



## "Country Doctor"

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# Coal opencasting and health

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### FRONT PAGE

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My work has been peer reviewed by professors in the UK and USA and describes just a small portion of the health damage and cumulative effects caused by opencast mining. For 13 years I have researched industrial air pollution (including opencasting) with its consequential health damage of illness and premature deaths. Published research confirms that both PM1 & PM2.5 particulates produced by opencasting of coal, especially if toxic waste is present due to known or unknown tipping, CAUSE new cases of asthma to develop in children and adults as well as exacerbating those who already have it. There will also result increased incidence of chronic pulmonary obstructive disease, heart attacks, generalised premature deaths, strokes, type 2 diabetes, clinical depression and in addition other conditions resulting from any toxic waste contaminating the site which would include cancers, hormone disorders, birth defects, skin rashes, eye inflammation, nausea etc. due to pollutants such as organic compounds, heavy metals, dioxins (rife in North East Derbyshire), and even radio-active matter. Fuel quality used by equipment & vehicles is also critical as fuel used is normally of much inferior quality than city diesel.

Studies in NE Derbyshire (1994-2000) comprising school medical records, school asthma inhaler use, microscopy of dust outside and within buildings, and PM 2.5 monitor readings with filter analysis, at 5 schools covering a 3 year period, all confirm a rise in asthma to affect 33% of primary school children living within one mile, a cumulative rise to 21 % at two miles and even up to 12% at three miles. Welsh Office studies at Gwaun Cae Gurwen also discovered 33% of children in three schools to have developed asthma at one mile, based on peak flow readings. West Glamorgan studies found coal particles plus diesel particles in the PM10 filters partly upwind of the opencast, over the top of a large mountain. Peakflow measurements and asthma inhaler use worsened as particulate levels rose in direct proportion, and this happened irrespective of home conditions and social factors. A Lanarkshire study (1998) proved that hospital admissions for asthma rose with opencasting of coal, again within three miles or so, with cumulative rises year after year, falling when opencasting ceased. A Liverpool University study even showed a rise in asthma in schools within 2km of moving coal at the docks, irrespective of smoking habits and unemployment. Hospital admissions for asthma in the Tinsley area, since opencasting began at Orgreave, rose to 11 per 1000 population as against 3 per 1000 at Sheffield City Centre and 1 per 1000 in Worcestershire. All three areas have motorways. GP doctors in the area of SE Sheffield, namely Handsworth area etc., have noted a large rise in asthma

incidence in their area since Orgreave opencasting began. They are clear of the M1. The rise at Tinsley is not confined only to asthma, also diabetes, due to possible dioxin or arsenic contamination contained in PM2.5 particulates (which produce oxidative radicals). In London 0.5 miles away from the millennium dome site, the asthma incidence rose from 11.9% of school children in early 1996 to some 50% in November 1998, with the only change being the "opencasting" of that waste site development at Greenwich, which would have contained nickel, phenols etc.

Findings of microscopy and particle analysis, presented at the Royal Microscopical Society in London in July 1998, revealed that asthma caused by opencasting is due to:

a) Cut quartz particles of which 36% were found to be less than PM0.3, which are second to asbestos in terms of serious effects on the lungs. The body has to wall off these particles, causing fibrosis, which was called silicosis in underground miners, but which equally applies above ground.

b) Coal particles around PM 1 in size that cause an inflammation in the lungs lasting up to seven days after each dose. Repeated doses then lead to fibrosis, which is called pneumoconiosis in miners. That is why it only needs a weekly dose of fine coal dust from the workings to keep asthma active in the population living within three miles. That is just say one or two days a week, with the weather and wind determining who breathes the dust. Macrophages can only cope with a small amount of PM1 & PM2.5 particles at a time. All excess gets walled off producing COPD, even at age 10 years, as was discovered in a large study of Californian children. Coal dust by opencasting is as small as PM 1.

It only needs an increase of 14.3ug/m3 of PM2.5s for 3 hours to cause a heart attack in a vulnerable patient. Peak levels of PM2.5 in Derbyshire exceeded 150ug/m3 in 2000. Peak levels of PM1 measured at Hollingdean (Brighton) brown field site development by 4 bulldozers June 2007 reached 1100ug/m3 of PM1 (safe level around 5ug/m3) and 375 ug/m3 of PM2.5 (safe level around 7ug/m3 Remember only particles smaller than PM3 (3 microns) get into the depths of your lungs. The UK only measures PM10 with monitors checking PM4 to PM10 none of which get into the lungs and most instruments can be adjusted downwards. It must be stated that readings of PM2.5s rise and fall entirely separately from PM10s so PM10 monitors are totally useless in the UK for commenting on health damage.

c) Fuel emission particles of acidic carbon with heavy metal contaminants of the fuel, especially nickel sulphate, cause a lung inflammation lasting several days (maximum effects on the heart arising second day) plus heart attacks, strokes, cancers years later from the cocktail of metals including arsenic and cadmium. Analysis of PM2.5s in Derbyshire discovered high cadmium levels plus substantial levels of arsenic and mercury from a brown field site, mixed in with coaldust. Two polyaromatic hydrocarbons emitted in the vehicle exhaust fumes are carcinogenic, as happens from smoking cigarettes, due to disruption of the p53 gene allowing cancers caused by the heavy metals, PAHs and dioxins to grow. The particles leaving the engine are as small as PM0.02 but coalesce together to PM0.2, and then finally coalesce to PM1 to PM2 size, all of which remain in the lung when inhaled. Even healthy human volunteers revealed significant increases in white cells, histamine etc, in the lungs by just 6 hours after

inhaling road diesel exhaust, with increased white cells and platelets in the peripheral blood. A rise of just 14.3ug/m3 of PM2.5s for 3 hours has been proved to increase heart attacks (p=0006). The fuel used by such heavy equipment in the UK is normally industrial diesel, which can contain toxic waste oils and solvents. The solvents can cause brain damage and any heavy metals and/or other contaminants cause cancers. A single earth moving machine could release as many as 145 million billion ultra fine particles per minute, equivalent to some 900,000 Volvo V70 petrol cars. That is why the PM1 reading in Brighton 2007 reached as high as 1100ug/m3 away from the site. Young babies die of the inflammation set up by raised PM1 to PM2.5 levels.

An American study involving x-rays annually for twenty years, showed 55% of opencast workers had developed lung damage, proven by x-ray by year twenty. The UK government is paying compensation to above ground miners for this same lung damage caused by coal dust inhalation. In USA many dozens of train staff have been paid compensation for COPD caused by inhalation of emissions from diesel fuels. The USEPA has brought in laws to improve this off-road diesel quality including reducing sulphur content by 99%.

Local government named planning officers and councillors who vote for a proposal, ignoring this evidence, could be sued by victims who live within a three-mile radius of an opencast site.

At a public inquiry in early 1997, concerning Shortwood Farm, Nottingham my

evidence was tampered with and rewritten with different conclusions in the inspectors' report to my document agreed and accepted when I gave evidence. Furthermore RJB Mining had illegally been allowed to insert in their submission in the inspectors' report an allegation about my map being concentric, which had not been brought up when I was cross examined and hence was added after the inquiry, as admitted by their barrister at the Hoodcroft public inquiry, which I won. In fact in the Dolk Report in the Lancet 1998, the graph revealed a concentric critical distance of 3 miles radius around waste sites for a rise in birth defects. The inspector recommended approval of the opencast and public footpath applications, but in early 1999 at a high court challenge, the DETR admitted that decision was incorrect, overturned the approval, and offered costs to the councils involved.

In Wales in February 2008 the Minerals Planning Policy Draft Minerals Technical Advice No. 2:Coal was published for consultation with responses closing 23 May 2008 . They insist on a health impact assessment (done by Cardiff University for Kenfig Hill proposal which led to refusal) and a 350m buffer zone (likely to become 500m). But a true buffer zone should be I maintain 3km downwind at least to protect public health.

All my medical evidence concerns PM2.5 particles and below. These are man-made, and are the ones that enter the lung. PM10 printouts in the UK cannot be relied upon for accuracy or to comment on health effects. The DETR has admitted that the figures are massaged down and are not accurate. This is confirmed by the Environment Agency who also has admitted that their data is not always "accurate, complete, up-to-date or valid." If PM IOs are an issue, then note that Professor Harrison's latest survey (1998) shows a contribution from the continent of Europe reaching the UK, of PMIOs around 1 ug/M3 only, certainly regarding NE Derbyshire. Also note that PM IOs recorded in January 1997 for this area, showed figures between 46 and 60ug/M3. Later months cannot be relied upon for reasons given. The highest PM 10 figures in the UK have been not from the highway traffic but from sites such as the opencasting of brownfield land in Brighton and of a burning coal tip in Standish and around the Castle Cement plant at Clitheroe (recordings of up to 250ug/M3 were found in a hospital and 600ug/m3 of PM2.5s downwind outside in the open).

Experience gained at Arkwright proves that the alleged ability to control dust by opencasters is a fallacy. I was present when the television filmed the emissions at Arkwright with separate clouds of coal-dust and vehicle emissions. PM2.5s rise, and can stay suspended in the air for up to one week while travelling downwind, totally dependent on the weather as to where and when they land to ground level where they may be inhaled into the lungs. Maximum grounding takes place at 11 pm and 4am when the air is cooled, confirmed by monitors. Motorways nearby increase the problem, by adding more vehicle emissions and generating heat, which keeps the particles suspended for a longer period, facilitating spread. The alleged developers mitigation measures are almost irrelevant as they do not resolve the real problem which includes use of non road diesel quality fuel. The PM2.5 and PM1 dust cannot be controlled.

PM2.5 measurements in the latter half of September 1998 have revealed higher levels at Grassmoor and Hasland, Derbyshire some 2.1 miles from Arkwright opencast than at Tupton and Wingerworth at about 3.1 miles from Arkwright. Levels at Grassmoor were as high as 42.5ug/M3, which is 4.25 times the WHO and US EPA recommended maximum levels. Peaks of PM2.5s in Oct. 1998 reached 80ug/M3 in the Hasland area and 150ug/m3 in 2000. This confirms the cause of the higher asthma incidence at Grassmoor and one could now expect those exposed to that sort of level, to have their lives shortened by some six years. Interestingly those in Arundel live some six years longer. Analysis of the filter heads confirmed that coal dust was the main ingredient.

The NHS is paying the bill. With cost limited frozen budgets now affecting both hospitals and PCTs, which patients will be denied treatment to pay for those made ill or who die, through opencasting? In USA , costing for health damage is being added to production costs prior to decision-making. Dare we?

What knowledge of medicine and toxicology has a Mineral Planning Authority got? What training has an environmental services department in medicine and toxicology? What postgraduate tuition in toxicology have public health directors received and from whom? COMEAP have huge conflicts of interest and their references are years out of date. Dr. Pless-Mulloli admitted 1997 at CwmBran

that her Newcastle report was "all fraud" and "political" and proves nothing due to a fraudulent protocol and methodology. There was no before-during-after data. There were no peak flow measurements. Ages 1 to 11 were supposed to fill in forms. In area 5 medical records of 38 patients

were taken when parents had DECLINED consent. Figures were "adjusted" and "cleaned" and discarded to suit. The controls were chosen with equally bad pollution & one overlapped. There were no coal or diesel particles found in filters. Numbers of returned forms were very low despite reminders. GPs were not involved. In one area GP data revealed 28 children were asthmatic but the report stated none. A PM10 reading of MINUS 4.9ug/m3 was rewritten as PLUS 0.5ug/m3. There were found numerous errors in numbers, scale, directions etc. totalling over 100.

Will named councillors and public health doctors who pass unsafe applications be forced to compensate? Article 8 of the Human Rights Convention should be used in the courts to force disclosure of raw pollution data and relevant health authority data, in consideration of public health risks of imposition by government agencies. The GMC in March 2002 stated that disclosure of health data in the public interest is justified. Article 16 states that use of one's rights (to opencast etc) must never ruin somebody else's rights. Article 2 provides for right to life.

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References and data were obtained from Dr D Williams, Mr P Ordidge, Royal Microscopical Society Conference July 1998, Epidemiology July 1995, West Glamorgan HA, Lanarkshire HA, Ken Coates MEP, English Partnerships (and CPL data), USEPA Research & Harvard School of Public Health, Health Effects Institute report May 2000, Respiratory Morbidity in Merseyside School Children exposed to coal dust and air pollution, in Archives of Disease in Childhood 1994;70:305-312 & Doctor Salvi et al AM J RESPIR CRIT CARE MED 1999; 159: 702-709. Also Proc R Coll Physicians Edinb 1999; 29;1115- "Health Effects of Respirable Dust from Opencast Coal Mining" by Doctors Munro and Crompton. This article backs up my research. What Car magazine of June 1999 contains an article analysing vehicle particle emissions from PM0.01 to PM 1, which reveals the scale of the problem just from ULSD. How much worse in content must emissions be with industrial vehicles using lower quality diesel/fuel? I append 218 relevant references.

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