Learmount
Operationally speaking

http://www.flightglobal.com/blogs/learmount/2012/11/aerotoxins-the-fog-begins-to-c.html

Aerotoxins: the fog begins to clear

The fight against aerotoxic syndrome has taken a major step forward with the announcement that a scientist at the University of Washington, Seattle, has identified modified chemicals that will reduce the toxicity of anti-wear additives in aero engine oil without reducing the lubricant's effectiveness.

Several recently reported incidents of cabin air contamination in German airlines (see previous blog entry) have brought to the fore once again the risks of engine oil fumes harming human health, particularly pilot cognitive and motor capacity while they are flying the aeroplane.

This discovery has the potential to reduce the health risks of oil fume events in aircraft, which has been an intractable problem since identified first in the 1950s.

The organophosphate tri-cresyl phosphate (TCP) is an essential component of the oil anti-wear additive, and it has long been accepted that certain organophosphates are neurotoxins.

Clement Furlong, Professor of Genome Sciences and Medicine at Washington University's School of Medicine explains that he has proven a test for identifying which are the neurologically harmful isomers of TCP, and which the less harmful.

What he has found turns accepted beliefs upside down.

Tri-ortho-cresyl phosphate was believed to be the only harmful TCP isomer, so the oil manufacturers tried to reduce its presence in the additive to very low levels, but it turns out that other TCP isomers which they believed harmless - and are present in large quantities in the oil- are in fact the culprits.

In simplified scientific terminology Furlong explains what he and his team have achieved: "An in-vitro screening protocol for potential toxicity of triaryl phosphates was developed...Isomers of tert-butyl phenyl phosphate produced the least BChE inhibition in vitro." BChE is butyrylcholinesterase, a naturally occurring and essential chemical in the human neurological system the beneficial action of which is inhibited by certain isomers of TCP, leading to neurological damage.

When also tested on mice, Furlong explained: "BChE inhibition by in-vitro bioactivated TAPS correlated with esterase inhibition in vivo [in the live tests]". TAPS are toxic triaryl organophosphate anti-wear lubricant additives.

In other words, this is the chemical and biological proof of their neurological harmfulness, which large sectors of the industry still deny.

Finally, Furlong has discovered that extracts from grapefruit can reduce the harmful effects of TCPs, and has demonstrated their effectiveness: "The flavonoid naringenin reduced bioactivation of TCPs into BChE inhibitors."

Eureka.

Clem, you are a genius!

AND CLEM HAS ALREADY CORRECTED ME AS FOLLOWS:

I should clear up a couple of points from the text below.

1) BChE is not a necessary enzyme for neurological function. It is just a good biomarker protein.

2) I don't know if the tri-tertbutyl phenyl phosphates have good lubrication properties. Only Eric Piveteau [Nyco Oil] will know.

3) While naringenin can block the in vitro conversion of TAPs into toxic esterase inhibitors, we should not push the concept that it can protect against exposures without important follow-on in vivo experiments.

It is important not to claim progress that industry could easily refute or criticize. The generation of potent enzyme inhibitors from D125 and from tri-p-cresyl phosphate are indeed important contributions. The naringenin inhibition needs in vivo follow-up experiments. It will be great if it blocks the toxicity of the exposures, but it is important to demonstrate in vivo.

Kind regards,
Clem Furlong