

**CHRYSOTILE MESOTHELIOMA CAUSATION IN TRIAL –
MEDICINE/SCIENCE v. TRIAL/BLAME A DEFENSE PERSPECTIVE**

The Forum: Trial is inherently not a scientific arena – plaintiffs count on that fact, defendants attempt to combat that fact

The Question: Are plaintiffs successfully shifting the burden of proof to defendant?

The Issues at Trial: Is the focus really on scientific causation, or on increased risk only? What's the difference? Is every exposure really causative? Does each exposure really contribute? How can you differentiate between substantial contributing exposures? Must the exposure be quantified/classified? How can it be?

1. Scientific Evidence v. Regulatory Pronouncement

A. Scientific Articles

(1) Epidemiology

- (a) *McDonald, Camus, et al* – the Quebec miners and millers studies

Is the Chrysotile contaminated? Is there an Amphibole explanation?

- (b) *Dement, Stayner* - the S. Carolina textile plant studies

Is fiber length the difference?

- (c) *Yano* – the Chinese mine study

But the Chrysotile was contaminated, per *Tossavainen*

- (d) *Piolatto* – the Balangero mine study

Is it truly a pure Chrysotile study if cannot rule out the role of Balangeroite (an asbestiform contaminant similar to Crocidolite)?

- (e) *Henderson* – Australian Registry

What is this, anyway? Does it mean anything?

(2) Case studies/reports

(a) *Iwatsubo* - 1998

- Doesn't distinguish between fiber types
- Low dose – “sporadic” exposures not at higher risk
- “Intermittent” exposures not at higher risk

(b) *Rodelsperger* - 2001

- Doesn't distinguish between fiber types (“in spite of its well-known importance”) – and it's an amphibole study
- Occupational models don't fit all exposures
- “Intermittent” exposures not at higher risk

(c) *Rolland* - 2006 abstract

- Doesn't distinguish between fiber types
- Never published – abstract only despite over 3 years out
- Occupational models don't fit all exposures

B. Fiber burden analysis and evidence-based medicine

(1) What can/do you find

Amphibole exposure – often contrary to purported exposure history

Roggli/Churg studies

(2) What do the findings mean

Actual evidence – remember work histories are “unreliable”

Do the Chrysotile fibers remain – and if they don't does it mean they weren't harmful

(3) Lung tissue v. pleural tissue, lymphatic tissue

Does it matter where the fibers are found? If there are Chrysotile fibers in the pleura, do we know if they did anything once they arrived?

(4) Pleural plaques, asbestosis – asbestos markers

C. Dose Calculations

Industrial Hygienist retrospective dose calculations v. medical “expertise”

D. Meta-analyses – relative potency/toxicity

- (1) *Hodgson and Darnton, 2000* - 500-100-1 range
- (2) *Berman and Crump, 2003* - 800-1 range
- (3) *Peto and Hodgson, 2005* - no weight to Chrysotile
- (4) *Yarborough, 2006* - zero potency for Chrysotile
- (5) *Berman and Crump, 2008* - zero potency for Chrysotile

E. Expert Panel “Consensus” Documents

- (1) *Helsinki Expert Panel Consensus - 1997*
- (2) *EPA - 2003*
- (3) *ATSDR - 2003*
- (4) *Chrysotile Consensus Expert Panel - 2007*

F. Regulatory Pronouncements

OSHA, EPA, WHO, etc. etc. – it’s not science, so who cares? Juries v. scientists/physicians

2. Judicial Opinions on Low-Dose Causation

A. Judge Colville’s *Frye* opinion

Remanded yes – but overturned?

B. Judge Tereshko’s *Frye* opinion

Still intact

C. The Washington bench *Frye* opinions

Judge Barnett, Judge Erlick both excluded extrapolation-down testimony

D. *Borg-Warner* opinion

Must show retrospective dose for the individual