Asbestos as ‘toxic short-circuit’ optic-fibre for UV within the cell-net: — Likely roles and hazards for secret UV and IR metabolism

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Abstract. The most toxic asbestos fibres have widths 250 nm - 10 nm, and this toxicity is “physical”, which could mean either mechanical or optical: Tangling with chromosomes is a mechanical hazard occasionally reported, and fibres <100 nm wide would probably be most knife-like. Our other concern here is optical: Calculations for fibres ≤300 nm reveal such a transmission possibility, but only when the amphibole fibres (brown and blue asbestos) are >100 nm wide — or chrysotile (white asbestos) is >150 nm. In both cases, UV A/UVB -transmission would then predominate. (Chrysotile 150 nm might be benign — escaping both mechanical and optical!). But what would generate such UV, and why would its transmission be toxic? Thar and Kuhl (J.Theor.Biol.:2004) explain that the long mitochondria on microtubules may be able to act as UV-lasers, (and many observers since Gurwitsch 1923 have reported ultraweak UV emissions escaping from all types of living bio-tissue). That all suggests some universal secret role for UV, apparently related to mitosis. Insertion of fibre “short-circuits” could then cause upsets in mitosis-control, and hence DNA irregularities. Such UV-control could parallel similar lower-powered Infra-Red control-systems (as considered elsewhere for coaxial myelin; or as portrayed by G.Albrecht-Buehler’s online animations etc.); and the traditional short mitochondria seem better suited for this IR task.

1. Introduction — Are we dealing with Toxic Geometry?

1.1 “Physical damage” rather than chemical
Asbestos fibres are clearly toxic — but why exactly? It seems universally agreed that their effect is non-chemical: — In 1978, Stanton and Layard [1] showed persuasively that the pathogenic properties of such fibres did not correlate with the chemistry of the fibres, but rather to their physical dimensions: — and they went on to list some of the geometrical correlates shown to

† or, if obsolete, consult the website: www.ondwelle.com
exist: Thus the stronger causal effects occurred with **diameters of 10-250nm**, and moderate effects with 250–1500nm thicknesses.\(^1\)

“Non-chemical” implies “physical” — but in what way? — It is easy to assume that this means “mechanical damage”, and there is evidence that this is sometimes true, as we shall see. But a more interesting possibility is that the cause is **optical**, since crystal-like solids often do feature in our dealing with light, though it may not be immediately obvious just what damage might arise in this way.

(That probably exhausts the list of feasible primary-cause possibilities, though we should remain open to the unexpected, and more of the less-likely options are mentioned later. For instance one could imagine acoustic damage, but it is difficult to see that as plausible. So let us concentrate mainly on the two existing suspects.)

1.2 **Damage to what?**

We are not just talking about membrane-penetration, or blood-letting lesions caused by a needle — the damage in question will affect future growth at that site, and maybe later at secondaries elsewhere. So the damage will almost certainly relate to the DNA, chromosomes, and mitosis. Thus the question narrows to asking **“What mechanical or optical effects could cause such chromosomal disruption?”** — and indeed **“How?”**

1.3 **What do we know about toxic fibres anyway?**

First note that, since our concern is with the physics and geometry of the fibres (and not their chemistry), we should extend our study to any other fibres with similar physical properties — notably glass fibres. That should come as no surprise, as such fibres have already been under suspicion for some time. If their toxicity seems less, that could well be due to their geometry being less extreme (and more under design-control) than the asbestos fibres provided by nature.

Thus we may provisionally assume that two fibres with the same geometrical configuration, electro-optical properties, (and perhaps density and stiffness) will be equally toxic-or-benign, regardless of any chemical difference between them. Hence this study will not be confined to asbestos alone.

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\(^1\) They also raise some other geometrical issues which we will not pursue here: — (i) the length and/or aspect ratio may also be significant in a less simple way. But if geometry is part of the cause, it is interesting to also see it as part of the effect, and that might be seen as significant. Thus: (ii) “cancer can be viewed as a disease of geometry. Tumor tissue results from growth, which is not patterned appropriately, because it is unable to perceive or execute morphogenetic cues.” — quoting Levin.
Table I
(adapted from [2], with permission)

Summary of the various supposedly-homogeneous dielectric fibres which can be seen as an “asbestos-like” toxic hazard — especially if their diameters are very fine (10–250 nm). There are two polarized opinions about the relative toxicities of chrysotile and the other (amphibole) asbestos types, as suggested by the two examples depicted in columns F & G.

| A | Names of dielectric fibre-types. | B | common name | C content-biases, or chemical formulae where: “♣” = Si₈O₂₂(OH)₂ | D structural pattern | E Refractive Index min – max | F [3] toxic? | G toxic? Hesterberg (1987) [4] | soluble? |
|---|---|---|---|---|---|---|---|---|---|
| 1 | crown “E glass” *v | (textile) | incl. Ca, Mg, Al, +maybe Na,K,B,F,Ti | amorphous | 1.508 – 1.549 | — | | |
| 2 | crown glass-wool *v | (insulation) | incl. Na, Ca, B, Mg, (+maybe Al, K) | amorphous | 1.530 – 1.587 | — | | |
| 3 | Flint glass *v | lead glass incl. Pb, (or Ti, Zr) | amorphous | ... 2.1 | — | No |
| 4 | (Ortho)chrysotile = mitaxite *v | white asbestos (note its uniqueness) | Mg₃Si₂O₅(OH)₄ | orthorhombic prismatic | 1.569 – 1.57 | slight? | 6X query! yes |
| 5 | Grunerite = Amosite = ferro-ruebeckite = (..)-crocidolite *v | brown asbestos | Fe³⁺ –♣ w | monoclinic prismatic | 1.663 – 1.709 | XX | ? No |
| 6 | Magnesio-ruebeckite. = (..)-crocidolite *v | blue asbestos | (Na₂Mg₂Fe³⁺₂) –♣ w | monoclinic prismatic | 1.68 – 1.70 | XX XX | X No |
| 7 | Tremolite, (Actinolite) *y | Ferro-actinolite (rare) | Ca₄Mg₆ –♣ y,w Ca₄Fe³⁺₂Mg₃ –♣ w | monoclinic prismatic | 1.599 – 1.626 | XX? | ? No |
| 8 | Cummingtonite = Anthophyllite, (rare) | 1. Mg₇ –♣ w (some Fe) | orthorhombic | 1.598 – 1.685 | slight | X No |

* Tennent [5] | * webmineral.com [6] | * Shelley [7] | * Mineralogists’ usage (1997) [8]

Secondly note that most asbestos types are classed as “amphiboles” with similar physical properties (and similar chemical formulae — See Table I). These amphiboles notably include the notorious “blue asbestos” (crocidolite), and the “brown asbestos” (amosite) — both with a refractive index of about 1·7, and both producing relatively straight and “tidy” fibres.

In contrast, there is “white asbestos” (chrysotile, n ≈ 1·57, often as “untidy” fibres, very slightly soluble) whose toxicity is in dispute, and which is still being used commercially in some countries. This uniqueness raises some interesting questions which we will encounter several times.

2. **What Physical Causes should we consider seriously?**

2.1 **Primary causes rather than secondary**

It seems sensible to restrict our search to whatever it is that *initiates* the toxic chain-of-events. — After that, the main damage has already been done (even though it may still be curable later on).
Indeed whatever the physical damage from asbestos may be, it will next have biochemical consequences, which are then themselves causes — secondary causes (like inflammation) which will probably affect other subsystems further down the causal chain. That constraint helps to clarify the issue — and limit the short-list of likely candidates somewhat:

2.2 Mechanical and Optical taken as the most likely toxic effects
In this context, “physical” is perhaps usually assumed to mean “mechanical”, as if each fibre were a miniature knife. That has some justification of course, so it deserves our attention.

But physics extends further than that, so we could plausibly blame direct-optical effects instead — especially if we view asbestos fibres as “windows onto a radiation landscape” (though it is then our duty to identify that radiation-hazard if possible). Such window-like properties would occur if we accept asbestos fibres as either crystals or potential optic fibres — and their geometry obviously suggests the latter.

2.3 Other conceivable toxic effects from asbestos
Toyokuni [9] lists three physically-toxic possibilities: (i) oxidative stress” from “frustrated macrophages” trying to dispose of the intruding fibres (and perhaps we can class each such fibre as presenting a physical “spanner in the biochemical works”); (ii) chromosome tangling (a clear case of the mechanical damage already mentioned); and (iii) adsorption onto the fibre, which could then concentrate any toxic proteins. — We might see the two cases (i) and (iii) as pseudochemical though still perhaps “arguably physical”. That does not mean they are necessarily wrong, but they (and other remote possibilities) will not be considered further here. Meanwhile (ii), already on the list, is now discussed further:

3. Mechanical Damage as one Key Cancer-trigger
There is some good evidence that this occurs when very narrow fibres (100nm–10nm) physically tangle with chromosomes. Thus:
• Wang et al [10] point to more polyploidy caused by chrysotile which seems inclined to “sever, puncture, or adhere to and mingle with chromosomes, causing deformities”
• Ault et al [11 (p796, fig5)] : “Chromosome breakage in a mitotic cell containing…fibers. …As the spindle rotated,…[chromosome] arms were dragged through…fibers. …break… and the resulting chromosome fragment…was left behind at anaphase.”
• Jaurand, et al., (1987), quoted by Voytek [12] “In rat mesothelial cells, chrysotile… with 84%... having a diameter of less than 0.058 μm, caused chromosomal aberrations... predominantly chromatid breaks...”

Such entanglement seems less likely for thicker fibres, which would increasingly appear to be like a perhaps-harmless blank wall — and therefore less like needles or thorns for the 30nm DNA/histone chromosome-threads. Nevertheless a mobile “blank wall” could at least be disorganizing.

4. Optical Damage as the other Likely Primary Cause
My own interest in the topic started when I pondered whether asbestos fibres might be transmitting infra-red — an idea prompted by the still unproven hypothesis that myelinated nerve fibres do just that (in addition to their acknowledged role of carrying action-potential pulses). [13] Some simple calculations and assessments quickly revealed that IR was absolutely impossible for these much smaller (and non-coaxial) asbestos fibres, though UV was a not-too-implausible
candidate, especially as it had much more destructive power (in some unspecified way) — but it was far from clear where such UV might come from.

This present revised version of the Optical Damage proposal builds on (i) the now-prolific evidence that living tissue universally emits occasional ultra-violet photons — and (ii) a more-specific recognition of the fact that such UV photons have uncharacteristically high quantum energy which is likely to do DNA-damage if misapplied.²

The perhaps-inevitable conclusions were eventually (i) that UV-emissions must be serving some (universally?) important metabolic role, but (ii) such photons needed to be carefully controlled and directed lest they cause damage, and perhaps miss some crucial target as well). So — (iii) If asbestos happened to act as fibre-optic “short-circuits”, it could seriously disrupt that UV system — causing DNA damage, and hence sometimes initiate cancer.

That leads to the crucial question:

4.1 Can asbestos actually transmit UV? — Experiments needed

Ideally that question calls for systematic laboratory investigation (and let us hope this will occur in due course, despite the obvious expense (♠3)), but meanwhile we must make do with theoretical calculations:

4.2 Instead: Calculated UV transmissibility under various Conditions

First note that as we go deeper and deeper into the UV range, wavelengths will shorten down towards 200 nm — and the waves will then cease to be transmitted through any material at all, because such radiation is all absorbed (due to resonance with the outer electron shells of atoms, [14 (p.483)] — so no calculations are needed in that extreme! However this effect does also start to make calculations difficult for nearby wavelengths — see [2].

First note that asbestos fibres are not those specially-designed optic-cable fibres which have a denser axial core for “steering” any light-beam away from the boundaries. (Nor are they coaxial cables, in the way that myelinated nerves arguably seem to be). Instead any UV/asbestos transmission would have to be by some form of “zigzag” reflection to-and-fro across the fibre — and that can only occur under certain conditions: (i) The refractive index ratio \(n/n_{aq}\) (fibre/aqueous-environment) must be high enough. (ii) The fibre-diameter \(D\) must be large enough for a half-wavelength loop \((\frac{1}{2}\lambda)\) to fit across it and leave some room for other complications.

Thus if \(D=\lambda\) it will be a borderline case. With that in mind we can sometimes get a good idea of a fibre’s optical capabilities without the need to calculate exactly. For instance, if a certain natural fibre has diameter \(D=300\, \text{nm} (0.3\, \mu\text{m})\), we can confidently guess that it will not be able to transmit visible light, for which \(\lambda>400\, \text{nm}\); but it might well be able to transmit UV. So we might then wish to investigate the situation further.

To calculate the transmissibility “should” be a fairly simple task — the above-mentioned use of “\(n/n_{aq}\)” within Snell’s law should ensure the right conditions for TIR (total internal reflection) so that the light does not escape from the fibre (or slab etc). The trouble is that we need to know

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² as is shown (obviously) by skin-cancers caused by excess UV exposure from sunlight or solariums.
³ As in the previous paper [15], I have used “♠” within the text to indicate points which now seem to be testable-or-retestable experimentally (or important enough to put onto the list anyhow!). Once again such items appear in an Appendix.
actual refractive indices \( n \) (inside and out) — but these \( n \) values change increasingly violently as we approach the absorption wavelength (\( \lambda_0 \approx 200 \text{nm} \)) — see Figure 1.

Nevertheless, the problem can be solved for this asbestos-case [2] though the procedural details need not detain us here. It may however be helpful to note the main assumptions made:

(i) The validity of Sellmeier’s formula.\(^4\) This is used to calculate \( n \) at any relevant wavelength (as in Fig.1), given the value for \( \lambda_0 \) — and “\( A \)”\(^5\).
(ii) For want of better knowledge, \( \lambda_0 \) was taken to be exactly 200\( \text{nm} \) for every relevant medium.

4.2.1 The Raw Results

The calculated figures are set out in Table II (adapted from the earlier report [2]). Thus, e.g., from the “fail” entries, we can see for chrysotile (within either of the two aqueous media) there can be no transmission at all for fibres with diameters of 150\( \text{nm} \) or less. — but a 200\( \text{nm} \) fibre within the “\( n_{\text{aq}}=1.35 \)” environment can just manage to transmit a maximum wavelength size of “325.38\( \text{nm} \)” (which happens to fall within the UVA range of 400–320\( \text{nm} \)).\(^6\)

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4 Sellmeier’s formula [14 (p469)] states, for the refractive index \( n \):
\[
n^2 = 1 + (A\lambda^2 / (\lambda^2 - \lambda_0^2)) \quad \text{— where } \lambda_0 \text{ is the resonance-wavelength (at which absorption is theoretically infinite — and so is } n, \text{ as indicated by the denominator “} \lambda^2 - \lambda_0^2 \text{” becoming zero).} \]

“\( A \)” is the Sellmeier constant, specific to the medium in question.

5 One can calculate \( A \), given some definite value of \( n \) at some specified wavelength, (usually using standard sodium light, for which \( \lambda = 588-995 \) [14]).

6 It is helpful to recall these wavelength ranges: …… \( \text{NIR} > [0.78\mu\text{m}=780\text{nm}] > \text{Red} > [610\text{nm}] > \text{Orange} > [590\text{nm}] > \text{Yellow} > [570\text{nm}] > \text{Green} > [500\text{nm}] > \text{Blue} > [450\text{nm}] > \text{Violet} > [400\text{nm}] > \text{UVA} > [320\text{nm}] > \text{UVB} > [280\text{nm}] > \text{UVc(normal)} > [200\text{nm}] > \text{UVc(vacuum)} > [10\text{nm}] ……
Clearly that rules out visible light and IR for any such chrysotile fibre of that width, but it does theoretically allow any available shorter waves, including UVB (320–280 nm) — though there is probably a practical limit as we shall see shortly.

Note that the rows of the table are spaced out (within practical limits) as a gesture toward making the refractive-index intervals more numerically-uniform and graph-like — as gauged by shading “1.5, 1.6, 1.7,” etc. (integer/10 figures in the right-hand n-column), and by a green dot half-way between each pair. The main asbestos-fibre types are emphasized, which incidentally makes clear much of the distinction between chrysotile (white asbestos) and the other “amphibole” types commonly threatening our health.
| Material                  | n<sub>eq</sub> (sodium light) = 1.35 | n<sub>eq</sub> (sodium light) = 1.45 |
|--------------------------|-------------------------------------|-------------------------------------|
| Fibre-Diameters (nm)    | refRACT<sup>nd</sup> index | refRACT<sup>nd</sup> index |
| Wavelength in vacuo (λ nm) | <p align=center>300</p> | <p align=center>250</p> | <p align=center>200</p> | <p align=center>150</p> | <p align=center>100</p> | <p align=center>300</p> | <p align=center>250</p> | <p align=center>200</p> | <p align=center>150</p> | <p align=center>100</p> |
| Max?                     | n  | λ  | λ  | λ  | λ  | λ  | λ  | λ  | λ  | λ  |
| Flat glass               | 2.1 | 799.94 | 675.72 | 533.74 | 435.86 | 326.58 | 779.76 | 659.14 | 540.79 | 426.62 | 321.11 |
| Alumotantite             | 2   | 738.14 | 624.97 | 514.17 | 407.69 | 309.99 | 716.22 | 607.00 | 500.21 | 397.80 | 304.22 |
| Klebelsberqite           | 1.95 | 706.44 | 598.99 | 493.99 | 393.40 | 301.67 | 683.50 | 580.22 | 479.43 | 383.14 | 295.74 |
| Lammerite                | 1.9  | 674.11 | 572.53 | 473.49 | 378.95 | 293.34 | 650.04 | 552.86 | 458.28 | 368.29 | 287.23 |
| Parwelkeite              | 1.85 | 641.06 | 545.53 | 452.62 | 364.33 | 285±1 | 615.69 | 524.85 | 436.69 | 353.23 | 1.85  |
| Roussite                 | 1.8  | 607.16 | 517.90 | 431.35 | 349.51 | fails | 580.31 | 496.07 | 414.60 | 337.92 | fails |
| Warikahnite              | 1.75 | 572.27 | 489.54 | 409.60 | 334.47 | fails | 543.70 | 466.38 | 391.92 | 322.34 | fails |
| Amosite brown            | 1.709 | 542.78 | 465.63 | 391.36 | 331.95 | fails | 512.87 | 441.22 | 372.82 | 309.33 | fails |
| Crocidolite blue         | 1.7  | 536.19 | 460.30 | 387.30 | 319.18 | fails | 505.59 | 435.58 | 368.55 | 306.44 | fails |
| Anthophyllite (rare)     | 1.685 | 525.09 | 451.33 | 380.48 | 314.53 | fails | 493.81 | 426.10 | 361.38 | 301.60 | fails |
| Crocidolite blue         | 1.68  | 521.37 | 448.32 | 378.20 | 312.98 | fails | 489.84 | 422.91 | 358.98 | 299.98 | fails |
| Amosite brown            | 1.663 | 508.57 | 437.99 | 370.37 | 307.67 | fails | 476.20 | 411.95 | 350.73 | 294.44 | fails |
| Attakolite               | 1.65  | 498.06 | 430.00 | 364.33 | 305.59 | fails | 465.60 | 403.44 | 344.34 | 290.16 | fails |
| Tremolite (rare)         | 1.626 | 480.03 | 405.02 | 353.04 | 295.99 | fails | 445.59 | 387.44 | 332.37 | fails | fails |
| Anthophyllite (rare)     | 1.598 | 457.72 | 397.13 | 339.61 | 287.00 | fails | 421.46 | 368.20 | 318.07 | fails | fails |
| E-Crown Glass (textile)  | 1.5874 | 449.08 | 390.22 | 334.45 | 286.22 | fails | 412.06 | 360.74 | 312.55 | fails | fails |
| Chrysotile white         | 1.569 | 433.83 | 378.05 | 325.38 | fails | fails | 395.39 | 347.54 | 302.82 | fails | fails |
| Arsenite (n=1.552)       | 1.55  | 417.70 | 365.22 | 315.86 | fails | fails | 377.61 | 333.53 | 292.57 | fails | fails |
| Other Crown Glass wool-glass | 1.5487 | 416.58 | 364.33 | 315.20 | fails | fails | 376.37 | 332.56 | 291.86 | fails | fails |
| E-Crown Glass (textile)  | 1.5302 | 400.41 | 351.52 | 305.75 | fails | fails | 358.40 | 318.47 | 284.17 | fails | fails |
| Other Crown Glass wool-glass | 1.5076 | 380.02 | 335.42 | 293.95 | fails | fails | 335.46 | 300.62 | 249.50 | fails | fails |
| Bihinite                 | 1.5   | 372.98 | 329.89 | 289.92 | fails | fails | 327.47 | 294.43 | 237.41 | fails | fails |
| Gearvsstite              | 1.45  | 323.91 | 291.69 | fails |

**Table II.** The longest wavelengths calculated as able to travel through fibres of various types (and 2 different environments).
4.2.3 The Effect of Substance-as-such

Apart from that, asbestos is here treated just the same as glass and the various other minerals (each often chosen simply as a convenient exemplar for its specific $n$-value). This implies the tentative hypothesis that, since we are looking for “diseases from geometry+optics” and not from chemistry, the toxic properties are to be gauged entirely from physical variables (mostly represented by the substance’s position within the table), and not by its identity-as-such.

What saves us from all those other substances is presumably the fact that they either do not readily appear as fibres at all, or their diameters are so comparatively wide that any UV within them has too much freedom, and thereby loses any dangerous laser-like unanimity-and-focus. Thus consider warikahnite (at $n=1.75$ on the table): I am not familiar with that mineral, but I presume it never naturally occurs as thin fibres comparable to those discussed here. However if it did, then its 200–150 nm fibres might well be just as toxic as amosite. Then again, that extreme type of flint-glass (with $n=2.1nm$), if made into fibres 150–100 nm, could well be particularly toxic; — but probably no-one has ever thought of producing such fibres out of “crystal glass” like that, and indeed such manufacture might be technically impossible or at least prohibitively expensive.

Regarding this particular set of calculated values. It would of course have been more ideal to work with accurate, systematic and comprehensive sets of reliable refractive indices (in the UV range!) for the various asbestos types; and maybe we can look forward to that in the future when such data becomes readily available; (♠). Better still would be some direct tests for UV transmissibility within actual fibres (as already suggested ♠). Meanwhile these present calculations might suffice for this early stage of the investigation — a talking point which might inspire more thorough investigations.

4.2.4 Interpreting the Results more generally

Additional shading has converted Table II into two sets of area-graphs to highlight UV-transmissibility. The boundary lines for these shapes merely reflect the figures on the table itself, but they may help our interpretation of it. The right-hand boundaries are just a “joining of the dots” trace of where a particular value does-or-would fall within the table. — Thus the right-most line joins the extreme figures (285, 286, 289.92, 291.69 nm — top-to bottom for the $n_{aq}=1.35$ case — and 287.23, 290.16, 291.86, 294.45 for the $n_{aq}=1.4$ case, on the right). As it happens, these figures all approach the UVB/UVC border of $\lambda=280$ nm, without quite getting there.

In principle then, these $\approx 290$ nm waves should also fit into the wider fibres further to the left, except that there is a PRACTICAL LIMIT as mentioned earlier (when the fibre becomes too spacious, and control is lost). There is no strict cutoff in this case, but for reasons explained previously [2 (§3.3.4., p.14)] it is convenient to deem this limit to be when the wavelength has decreased to about 70% of the maximum — or, as in this case, when the available space has increased by (100/70), i.e. $\times1\cdot429$. That gives us the means to draw the left-hand boundary, seeking cells where correspondingly longer

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7 If so, that apparent laser-like attribute is a partial clue in our search for their source, as we shall see.

8 However if the manufacture of such bizarre fibres does ever turn out to be feasible, (and diamond would be offer another such example, with $n=2.417$), then this could make for some interesting experiments. ♠. (Moreover the thin-layer depositing of artificial diamond has already been achieved).

Incidentally there is another way in which bizarre manufacturing could help: One frustration when trying to assess geometrical measurements is that published results comparing width (or length, etc) have always depended on sample fibre-populations with their measurements defined only statistically (at best) — thus blurring any sharp effects like cutoff which can occur in optics. If feasible, I would encourage the manufacture of asbestos-like glass fibre-sections — all with uniform dimensions and optical properties within each sample. It could then be interesting to replicate many of the existing studies. ♠.

9 The “dot” (aimed at in this plotting) is deemed to be just below the printed value (if any), and central to it.
wavelengths are only just becoming permitted — namely \((\approx 290\text{nm}) \times (10/7) \approx 414\text{nm}\) for the areas in question. That gives us the pale-green area for each of the two cases, \((n_{aq} = 1.35 \text{ and } 1.4)\) — denoting those fibres deemed to be most toxic if \((\approx 290\text{nm})\) radiation (high-energy UVB) were applied to them.

If instead we are interested specifically in UVA at its border with violet \((\lambda=400\text{nm}, \text{ arbitrarily fixed this time})\), a similar process gives us the right-hand boundary for the pink area; — and \(400\times(10/7) = 571.4\) gives us the left-hand boundary in the two half-tables. Then again, the intermediate case for the UVA/UVB interface at \(320\text{nm}\) gives us an intermediate area (plotted in pale-yellow, and barely visible apart from its outline).

Thus any fibre-configuration which falls within any of these areas is (hypothetically) a passive possible-carrier of UV (of one type or another, and some of these will be more toxic than others).

4.2.5 The Effect of Diameter

Perhaps the most important feature is the absence of any values at the bottom right of both half-tables. The equations were simply not solvable in those cases. Thus some fibres are just too narrow to carry UV of any type; so if they are toxic at all, it must be for other reasons — and we have already seen their likely role in mechanical damage.

Apart from that however, there is almost nowhere to hide if we insist on using fibres as narrow as those depicted! (Except perhaps in the top left-hand corners, and even that depends on the somewhat arbitrary “PRACTICAL LIMIT” which we might hesitate to trust in life-and-death situations). On the other hand, if the problem is one of interference in some internal metabolic process, the degree of damage may well depend on just what wavelengths are involved metabolically — so exact tuning could turn out to be important, such that some areas of the table could be more important than others after all.

On a more quantitative level, we may recall Stanton and Layard’s summary (1978)[1] (already mentioned) that — the stronger causal effects occurred with diameters of 10-250nm, and moderate effects with 250–1500nm thicknesses. That appears to be consistent with Table II if we (i) blame mechanical damage exclusively when the fibres are <100nm (amphiboles) or <170nm (for chrysotile); (ii) blame UVB for most of the rest of the <250nm cases; and (iii) blame UV in general (sometimes bypassing the “PRACTICAL LIMIT” putatively offered in wider fibres) for the “moderate effects with 250–1500nm thicknesses”

On top of all that, is the complication that there is the frequent “untidiness” of chrysotile fibres. On the one hand, that would tend to upset any tendency to transmit UV; and on the other hand it probably favours mechanical damage (e.g. with fine fragments breaking away from other pieces).

4.2.6 The Effect of the External Refractive Index

For the two cases considered, \((n_{aq} = 1.35 \text{ and } 1.4)\), there is clearly no great difference (except perhaps for chrysotile) — but it is not clear how much we can validly extrapolate from these cases. Meanwhile it would be helpful here if more research (●) could be devoted to measuring the optical properties of the various aqueous and other bio-media. (That need for the same data also applies to the mitochondrial discussion raised by Thar & Kühl [16], who used the same two \(n\) values as a necessary speculative guess).

Actually this situation could turn out to be unexpectedly complicated, due to findings such as those of Preoteasa & Apostol [17] and later work — that local “coherence domains” (CDs) could cause inconstancies in the optical properties of liquids, especially near boundaries. As such inconstancies would tend to be dynamic, and if some fibre-optic transmission is only just on the borderline of success (depending on an \(n\)-value outside), then that sets the stage for a triode-like situation which can switch the transmission on-and-off — or indeed serve as an amplifier. I had
contemplated investigating this possibility further, but in view of the apparent slight \( n_{\text{aq}} \)-effect shown in Table II, it seems sensible (at least temporarily) just to await events in this area!

5. Politics and Lobbying over White Asbestos (Chrysotile)

Chrysotile is different, and commercial apologists like Bernstein are quick to point out ways in which it is supposedly benign.[18,19] Table II (if valid) partly supports that claim, but only partly, and rather weakly: To have a comparable UV-transmission, these fibres would apparently need to be some 50\( \text{nm} \) thicker than their amphibole counterparts, perhaps leaving a “harmless-diameter” interval at about 150\( \text{nm} \) before the mechanical toxicity begins. (It might be interesting to test that specific point, with stable fibres of that 150\( \text{nm} \) size♠)

At the same time, that gap (if it exists) could help to confuse the evidence, especially if we fail to recognize a likely double-cause phenomenon: In chrysotile's case, the two causes could well be of about equal potency (on different occasions), whereas for amphiboles it is likely that one cause (the UV) would largely eclipse any slight “mechanical” effect. It might thus be worthwhile bearing this complication in mind when assessing the politics of the situation — and careful locally-sensitive reviews of such lobbying might be considered, giving due weight to economic realities, and to those participants (on both sides) who are less well funded. (♠).

One point in partial favour of chrysotile is that it is slightly soluble (as Bernstein is keen to point out); so it tends to disappear within weeks [18]. That seems desirable, though it might not stop crucial damage which could occur meanwhile. Moreover there is a possibility that, while shrinking, it might briefly expose new micro-sites to “short-circuiting”!

6. Mitochondria and their likely UV communication

It is no secret that I support the hypothesis that the human/vertebrate nervous system carries a double set of signals: — “[A]” the orthodox Action-potential system — but also the yet-to-be-confirmed Infra-Red + RNA system “[R]”.

Thar & Kühl’s very interesting paper [16] (and the many other minor clues about the separate importance of UV), now raise the possibility of yet another communication system — even if it seems to be much more parochial in its reach. So let us think of it tentatively as the “[U]” system, serving the individual cell and its close environment — apparently via UV, and (at least when necessary) sending a directional UV laser-beam in a (perhaps) highly directional signal-stream or energy-boost.

If that is the case, then it is easy to see that such an arrangement could occasionally be severely upset by anything which misdirected such signal flows — and of course that is consistent with ideas put forward earlier in this paper.

It is perhaps significant to compare long and short mitochondria. For the long version, Thar and Kühl [16] argue that the reflection necessary for laser-production could be provided by a particular optical trick of multi-layer pseudo-mirrors (which, here, could only work for UV), whereas Cope [20] had argued in 1973 that the short\(^{10} \) mitochondria were reverberation chambers for IR, using some sort of orthodox reflection (though he did not even mention that such reverberation could generate laser-beams). Meanwhile his account of the supposed reflection did not seem very convincing if applied to visible light (and it certainly would not have worked for UV), nevertheless he may have been right for the case of IR. To sum up then: It may be the case that long mitochondria are “tailor-made” for UV, while short mitochondria are “tailor-made” for IR. That raises the intriguing-but-peripheral question

\(^{10}\) Long mitochondria were not identified until recently, so Cope would have known only short mitochondria at that time (1973).
of whether these are just the same sort of mitochondrion, reconfigured for the particular task in hand — or whether they would have permanently differentiated beforehand.

In case the [U]-system idea seems a bit far-fetched, it may help to look first at Table III depicting advances in our social communication systems, and then compare it with Table IV which outlines ideas (which seem to correspond) about biological communication. Both tables illustrate the thought that, just because we have already found one or two ways of doing things, that may not be the end of the story — there may be more to come. Of course that is not proof, but it should encourage serious investigation and support on such matters.

| Table III  Four types of Social Communication |
|-----------------------------------------------|
| ![Table III](image) |
## Table IV  Four types of Biological Communication, (equivalent to Table III?)

| [M] | [A] | [R] e.g. see [15] | [U] speculative! | # |
|-----|-----|------------------|-------------------|---|
| Biological communication-system: | Endocrine system (including selective coding) | Chem $\rightarrow$ ElecSig $\rightarrow$ Chem at Synapses. Action potentials | RNA $\rightarrow$ IR/ Nerve-myelin $\rightarrow$ RNA. Local and between nodes of Ranvier | Cell-network of microtubules and mitochondrial UV-lasers(!?) [16] | B |
| memory storage (biological) | Immune system? | Synaptic configuration | RNA-network of “schèmes” | DNA? centrosome? nucleus? | B1 |
| Bio-code | Protein shapes | ?? | IR modulation? | Epigenetics? UV-modulation? | B2 |
| Signal carrier, (biological) | Blood / lymph systems | Nerves, with or without myelin | Myelin (or chitin?) Microtubules$^{11}$ + long mitochondria | Long mitochondria in the cell [16,21] | B3 |
| Signal emitter | Endocrine gland | Neuron-body; then synaptic bouton | ?glia? using short mitochondria. [20,21] | | B3 |

### 7. Conclusions

There can be no firm inferences at this stage, but the collective evidence does seem to be coming together to suggest a coherent overview. Two areas deserve comment:

#### 7.1 “Secret” metabolism involving UV and/or IR

It is usual to assume that metabolism is just a matter of chemistry. (That view is traditional, and the bias is indeed reflected in the pro-chemistry training of medical researchers). However it is becoming apparent that there are important and sophisticated physics-based aspects to metabolism. That can now hardly be doubted regarding IR, thanks to the work of Albrecht-Buehler — e.g. [22].

However our present concern is with UV, and here the evidence is still largely theoretical and circumstantial, as suggested in Tables III and IV — plus investigations such as those of Thar & Kühl [16]. Nevertheless that should suffice to induce us to take the topic seriously — and the corollary that upsetting such systems could have significant consequences.

In comparing UV and IR systems, it is worth bearing in mind (i) that a typical UV photon has about ten times the energy, and hence is more likely to do damage if misplaced; and (ii) by the same token it is more suited to any changes to more stable configurations such as DNA-complexes, (whereas IR probably confines itself to modifying those comparatively benign changes of day-to-day biochemistry). Meanwhile (iii) the UV photon’s wavelength and other linear measurements will be about 1/10 compared to IR — and that seems appropriate if UV’s role is ultra-micro.

#### 7.2 Some toxicity may entail a signals-upset within UV metabolism

As shown in Table II, asbestos seems theoretically able to act as a conduit for any UV signals which happen to enter the ends of its fibres. The postulate then follows that such conduits would then

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$^{11}$ Microtubules need not be bound by the constraints imposed by Table II. (Table II applies to fibres deemed to be optically uniform internally, and therefore compelled to use the “zigzag” transmission modes which lead to the constraints). In contrast, coaxial cables, *commercially-designed optic fibres, and (perhaps? ♠)* microtubules, can circumvent this constraint. In those circumstances, they could then transmit the usually-preferred “TEM” mode more-or-less as if it were travelling (unconstrained) in free space.
sometimes act as “short circuits” — delivering messages to inappropriate places, and thereby upsetting
the normal metabolism. Alternatively they might just degrade the efficiency of normal UV activity
— whatever that may be.

7.3 Other toxicity seems due to asbestos’s mechanical damage to DNA
It is clear that this can happen with very narrow fibres, (though it is less clear just how common that
might be in natural conditions). It has here been assumed that thicker fibres would be less likely to be
“knife-like” in this way — but it might be prudent to test that assumption: One perhaps-useful test ♠
would be to manufacture opaque fibres (to eliminate any UV-causality), and then do in-vitro studies in
the standard way — though no doubt there would be practical difficulties.

7.4 New experimental studies are needed, and perhaps in new directions
Further directed experimental investigations are obviously called for, and some suggestions are
offered in the list which follows:

8. List of Suggested Experimental Investigations
Once again, as in the previous paper [15], I offer a list of items, (marked “♠” within the general text)
which already seem amenable to experimental testing (or will ultimately need testing despite
problems); and I invite those with the relevant resources to investigate them — with or without my
collaboration.

| §    | Footnote | Description of suggested area for experimentation, marked “♠” in the text |
|------|----------|--------------------------------------------------------------------------------|
| 4.1+ 4.2.3 | (3)      | Direct test for the supposed ability of asbestos fibres to transmit UV (under predicted conditions) |
| 4.2.3 |          | Test the hypothesis that fibre toxicity depends entirely on its geometry and optical properties — and not (as such) on its chemistry, or its classification into “asbestos” or “glass”, etc. |
| 4.2.3 | 8        | If possible obtain manufactured fibres of unusual media (e.g. with \(n>2\) — such as extreme types of flint glass, or diamond), and test such fibres for UV transmissibility |
| 4.2.3 | 8        | If possible obtain manufactured fibre-samples in which there is high uniformity of fibre-properties, thus eliminating much of the current uncertainty which comes with statistical studies of toxicity. |
| 4.2.3 |          | Compile basic tables of refractive indices within the UV range — especially for asbestos |
| 4.2.6 |          | Compile better basic tables of optical properties (including the UV range) for bio-media — especially aqueous solutions and lipids. |
| 5    |          | Test the prediction that 150\(\mu\)m-wide chrysotile might be less toxic than if wider or narrower |
| 5    |          | Re-assess the public-health hazard and local politics of chrysotile, given the likelihood that it may have two different causal effects (which may have confused the evidence in the past). |
| 6: Table IV  | 11       | More intensive study of the optical properties of microtubules (and other features related to mitochondria) |
| 7.3  |          | If possible get opaque asbestos-like fibres (to eliminate UV effects) and then do standard carcinogenic tests via the other causes — especially mechanical damage, and whether it decreases as fibre diameter increases. |
|      |          | Systematically collect and collate more-exact wavelength details about ultraweak UV emissions |
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