

Children's Health since the Mine Fire

Hazelwood Inquiry Supplementary Submission

Dr Joanna McCubbin

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Jo McCubbin is a Paediatrician based at Gippsland Paediatrics in Sale, seeing ambulatory patients from East, West and South Gippsland.

She teaches Environmental Medicine to Monash Medical Students in Sale and has a particular interest in Air Quality Toxic Waste and Mercury in the Gippsland Lakes catchment.



ABSTRACT

The major concern for Gippslanders, following the mine fire, is about their health.

They are not only concerned about excess deaths (mortality) but about excess illness (morbidity). It is also important to consider chronic illness in terms of a slow death, or shortened life span.

I have even had parents from East Gippsland, raise the possibility that their children's asthma may have been triggered by travelling through the mine fire smoke, returning from Melbourne, after the fire started. In that instance, reassurance is in order (but the fires in East Gippsland, throughout the time of the Morwell Mine Fire were possibly, more likely implicated).

Interestingly just this morning (3/8/15), local ABC radio announced that Gippslanders were bad at treating their asthma compared to the rest of the country because they present to hospital with asthma, more frequently. The spokesperson from Melbourne, seemed to have little idea of the other reasons, why Gippslanders might have severe asthma! It is laughable that residents are considered negligent, when the air quality is beyond their control.

Parents closer to the fire have more cause for concern. I am aware that many Morwell parents feel that their children are not as healthy as they were prior to the mine fire. Asthma, stomach aches and behavioural concerns have been mentioned. Their concerns are not unreasonable, since the evidence suggests that fine particulates are implicated in inflammation, which may cause both lung irritation but also brain inflammation leading to cognitive and behavioural effects as well as mental health issues.

The most studied particulates, in this situation, are Poly Aromatic Hydrocarbons (PAH's) and in particular benzo{a}pyrene (BaP). Benzo{a}pyrene has long been known as a carcinogen, originally linked to scrotal carcinoma in chimney sweeps (200 years ago). It is released by burning coal and diesel as well as wood fires. Studies in Poland and The Czech republic (also brown coal) have linked inhaled PAH's to pre cancerous cellular changes in buccal swab cells of children.

For pregnant women there are now many studies linking pre natal exposures to PAH's, with reduced IQ, autism, congenital heart defects and reduced birth weight. New information is coming to light, about genetic and epi genetic effects of poor air quality, as

well. Reduced birth weight, in turn is correlated with increased risk of cardio vascular disease, and diabetes as well as anxiety and depression.

Much more could be done to meet the concerns of parents. A Health study which ***eventually*** provides information on health risks, is not helpful to people who are already concerned about existing health problems. These parents need a service which can provide reassurance, toxicity testing, where appropriate, and treatment options to be fed back to the GP.

In the US they have a network of Paediatric Environmental Health Specialist Units, coordinated through the CDC. A similar service in the Latrobe Valley would provide real evidence of care from the Government. It could also be a hub for assistance to help children recover better. It could also be funded via mine security payments from the polluters or some such. An expanded service, might also include toxicology for agriculture, water quality etc.

RECENT INTERNATIONAL CHILD HEALTH STUDIES

At the recent Australian Environment Ministerial meeting, agreement has been reached on strengthening air quality

My colleagues and I agreed in-principle to two key actions under the Agreement's initial work plan.

In recognising the health impacts of airborne particles, it is our intent to strengthen the reporting standards for particles (PM2.5 and PM10) in the National Environment Protection (Ambient Air Quality) Measure.

We agreed in-principle to adopt annual average and daily PM2.5 reporting standards of 8 µg/m³ and 25 µg/m³, respectively, with a move to 7 µg/m³ and 20 µg/m³ in the longer term.

standards, as delineated on Minister Greg Hunt's website:

This suggests that both the Federal and State Governments acknowledge the need to tighten standards, in the face of mounting International evidence of harms from poor air quality.

A 2013 paper from The Czech Republic looks at children from heavily polluted industrial areas and compares them with a group of

children from a less polluted area.
They found higher asthma rates but also raised issues with PAH interactions with DNA¹

The BMJ reports a planned study, underway in Italy specifically looking at markers of genetic damage in children in polluted areas in the Po Valley. This was announced in September 2014. The analysis of results is to be shared with the public and policy makers and is an area that may be worth following as part of the Mine Fire Health Study. Importantly, this study proposes to use mouth swabs to collect cellular evidence of harmful exposures in children. Leukocyte micronuclei are seen as indicators of mutagenic risk for subsequent cancers². An awareness that air pollution may have genetic consequences is not new³

The fetus is at particular risk: indirect genetic effects are possible, with the potential for subsequent developmental disorders and other diseases with onset occurring in childhood, or later, or even in subsequent generations. Some animal studies suggest that effects may be seen for 3 generations, in some instances (mice fed BaP)⁵. This is partly related to the endocrine disruptor effects of PAH's. The precursor testicular and ovarian cells, of the embryo, and fetus are highly susceptible to epigenetic dysregulation by environmental chemicals, which can thereby exert multiple adverse effects

There has long been an association of low or lower birth weight with increased risk of adult onset cardiovascular disease, type 2 diabetes mellitus, depressive disorders and certain cancers.

With respect to prenatal exposures, there is an increasing body of evidence that diverse pollutants alter epigenetic programming and disease risk over several generations, so that grandchildren may bear the effects. These include heavy metals, indoor air pollutants like tobacco smoke, outdoor air pollutants, and endocrine disrupting chemicals⁴.

There is also data which indicates that BaP and other PAHs disrupt several neurotransmitters in the brain, such as via the glutamate pathway and the dopaminergic and serotonergic systems, correlated with observed disturbances of learning and emotional behaviour⁴

However care needs to be taken in interpreting data, as importantly, when you consider many households, three generations (foetus, mother and grandparents who are often carers) at once, are exposed to the same environmental conditions (diet, toxics, hormones, etc.). In order to provide a convincing case for epigenetic inheritance, an epigenetic change must be observed

in the 4th generation.

Wright 2010, has reviewed the evidence that psychosocial stressors and physical environmental toxins can combine (synergistic) to disrupt immune and endocrine pathways involved in respiratory and cognitive development and function. Oxidative stress is recognised as a pathway that may influence health. Pro-inflammatory immune pathways and autonomic disruption may occur. Both psychological stressors and environmental pollutants such as tobacco smoke and air pollution, all generate reactive oxygen species, which may change the internal milieu. Further, by causing disruptive behavioral states such as depression and anxiety, psychosocial stressors may produce long-lasting effects on physiology and thereby increase risk from pollutant exposures⁶.

Importantly, Wright notes that animal and human studies have also shown that environmental enrichment can reverse the effects of early stress .

There is a growing body of evidence that low birth weight and air pollution may influence mental health. Long term studies of low birth weight have indicated risks of depression in older males⁶ and young women⁷. There are some conflicting results but this is clearly an area where more research is needed⁸. However, anecdotally there was a doubling of adolescent depression in the year following the Black Saturday fires, noted both in the Latrobe Valley and further East within Gippsland, (personal communication with Gippsland Child and Youth Mental Health Service).

WHAT CAN WE DO TO IMPROVE OUTCOMES?

In the US there is a collaboration between the EPA and The Centers for Disease Control, to run Paediatric Environmental Health Specialist Units. These have a dual role, of responding to environmental issues and importantly, educating the health work force. Something similar, which provides access to paediatric assessment and also enrichment to affected children, would be two options that would help the community to cope⁹.

In response to parental anxieties, Premier Daniel Andrews, offered to send paediatricians from Melbourne to see patients.

This is not really a solution, as we need a permanent presence, with the ability to follow up children with continuing issues. There also needs to be a range of professional groups present, including, but not limited to, public health specialists, statisticians, toxicologists, paediatricians, maternal and Child Health Nurses, psychologists,

teachers.

Such a centre must be local and as well as seeing children, it should be available to advise, and educate as well as to assess children and their environment.

It could also have a research arm, and feed in to the longterm Health Study.

Setting up such a centre, would also be a mark of goodwill from government. If funding was sourced from polluters or perhaps as part of mine clean up bonds, the coal industry corporations would also contribute. This kind of support would help the local community, which suffered so much.

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