J Neuroinflammation. 2020 Nov 6;17(1):334. doi: 10.1186/s12974-020-01977-0. PMID: 33158438; PMCID: PMC7645903.

[23] Adamkiewicz G, Liddie J, Gaffin JM. The Respiratory Risks of Ambient/Outdoor Air Pollution. Clin Chest Med. 2020 Dec;41(4):809-824. doi: 10.1016/j.ccm.2020.08.013. PMID: 33153697; PMCID: PMC7665094.

[24] Andrea Pozzer, Francesca Dominici, Andy Haines, Christian Witt, Thomas Münzel, Jos Lelieveld, Regional and global contributions of air pollution to risk of death from COVID-19, *Cardiovascular Research*, , cvaa288, https://doi.org/10.1093/cvr/cvaa288

[25] Thurston GD, Ahn J, Cromar KR, Shao Y, Reynolds HR, Jerrett M, Lim CC, Shanley R, Park Y, Hayes RB. Ambient Particulate Matter Air Pollution Exposure and Mortality in the NIH-AARP Diet and Health Cohort. Environ Health Perspect. 2016 Apr;124(4):484-90. doi: 10.1289/ehp.1509676. Epub 2015 Sep 15. PMID: 26370657; PMCID: PMC4829984.