

**There is an increasing requirement for more airports and runways as travel increases. As a consequence, more people are being exposed to Aviation fuel. What groups of people might be exposed, and is there any toxicity associated with exposure to aviation fuel?**

With Aircraft travel on the increase and flight paths changing more frequently since September 11<sup>th</sup> 2001, a larger number of people are being exposed to aviation fuel pollution. It seems that aviation fuel is taking up a large slice of the pie of pollution caused by transportation. Indeed, the United Nations has released a report stating that aviation is responsible for over half of the pollution caused by transportation. In comparison to ground transportation with its millions upon millions of vehicles, there are surprisingly few aircraft (34,444 US-civil, 5,778 US-commercial). Thus, one can only imagine the massive amounts of pollution they emit. A loaded jumbo 747, for instance, uses tens of thousands of pounds of fuel on merely take-off. The pollution from just one, two-minute 747 takeoff is equal to operating 2.4 million lawnmowers simultaneously!

So which groups of people may be exposed to this pollution? There are many different groups of people with varying levels of toxicity. Airport staff such as ground staff, signallers and baggage handlers are exposed to both aviation fuel liquid and vapours from aviation fuel if spilt accidentally. In addition to this they will be exposed to the products of combustion of aviation fuel. Staff who work on actually refuelling the aircraft may be contaminated with aviation fuel if it is spilt onto the skin and absorbed percutaneously or accidentally taken orally. There are also a large number of people who live surrounding airports and are below various flight paths who may suffer from toxicity from the products of combustion of the aviation fuel. Another large group of people with occupational exposure to aviation fuel are those who work in the Royal Air Force in the UK and other aviation defence units in other countries.

I will look first at probably the largest group of people affected by aviation fuel; the millions of people worldwide who live within close proximity of an airport. A report produced by the Seattle- King County Department of Public Health in 1997 compared residents of Georgetown, an area of Seattle which surrounds King County International Airport to King County which surrounds greater Seattle. When comparing hospitalisation rates for Georgetown to those of King County Georgetown residents were found to have:

- a 57% higher asthma rate
- a 28% higher pneumonia/influenza rate
- a 26% higher respiratory disease rate
- an 83% higher pregnancy complication rate
- a 50% higher infant mortality rate
- higher rates of genetic diseases

The average life expectancy was found to be 70.4 years in Georgetown a rate comparable to that of many developing countries whereas Seattle's average life expectancy was 76.0 years. Mortality rates for all causes of death were found to be 48% higher in Georgetown compared to King Counties. These findings show alarming differences. However, a

study has yet to show a causative link between the pollution of aviation fuel and the higher mortality and morbidity rates found in the report produced by the Department of Public Health. This preliminary epidemiological study does seem to indicate the need for more specific research to be carried out regarding the toxicity of particles produced by the combustion of aviation fuel.

In August 2000 a study by Environ International Corporation detected 219 volatile compounds in the air around Chicago's O'Hare International Airport. 78 of these chemicals were at "increased levels". The estimated resulting cancer risk for people living near the airport was five times higher than the regional average proving just how dangerous it can be to live near an airport. In order to look more specifically at what factors could be causing the increased risk of cancer I am going to look at the specific chemicals found in aviation fuel and their ability to cause cancer and other diseases.

Aviation is responsible for emissions of nitrogen oxide, hydrocarbons, sulphur dioxide, naphthalene, benzene, benzo[a]pyrene, formaldehyde and dust particles to mention but a few. These chemicals may affect the body in various ways depending on the mode of transmission, for example, naphthalene may dissolve from the air into the lacrimal secretions of the eye. The eye may be a target organ as a result of its external position in the organism and direct exposure. Exposure of the eye to naphthalene may damage the lens and trigger the process of cataract formation. Naphthalene is also toxic to the lung when inhaled. It is metabolised by cytochrome P-450 in Clara cells (non-ciliated bronchiolar epithelial cells) to a cytotoxic epoxide intermediate. This epoxide is then able to bind to DNA forming an adduct which distorts the macromolecular structure of the DNA and hence its ability to function correctly. This change causes carcinogenicity of the pulmonary cells.

Another common pollutant of combustion of aviation fuel is nitrogen dioxide. This has been said to be found in the air in increased levels up to a 20 mile radius away from most major airports. Nitrogen dioxide can cause peroxidation of cellular membranes causing direct damage to cells of the respiratory tract as airborne particles. This damage to the cellular membranes in the upper airways (trachea or bronchi) can cause a change in permeability and extensive oedema. When this is coupled to the irritant effect of sulphur-dioxide and dust particles, also present in aviation fuel pollution, lung function may be compromised. If considering a patient with an underlying condition such as a child with asthma, lung function may be dangerously compromised. The sulphur-dioxide and dust particle irritants may cause an allergic asthmatic response causing IgE mediated inflammation and sympathetic constriction of the airways. A Swedish study looked at the effect of NO<sub>2</sub> exposure and wheezing rhinitis and found a link between increased NO<sub>2</sub> levels over a day care centre and the symptoms suffered by the patients living there (Pershegan et al 1995). This would suggest that people living in the surrounding area of an airport with increased levels of NO<sub>2</sub> due to the combustion of aviation fuel would also suffer more frequent symptoms of wheezing rhinitis than the general population.

It has been noted that in addition to the obvious effects on the lung of airborne pollutants there are also many systemic effects seen in people exposed to air pollution such as that

created by combustion of aviation fuel. The American Heart Association carried out research to try and find an explanation for general extrapulmonary effects of pollution by particulate matter. Pollution by particulates has been consistently associated with increased cardiovascular morbidity and mortality. However, the mechanisms responsible for this have never been well exemplified. In this experiment the American Heart Association labelled carbon particles with  $^{99m}\text{Tc}$ . Radioactivity was detected in the blood at 1 minute and reached a maximum concentration between 10 and 20 minutes and remained at this level until 60 minutes. Gamma camera images showed substantial radioactivity over the liver and other areas of the body. This seems to account for the hepatotoxicity and other systemic toxicity caused by some pollutants. The pollutants seem to be taken up in the lung where the majority of the pollutants cause cytotoxic damage, eg benzo[a]pyrene and naphthylene and then pass rapidly into the circulatory system due to the fact that the lungs receive 100% of the cardiac output. Pollutants, notably carbon particles as in this experiment, and other membrane permeable airborne pollutants are then free to pass into vascularised organs, for example the liver and kidneys. This goes some way to explain the systemic effects of some airborne pollutants.

I then went on to look at specific extrapulmonary symptoms caused by particulate matter pollution. It has been suggested that the increased pollutant levels can contribute to the onset of acute myocardial infarctions (MI). A study by the American Heart Association looked at hourly concentrations of particulate mass  $<2.5 \mu\text{m}$  ( $\text{PM}_{2.5}$ ) a product of aviation fuel combustion, carbon black and gaseous air pollutants. A case-crossover approach was used to analyse the data for evidence that these pollutant levels could trigger the onset of a MI. The study looked at exposure to fine particulate matter in the previous 2-hour period and MI after a delayed response associated with 24 hour average exposure 1 day before the onset of symptoms. Multivariate analyses considering both time windows jointly revealed an estimated odds ratio of 1.48 associated with an increase of  $25 \mu\text{g}/\text{m}^3$   $\text{PM}_{2.5}$  during a 2-hour period before the onset and an odds ratio of 1.69 for an increase of  $20 \mu\text{g}/\text{m}^3$   $\text{PM}_{2.5}$  in the 24-hour period 1 day before the onset (95% CIs 1.09, 2.02 and 1.13, 2.34, respectively). These results were statistically significant but the study may have been improved. The experiment relied on self-matching results which meant that risk factors were stable over time within an individual but differed between study subjects. In its favour, the study did have specific criteria for positive inclusion in the study and looked at a large number of cases (772 participants). In conclusion the study showed that high levels of  $\text{PM}_{2.5}$  (such as those produced by aviation) could precipitate a MI in individuals with predisposing factors. For example; hypertension, high low density cholesterol (LDL) levels, smoking, obesity.

Benzo[a]pyrene is a polycyclic aromatic hydrocarbon (PAH) and is an extremely potent carcinogen. Many PAH's including benzo[a]pyrene are present in aviation fuel. When taken in to the body PAH's go through a wide variety of metabolic transformations catalysed by the microsomal mixed function oxidases. The ultimate carcinogen is an epoxide of a dihydrodiol metabolite where the epoxide is adjacent to the "bay-region". It has been demonstrated that the 7,8 dihydrodiol of benzo[a]pyrene reacts covalently with

nucleic acids in vitro, provided that the microsomal enzyme systems necessary for its activation are present, causing mutagenic DNA. The benzo[a]pyrene can form an adduct on to the p53 tumour suppressor gene which then leaves open a window for mutated cells to replicate and a cancer to form.

A molecule in aviation fuel which may be absorbed by people working in close contact with aviation fuel, for example, the people who refuel the aircraft, is benzene. Incidental exposure or accidents can allow acute toxicity of benzene. Acute toxicity can cause symptoms of euphoria, headaches, nausea, staggering gait eventually leading to coma, convulsions, respiratory collapse and circulatory arrest. Obviously these symptoms can cause fatal illness if the worker is not removed from the source of exposure of benzene quickly. However, probably a more worrying consequence of working with benzene is chronic toxicity. Many staff at airports and people working in the RAF may all be exposed to small doses of benzene on a frequent basis. Benzene is a highly lipophilic molecule. It is readily hydroxylated within the cell. The major product of benzene hydroxylation are phenols. However, catechols and quinols may also be formed from further metabolism. One of the toxic effects of chronic exposure to benzene is that it may cause aplastic anaemia. Benzene can be metabolised in vivo by cytochrome P-450 to form benzene oxide this may then be converted to a phenol and then hydroxylated to either a catechol or hydroquinone. The catechols and hydroquinones may then be converted to a-benzoquinone and b-benzoquinone respectively. Both a-benzoquinone and b-benzoquinone are thought to be able to attack and damage the DNA of pregenitor cells leading to the development of leukaemia and aplastic anaemia.

I have looked at various people who may be affected by aviation fuel and it's possible toxic effects. The toxic effects may vary depending on whether the dose is toxic or chronic, e.g. acute benzene toxicity and aplastic anaemia. Also any protective clothing worn could affect the outcome to exposure of toxic compounds in aviation fuel e.g. formation of pulmonary cancers if naphthalene is inspired and the formation of cataracts if it is spilt into the eye or if vapours dissolve into lacrimal fluid. Pulmonary and extrapulmonary toxicity may be caused by inhalation of products of aviation fuel combustion by passengers, airport staff and people living in areas surrounding airports. Aviation fuel seems to be a causative agent in many health problems and its use may have to be contemplated by Departments of Health over the world. Various suggestions by campaigners such as Jack Saporito, President US-Citizens Aviation Watch Association, and Executive Director of the Alliance of Residents Concerning O'Hare for a decrease in the use of aviation fuel have been to put limits on minimum passengers for each flight, decrease frequency of internal and local flights and to encourage use of alternative transport such as trains.

### References

- Principles of Biochemical Toxicology; Timbrell, John A (2000)  
"Increased Particulate Air Pollution and the triggering of Myocardial Infarction"  
(Circulation 2001; 103:2810) American Heart Association  
Seattle - King County Department of Public Health Report (1997)  
Environ International Corporation Report (August 2000)

Nitrogen dioxide exposure and wheezing rhinitis. Pershegan et al (1995)  
"Passage of inhaled particles into the blood circulation in humans" *Circulation* (105:411)  
American Heart Association (2002)