THE McTEAR CASE: THE ANALYSIS

08/03/2012

Introduction.

It is not easy to find a precise statement about the history of this case, and it is not very important anyway, but I have searched briefly and found what I think is enough information.

In 1992, Mr McTear was diagnosed with squamous cell carcinoma. This tumour was situated around the place where the bronchial tube (windpipe) divides, one tube going into the left lung and the other into the right. It was inoperable. The word 'squamous' means 'scalelike', as in 'fishscales' or the scales of snakeskin. That is, they are flatish. They are particularly prevalent in the 'epithelium' of the bronchial tubes. The epithelium is a sort of 'skin' which lines the inner surfaces of organs of the body, in a similar way that your skin covers the outside of your body.

At the time, ASH was advertising for smokers with lung cancer to contact them with a view to suing Tobacco Companies. I have since discovered that 300 people contacted ASH. Mr McTear was one.

The preliminaries for bringing an action began straight away, but Mr McTear was becoming weaker and so he gave evidence (presumably at his home) in the form of a deposition. The deposition covered such matters as when he started smoking, what type of cigarette he smoked, when he changed to roll-ups, what roll-up tobacco he used, when he tried to stop smoking and how often, and so on.

In 1993, Mr McTear died, and so his wife, Margaret McTear continued the action. Death is such a strange thing – a few weeks before he died, Mr McTear went on holiday with his wife to Malta for a fortnight. Death is such a strange thing.

Another peculiar thing, in the circumstances, was that Mrs McTear refused to permit a post mortem on the grounds that Mr McTear did not want it to happen. Very odd in the circumstances.

In 2001, Lord McCluskey agreed that there was enough 'good reason' to allow the action by Mrs McTear against Imperial Tobacco to proceed.

I am not sure when exactly the action started. There are a lot of preliminaries to be addressed before the action actually goes to court. Both sides have to prepare their cases and produce documents and reveal their arguments to the other side. Witnesses need to be prepared and expert witnesses engaged. As far as I can see from this document, the real court action started in 2003, but spread over into 2004 somewhat. The critical event for this summary was:

31st May 2005: Opinion of Lord Nimmo Smith:

In the cause of:

Mrs Margaret McTear (persuer):

Against:

Imperial Tobacco Limited (defenders).

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We must remember that THE OPINION was not a transcript of the whole trial. Lord Nimmo writes down in the Opinion those matters which were important in his decision-making process. However, he quotes huge chunks of verbatim statements from the witnesses, so, to that extent, it is almost a transcript.

I have pondered what might be the best way to bring things together because some of the Judge's comments on witnesses' statements appear right at the end. Although the vast majority of the Judge's comments will have to stay where they are (because they inter-relate), I have decided that this summary will be easier to read and understand if I bring forward some 'later revelations' (to show where a line of questioning is leading, for example).

Where it seems appropriate, I have indicated the 'Part and Paragraph' in brackets alongside the quote or segment. Thus, (5.123) indicates part 5, paragraph 123. I have had to be very selective, otherwise this piece would be almost as long as the original.

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(THROUGHOUT WHAT FOLLOWS, I SHALL USE THE WORD 'McTEAR' TO INDICATE 'COUNSEL FOR….' AND SUCH, AND 'ITL' TO INDICATE 'COUNCIL FOR IMPERIAL TOBACCO' AND SUCH.)

PART 1: PRELIMINARIES.

There isn't an awful lot that we need to concern ourselves with in this Part, but I will quote this:

(1.5)

The pursuer can succeed in this case only if she proves all of the following:

(1) that cigarette smoking can cause lung cancer;

(2) that cigarette smoking caused Mr McTear's lung cancer;

(3) that Mr McTear smoked cigarettes manufactured by ITL [Imperial Tobacco] for long enough and in sufficient quantity for his smoking of their products to have caused or materially contributed to the development of his lung cancer;

(4) that Mr McTear smoked cigarettes manufactured by ITL because ITL were in breach of a duty of care owed by them to him; and

(5) that such breach caused or materially contributed to Mr McTear's lung cancer, either by making at least a material contribution to the exposure which caused his lung cancer or by materially increasing the risk of his contracting lung cancer.

But, there is one other consideration, and that is that if ITL rely upon the legal maxim *volenti non fit iniuria* (meaning that Mr McTear smoked 'voluntarily' and thus cannot claim 'injury'), they must themselves prove that assertion.

I think that it might be as well dispose of the last para right way. At the very end of his Opinion, Judge Nimmo (I suppose that I should say 'Judge Smith', but I like the sound of 'Judge Nimmo' better – and, it sort of 'stands out', doesn't it?) says this:

(9.13) *On my interpretation of the law relating to the maxim volenti non fit iniuria, and in the circumstances of this case, I would not have been disposed to sustain the fourth plea-in-law for ITL, if the pursuer had otherwise succeeded on the foregoing issues (paras.[7.204] to [7.208]).*

Which means that, had Mrs McTear succeeded in proving that ITL 'caused' Mr McTear's lung cancer, Judge Nimmo would not have accepted that 'Mr McTear contributed to his own problems', in which case ITL would have been liable.

(I trust that people can now see the value of jumping backwards and forwards! Much is simplified and we can disregard many legal arguments about the "volenti…" maxim)

Nimmo goes on to say that 'public health matters concerning smoking are not involved and that only evidence before him matters.

MCTEAR had been refused legal aid. The reason for this (among others no doubt) was that ASH had made statements which suggested that the case was being brought 'for the benefit of others', besides McTear:

(1.20)"we just need one breakthrough, we just need one victory. [...] We just have to win one case to win everything".

(1.25) The judge agreed that ITL had greater resources than McTear, but this was to be expected. However, he would not allow ITL to unreasonably drag the case out.

There then follows a long list of legal and non-legal documents referred to in the case.

PART 2: THE PARTIES' POSITIONS ON THE MAIN FACTUAL ISSUES.

<u>McTear's position</u>.

MCTEAR claimed: that Government and WHO agreed that smoking 'causes' lung cancer; that Mr McTear's lung cancer was caused by smoking; that, in 1964, when he started smoking, he did not know that smoking was dangerous; that, when he did become aware, he was addicted and could not stop smoking.

MCTEAR suggested that the case was a straightforward one since *Donoghue* v *Stevenson 1932* had established the principle of 'product liability'. But the judge said:

(2.4) *As will be seen, this approach has not found favour with me; though no doubt, if it had, I might have spared myself, and the reader, many hundreds of the pages which follow.*

Here is something that I did not know. It seems that 'tobacco products' were specifically excluded from the Consumer Protection Act 1987. This came out when the judge asked MCTEAR why no action had been brought against Tobacco Companies before, if it was so straightforward. MCTEAR said: *"They [TCs] were a very well-funded industry who were able to fight off attacks. This was how they had obtained the exemption for tobacco under section 10 of the Consumer Protection Act 1987."*

ITL's position.

(2.7) ITL held that: smoking had not been proved to cause lung cancer; that smoking was a habit and not an addiction; that, in 1964, the possible dangers of smoking were well known among the public; that it was well known that the habit was difficult to break.

In the next section, MCTEAR claimed that ITL had, more or less, admitted the harm of cigarette smoking in letters to the Health Committee of the Commons. ITL said that it had not agreed 'more or less' as was illustrated in this quote from a letter to the Heath Committee:

(2.13) "*This is why, whatever our views on these complex issues [tobacco and health], Imperial does not challenge the public health message. It has not done so for almost forty years and intends, in the future, to continue its policy of not challenging the public health message that smoking causes these diseases.*"

(2.14) Prof Sir Richard Doll, Mr Gareth Davies (CEO of ITL). Prof James Friend and Prof Gerad Hastings gave oral evidence at a meeting of the Health Committee in 2000. This event was brought up during the present action as putative evidence that ITL had admitted that smoking caused various diseases. Although this section is quite long and detailed, I think that we can miss it out. Essentially, for various reasons, Doll said that ITL admitted it, but Davies said that ITL had only agreed that smoking *might* cause diseases, but ITL did not *know*. ITL did not contest the public health messages.

(2.62) ITL then had the chance to tell the Judge about what it did when the suspicion arose of a connection between lung cancer and smoking. Researchers had attempted to cause lung cancer in animals from tobacco smoke, without success. It was right, therefore, for ITL to 'withhold judgement' as to whether or not tobacco smoke caused lung cancer.

PART 3: PUBLIC AWARENESS.

(3.2) Copies of newspapers from around 1964 and onwards were produced showing articles about smoking danger. Ministerial pronouncements were itemised. Many direct quotations from newspapers were itemised by the Judge up to 1964. TV advertising of cigarettes was banned in 1965 and publicised widely. Many newspapers published articles about scientific research around that time.

PART 4: MR AND MRS MCTEAR: QUESTIONS OF FACT.

Judge Nimmo said:

(4.1) *The personal history of Mr McTear, and consequently also that of Mrs McTear, was brought out in remarkable, but by no means unnecessary, detail.*

Indeed it was, but there is no need for us to provide the detail. It is sufficient to say that Mr McTear was not the most responsible of persons. He joined the army but was constantly going AWOL (absent without leave). Eventually, he was discharged. He had many jobs, but tended to cry off sick after a couple of weeks or simply pack the job in. He was reckless, appearing frequently before the magistrates and spending some time in jail. He drank hard. On a couple of occasions, he deliberately set fire to his home.

It would be advantageous if I say now that Mr McTear's lifestyle was more important than merely behavioural. Later, an expert witness for ITL spoke of other causes of cancer, such as personality, alcohol, diet, the lack of vitamin A (a vitamin of importance to the cells of the epithelium (the 'skin' which coats internal organs of the bronchus), genetics and so on. Of particular significance was his history of 'throat infections' (4.78), which were recognised as damaging to the epithelium and possibly significant in the later formation of cancer.

We can skip this section since it is adequately summarised above.

Next followed the evidence of Mr McTear himself 'taken on commission' (whatever that may mean). I suppose that his health condition was bad, and so he 'made a statement' and was asked questions. He described when he started smoking, what cigarettes he smoked and the type of tobacco he used when he started to use roll-ups. He tried to quit on a number of occasions but started again. He finally did quit when he was diagnosed with lung cancer.

He was cross-examined at the time, but that evidence was not read out at that time. ITL counsel said that he would prefer to refer to it as and when it was relevant. (4.111)

Mrs McTear then gave her evidence. Again, we need not dwell on this section. It is mostly a re-iteration of Mr McTear's evidence. There were some disparities, but not

anything serious (as I see it!). One thing, however, was that the Judge was not willing to accept that Mr McTear would not have started smoking had he been aware of the dangers. For various reasons to do with his behavioural attitude, the Judge did not regard his 'statements of intent' and motivation as reliable.

In the Judge's 'discussion' of the evidence of Mr & Mrs McTear, a curious piece of evidence emerged. The McTears had claimed that Mr McTear had been smoking 'John Player Special' since 1964 or shortly thereafter. However, it came to light that 'JPS' did not appear on the market until April 1971.

PART 5: THE EXPERT EVIDENCE.

This is probably the most important section.

(5.1) *"I turn now to the evidence bearing on the pursuer's averments that cigarette smoking can cause lung cancer, that it did in fact cause Mr McTear's lung cancer, that tobacco is addictive (in the sense defined by the pursuer) and that Mr McTear was addicted to cigarettes. The burden of proving these averments rests, as I have said, on Mrs McTear. The evidence in question is principally that given by expert witnesses called for both parties." Judge Nimmo.*

First, the judge referred to the law relating to the duties of expert witnesses. Of particular importance was that they should stick to their own field of expertise and that they should only refer to 'studies' and 'research' in their own field, except in certain special situations. An example of a special situation was one where a medical expert witness referred to epidemiological studies. This situation might arise where the epidemiology was actually very clear and apposite. But, even then, judges were required to examine such epidemiological evidence with great care. A specific case in the past was one which arose in connection with the effects of asbestos dust. Nimmo also strongly made the point that the purpose of expert witnesses was to enlighten *him*, the judge, and guide him through the intricacies of, for example, epidemiological studies.

In this section, Judge Nimmo said that the fact that ITL expert witnesses were paid for their efforts in preparation for the case could not be held against them. It was the content of their evidence which was important.

<u>Expert Witnesses for Mrs McTear</u>.

[These were: Dr McCarroll, Prof Friend, Prof Sir Richard Doll, Prof Hastings and Dr Kerr]

<u>(5.20) Sheila McCarroll.</u>

She was Mr McTear's GP and was qualified to be so.

She gave evidence that she was made aware of the dangers of smoking in medical school and had read various Health Dept publications which stated that 'smoking kills' and that over 100,000 people per an die as a result of the various illness caused by smoking. She had witnessed in her own surgery smokers presenting with such diseases (lung cancer and such).

In cross-examination, she agreed that she had not studied the literature extensively, but relied upon medical school and journals such as the British Medical Journal. A question was put to her about the possibility that the cause of a death could be recorded on a death certificate as 'smoking'. She agreed with Peto and Doll that it was not possible to ascribe *any specific individual's* death to smoking.

One of the strange things that we amateurs have to contend with is that we do not know about 'legal procedures'. For example, it is not the responsibility of the Judge to bring forward the arguments in favour or against a proposition. It is the duty of the counsel on either side to produce the legal arguments. Only after the legal arguments have been made on both sides might the Judge comment on the arguments and, possibly, introduce legal arguments of his own. With this in mind, we can easily understand how the Judge dismissed the evidence of McCarroll about 'the cause' of lung cancer as follows:

(6.62) *Counsel [ITL] submitted, without intending it as a criticism of Dr McCarroll, that as a general practitioner her views on these matters were of no assistance to the court.*

Interestingly, it turned out that: " …..*she had not heard of Doll or Peto"*. That is, that she knew nothing about the actual epidemiological evidence which supported the claim that 'smoking causes lung cancer'.

It is important to understand the implications of this: essentially, the implications are that doctors, generally speaking, only accept that smoking causes lung cancer because they have been told so ("brainwashed", if you like).

<u>(5.27) Professor James Friend</u>.

Prof Friend was, until he retired in 2002, an Emeritus Professor – a big noise indeed. He was also a 'big noise' in ASH.

We can save ourselves an awful lot of time, however, if we accept his own description of himself as just a clinician. That is, not a pathologist, not an epidemiologist, not a statistician. During his career, he had personally been involved with doing biopsies for some 3000 cases of lung cancer. And yet, despite being just a clinician, he became: *Among his current appointments, he had been Chairman of the Government Scientific Committee on Tobacco and Health since 2000, a Member of the National Cancer Task Force of the Department of Health, also since 2000, and Chairman of the Tobacco Policy Review Group for Grampian Health Board.* (5.27)

(5.34) Professor Friend was asked by McTear's counsel to comment on a number of passages in IARC 1986. (IARC is short for "International Agency for Research on Cancer") The document, IARC 1986, was almost exclusively concerned with the evil

of tobacco smoke. For all intents and purposes, he agreed with everything that IARC 1986 said about the evil of tobacco smoke. This part of the Opinion is very long indeed, but essentially it comes down to: tobacco smoking causes cancer; Mr McTear smoked and thus his cancer was caused by smoking; smoking is addictive and Mr McTear could not stop smoking – but 'the proof' required masses and masses of studies by IARC and others. Prof Friend was led by MCTEAR to discuss reports from the Royal College of Physicians and the Medical Research Council and others (5.84 and 5.85). He agreed with everything that these reports said about smoking and health.

It seems that Judge Nimmo became a little frustrated by the constant repetition of "I agree, I agree, I agree" and asked:

(5.95) *"Lord Nimmo Smith: Professor, I am just wondering what I should be making of this part of your evidence. Are you in a position to express any expert opinion about epidemiological studies?*

A. I would not claim to be an epidemiologist but I have been very impressed by these studies, particularly the Doll and Hill study, which [...] was conducted in a very simple manner on British doctors – where every doctor on the British Medical Register was written to and asked about their smoking habits – and, ever since then, Doll and Hill, and latterly Doll himself, have really sat receiving the death certificates of the doctors who participated in that study. And [...] the paper reported here represents the early results, but he has continued to accumulate those results and they have been utterly consistent. I find that very persuasive, although, as I say, I am not an epidemiologist, but they seem very convincing to me.

Lord Nimmo Smith: I am just wondering what to make of it, because Mr McEachran [counsel for McTear] is reading out quite long passages to you and you say 'Yes' from time to time. When you say 'Yes', does that mean you see that this is what is written on the page, or does it mean also that you accept it as accurate?

A. I would say what is stated in the document is in accord with my clinical experience and with my beliefs from a reading of the literature, but I have not had an in-depth reading of the literature [...] in the way a professional epidemiologist would have had."

[5.96] In view of this, I do not propose to do more than refer briefly to further passages read out by Mr McEachran from Chapter 9"

I suggest that the last sentence means that, as a person who was not an epidemiologist, Prof Friend's opinion about the epidemiological results was irrelevant. That is, most of the stuff to which he was agreeing what not something that he was an expert in. (Ref: "Duties of expert witnesses" earlier)

(5.100) Cross-examination of Prof Friend.

Prof Friend had presented a report. As a clinician, his principle source of information was the medical records of Mr McTear. The final part referred to information from Public Health documents (such as IARC (International Agency for Research on Cancer), RCP (Royal College of Physicians) and USSG (US Surgeon General)).

He was asked about his relationship with ASH. He had been a board member, among other things. He described how ASH was set up. An interesting point came out of this – it seems that ASH does not have 'members' as such. It has 'supporters' and 'donors', but not 'members'. (What does that say about their Charitable Status?) He was asked about the strategy of ASH – the production of epidemiological studies, pressure on civil servants and politicians, media advocacy, changing public attitudes and CREATIVE epidemiology. ' Creative' meant making studies easier for the public to relate to, he said, such as:

"*Every year in this country about 110,000 people die because they smoked cigarettes. It's as bad as a jumbo jet crashing every day of the year, killing all the passengers. [...] Every two hours someone dies from lung cancer in Scotland – the highest rate in the world."* (5.110)

He was asked about publications of his own in which he condemned tobacco companies for 'selling tobacco to children', and such.

In his report prepared for the court, he referred to evidence produced by Dr Kerr. Dr Kerr was a pathologist. He said that a biopsy on Mr McTear had revealed 'squamous dysplasia' – considered to be a precursor to lung cancer, but not always. Oddly:

He did not know that it could be found in the lungs of those who had succumbed to influenza. (5.113)

(5.115) There was a reference in this para that Prof Friend thought that persons who did not smoke could be 'infected' by Second Hand Smoke.

(5.116) He was asked about Enstrom and Kabat 2003. He knew about the study which had compared the incidence of lung cancer and heart disease in a very large number of people over some 40 years, and which had found no significant increase in these diseases in spouses who lived with smokers as compared with those who did not. He dismissed these findings on the grounds that the study was funded, in its later stages, by an organisation supported by tobacco companies (even though it had been peer reviewed and published in the BMJ).

He was asked about a study which revealed that the amount of carcinogens in tobacco smoke was far too low to produce cancer. Another study mentioned said this:

"It is now accepted that smoking increases the risk of lung cancer, and that the heavier the smoking the greater the risk. But nowhere has it been claimed that the heavy smoker is stricken with cancer earlier than the light smoker. If lung cancer in smokers is a result of direct carcinogenic action, one would certainly expect this to happen; for experiment has shown beyond question that a potent carcinogen induces tumours early."

Prof Friend said that he did not know the author of that study (Passey 1962). Passey was Emeritus Professor of Experimental Pathology at the University of Leeds, and was writing from the Chester Beatty Research Institute at the Royal Marsden Hospital in London. Friend said that 'such people were no longer with us in the sense that they were only a small minority'.

(5.119) Judge Nimmo went on to say:

He did not know that experiment had shown beyond question that a potent carcinogen induced tumours early. He said:

"I should emphasise [that] I am not a toxicologist, I am not an epidemiologist, I am a clinician who obtains information about the cause of disease from my reading elsewhere."

It may be as well if I indicate here the reason for asking Prof Friend all these questions. It will make it easier to understand what is going on. Only later in the text does this reason emerge. Prof Friend had implied in his report that 'the science was settled' and that he knew what he was talking about. It is becoming apparent that the science was not settled and that he did not personally have the expertise to make the comments which he made in his report. Viz, this statement by Friend:

He was not aware when he prepared his report that every single statement in it had to be supported by a reference to the literature. (5.152)

Succeeding paras go on to emphasis that the science was far from settled. There are references to studies which show that lung cancer can strike at any age, regardless of whether or not a person smoked, and that the number of cigarettes smoked and the duration of smoking by any particular individual did not produce consistent incidents of disease. Prof Friend did not know much about these studies. (5.129)

Again we see Prof Friend's ignorance here:

(5.161) *Asked whether he stood by* ***the statement in his report*** *that in lung cancer there was only limited evidence for familial susceptibility.…..*

We see there that Friend had been stating, in his report, as *facts*, results which he was only aware of via 'approved' public health literature. He opened himself up to questioning about the extent of his knowledge and was found wanting. In fact, as regards lung cancer in family histories, there was plenty of evidence that lung cancer susceptibility was strong in such families. Samet *et al.* 1996 revealed an odds ratio of 5.3. Dong and Hemminki 2001, studying a population of some five million, discovered, in some circumstances, a ratio of 13.65 for lung cancer. Ooi *et al.* 1986 discovered a ratio of 2.4. All the above had allowed for confounders such as smoking.

(5.160) *In Gauderman et al. 1997 at pp.208-209 the authors stated:*

"The results from these analyses support previous findings that a major gene plays an important role in lung cancer risk. An additional finding not previously observed is

that there is no apparent interaction between the putative lung cancer gene and smoking."

Professor Friend said that he did not have enough expertise to make a judgment upon this statement.

The above sections relating to family history turn out to be very significant later because Mr McTear's mother had died from lung cancer, and so had a couple of other family members.

(5.162) Prof Friend was then asked about the addictive qualities of tobacco. I propose to skip most of this section, but I will point out this. The 'authority' which Friend relied upon for the addictive qualities of nicotine was US Surgeon Gen 1988. But a book examining the data which the SG relied upon had this to say:

(5.166) *He was asked about a number of other passages in the book. At p.177 the authors wrote:*

"[T]he flaws we found in the nicotine research literature are of such magnitude and occur in such a regular fashion that they demand an explanation. A partial list of the methodological shortcomings compiled in this book includes:

- *I Systematic exclusion of subjects from statistical analyses*
- *Absence of saline control groups for injected drugs*
- *Result-biased selection of number of sessions to test manipulations*
- *Absence of statistical comparisons [...]"*

Also, from the same book:

"Thus, nicotine's role in maintaining the smoking habit bears no similarity to the role played by genuinely addictive drugs such as heroin, barbiturates, alcohol or other drugs to which nicotine is routinely compared." (5.165)

Discussion with Friend then moved on to 'causation'.

(5.172) *The next passage of cross-examination related to Professor Friend's* ***statement during his evidence-in-chief*** *that he agreed with the statement in Doll 1997 that following RCP 1962 and USSG 1964 the idea that smoking was a major cause of lung cancer ceased to be seriously challenged.*

Unfortunately for him, it had been seriously challenged. Sir Ronald Fisher, the 'Father if Statistics' no less, had cast doubt upon causality. But he was not alone:

(5.174) *Finally, Professor Friend was asked about evidence given by a number of witnesses to sub-committees of Committees of the United States House of Representatives. In Sommers 1972, Dr Sheldon C Sommers, Chairman of the Scientific Advisory Board to the Council for Tobacco Research, a physician specialising in pathology and Clinical Professor of Pathology at Columbia University*

College of Physicians and Surgeons and University of Southern California School of Medicine, gave evidence which included this statement, at p.96:

"[S]tatistical mathematics can never prove cause and effect. All they show is a relationship requiring further study, usually experiments in animals, to find out the meaningfulness biologically of this relationship. I really believe that ***among the active researchers*** *in these fields, there is no great preponderance of feeling that cigarette smoke is carcinogenic****."*** [My bold]

The bold indicates that not everyone agreed by any manner of means.

And here is something of a killer:

(5.175) *In Sommers 1976 Dr Sommers said at p.269:*

*"Now, as to lung cancer, there is a statistical association between cigarette smoking and lung cancer. But at present the nature of the association or whether it is causal are not known. The test of the original Surgeon General's report [USSG 1964] deals with the difficulties of assigning causality****, but the summary and conclusions brush these aside****, and assign a causality not demonstrably evident in the text. It is widely known that a statistical association is not by itself proof of causation. A statistical association may point to experiments that will help to determine whether there is cause involved."*

Another:

(5.176) *In Furst 1982 Dr Arthur Furst, Director Emeritus of the Institute of Chemical Biology at the University of San Francisco, said at p.512:*

"For many years, I tried to induce lung cancer in animals with cigarette smoke, with no success, despite the most sophisticated smoking machines available. Not only were my colleagues and I unsuccessful, but so was every other investigator. There have been a very small number of published reports of lung cancers occurring in experimental animals during smoke inhalation experiments. Anyone attempting to interpret these as showing that smoking causes lung cancer must understand that animals, like humans, do spontaneously develop lung cancer even in the absence of any suspected carcinogen."

Several more paras introduced other 'physicians of note' who disagreed with the causal hypothesis. The cross-examination of Prof Friend then concluded.

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Prof Friend was then re-examined by his own side. Some comment were made about Sir Ronald Fisher, but nothing of note came out of the re-examination. Friend continued to believe that smoking caused lung cancer.

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<u>Professor Sir Richard Doll</u>.

Judge Nimmo outlined Doll's CV. In 2005, Doll was 91 but still active in tobacco control circles. He was 'pre-eminent' in that field.

(5.190*) Sir Richard was next asked to consider IARC 1986. [Int Agency Research Cancer] He agreed that this monograph was more than 400 pages long. He was chairman of the working group which wrote it.*

He said that the members of the group were international experts. He knew about half of them.

The group concluded that:

- Lung cancer kills the greatest number of people.
- The major cause is smoking.
- Duration and amount are critical.
- In heavy, long-term smokers, causality is 90%.

Sir Richard said that this was the conclusion of all twenty-seven members of the working group, with no dissent.

He went on to say:

(.5.192) *At p.314 the working group set out their evaluations:*

"There is sufficient evidence that inhalation of tobacco smoke as well as topical application of tobacco smoke condensate cause cancer in experimental animals.

There is sufficient evidence that tobacco smoke is carcinogenic to humans.

The occurrence of malignant tumours of the respiratory tract and of the upper digestive tract is causally related to the smoking of different forms of tobacco [...]."

Asked whether, after all the work they had done, this was quite a modest way of putting their conclusions, Sir Richard said that it made them clear for any reader to understand. So far as he was aware there had never been any challenge to these evaluations.

He was then asked about IARC 2004, which was substantially the same. His attention was drawn to two specific statements:

"The most compelling evidence for a positive carcinogenic effect of tobacco smoke in animals is the reproducible increase observed in several studies in the occurrence of laryngeal carcinomas in hamsters exposed to whole tobacco smoke or to its particulate phase."

"Tobacco smoking is addictive, and nicotine has been established as the major addictive constituent of tobacco products."

He said that the group had so concluded.

(5.194) He was asked about a lecture which he had given in 1997. In the lecture, he re-iterated his statements in greater detail.

Cross-examination of Sir Richard Doll.

(5.211) Doll was asked about this statement from US Surg Gen 1964:

"Statistical methods cannot establish proof of a causal relationship in an association. The causal significance of an association is a matter of judgment which goes beyond any statement of statistical probability."

He agreed with it.

He was asked about a statement from Doll 1950:

"*The paper obtained much less publicity than we had expected…."*

And this from Doll 1998 (seems to have been some sort of memoirs):

"*To Doll and Hill, it seemed clear that evidence of a different type would have to be obtained, if [critical] reactions to the findings in the case-control studies were to be changed*."

For these reasons, he and Hill decided to start the Doctor's Study (involving every doctor on the medical registry and concerned with their smoking habits). The study was followed through until 1998 (?) and Doll and Hill saw the death certificates of those doctors who died, apart from those who dropped out.

Doll wrote (in the same 1998 paper) that, following the publication of Doll 1954 and 1956, "*scientific opinion rapidly changed*". But two leading statisticians were unconvinced. Berkson from the USA thought that the first study might be biased. Doll said that he was wrong. Sir Ronald Fisher thought there could be genetic influences. Doll thought that Fisher's objections were not valid.

He also said in the same volume:

"*Following these reports, the idea that smoking was a major cause of lung cancer ceased to be seriously challenged, except by the tobacco industry outside the UK (where it had been quietly accepted)* ***and by a few eccentric individuals such as Burch*** *who, however, raised no material objections."* [My bold]

"*Asked by counsel for the identity of other "eccentric individuals", Sir Richard spoke of an Irishman, an American and an English psychiatrist; he could not remember their names or those of any of the others.*"

As regards Sir Ronald Fisher, he said:

"*[Doll] said that it seemed a pretty bizarre idea to him and to a lot of people. He was not aware of any epidemiologist, anybody experienced in his field, who took it seriously. There could have been some American geneticists who took him seriously, but how they came to do it he could not imagine."*

Doll also said:

"*Sir Ronald thought that he himself was "fair-minded", but nobody else did*."

In connection with Fisher's queries about the odd fact that smokers who inhale same to suffer less than those who do not, Doll said:

"…..of course Fisher was ignoring a lot of published data and he was quite wrong in making that conclusion in any case. He refused to look at the data when he made that remark. Sir Richard agreed, however, that this was the view that Fisher expressed."

And finally, Doll about Fisher:

"*He described Fisher as an "ignorant geneticist" in expressing this extreme view which other geneticists would not have taken. In relation to smoking, Fisher was an ignorant geneticist."*

It would perhaps be as well to say now where this line of questioning was going. In the first place, it had been said over and over again that the medical fraternity, almost to a man, totally agreed with the proposal that smoking caused lung cancer. But here was 'scientist' after 'scientist' being shown not to agree. Secondly, Doll was shown to be dismissive of anyone else who deigned to disagree with him, sometimes being rather insulting. He dismissed any other hypothesis as being 'far-fetched' or 'wrong-headed'. Thirdly, doubt was being cast upon the impartiality of various groups like IARC, the RCP and the USSG. The previous evidence of Prof Friend and Mr McTear's GP suggested that they themselves simply accepted as true what a comparatively small group of 'insiders' was telling them about smoking and lung cancer. Fourthly, although Doll dismissed any specific effect of another cause of lung cancer as having only a small effect, there were many such possible other causes, which, added together as possibilities, could prove to be rather large confounders.

We shall see later what Judge Nimmo thought of Doll's evidence.

The questioning went on in a similar vein for some time. Quotes from the work of many other credible people (with positions in universities all over the world) were brought out and equally dismissed. There was quite a lot of stuff about experiments with mice, hamsters, rats, rabbits and dogs. Attempts to produce squamous cell cancer in the epithelium ('inside skin', remember?) failed again and again, although certain Syrian golden hamsters did contract cancer of the larynx.

The relevant paras are about 5.220 to 5.303.

(5.304) The re-examination of Doll was very brief. I shall show this quote from the Judge's description:

Doll said:

In the case of occupational hazards, if a chemical agent was removed and the disease from which the workers were suffering disappeared, the causal relationship was confirmed. As regards smoking, if smoking was reduced, the diseases due to smoking became less common. "So what you have to do is decide that something is proved beyond reasonable doubt and then test it in practice, that your conclusion is correct." This was the standard he had used throughout his career as an epidemiologist.

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Professor Gerad Hastings.

(5.305) "*Professor Hastings, PhD, BSc, aged 49, was Professor of Marketing at the University of Strathclyde. He had been Director of the Centre for Social Marketing and the Centre for Tobacco Control Research there since 1987. He said that the Centre for Social Marketing was previously known as the Advertising Research Unit and was founded in 1980 by his predecessor. Once Professor Hastings had taken over the directorship, in 1992, to reflect the broadening of its advertising to look at the marketing process more generally, its name was changed to the present one. The Centre for Tobacco Control Research was founded in the late 1990s, with funding from the charity Cancer Research UK and other non-profit and Government organisations. He said that the two research centres really provided a channel for his research activity as an academic, which was an important part of his job*".

A strange sentence appears:

(5.306) *Professor Hastings served as Head of Department in the Department of Marketing from **1996 to 1992**.* 1996 to 1992???

Which shows that even judges can make mistakes! But the info is not relevant. I suspect that it should read '1986 – 1992.

Here is an interesting thing about Prof Hastings:

(5.308) *Professor Hastings said that in addition to having been Special Advisor to the House of Commons Health Committee on the Tobacco Industry and the health risks of smoking, he was currently Special Advisor in connection with an inquiry into **obesity**.* **[**My bold]

Well! Well! We **knew** that activists were multi-tasking, but there it is in print! Well! Well! And that was in 2003!

Whatever – press on.

(5.309) *Professor Hastings's report continued:*

"The health evidence about tobacco was therefore well known and publicised by the 1960's. For Mr McTear, however, the effects of this health information have to be balanced against three powerful counter-veiling [sic] forces emanating from the industry in general and Imperial Tobacco in particular:

1. Tobacco advertising

2. Health risk denial

3. A reluctance to introduce health warnings"

The imput of Prof Hastings is really weird. Here is another quote (remember that this is what Judge Nimmo describes):

Alan Marsh earlier in the 1990s

"He [Alan Marsh, a researcher] put the prevalence of smoking among poorer people down to "the sheer disappointment of being poor in Margaret Thatcher's Britain." He tied it in very closely with the problems of having very low aspirations, hopes and capabilities. Professor Hastings said he did not know about the reference to Margaret Thatcher, but he thought the principle was a good one. He had done focus groups in Jarrow in Northumberland with single mothers in very straitened circumstances, and tobacco provided one of the few pleasures they had in a life that was otherwise fairly bleak. "So it seems perverse but it does seem to provide people with some sort of support and tobacco promotion has actively exploited that."

So, as far as Hastings was concerned, it was desirable to withdraw one of the few little pleasure that poor people had in order to save them from…..what?……And to enable…..what?……and to stop tobacco companies doing…what?….and to put up taxes on fags to achieve…..what? More and more misery?

Thus we see the mindset of Hastings.

(5.319) A strange little disagreement arose. MCTEAR wanted to change a previous submission about tobacco advertising. ITL objected. Essentially, what MCTEAR wanted to do was to change a previous claim about 'what tobacco companies wanted to achieve by advertising' from the general case to add: "*The defenders' tobacco advertising since 1960 has encouraged people, including Mr McTear, to take up and continue smoking."* The Judge refused permission to change the previous submission, but MCTEAR continued to make references to ITL advertising intentions and so the Judge removed from the transcript any such references. Quite amusing, I thought.

Hastings goes on to make the most curious claims:

"*If Imperial Tobacco were genuine in their desire for Mr McTear to know about the health consequences of smoking they would not only have refuted this stance, but also moved to put health warnings on their products and advertising as soon as the health risks were established. The Royal College of Physicians Report in 1962 would have been a suitable time, for example."*

So Prof Hastings thought that, because the RCP 'spoke', tobacco companies should have automatically complied? Weird, or what?

Cross-examination of Prof Gerad Hastings.

This should be fun.

People who have seen this blog before might remember the numerous occasions upon which I have mentioned that there must be 'AN ASH SPEECHWRITER'. Reading this case, I have become convinced that Hastings is ASH's speechwriter. There may, in fact, be a 'speech writing department' in ASH, headed up by Hastings. It would not surprise me one bit because his speciality is 'media savviness'. He is a self-confessed ASHITE.

(5.330) *Professor Hastings agreed that in addition to the appointments he had mentioned in his CV, he was a member of the Advisory Council of ASH, which was a campaigning body. He gave advice to ASH on his understanding of how marketing worked and the need to fully comprehend how multifaceted it was.*

It is really difficult to take this guy seriously. His statements say, again and again, that the poorest people are the ones most likely to smoke….and take drugs….and drink alcohol. (5.333) Like….so what?

It is hard to understand how Hastings became 'an expert witness'. All he said was that advertising of fags was pernicious. But advertising of fags on TV had been banned since 1965, so what was his point? The pointlessness is illustrated by the fact that there was no cross-examination or re-examination.

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Dr Keith Kerr.

(5.339) *Dr Keith Kerr, BSc (Hons), MB, ChB, FRC Path, aged 47, was a Consultant Histopathologist at Aberdeen Royal Infirmary, a post he had held since 1989. His specialist area of expertise was pulmonary pathology. He acted as Consulting Pathologist to the Medical Boarding Centre (Respiratory Diseases) for Scotland and Northern Ireland. He was a member of the UK Interstitial Lung Disease Pathology Panel and a member of the International Association for the Study of Lung Cancer. He served on an international expert panel of six pathologists, with two others from Europe and three from the USA, for the ongoing trials of screening detection of early lung cancer. He had published over fifty scientific research papers, book chapters and invited review articles, and many abstracts relating to research into, and the diagnosis of lung disease, particularly lung cancer and its development. He sat on the Editorial Consensus group for the forthcoming revised World Health Organization (WHO) Classification of Lung Tumours, of which he was a co-author.*

The reason that I have published all that is because it seems to me that Dr Kerr is the only actual 'expert witness' that MCTEAR produced, at least as regards 'the actuality' of lung cancer. At least he was straightforward in his beliefs:

(5.340) *He taught that there is a strong association between smoking tobacco and lung cancer and that there were things in tobacco smoke which caused lung cancer.*

(5.341) In this section, Kerr describes precisely the changes which he saw in Mr McTear's bronchi. Read it if you wish, but it contains the foreign language of medical terminology.

In my personal ignorance, at first, I could not understand how Kerr was able to decide about Mr McTear's cancer in view of the fact that Mrs McTear refused to allow a post mortem. But then I saw this:

"*…each with three sectioned levels of a bronchial* ***biopsy*** *specimen*"

So, on the face of it, Kerr was using biopsy specimens which were taken before Mr McTear died.

It is, however, perfectly clear that Kerr *saw* squamous cell carcinoma. No doubt.

As I read (for the third or fourth time) Kerr's evidence, a sadness came into my mind. Here, I thought, was an immensely skilful man who had been brainwashed. Why else would the next section occur?

(5.343) *Dr Kerr said that he had seen thousands of biopsies like this from other patients. Squamous cell carcinoma, he said, was one of two types of lung cancer which were most strongly associated with a history of smoking tobacco. When specimens were submitted to him for examination, he might or might not be told the smoking history of the patient. If he was given any history at all, it would more often be in the unusual situation where such a specimen was sent to him in a non-smoker, simply to highlight the fact that he was being asked whether the patient had lung cancer and ought to know that the patient was a non-smoker. This sort of information was highlighted, though not very often, because it was so unusual that there would be a suspicion of lung cancer in someone who did not smoke. Specifically, with squamous cell carcinoma, 98% of cases were seen in smokers. This was based on the literature, but also his own experience: it was most unusual to see a squamous cell carcinoma of the lung in a non-smoker. He could give a figure of 98% based on his own patients. Lung cancer, he went on, was rare in non-smokers, and when it did occur, the squamous cell type was very infrequent. The pre-invasive squamous dysplasia, the precursor lesion of invasive squamous cell carcinoma, was also frequently found in the bronchi of smokers and much less so in non-smokers.*

It is really quite sad. The reason is that there are big contradictions in that paragraph which were only pointed out much later. The biggest contradiction was: "How could Kerr know that 98% of the biopsies which he performed, which showed squamous cell cancer, were from smokers?" He said himself that he was only told occasionally whether or not the specimens were from smokers, and only then when the sufferer was thought to be a non-smoker. But it is really obvious that people, said to have been non-smokers, may have been smokers, or vice versa. How was he to know? If he was not told in every case, how could he decide upon a figure of 98%?

However, having said that:

(5.344) *Table 7-4 gave figures (modified from Rohwedder and Weatherbee 1974) for five histologic subtypes of carcinoma in smokers and non-smokers. For squamous cell carcinomas, 98% were stated to have been in smokers and 2% in non-smokers. Dr Kerr said that it was from this table that he derived his figure of 98%.*

As we have said before, so where are the deaths from SHS? "Squamous cell cancer in non-smokers was 'very rare' or 'very infrequent'? So where is the evidence for SHS-caused lung cancer?

But we must press on.

Kerr referred to other research which described the transitional stages by which normal cells could be altered, step by step, until they become cancerous.

Cross-examination of Dr Keith Kerr.

I think that it is worth quoting the next para in full. The reason is that it gives a simple physical description of the location of the body parts which are affected by lung cancer and how these cancers cause death.

(5.350) *Dr Kerr agreed that the word "cancer", in colloquial use, referred to a variety of different types of tumour. In its general sense, the tumour was any lump or swelling, but in a more technical sense it might be used to describe a neoplasm. This was an abnormal growth of tissue that had become independent of any outside stimulus. It might be benign, such as a wart, which was a benign epithelial neoplasm, or it might be malignant. A malignant neoplasm was characterised by, among other things, invasion of the surrounding tissue. A squamous cell bronchial carcinoma might invade through the bronchial wall and into the mediastinum. It could involve the pericardium, the membrane surrounding the heart, and the great vessels which passed through the mediastinum. Simply by virtue of its growth, it could impinge upon these great vessels or on the oesophagus, in a manner which could lead to their destruction. Any of these events could result in the death of the patient. Another feature of malignant neoplasm was that it might metastasise to sites removed from the primary seat of the tumour. Dr Kerr had written on the significance of lung cancer to the mediastinal lymph nodes. Primary tumours in the mediastinum were relatively rare.*

(5.357) I am going to quote this section because it is interesting:

Asked whether it was the case that we did not know what proportion of the population, who exhibited no clinical symptoms and who never went on to develop lung cancer or any other condition requiring biopsy, had lesions in their lungs, Dr Kerr said that there was arguably some evidence in the papers of Auerbach and colleagues. In the autopsy studies, some patients had lung cancer, some did not, some patients were smokers and some were never-smokers, so in a sense these were studies of samples of the population at large and gave some indication of how frequent or otherwise these changes might be in the airways. A variety of the changes were found in patients who did not have lung cancer, but the changes most likely to be associated with the development of lung cancer were very rarely found in patients who were never-smokers as opposed to those who were smokers. He agreed that these were

autopsy studies carried out on people who died in hospital, but said that they would be more or less typical of the population, depending on the parameter that was being examined. Auerbach worked at a veterans hospital, but Dr Kerr thought that the general population were treated at such a hospital, it was not just for ex-soldiers.

It is interesting because, as far as I am concerned, it is the first time that I have seen evidence of attempts to find out whether or not people who died for other reason might have had some form of undiagnosed cancer.

There is more about this in succeeding sections, but not a lot.

(5.362) *Kerr was not aware of ant studies which had examined the possibilities of other factors in the development of lung cancer such as alcohol, lack of vitamin A, etc.*

There then followed further discussion of statistics re smoker and non-smoker lung cancers and other cell changes.

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Re-examination of Dr Kerr.

This was very short and adds little.

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EXPERT WITNESS FOR ITL.

[These were Prof Gray, Dr James, Prof Idle, Dr Lewis and Prof Platz.]

(5.373) *Professor Gray, aged 69, was Emeritus Professor in the Department of Psychology at the Institute of Psychiatry, University of London. He described this as a tertiary education and research establishment, generally regarded as one of the best, and perhaps the best, in the world, in the field of psychiatry.*

(5.378) *As part of Professor Gray's research he had investigated dependence on both opiates and anti-anxiety drugs, and psychological treatments for anxiety, depression and opiate dependence.*

So we get a feel of the expertise of Gray. ITL have introduced a Psychologist. He described research on the behaviour of animals like rats and monkeys as they might mimic human behaviour.

In further sections he described the many and various ways that smokers smoked and what they wanted from and how they were affected by the activity, including smoking de-nicotinised cigarettes.

Here is an interesting observation:

(5.380*) Conversely, cigarettes from which the nicotine had been removed reduced self-reported "craving" and increased ratings of smoking satisfaction and psychological reward. In the same experiment, moreover, an intravenous injection of nicotine failed to increase either satisfaction or reward.*

(5.387) Gray reported this interesting possibility: the 'depression' which smokers felt when trying to quit may not be a result of trying to quit. It may be the other way round. It may be that people smoke to alleviate mild depression, and that the condition returns when they stop smoking.

An interesting observation:

(5.394) *Professor Gray next referred to what he called the "addiction" view. He said that in contrast to the complexity of smoking behaviour already noted by him, it had become almost a point of dogma to treat the continuation of tobacco smoking as entirely dependent upon one particular compound: nicotine. At its simplest (the level, indeed, at which it was most often expressed), this view was summed up in two bullet points on the back cover of RCP 2000:*

"

- *Most adult smokers do not smoke out of choice but because they become addicted to nicotine [...];*
- *Cigarettes are highly efficient nicotine delivery devices and are as addictive as drugs such as heroin or cocaine."*

Professor Gray said that he did not agree with the view that smokers do not smoke out of choice but because they become addicted to nicotine.

Contrasting the 'nicotine habit' with hard drugs, he said:

"The drug-taker [hard drugs] now had two strong compulsions to continue taking the drug: craving for its euphoric effects and the need to avoid unpleasant withdrawal symptoms." Smokers did not experience 'the high' which heroin users did.

Another interesting observation:

(5.402) *Returning to the issue of self-report, Professor Gray said that smokers did, in contrast, report reduced "craving", increased smoking satisfaction and increased psychological reward when, after overnight abstention from tobacco, they smoked cigarettes* ***from which the nicotine had been removed****: Rose et al. 2000. Thus it was misleading to liken the pleasure derived from smoking cigarettes to that occasioned by cocaine or heroin; and the pleasure so derived did not depend principally upon nicotine.*

(5.405) *Abstinent smokers given nicotine replacement therapy did not normally continue to take nicotine for long periods, in sharp contrast to abstinent heroin users treated with the opiate substitute, methadone, who typically continued to take this drug for years. And, when patients were treated with nicotine for other medical conditions, such as ulcerative colitis, they did not exhibit nicotine-seeking behaviour,*

whether they were non-smokers or ex-smokers; nor did the ex-smokers revert to smoking as a result of this treatment.

About 'withdrawal symptoms':

(5.411) *There were marked differences between the "withdrawal symptoms" described upon quitting smoking and heroin, respectively. As well as diarrhoea, symptoms of heroin withdrawal included tears from the eyes, bleeding from the nose, nausea or vomiting, sweating, fever, muscle pain, joint pain and lethargy. The symptoms described by smokers were almost entirely psychological, above all irritability,*

Another:

One therefore needed to consider the possibility that the causal chain which led to the withdrawal symptoms in the person who quitted smoking might not involve nicotine at all, but might involve merely the habit of smoking: of carrying the cigarettes,

Dr Gray's testimony was very long, so I propose to move on. We can see the direction in which he is going. Towards the end, he mauled some of the statements in some RCP (College of Physicians) documents regarding addiction. He also commented on the difficulty of obtaining funding. Where significant funding was required, only so many sources were available. If funding was refused by medical sources, there was normally only one other source (regarding tobacco studies) which was tobacco companies. This caused a problem in that most institutions simply refused outright to consider any studies fund by TCs.

Cross- examination of Dr Gray.

First, Gray was asked about his funding for his studies. Some was from tobacco companies as he had explained. He was being paid for his time in the present case and expected 'five figures'.

He was asked about the statistics for quitting. He said that much of it was outside of his expertise. When asked if he agreed that nicotine caused the difficulties in quitting he said:

(5.440) "*If the quitting were quitting from nicotine use, rather than smoking, then there was no problem whatsoever: the quitting rates from nicotine delivered by any other mode were essentially 100% immediately. Smokers smoked cigarettes, as he had previously explained, for a variety of reasons*."

He said this about USSG 1988:

(5.443) *"A report such as this was essentially part of a strategy aimed at changing people's behaviour and was supplying messages concerning health and the measures that people could take. So it was a mixture of propaganda and the scientific evidence upon which it was based, but with the interpretations and the language used to*

describe those data biased towards the interests of the message that the report was conveying. With regard to the scientific standard of proof it was a biased report"

Re-examination of Dr Gray.

(5.447) *In re-examination Professor Gray pointed out* ***that Warburton had been involved in the preparation of USSG 1988. In Warburton 1988b he referred to criticisms he had published of the conclusions of the editors of the report.*** *Professor Gray said that the view he had reached on his reading of the evidence was not dissimilar, as regarded the addiction model, to that of Professor Warburton. Frenk and Dar 2000 expressed views with which he also agreed about the inappropriateness of describing nicotine as addicting.[My bold]*

That was it. Note well the bold bit. The authors of USSG 1988 had used it for 'health messages' purposes, beyond what the scientific evidence said.

Dr Derek James.

(5.448) *Dr James, aged 40, had been senior lecturer in Forensic Pathology at the University of Wales College of Medicine since 1977. He was Honorary Consultant in Forensic Pathology to Cardiff and Vale NHS Trust, also since 1997. In the latter post his responsibilities were to run the hospital consent autopsy service, in association with other pathologists, to teach the postgraduate pathology trainees and carry out other teaching duties……*

Here is a strange statement:

(5.450) *Dr James was one of the signatories of a letter published in the British Medical Journal on 3 October 1992, James et al. 1992. The letter started:*

"The reasoning behind the decision ***that doctors can now put smoking as a cause of death on death certificates*** *without the death having to be reported to a coroner is obscure."*

Was that ever put into effect? I have never heard that before. James said that there could be litigation implications since doctors frequently found it very difficult to say with any precision what the cause of death was.

An important plank in the arguments that smoking causes lung cancer is the perceived *fact* that lung cancers rose massively when cigarette smoking became common. As a person having been involved officially in government investigations of the accuracy of death certificates, he was qualified to speak on this subject.

In para 5.454, Judge Nimmo quotes a long passage which I will not produce. Suffice to say that there were many reasons that diagnosis of lung cancer in earlier times was

unusual and that modern equipment and modern hospitals made the task much easier. The massive increase may be unreal.

James went on to explain the difficulties at great length and also compared the position in Britain with that in the USA. The same factors held there also.

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Cross-examination of Dr James.

MCTEAR tried hard to shake Dr James, but failed.

I have mentioned in an earlier post the para which mentioned a '50-fold increase' in diagnoses of lung cancer. James dismissed it. He said that, since deaths from lung cancer were reported to be 1% of deaths in 1900, a 50-fold increase would mean 50% of deaths were from lung cancer, which was nonsense.

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Prof Jeffrey Idle.

(5.480*) Professor Idle, aged 53, was Professor in Medicine and Molecular Biology at the Institute for Cancer Research and Molecular Biology, Medical Technical Research Centre, Norwegian University of Science and Technology,…. He became a European Professional Biologist in 2000 also, his qualifications in biology in the United Kingdom thereby being recognised across the European Union.*

In other words, a bio-chemist.

(5.484) *Professor Idle was asked by ITL's solicitors to give an opinion based upon his own area of scientific expertise in answer to the following five questions:*

"(1) In your opinion was it 'almost universally accepted by scientists' in 1964 [as averred on behalf of Mrs McTear] that cigarette smoking can cause lung cancer?

(2) In your opinion had cigarette smoking been established as a cause of lung cancer as at 1964?

(3) In your opinion had cigarette smoking been established as a cause of lung cancer as at 1971?

(4) In your opinion had cigarette smoking been established as a cause of lung cancer as at September 2003?

(5) In your opinion did cigarette smoking cause Mr McTear's lung cancer?"

That is our starting point. We are now into the really, really important part – CAUSATION.

After reviewing the scientific literature for the relevant periods, his answer to the first four questions was "No" and for the fifth was "Don't know".

We should note this:

(5.485) *Professor Idle said that the complexity of tobacco smoke was something he had investigated in the course of researching for his report, as was DNA damage, which indeed he dealt with in his normal academic working life.*

(5.486) RCP 2000 had listed the diseases which the College of Physicians thought were 'caused in part' by smoking. "……*cancer at seven specified sites and also in unspecified sites, and myeloid leukaemia. Among other diseases were ischaemic heart disease, cerebrovascular disease, aortic aneurysm, myocardial degeneration and atherosclerosis, in the category of circulatory diseases; and ulcer of the stomach or duodenum, in the category of digestive diseases.*

Professor Idle said that in preparing his report he had given some thought to the biological plausibility of cigarette smoking being responsible for some of this range of diseases."

These ideas had been around for decades. In 1959, some researchers were calling these statements into question:

(5.487) *In Berkson 1959, the author stated at p.445:*

"For myself, I find it quite incredible that smoking should cause all these diseases. It appears to me that some other explanation must be formulated for the multiple statistical associations found with so wide a variety of categories of disease. And if we are not crassly to violate the principle of Occam's razor, we should not attribute to each separate association a radically different explanation.

One explanation is that the associations have a constitutional basis. This hypothesis has been formulated – and rejected – by Doll and Hill in the following terms:

'… it has been suggested that constitutional and psychological factors might have such an effect…. that persons of a certain "make up" are peculiarly liable to lung cancer and to smoke. We know of no published evidence to this effect.'"

To the quotation from Doll and Hill Berkson added a footnote, also at p.445:

"Cancer is a biologic, not a statistical, problem. Statistics can soundly play an ancillary role in its elucidation. But if biologists permit statisticians to become the arbiters of biologic questions, scientific disaster is inevitable."

Idle said that the epidemiological evidence constituted no more that 'a hypothesis' which would need to be confirmed by 'observation (physical research)'.

Idle goes on to talk about the biology of cancer in general:

(5.492) *Professor Idle then turned to the biology of cancer. He said that it was quintessentially a biological entity. It was a general term for more than 100 malignant diseases that were characterised by uncontrolled, abnormal growth of cells and the spread of these cells locally and through the bloodstream and lymphatic system to other parts of the body, by a process known as metastasis.*

Idle went on to describe historical research into the cause of cancer. He was emphatic about 'the scientific method' – controlled, physical experiments. He talked about:

- Chronic irritation.
- Viruses.
- Chemicals.

A quote:

(5.502) *By the end of 1971, therefore, the evidence suggested that viruses could cause human cancer and that respiratory virus infection might be a cause of human lung cancer.*

Another:

….one of the viruses was unable to cause cancer itself in the animal model but after irradiation with ***ultraviolet light*** *it was,…..[My bold]*

About cancers caused by viruses:

(5.507) *Estimates of the proportion of human cancers induced by viruses ranged from 10% to 25%. Anonymous 1986 stated, at p.1431: "[A] reasonable estimate suggests that at least 25% of all human cancer worldwide is likely to be virus-induced." Specifically, Professor Idle stated, with respect to lung cancer, two human viruses and a number of animal viruses had been proposed as aetiological agents: first, the T-cell leukaemia virus, HTLV-1, which was endemic in certain regions, was found to be associated with small cell lung cancer in Japan; secondly, several studies had reported a high incidence of lung cancer in HIV-positive patients; and, thirdly, human papillomavirus (HPV) had been suggested to contribute to the pathogenesis of human lung cancer based upon the finding of statistically significantly elevated HPV in the blood of 47.7% of 149 lung cancer patients, compared with 12.6% of 174 non-cancer controls in a study conducted in Taiwan: Chiou et al. 2003.*

Idle then talks about chemicals as carcinogens. In early experiment (1922), shale oil distillate ('tar', essentially) was painted onto the skin of mice and caused malignant cancers. (5.513)

(5.517) Much research was done on the chemical constituents of coal tar to try to discover the actual molecules which were the carcinogens. Attention concentrated upon benzo[*a*]pyrene. *Only benzo[a]pyrene was carcinogenic on mouse skin.*

Read that para yourself if you want to know more.

Several long paras then ensued as this matter was investigated over a long period of time, until we reach this point:

(5.534*) Cells were always thought to die by damage from the outside, undergoing a certain transformation during death that could be seen under the microscope, a process called necrosis. It was then observed that cells died, not just by necrosis, but by what was now called apoptosis. A signal was sent from the nucleus of the cell, usually to itself but also to other cells, saying that it was time to die. The cell shrank and a dissolution took place, enabling its materials to be used by other cells. Interference with either an oncogene or a tumour suppressor gene affected the division, differentiation and death of a cell.*

[An 'oncogene' is a gene which, when damaged, can cause a cell to become cancerous. A 'tumour suppressor gene' is one which can either stop the action of an oncogene or repair the damage (I think!)]

In some ways, the rest of this section seems to move into the realms of science fiction:

(5.535) *There were tens of thousands of modifications to DNA in every single cell of the human body every day.*

Of particular important was the P53 gene:

(5.537) *Professor Idle said that the tumour suppressor gene which was of the greatest interest and which had been the most extensively studied over the past twenty years was p53 (the protein coded for by this gene having a molecular weight of 53,000). The p53 gene served as a hub to contribute to the control of apoptosis, DNA repair, the cell cycle and differentiation pathways: many of the housekeeping and important functions of a cell. Robles and colleagues, writing in 2002, stated that since the discovery of this gene an overwhelming volume of literature had accumulated supporting its role as a central regulatory node in the protein network that mediated cellular responses to endogenous and exogenous stresses. Disabling this pathway through mutation or silencing of its components led to self transformation and was thought to contribute to tumorigenesis.*

A quote:

(5.542) *Under reference to a figure in his report, Professor Idle said that it had been reported that the p53 gene contained mutations in most types of human cancer, ranging from a frequency of between 13% and 14% in prostate carcinoma, up to more than 70% in small cell lung cancer. The frequency in non-small cell lung cancer, which included mainly adenocarcinoma and squamous cell lung cancer, with a frequency of about 40%, placed this group in the middle of the range, so it was no different from other tumours essentially. Professor Idle said that it was noteworthy that, while 40% of non-small cell lung cancer tumours were reported to have had p53 mutations, 60% did not. The data were drawn from an international database held in France and associated with IARC; they came from a relatively small sample of the population. The observation that certain proto-oncogenes and tumour suppressor genes were mutated in various human tumours had become a major focus of the current paradigm of chemical carcinogenesis.*

Some parts of this section become difficult to follow and to understand. Best thing to do is just read them and move on! Just read them and take out of them what you can.

(5.548) The *next theory discussed by Professor Idle was that of personality, emotion and stress. He said that this might be the most long-lived of the all the theories.*

I don't propose to examine the detail of this section. Suffice to say that the probable reason that stress can lead to cancer is its effect upon the immune system*. LeShan, writing in 1959, was one of the first to point to personal loss as a cause of cancer in humans, when he wrote that the most consistently reported, relevant psychological factor had been the loss of a major emotional relationship prior to the first-noted symptoms of the neoplasm.* (5.548) [I have personal experience of this. My sister died from breast cancer. Within twelve months, her husband was dead from stomach cancer (his father died from the same cause)].

I have moved on substantially through Dr Idle's evidence. It is very long, very detailed and very complicated. I arrive now at:

(**5.586**) *Professor Idle said that during the 1990s, there was still much discussion of the immune surveillance theory of cancer. The theory was restated in simple terms by Chowdhury and others in 1991, with the concluding statement that the survival and growth of the tumour cells depended crucially on how the tumours evaded the immune surveillance mechanism of the host and escaped the killer cells as well as the antibodies.*

The above seems to be related to the fact that the risk of cancer grows as people age. (5.585) *One of the strong arguments in favour of the immune surveillance theory of cancer, he said, was the mounting incidence of cancer with increasing age, an approximate doubling every five years after the age of 25, and the parallel decline in immune competence with increasing age.*

Idle then talks about the opinions of those who disagree, and the consequences.

At 5.600 Idle discusses oxygen 'free radicals'. He said that work continues on this subject *In Professor Idle's judgment, whether or not oxidative stress played a role in human lung cancer was not established, though work continued on this question.*

From 5.602 Idle discussed 'epigenetics'.

This term was coined by the eminent biologist CH Waddington, who was Professor of Genetics at the University of Edinburgh. He did not believe that genetics, embryology and evolution were separate sciences, and attempted to understand the link between the genotype and the phenotype of an organism. A phenotype was a genetically-determined characteristic of an individual, ***such as eye colour or blood group****. The genotype was the genetic information held in those genes that coded for the phenotype, so that the latter was a manifestation of the former. Epigenetics was the study of the processes by which the genotype gave rise to the phenotype, how the phenotype was manifested from the genetic information in the cell.*

As far as I can tell, there is an implication in what Idle says that cells are coded with many possible alternatives. For example, if a man with brown eyes and a woman with blue eyes have a child, the child may have brown eyes or blue eyes or some variant. The DNA of the child has the 'codes' for both. If the child's eyes turn out to be blue, it is because the 'brown code' has been suppressed, and not that it does not exist.

At 5.610, Idle turned to the subject of 'aneuploidy'. *He explained that aneuploidy was the state of having the wrong number of chromosomes in the cells, either too many or too few.*

I propose not to enter that subject, except to say that cancer cells seem to be prone to 'aneuploidy' (Too many or too few chromosomes).

(5.616) The next main section of Professor Idle's report and evidence related to laboratory investigations with tobacco derivatives and public health reports up to the end of 1964.

Another huge section. Idle takes the court through the masses of experiments on animals (mostly mice, hamsters and rats) concerned with tobacco smoke and tar condensates. I propose not to go into detail, but I will quote this para because it teaches us something:

(5.624) *The next comparison made by Professor Idle was that of mouse skin epidermal tissue versus respiratory epithelium. He said that the mouse skin, like most mammalian skin, was comprised of epithelial tissues, organised in layers. Within these layers were specialised tissues, such as various types of gland, hair follicles and so on. When the outer layers were damaged, they came off, and new layers rose up, hardened and became the outside surface. In the lung, while the tissue was epithelium, it was structurally very different. The cells were not arranged in flat layers, they were arranged in columns known as "columnar epithelium". On the top of each column, at least in the tracheobronchus, were little cilia, which wafted the mucus blanket up from the lung to the level of the larynx. Between these columns were goblet cells that produced the mucus. The epithelial cells of the skin arranged in layers, sometimes described as a "squamous" or "pavement" morphology. Thus, the epithelia of the skin were and the tracheobronchus were architecturally different and they served distinctly different functions for the host.*

Another portion is worth quoting:

(5.636) *Professor Idle was next asked to comment on passages in various public health reports. In RCP 1962 it was stated at p.26, para.37:*

"Skin cancer can be produced in mice by applications of tar condensed from tobacco smoke but the results obtained by various investigators have not been uniform and exposure of animals to tobacco smoke in inhaled air has failed to produce lung cancers."

Professor Idle said that these statements were accurate. The paragraph continued:

"Moreover the amount of cancer-producing substances in the smoke itself does not seem likely to be sufficient to account for the large number of cases of cancer associated with the habit."

Professor Idle said that he thought that the Royal College of Physicians were recognising at that point that workers had not been able to identify any substance or substances in the smoke in sufficient quantity to explain the statistical association between cigarette smoking and lung cancer. RCP 1962 stated at p.26, para.39:

"In addition to the known carcinogens which have been detected in tobacco smoke others as yet undetected may be present; possibly two or more in combination may reinforce each other in producing cancer. It is possible that tobacco smoke may contain substances which act in conjunction with substances generally present in the air we breathe to produce cancer, although neither substances might do so alone. Indeed the action of tobacco might be simply to produce chronic irritation which, as in other tissues, may increase liability of the lung to cancer. There is a wide field for further investigation here, but no ground for refuting the evidence from human experience."

Professor Idle commented that this passage contained speculation, unvouched by experimental evidence, and he agreed that there was scope for further investigation. In RCP 1962, at p.S5 of the summary, among the facts mentioned which might be considered to conflict with the conclusion that smoking was a cause of lung cancer was the fact that no animal had yet been given lung cancer by exposure to cigarette smoke. Professor Idle agreed that this statement was correct.

Are we seeing a pattern again and again? The 'science' says one thing and 'public health' says another.

Another quote:

(5.650) *Professor Idle's attention was next directed to IARC 1986. At pp.135-136, in discussion of the administration of tobacco smoke condensates, reference was made to the 1952 paper of Wynder and others. At p.136 it was stated:*

"Animal studies conducted prior to 1964 provided an important measure of support for the epidemiological demonstration that cigarette smoke is an important human carcinogen."

Professor Idle said that he did not agree with this view, for the reasons already given by him. This statement, in IARC 1986, was at variance with Wynder's statements about the purpose of his studies, which was to identify constituents that might be carcinogenic and to remove them, rather than to prove a causal connection between cigarette smoking and lung cancer. For the reasons he had given, studies in which tobacco smoke condensate was painted on to mouse skin were not designed to answer, and could not answer, the question of whether or not cigarette smoking was the cause of human lung cancer. Thus, in the period 1965 to the end of 1971, there was an increased focus on experiments that exposed experimental animals to whole cigarette smoke by inhalation. This was seen, at the time, as a model which more closely resembled human smoking than the mouse skin painting model.

Are there any depths to which ASH ET AL will not sink in order to promote their propaganda? The reference to the purpose of 'mouse skin painting' (to try to find out what actual carcinogenetic substances might possibly cause lung cancer and remove them) is verified by the fact that the condensate used was forty times stronger than the condensate (tar) in normal cigarette smoke.

(5.651) Professor Idle then turned to experimental animal inhalation models.

Again, I do not propose to examine the detail, but here is a straightforward quote:

(5.652) *First, there were studies in mice, the methodology and results of which were summarised in his report. He was asked simply to state the conclusion, that in none of them was squamous cell carcinoma of the lung induced. [Judge Nimmo]*

Again, trickery by ASH ET AL was exposed. The science was 'bent and twisted' to fit the phraseology.

There is so much stuff here that it is hard not to quote almost everything! But I have to be selective so as not to drag this post out too long. I have looked for information which might support ASH ET AL, but can find none.

(5.675) *Among studies in dogs, an unpublished industrial report from Hazleton Laboratories America, Inc in 1971, cited by Coggins in a review article in 2001, described a study in which 240 male beagles were exposed to cigarette smoke by tracheostomy for 108 weeks (the same procedure as that used by Auerbach and Hammond). There were no reports of neoplasia.*

Here is a quote with an interesting comment:

(5.677) *The largest and best design smoke inhalation study was that performed by Microbiological Associates, reported in CTR 1984. In this study, no squamous cell carcinomas were reported to develop in mice exposed to fresh whole cigarette smoke.* ***Positive controls demonstrated that the mice used in the study were capable of developing squamous cell carcinoma of the lung when exposed to the carcinogen 20-methylcholanthrene.***

See? It was not possible to claim that the mice simply could not develop squamous cell cancer since they did develop that cancer when exposed to 20-meth…..

(5.681) *The next topic was short-term mutagenicity testing. Professor Idle was told that Dr Kerr had given evidence that "there are things in tobacco smoke which cause lung cancer", a view based on, inter alia, reports that chemicals in tobacco smoke had induced changes in human cells, in tissue culture for example, recognised as being associated with the development of the malignant process. Professor Idle said that he recognised that this was a process that occurred.*

In this section, Idle discusses experiments with bacteria which could be 'genetically engineered' to show the effects of tobacco smoke.

(5.687) *Professor Idle then went on to consider research in the period 1973 to the present date into the individual chemical constituents of cigarette smoke with a view to understanding which constituent or group of constituents might cause lung cancer in cigarette smokers. Having reviewed various areas of research in this period, he concluded that as at September 2003, no constituent or constituents had been identified in cigarette smoke that accounted for lung cancer in cigarette smokers.*

Here is an interesting quote. It has to do with the possibility of checking every possible combination of the 4,000 chemical in tobacco smoke for carcinogenicity (even if only two at a time were combined):

(5.691) *With respect to cigarette smoke, with 4,000 chemical components, the "factorial design" would call for 2^{4000}-1 individual binary interaction experiments. This number might also be expressed as 1.32×10^{1204}. To perform the binary interaction experiments at two doses of each component, which was the recommended procedure, the number of experiments required rose to 1.55×10^{1908}. Professor Idle said that clearly this was neither feasible nor desirable. A rigorous, bottom-up, component-based, toxicological analysis of tobacco smoke was impossible.*

We are now at the end of Professor Gray's evidence. Here is a final quote:

(5.694) *In explaining his conclusions as at September 2003, Professor Idle said that it was his judgment that cigarette smoking had not been established as a cause of human lung cancer. Indeed, as explained by him, the cause of cancer was unknown. Moreover, the mechanisms by which lung cancer developed were not known. Researchers had not produced squamous cell lung carcinoma in laboratory animals by inhalation exposure to cigarette smoke. No constituent or group of constituents, as they existed in the complex mixture which was cigarette smoke, had been shown to be a cause of lung cancer in smokers. In view of this, it could not be determined whether or not smoking caused Mr McTear's lung cancer.*

(5.695) *Professor Idle said that he did not always hold the view that cigarette smoking had not been established as a cause of human lung cancer. He entered into the field of lung cancer as a young researcher from pharmacology, a background of pharmacogenetics,* ***and he adopted the received wisdom of the day that smoking causes lung cancer****. More than twenty years of association with studies and becoming familiar with the literature caused him to look at the subject in a serious way, and then to understand that there were so many components missing that he could not see where, in the history of the subject, it had been scientifically established by experiment using the scientific method. So his view had altered within the last ten years.*

<u>Cross-examination of Prof Idle</u>.

MCTEAR did their best shake Prof Idle, but failed completely. They took the opportunity to bombard him with the conclusions of various 'eminent bodies', such as the College of Physicians. He would not be shaken. It is impossible not to come to the conclusion that MCTEAR knew very well that they would not be able to shake Idle

and that their protestations of the eminence of the organisations were aimed at Judge Nimmo.

Re-examination of Prof Idle.

Idle was taken back through some of the evidence which he had given. Not a lot was added, but I can quote this:

(5.742) *Professor Idle was asked about the suggestion that had been made, in connection with animal inhalation studies, that animals could not be compared with humans because of their much shorter life spans. He said that he was sure that researchers were aware of this and took account of it in their study designs. As previously stated by him, Laskin and colleagues exposed rats to the irritant gas sulphur dioxide mixed with the polycyclic aromatic hydrocarbon benzo[a]pyrene and reported that some rats developed squamous cell lung tumours. This report established that the rat was a species susceptible to develop squamous cell lung carcinomas.*

Dr Arnold Cohen.

(5.745) *Dr Cohen, BSc, PhD, C.Chem, FRSC, MBIRA was aged 66. He was an Associate and thereafter a Fellow of the Royal Society of Chemistry (formerly the Royal Institute of Chemistry) and a member of the British Institute of Regulatory Affairs and had been admitted to the Register of Toxicologists of the Institute of Biology/British Toxicology Society and the register of Eurotox Registered Toxicologists.*

Cohen's evidence was very much a'back-up' of Idle's evidence. As a chemist and toxicologist, he confirmed Idle's analysis. He also added some observations of his own about 'confounders' in certain research studies. In particular, he addressed the problem of lifespans in mice, rats, etc as compared with humans when calculating the effects of carcinogens. He said that experiments had shown that these species were indeed susceptible to the carcinogens. He explained how control groups of animals were used (in the best way).

A quote of interest:

(5.759) *Dr Cohen then turned to rat studies. IARC 1986 stated at p.194: "In one study involving long-term exposure of rats to cigarette smoke, tumours of the respiratory tract were induced." This was a reference to Dalbey et al. 1980. In this study the authors stated that their choice of a particular strain of rat was heavily influenced by the induction of squamous carcinomas in the respiratory tract after intratracheal instillations of relatively small amounts of polycyclic hydrocarbons or after bronchial implantation of pellets containing cigarette smoke condensate. They stated at p.387:*

"We observed 10 respiratory tumors in 7 smoke-exposed rats. Nasal tumors occurred as 1 early adenocarcinoma and one squamous cell carcinoma. The pulmonary tumors were 5 adenomas, 2 alveologenic carcinomas and one squamous carcinoma [...]. One alveologenic carcinoma was observed in controls. [...]

Besides the description of laryngeal neoplasms in smoke-exposed hamsters [...], the present work is the only study in which an unequivocal tumor response in the respiratory tract resulted from long-term tobacco smoke exposure. The tumors in the respiratory tracts of smoke-exposed rats consisted of 5 adenomas and 5 adenocarcinomas, alveologenic carcinomas or squamous carcinomas, as compared to one alveologenic carcinoma in the control animals."

In Table 2 the authors stated that 9% of smoke exposed rats, i.e. seven individuals out of the original group of eighty, had tumours in the respiratory tract. Dr Cohen said that he believed that the authors should not have included certain tumours in that grouping, and should have shown the benign tumours and the malignant tumours in the lung not only separately but to indicate whether the animals that showed the adenomas also had adenocarcinomas. ***It was not known whether the lung adenomas and lung adenocarcinomas appeared in seven, six or five animals****, from the data presented in the paper****. The two nasal tumours should not have been included because the practice of the National Toxicology Program (NTP) guidelines at the time recommended that nasal tumours should not be lumped together with lung tumours in tumour analysis.*** *[My bold]*

A long quote, but necessary. The reason is that, as the bold bits indicate, the researchers were not following accepted procedures and that they had only stated the number of tumours without stating how many animals these tumours appeared in. Taken together, these faults could reduce the apparent incidence of tumours in the 'smoking animals' as compared with 'non-smoking animals' to unity (the same). That is, that the researchers did not know the rules, and, by implication, that they did not know what they were doing. We amateurs do not realise how MASSIVELY IMPORTANT these faults are.

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<u>Cross-examination of Dr Cohen</u>.

There is no need to spend much time on this. Again, MCTEAR tried to shake Cohen without success – epidemiology was not his sphere, etc. But I must quote this:

(5.768) *Asked about his criticisms of Dalbey et al. 1980, Dr Cohen said that if only five instead of seven animals developed tumours, that would not be statistically significant even compared with combined controls. He had consulted a statistician and five out of eighty compared with one out of ninety-three would not be statistically significant at the probability level of less than 0.05 (P<0.05). In carcinogenicity studies the importance should be on the number of animals showing the tumour, rather than the number of tumours, because some animals could have multiple tumours. This was the practice accepted by IARC.*

Do you see? IARC accepted the rules, and these had been broken, and yet organisations such as IARC were using this study!

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Dr Michael Lewis.

(5.772) *Dr Lewis, aged 55, stated that he was born in Germany and studied biology in the USA from 1970 to 1974, gaining a Bachelor of Science degree. He then went on to study medicine in Germany and became a doctor of medicine, followed by epidemiology in the USA and Canada, gaining a diploma in epidemiology from McGill University.*

An epidemiologist! Let the brown stuff hit the twirly thing!

Lewis assisted the court by describing how 'relative risks' works. For example, if 10 people in 10,000 get flu in the winter, year after year, that could be regarded as 'the normal risk' of flu in any given year. That could be regarded as '1' – equals 'normal'. If, on the other hand, in any given winter period, 20 people in 10,000 get flu, then 'the normal risk' is doubled. The 'relative risk' goes from 1 to 2. An 'epidemic' might be declared to exist if the numbers rose to 100 in 10,000. In that case, the 'relative risk' becomes 10 – meaning that the risk is ten times greater than 'normal'. It is easy to see how newspapers could shout out headlines about this 'relative risk' – "BEER DRINKERS TEN TIMES MORE LIKELY TO GET CANCER!!!"

For simplicity, I used the calculation 10 in 10,000 and 100 in 10,000, but the same headlines could be produced by a calculation of 10 in 1,000,000 and 100 in 1,000,000. The RR is still ten times!

It is very easy to use 'relative risk' to scare people to death, as we have seen above.

In connection with what I have just illustrated, look at this quote from Lewis:

(5.817) *In ACC 1981 at p.13 a table was given for age-adjusted death rates per 100,000 population for selected sites for forty-two countries, derived from statistics for the years 1976 and 1977. For lung cancer in males the rate for Scotland was 108.5, which made Scotland the highest ranking country. For Ireland the rate was 61.8 and its rank was sixteenth, and for Japan the rate was 28.3 and its rank was thirty-first. Dr Lewis added the comment that at that time Japan was known to have one of the highest smoking prevalences in the world.*

Roughly, the 'relative risk' of lung cancer for Scotland was 100 per 100,000. For Ireland, it was 60 per 100,000. For Japan, the risk was 30 per 100,000. Let us look at the simple ratio here. If we take the lowest 'relative risk' as 1 (Japan), then Ireland becomes 2 and Scotland becomes 3 and a bit. Wow! Scotland is three times worse than Japan! But there is a HUGE 'but' – smoking is more prevalent in Japan than in Ireland and Scotland!

But we can see here something very curious. It seems that the ‘smoking problem’ in Scotland is so bad that it needs an ASH ET AL presence three times greater than England and Wales.

Whatever – we have not yet finished with THE JUDGEMENT.

The re-examination of Prof Lewis continued, but MCTEAR could not shake him. In fact, the cross-examination afforded Lewis even more space to contradict the received wisdom that smoking ‘causes’ cancer.

We can move on.

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Prof Charles Platz.

(5.847) *Professor Charles Platz, aged 66, was a retired surgical pathologist. After completing his education, which included the degree of MD from the University of Chicago and postgraduate posts held there, he held a series of academic appointments.*

We now have a surgical pathologist.

Platz was was asked to comment on Auerbach *et al.* 1957 and Auerbach *et al.* 1961.

(5.855) *At p.254 of the 1961 paper the authors stated:*

“Ever since 1954 the tracheobronchial tree has been dissected out of the lungs of every person examined at autopsy at the Veterans Hospital, East Orange, New Jersey. More recently, a similar arrangement was made with 11 other hospitals, most of which are in upstate New York. All this material is imbedded in paraffin and is available for study.”

Altogether 402 white males, including the 117 subjects in the 1957 paper, were included in the 1961 study.

The authors of the study came to the conclusion smoking was very definitely associated with squamous cell cancer. But Platz said the type of person using that hospital was not typical of the population as a whole. The hospital was an army veterans hospital. Many of the people who died there may have been subjected to poison gas in WW1, also, lots of them were poor and undernourished and suffering from a variety of illnesses. The authors of the study had not allowed for these ‘confounders’. He did not agree with their conclusion that smoking was ‘the cause’ of the cancers.

Judge Nimmo’s notes are very brief concerning the evidence of Platz. Essentially, Platz agreed smoking *could* cause cancer but he did not *know* so.

That concludes the evidence of the expert witnesses. We are now moving towards the end of the case. At this point, it is pretty clear that ASH ET AL were unlikely to be

totally successful. Their evidence was rather flimsy whereas ITL's evidence was substantial. However, as happened in the American case which resulted in the Master Settlement, ASH ET AL did not need total victory. It required only that the Judge find ITL a little bit 'guilty' of negligence for the floodgates of compensation to be opened.

PART 6: CIGARETTE SMOKING, LUNG CANCER AND ADDICTION.

Judge Nimmo began his 'summing-up'.

Causation and the Law.

(6.1) "*Before I discuss the conclusions to be drawn from the foregoing expert evidence, I propose at this stage to discuss counsel's submissions about the concept of causation. This is relevant to three averments for the pursuer: (1) that cigarette smoking can cause lung cancer; (2) that Mr McTear's lung cancer was caused by his smoking; and (3) that his lung cancer was caused by the fault and negligence of ITL.*"

Nimmo then outlined a number of Judgements regarding the subject of causation in general. The cases that he quoted covered many different situations where employees had sued their employers for negligence. We can skip this section mostly.

Submissions for Mrs McTear.

(6.22) *Mr McEachran [counsel for MCTEAR] submitted that it was not necessary for the pursuer to demonstrate that a particular packet of cigarettes gave rise to Mr McTear's lung cancer. It was enough for her to establish that the smoking of John Player cigarettes played a material part in the smoking which caused his death.*

MCTEAR said that the law as stated in *Wardlaw* applied. In *Wardlaw,* a group of workmen sued their employer because they had contracted pneumoconiosis as a result of silica dust which came from grinding machines. The importance of this case was that the judge in the case found that the workmen did not have to prove precisely where the dust came from. It was sufficient that it was in the atmosphere. In the McTear case, the significance is that the same could be said about which brand of tobacco he used.

Submission for ITL.

(6.24) *Mr Jones [counsel for ITL] submitted that the most relevant authority was Wilsher, approved and reinforced by Fairchild. The question, in counsel's submission, came to be: if it was held that smoking could cause lung cancer, did it cause Mr McTear's lung cancer? Or, as it was put in Fairchild, could it be held that but for his smoking Mr McTear would not have developed lung cancer?*

In *Wilsher,* a baby was born prematurely. It was not able to breath properly and so doctors decided to oxygenate its blood directly via a catheter inserted in an artery. Unfortunately, they put the catheter into a vein instead of an artery. The baby survived, but went blind. The problem was that there were several other possible

causes in one so premature. The case went all the way to the House of Lords where it was decided that previous courts had not examined the other possibilities carefully enough. The case had to be re-tried. I don't know what happened subsequently – perhaps the matter was settled out of court.

Nimmo also mentioned another case which was relevant in that it concerned the use of statistics. It was held that statistics alone were insufficient to prove causation.

Judge Nimmo said:

(6.29) *The conclusions I draw from these authorities, as applied to the evidence in the present case, are as follows:*

(1) On the matter of general causation, the burden is on the pursuer to satisfy me, on the balance of probabilities, that as a matter of fact cigarette smoking can cause lung cancer, in the sense that both in the general population and in any individual case it can be said that but for the smoking of cigarettes, lung cancer would probably not have been contracted.

(2) If so, on the matter of individual causation, the burden is also on the pursuer to satisfy me, on the balance of probabilities, that but for Mr McTear's having smoked cigarettes he would probably not have contracted lung cancer.

(3) If so, on the matter of fault causation, the burden is on the pursuer to satisfy me, on the balance of probabilities, that negligence on the part of ITL, in one or other or both of the respects averred in the pursuer's pleadings, caused or materially contributed to Mr McTear's lung cancer either by making at least a material contribution to the exposure which caused his lung cancer or by materially increasing the risk of his contracting lung cancer.

<u>General causation and individual causation</u>.

<u>Submissions for Mrs McTear</u>.

(**6.30 and on)**

Nimmo said that MCTEAR wanted him to accept that *(1) that cigarette smoking could cause lung cancer and (2) that Mr McTear's lung cancer was caused by cigarette smoking.* (6.30)

This section is very long and detailed. Nimmo goes through the evidence given by the expert witnesses for MCTEAR. I shall simply pick out the main points in brief.

Doll quoted the various studies and reports (College of physicians, etc). He claimed that 89% of lung cancers were in smokers. McCarroll (Mr McTear's GP) thought that smoking caused Mr MeTear's lung cancer as she had been told at college and as stated in the health literature. Friend said that he had seen 3,000 people with lung cancer and 89% were smokers (there was a study which said 90%). He thought that the more a person smoked, the more likely he was to develop lung cancer. He claimed massive support from doctors and 'the literature' (IARC etc). Although the exact way

in which cells became cancerous was not known, the epidemiological evidence was strong enough. The few who disagreed were small in number. He also said that the risk of LC in smokers diminished when they quit until, after some 15 years, their risk was the same as non-smokers. He thought that that was very strong evidence.

Doll had explained that members of the working groups were eminent people and that all agreed that smoking caused cancer. The small number of people who disagreed were just eccentric or wrong. ITL suggested that Doll was simply saying 'you must accept my word for it', but MCTEAR said of IARC 1986:

(6.38) "*It is 600 pages-worth. If they want to challenge it, they have to go into every page and see if it is wrong. But this is a report by some twenty-seven doctors from all around the world reaching this conclusion. It is extremely powerful evidence for what they conclude and their evaluation.*"

Kerr had said that 98% of squamous lung cancers were in smokers. Such cancers were rarely seen in non-smokers. *"…it was most unusual to see a squamous cell carcinoma of the lung in a non-smoker"* (6.39). Other 'pre-cancerous' conditions were also seen most frequently in smokers. ITL had accepted the strong association. Smokers were said to be 20 to 40 times more at risk than non-smokers. The agreement of groups of scientists was strong evidence. MCTEAR complained that ITL had not suggested any other cause of Mr McTear's cancer and that they should have if they thought that it might not be smoking.

(6.48) *Counsel referred to USSG 1964 conclusion 1: "Cigarette smoking is causally related to lung cancer in men. The magnitude of the effect of cigarette smoking far outweighs all other factors." It was probable that Mr McTear was one of the 90%, and because he was such a heavy smoker it was more than probable. Accordingly it was established that smoking caused his lung cancer.*

MCTEAR said that it was revealing that ITL had to go to Berlin, Prague and Iowa to find experts to represent them and that these people were not particularly 'eminent'. No evidence was put forward by ITL which suggested any other cause than smoking. MCTEAR pointed to the large fees paid by ITL to its witnesses and suggested that there may therefore be bias, but Judge Nimmo said, "*Counsel was, however, unable to point to any part of Professor Idle's report or of his evidence which displayed bias induced by the fact that he had been paid a fee"* (6.53).

MCTEAR insisted that there had been a massive increase in lung cancer since 1900-ish.

The final para in this section is this:

(6.56) *Mr McEachran [counsel for MCTEAR] said that Dr Cohen was another witness who did not know whether smoking killed. He concluded that laboratory studies using whole cigarette smoke had not produced squamous cell carcinoma of the lung in experimental animals. This was not disputed, but one wondered where this evidence took the defenders when Mr Davis accepted that there was no such thing as a safe cigarette and that smokers were far more likely to develop serious diseases like lung cancer than non-smokers.*

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Submissions for ITL.

(6.57) *Mr Jones [counsel for ITL] said that when he had looked at the whole evidence he would ask me to make no finding on the question whether smoking could cause lung cancer, on the basis that I had not seen sufficient, appropriate, reliable evidence to allow me to justify my making this finding. Ultimately, the pursuer had failed to prove that smoking caused Mr McTear's lung cancer.*

ITL rejected the attacks on its witnesses because of their fees and funding.

Dr James was not asked if smoking *could* cause lung cancer; he was asked if it *did*.

Prof Idle had looked at all the literature of the 20th century plus the most recent about lung cancer and other cancers in relation to smoking. He had mentioned reports from both sides of the debate.

Prof Platz was specifically brought in to counter the arguments of Dr Kerr re the Auerbach studies in the veteran hospital with which he was very familiar. He pointed out the methodological flaws.

Re the MCTEAR witnesses, it was suggested that Prof Friend had been used as a conduit to produce 'evidence' which was not in his field of his expertise. Friend was an anti-smoking advocate and much of what he said was advocacy and not evidence. He was an advocate for ASH. Friend raised doubts about Enstrom and Kabat purely because that study was partially funded by tobacco companies.

[I have read elsewhere that Enstrom and Kabat were not initially funded by tobacco companies. Funding was withdrawn towards the end of the study which was when they turned to tobacco companies to finish the study]

Friend had stated certain propositions, but had not produced evidence for them. Specifically, he mentioned *RCP 1962 and USSG 1964,[which] he had not read.* (6.65).

(6.67) *The statistical evidence, spoken to by Dr Lewis, was that the lifetime risk of developing lung cancer was approximately 1:100 for non-smokers and 10:100 for smokers. The diagnosis of lung cancer in non-smokers on these statistics was therefore unusual and the diagnosis in smokers less unusual, but we learnt nothing from this about what might have caused cancer in an individual smoker or in an individual non-smoker.*

Friend suggested that those persons who were not smokers who got lung cancer could have done so through inhaling tobacco smoke at work or at home, but he produced no evidence. He had said that tobacco smoke contained carcinogens, but, again, had produced no evidence. When pressed, he said that he was not a toxicologist. He claimed that the effect of smoking depended upon how much for how long, but, again, this was not within his area of expertise. *Once again, this evidence from Professor Friend was no more than his ipse dixit. The court had no criteria by which*

to form its own judgment on the question why an individual's consumption of tobacco put him at a greater risk of developing lung cancer than that of a non-smoker or someone who smoked fewer cigarettes. (6.73)

It was suggested that no evidence was brought which said that 'population studies' could be related to individuals. In fact, Doll himself had said that not. Only Friend and McCarroll had said that smoking caused Mr McTear's cancer. They had claimed that Doll's Doctors Study indicated this. But this could not be inferred from the Doctors Study (different lifestyles?). It was said that a person might smoke 20 cigarettes per day and get lung cancer while a person who smokes 50 per day might not. Also, as regards dose relationship, there was a study from central Scotland which showed that, at a certain level of smoking in heavy smokers, the risk did not increase. Further, as regards the diminishing of risk when a smoker quit, Friend had not supported this assertion with evidence. He just said it.

(6.77) *Professor Friend then said that not all smokers would develop lung cancer. It had been estimated that about 16% of smokers who continued to smoke would develop lung cancer before they reached the age of 75 years. Again, this was an estimate. It was not stated whose estimate it was, when it was made, or where it was made. The court was not told about the data on which it purported to be based, or the methodology applied. Again it was just an assertion, unsupported, from a witness who was not qualified to make a pronouncement.*

Friend then said that 89% of lung cancer mortality in men was due to smoking (RCP 2000). ITL made four points about this: 1) that the estimate was not backed up by evidence before the court; 2) that the figure of 89% was just a fraction/number. Friend had misunderstood the nature of 'attributable fractions' – in any particular individual, the 'cause' of that person's lung cancer could be 100% due to smoking, or 100% due to genes, or 100% due to alcohol, etc; 3) the 89% fraction came from the Callum study – Callum himself said that the fraction could not be related to individuals; 4) it was for MCTEAR to exclude other risks, which they had not done. One specific was heredity. His mother had died from lung cancer as had a half-sister and a half-brother (his father's children by another woman). Nor had other risks been excluded – those relating to his lifestyle. (6.82)

(6.83) *In addition to not having read RCP 1962 or USSG 1964, Professor Friend had not read USSG 1988. He said earlier in his evidence that he had read the expert reports produced on behalf of ITL, including Professor Idle's report, but it transpired in cross-examination that he had not read it. He was also unable to provide references for any of the data on which he relied in his own report, and he had forgotten the results of his own studies on smoking cessation, which came to the opposite conclusion from the proposition that the heavier the smoking, the more difficult it was for the smoker to stop smoking.*

ITL then moved on to Prof Doll.

Prof Doll was convinced that smoking causes cancer and other diseases. His approach was general. He did not relate his evidence to Mr McTear. He dismissed anyone who disagreed with him *as being an eccentric, corrupt, or other descriptions.* (6.86) Doll

also gave the court an 'ipse dixit' (He himself says'). He brought forward no specific data for the court to examine.

(6.89) *While the fact that Sir Richard's views had come to be generally accepted by a majority of those working in the field might have some weight, nevertheless, without knowing on what the views were based and why they came to be generally accepted, it was not possible to say whether or not they were right, despite the very great eminence of Sir Richard.*

I think that there is an important inference in that sentence. Why did physicians generally accept that it was true that smoking caused lung cancer? Did they personally peruse the evidence, or did they just accept the word of 'the eminence'? In fact, Nimmo said: *"This did not take me anywhere, counsel submitted, when I had to address that question as an issue in this litigation. In other words, I did not know on what basis this consensus had been reached*" (6.89).

(6.90) suggests that the College of Physicians' publications were for the benefit of Parliament and not intended to be 'scientific'. They were for public health purposes and not 'evidence' as they had been projected to be by MCTEAR in this lawsuit. Doll's data should have been produced, but it was not. Doll was insistent that his view were correct. He dismissed the studies and opinions of *Fisher, Berkson, Seltzer, Yerushalmy, Eysenck, Burch, Oldham, Stern, Gwynne Jones, Feinstein, Passey, Little, Tokuhata, Hueper and Rosenblatt.* (6.94).

Judge Nimmo was invited by ITL to reject Doll's evidence more or less completely.

Dr Kerr's evidence was next.

Kerr relied upon his own observations that 98% of squamous cell cancers were in smokers, even though he mostly did not know whether the patients were smokers or not. He also relied upon certain studies, but accepted the figures uncritically.

In the paras which followed, ITL went into some detail about the epidemiological studies. There seem to have been numerous faults.

Here is a para worth quoting:

(6.104) *Dr Kerr agreed that in Auerbach et al. 1975 the study comprised 662 autopsies of men, of whom six were non-smokers. In counsel's submission, since 99% of the population studied were smokers, one could not take anything as being significant from the fact that 100% of the squamous cell carcinomas were found in smokers. This again was to do with populations. The authors of the paper did not claim that what they found was statistically significant. If the purpose of referring to Auerbach et al. 1975 was to support the figure of 98% in Colby et al. 1995, then it did not give support.*

There follows then statements from ITL to the effect that much of the evidence for MCTEAR was unsubstantiated as required for the court to make a decision. There was also criticism of MCTEAR's expert witnesses in that they seemed not to have complied with the 'rules' regarding expert witnesses. There was much criticism of

reports from IARC and RSP etc. Their reports had purposes which did not require the standards of proof required by a court of law- they were public health documents.

(6.123) *Perhaps one of the most representative criticisms of the public health reports was put to Professor Friend, and he was not able to refute it. This was the view expressed in Sommers 1976 that USSG 1964 dealt with the difficulty of assigning causality, but the summary and conclusions brushed these aside and assigned a causality not demonstrably evident in the text.*

In (6.126), ITL suggested that caution was especially required where the number of words and the number of supporters of those words was intended to impress the court. The court could not make its decisions on the basis of 'a majority vote'.

Continuing, ITL pointed out the contributions from its own expert witnesses, which I have outlined above in some detail.

(6.140) ITL introduced a consideration in the evidence of Lewis:

(6.140) *Finally, Dr Lewis gave evidence about the relationship that might be expected between the incidence of lung cancer and a decrease in the prevalence of smoking. In the MRFIT (see para.[5.845]) no difference had been found in lung cancer mortality, or in overall mortality, when a comparison was made between the "special intervention" and the "usual care" groups. At least, he said, this showed that the attributable fraction calculation was not valid.*

Put simply, one group of lung cancer patients was given 'special intervention' while another was given 'usual care'. Both groups experienced the same level of mortality. Although not clearly stated, it seems that the 'special intervention' group stopped smoking, but the mortality rate did not 'improve'. It is not stated for what period of time this 'special intervention' existed. Assuming that the period of time was many years, then, in theory, the 89% 'attributable fraction' should have diminished, year-on-year, in the 'special intervention' group, but it did not. There are repercussions, but this is not the place or time to go into them.

There is a final submission by ITL, in response to a question from Judge Nimmo. Nimmo's question was, "How shall I proceed if the evidence is weak from both sides?" He quotes the well-known saying from Sherlock Holmes: "If every other possibility is found to be wrong, then the remaining possibility, no matter how unlikely, must be correct". Are there any logical faults in that reasoning from a legal point of view? There are three:

1) A judge can properly say that none of the possibilities have been sufficiently proven to be correct.

2) Every other and all other possibilities must have been eliminated.

3) The use of 'common sense' dictates that, if an event is extremely unlikely, the fact that it is less unlikely than another event is neither here nor there. It is still extremely unlikely.

Therefore, as regards ‘liability’, MCTEAR must ‘prove’ that there is liability. Nothing less is acceptable. (6.148).

Discussion (1) General causation.

Judge Nimmo starts to describe his own ‘Opinion’.

(6.149) *I propose to say at this point only a brief word about the expert witnesses. The demeanour of only two of them requires comment. Sir Richard Doll (who, of all the expert witnesses, was the only one not to avail himself of my invitation to be seated while giving evidence), made clear by his demeanour as well as the content of his evidence with what disdain he regarded those individuals who disagreed with his conclusion that the causal connection between cigarette smoking and lung cancer was proved.*

Professor Hastings, who regarded himself as an advocate for greater measures of tobacco control, carried this into his courtroom manner and his tendency to argue with counsel rather than to answer questions. I am bound to say that none of Professor Friend, Sir Richard Doll and Professor Hastings seemed to me to be mindful of the need to be independent (see para.[5.18]), and each appeared to me to engage in advocacy to a greater or lesser extent. Beyond these comments, my impression of all of the expert witnesses was that they gave evidence in a manner appropriate to their professional standing and the context of the proof. I propose therefore to concentrate principally on the content of the expert evidence, to the extent necessary to explain the views set out in the paragraphs which follow.

Thus spake Judge Nimmo.

Note that, from now on, it is Judge Nimmo who is speaking directly.

MCTEAR relied upon the epidemiological evidence. There was no other evidence. Animal experiments had proved to be useless. Very generally, there was no physical evidence that smoking ‘causes’ lung cancer.

In 6.152, Nimmo says that Prof Idle showed that, apart from epidemiology, no branch of science showed that smoking ‘caused’ lung cancer. Enormous effort and expense on animal research had not shown ‘causation’ in respect of smoking and lung cancer.

6.153. Cohen’s evidence. A particular study said that squamous lung cancer had been induced in animals. This turned out to be false. There was no other such evidence.

6.154. Animal experiments do not necessarily transfer to humans, *but* there would be no point in the expenditure of time and effort and money if it was not expected that there would be similarities. The absence of confirmation from animal research means that only the epidemiological evidence that smoking causes lung cancer exists. It follows, therefore, that the epidemiological evidence must be ‘foolproof’.

6.155. In order to accept the epidemiological evidence that ‘smoking causes lung cancer’, there must be little doubt. The evidence must be very, very strong. It must convince the Judge.

Nimmo goes on to describe his thinking about the difference between epidemiology as a study of factual things (such as the incidence of lung cancer) and epidemiology as a study of possibilities (although he does not put it in that way). The later is what 'public health' relies upon. The first gives rise to 'relative risks' which are minuscule in a large population. The second emphasises the difference, which might be large, but is, nevertheless, still minuscule (although he does not put it that way). (6.159) states much the same thing. Essentially, if 1 person in a million is at risk, multiplying that risk by 100 means that the risk is still minuscule for any particular individual. 100 in a million means 1 in 10,000, which is unutterably minuscule – imagine a red table tennis ball in a huge box also containing 9,999 white balls. What is the chance of you dipping your hand into the box and selecting the red ball? Remember also that every time that you 'dip in', the chance of selecting the red ball is exactly the same as the previous time – 1 in 10,000. The Holy Zealots emphasise the 'relative risk' and ignore the 'absolute risk'.

Here is an awfully difficult para to understand:

(6.159) *The concept of relative risk requires discussion. This, as I understand it, is used to compare the incidence of a disease or condition in a group with a particular exposure to those without it. It is thus related to the concept of association, and is neutral: it does not connote that a causal connection is established. The use of the word "risk" in epidemiology appears to have led to some misunderstanding on the part of those unfamiliar with the terminology of this discipline. In ordinary language, "risk" in its primary sense refers to exposure to a hazard or danger, and carries the connotation of a potential causal connection between the risk and the subsequent misfortune or loss, should that eventuate. This has, in my view, led to a serious misunderstanding on the part of Professor Friend, who seems to have regarded the relative risk derived from a comparison of the incidence of lung cancer in smokers and non-smokers, which is such as to yield a positive association between the exposure and the disease, as connoting the establishment of a causal connection between the two. This is not so, though the relative risk may be of a magnitude such that a positive association may be judged to be strong enough for a causal connection is established.*

A tricky para to understand, yes? But the meaning is actually simple! If, in the whole population, there is a minuscule risk, it does not become much greater if it is multiplied by 10 or 100 – it is still minuscule. As applied to the development of lung cancer, the risk (via smoking) is so small in any particular individual that other factors, such as heredity, become just as important, if not more so.

Nimmo now explains what is meant by 'the literature' re epidemiology. 'Primary lit' is the 'archival data' – it includes the numbers, the statistical methods, the confidence interval, etc. This needs to be transparent so that other may scrutinise the material and replicate it.

(6.160) *A good example of this process may be found in Dr Lewis's discussion of Doll et al. 1994 at paras.[5.832] to [5.838], including the statement at para.[5.834] that he found the paper difficult to understand because he did not see the numbers behind it.*

The ‘Secondary lit’ is meta-studies which accept the validity of the primary lit and accumulate the information. Essentially, it arrives at ‘an average over all individual studies’. The ‘tertiary lit’ is that which uses the secondary lit to inform. In that category fall reports of the College of Physicians, IARC, USSG, and so on.

The next para is an important one. I quote in full:

(6.162) *I can say with confidence that no evidence was led about the primary literature which was sufficient to impart to me special knowledge of the relevant subject-matter and to enable me to form my own judgment about it and the conclusions to be drawn from it. This could have been done: it is clear that the survey of British doctors, on which Sir Richard Doll and colleagues have worked for many years, is regarded as a classic of its kind, both because of the pioneering nature of the research, a preliminary report of which was published as Doll and Hill 1950, and because this has been followed up with subsequent papers over several decades. I could at least have been shown these papers, which I assume disclosed the data, the statistical techniques and all the other considerations which led to the authors’ conclusions, so that I could see for myself whether these conclusions were soundly based. The opportunity was there, with Sir Richard Doll in the witness box, and indeed Professor Friend for one thought that evidence would be given about this survey. Warning had been given on behalf of ITL, as early as the specification of documents referred to at para.[1.24], that Sir Richard Doll’s data were of potential interest to the court. But in the event no attempt was made to show me the data.*

Nimmo criticises MCTEAR for not showing him the primary literature, especially the British Doctors Study. Doll had the opportunity to explain why those who disagreed with his conclusions but did not do so. *I can find little in these passages beyond assertions that those who disagreed with Sir Richard were wrong, coupled from time to time with epithets which I quite frankly found it unbecoming for a man of his stature to have chosen to use. If Sir Richard succeeded in winning over any of those whom he had previously failed to persuade, it cannot have been with these words.* (6.164)

(6.166) *Mr McEachran’s (counsel for McTear) main argument, as I understood it, was that the conclusion that cigarette smoking could cause lung cancer had met with general acceptance in the scientific community by the late 1950s, was accepted by the media in the 1970s, was taught at medical schools and reflected in textbooks…*

Nimmo says: *This is all very well, but I have to say that I am reminded of the Bellman in Lewis Carroll’s The Hunting of the Snark, who said: “What I tell you three times is true”*.

He goes on to say that, despite the vast amount of primary literature, he was shown none of it. He was expected to make his decision on the basis of ‘oracular pronouncements’. He said that Prof Friend was not qualified to help him re the epidemiology and that Doll had missed the opportunity. *…no doubt it was because of this that the decision was taken by Mr Jones not to ask Dr Lewis, when in due course he gave evidence, about the issue of general causation: there was by then no case for ITL to answer* (6.167). He next states that there was uncertainty that there was a huge increase in lung cancers between 1900 and 1950. Again, a lack of evidence and the

various changes in medical practice during that period. The putative increase was an important plank in MCTEAR's case in that it could be said to indicate 'general causation'. He criticises the tertiary literature for accepting the primary and secondary literature without scrutiny.

(6.170) For all these reasons, and in addition for the reasons advanced in detail by Mr Jones, which I accept, in my opinion the pursuer has failed to discharge the burden of proving, in accordance with the requirements of the law of evidence relating to expert witnesses, that cigarette smoking can cause lung cancer. I am forced to say, following Lord Brandon in Rhesa Shipping Co S.A. v Edmunds, referred to at paras.[6.147] to [6.148], that the state of the evidence is such that I simply cannot decide one way or the other whether cigarette smoking can cause lung cancer. Since the burden of proving this rests on the pursuer, she has failed to discharge this burden, and accordingly this branch of her case fails.

(6.171) I conclude this passage by emphasising that I am in no way finding that cigarette smoking cannot or does not cause lung cancer: I am simply saying that, approaching the evidence with an open mind, as I am bound to do, and applying the law relating to expert evidence, I am unable to find it proved that cigarette smoking can cause lung cancer.

It is very important to note that Judge Nimmo is not only saying that the evidence is not strong enough to 'prove' that smoking *does* cause lung cancer, he is also saying that it is not strong enough even to 'prove' that smoking *could* cause cancer.

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(2) Individual Causation.

Did smoking cause the lung cancer of the specific individual, Mr McTear?

(6.172) *"I turn now to the question of individual causation, that is to say whether it is proved on the balance of probabilities that cigarette smoking caused Mr McTear's lung cancer, in the sense that but for his smoking of cigarettes he would not have contracted it."*

One would assume that, since 'general causation' had failed to be proved, there would be no point in examining 'individual causation', but Nimmo is required to assume that general causation may have been successful. I think that this is a matter of being thorough, so that this case becomes a definitive one.

Essentially, the question is: is it acceptable to argue that, because 90% of lung cancer is, *in general,* caused by smoking, then Mr McTear's cancer was caused by his smoking.

A point raised by Nimmo was that it is not possible to look at a lung cancer and point to some specific element of that cancer and say that that element proves that the cancer was caused by tobacco smoke. There is no 'residue' which could be observed.

In the next section, Nimmo explains why it is that epidemiological 'proportions' cannot be applied to individuals. Even a 90% probability does not exclude the other 10%. Mr McTear could easily be in the other 10% for reasons associated with his family history, his lifestyle, and so on – each element of which could be 100% the cause of his cancer.

Here is a critical legal point:

I do not agree with Mr McEachran's submission that it was for ITL to prove that Mr McTear's lung cancer was due to some cause other than cigarette smoking. The burden of proving individual causation is on the pursuer, and in assessing the evidence of Professor Friend I am entitled to have regard to the extent to which he considered and, if so, found reasons for discounting other possible causes. (6.181)

Finally:

(6.185) *In the result, I am not in a position to hold, and do not purport to hold, that Mr McTear's lung cancer was not caused by his cigarette smoking. But I am not satisfied, on the basis of the evidence led before me, that it has been proved that it probably was caused by his smoking, in the sense that it is more likely than not that but for his smoking he would not have contracted lung cancer. The pursuer fails on this branch of the case also.*

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Addiction.

(6.186*) I turn now to consider the averment for the pursuer that:*

"Tobacco is addictive in the sense that once individuals such as [Mr McTear] have started smoking it is difficult for them to wean themselves off the habit. It is more addictive than cocaine." (Closed Record p.22D-E)

*This is supported by a reference in the pursuer's pleadings to USSG 1988. ITL deny these averments and aver that smoking is correctly regarded as a habit and not an addiction. They go on to aver that people choose to smoke for a variety of reasons, that smokers derive benefits from smoking**, that over the years many millions of smokers have stopped smoking through choice**, and that while some smokers may find it difficult to stop smoking, smokers who choose to stop smoking are able to do so.* [My bold]

Fatal flaw?

Submissions for Mrs McTear.

MCTEAR had said previously that it was nicotine which caused the craving. The evidence of Friend and Hastings was most pertinent. Mr McTear might have had particular difficulty in quitting. Advertising played an important part in continuing dependence. MCTEAR said that Gray [for ITL] had admitted that smoking was addictive in the common sense of the word. Although Gray disputed that nicotine was

the addictive substance, he was ‘a tobacco company man’ and was one of only a very few who said that.

Submissions for ITL.

(6.194) *Mr Jones [for ITL] submitted that it was quite inappropriate on the evidence to seek to dismiss Professor Gray’s evidence, as Mr McEachran had done, by calling him a “tobacco man”. He was the only witness who was qualified to give opinion evidence on the question of addiction. He was in fact a distinguished Professor of Psychology from a leading academic institution and had researched and published extensively on a range of subjects to do with the brain and its relationship to behaviour.*

ITL said that it was wrong to dismiss Gray’s evidence because he received some funding from tobacco companies at a time when it was common and accepted to do so. One research project which was funded by tobacco money was in connection with Alzheimer’s disease. Gray was supported by others (Warburton 1988a and 1988b, and Frenk and Dar 2000).

(6.194) *In counsel’s submission, what was important was not the “head count”, but the cogency of Professor Gray’s reasoning for his conclusions, which was not effectively challenged either in evidence or in submission.*

(6.196) *Mr Jones went on to submit that ITL’s averments were proved. Mr McTear stopped smoking. Mrs McTear stopped smoking. Sir Richard Doll, having been a smoker for twenty years, stopped smoking “without difficulty”. USSG 1988 at p.466 stated:*

“[T]here are approximately 41 million former smokers in the United States. Approximately 90 percent of former smokers report that they quit smoking without formal treatment programs or smoking cessation devices [...].”

USSG [Surgeon Gen] 1988 quoted the authority of Henningfield 1984 and others to claim that nicotine was similar to drugs like heroin and cocaine in providing a euphoric effect. Prof Gray had criticised the methodology used in those studies, referring to Warburton 1988a. *Another aspect of this was that Henningfield was one of the scientific editors of USSG 1988 and he was a contributor to RCP 2000. RCP 2000 was also criticised in relation to its use of statistics on quitting.* (6.198)

ITL went on to submit that, in effect, MCTEAR were simply stating that ‘some people found it difficult to stop smoking’, which was not disputed by ITL. No actual evidence had been produced to support the contention that *“tobacco is more addictive than cocaine”* (6.201)

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Discussion.

Nimmo said straight away that there was no evidence that tobacco is more addictive than cocaine. He declined to accept Prof Friend's evidence since it was outside his area of expertise.

(6.203) *By contrast, Professor Gray appeared to me to be well qualified to give evidence about addiction, and I found his evidence to be persuasive. I reject the criticism of him that he was a "tobacco man". I detected no bias in his evidence, which appeared to me to be presented with moderation, internal coherence and appropriate reference to authority. Rose et al. 2000 gave support for his view that, as far as the pleasure and satisfaction of smoking were concerned, the principal route did not involve nicotine, but lay somewhere else in the complex behaviour that was smoking a cigarette. Support for the view that smoking had an intrinsic anti-depressant action was found in Kendler et al. 1993, and there was laboratory evidence that nicotine could have the effect of reducing anxiety. Accordingly Professor Gray's overview that nicotine and smoking tobacco had a remarkably broad spectrum of activity in alleviating negative mood and emotion, including depression, irritability and anxiety was supported by the literature. This justified the functional view which he favoured.*

Nimmo particularly pointed to the 'errors' in Henningfield 1984. In that study, the effects of various drugs were plotted (on a graph?) **but the graphs were not on the same scale**. When plotted correctly, nicotine was, at best, only a weak euphoriant. The contrast between withdrawal symptoms of heroin and nicotine was also noted. It was not possible to say why some people had difficulty in quitting while others did not. It was noted that people in lower socio/economic groups (like Mr McTear) smoked more and found it hard to quit.

Finally, on addiction:

(6.208) *The evidence of Professor Hastings appears to me to add nothing for present purposes. The fact that individuals may be exposed to advertising, and even influenced by it, does not mean that they are precluded from exercising a free choice. Advertising simply adds to the complexity of the individual's decision-making process. People are well-accustomed to weighing up mixed messages, and to making their own decisions and choices. In any event Professor Hastings himself disclaimed any such mechanistic account of the influences to which an individual such as Mr McTear might be exposed. For an individual to say that he has found difficulty in altering or giving up a habit, as Mr McTear did of his smoking, because he is "addicted", appears to me to be little more than an attempt to absolve himself of individual responsibility for his own decisions and choices. In my view a smoker such as Mr McTear makes a deliberate choice as to whether to start smoking, whether to continue smoking or to stop smoking, and indeed whether or not to smoke a cigarette on any particular occasion. The fact that smokers such as Mr McTear may find it difficult to give up does not appear to me to deprive them of the element of free will which is fundamental to the individualist philosophy of the common law.*

The comments there about the effects of advertising also have relevance today to display bans and plain packaging.

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PART 7: LIABILITY.

Submission for Mrs McTear.

Despite the failure of causation, Nimmo was obliged to consider liability as if McTear had succeeded.

MCTEAR led the judge through a whole series of court decisions regarding the liability for harm as regards manufactured goods and other things. Since this section is concerned with 'legal niceties', we need not trouble ourselves with it. MCTEAR claimed that as soon as it was discovered that smoking seriously damaged health, tobacco companies should have stopped making cigarettes. Manufacturers of other products had done so. Had ITL stopped in 1957, when the danger was discovered, Mr McTear could not have started smoking and might not have got lung cancer.

Submission for ITL.

Again, lawsuits were cited which ITL suggested were the ones which should apply in this case. No need for us to go through them. We should be thankful for that since this section is very long. I read them all, which is why I can say that we can skip them.

We resume at (7.133).

MCTEAR had suggested that a cigarette was 'a defective product'. ITL said that not – there was nothing defective about a cigarette. It was just a cigarette. A cigarette could be compared with, for example, fatty food. ITL suggested that it was for individuals to decide whether or not they wanted to take the risk, if any. It was not for the court to decide whether or not a product should be banned. In any case, the science in 1964 (when Mr McTear started smoking) was far from clear and disputed by many.

(7.134) *Cigarettes were at all relevant times widely enjoyed and socially acceptable. In the 1960s, the majority of adults in the United Kingdom smoked. USSG 1964, referred to in Professor Friend's evidence, discussed the significant beneficial effects of smoking. Professor Gray described the benefits which smokers might derive from smoking: most smokers reported that they enjoyed smoking. Mr McTear's evidence, given on commission, was that this was why he smoked. Smokers described how they smoked for one or more of a range of beneficial effects related to sensory enjoyment, mood control and increasing alertness. In MacAskill et al. 2002, of which Professor Hastings was one of the authors, it was found that smoking was, for many of the subjects of the study, their main pleasure, and met a multitude of needs in the lives of the study population.*

(7.136) *The attitude of the Government and the public health authorities was relevant to the issue of reasonableness [re not stopping making cigs] in not withdrawing the product from the market and not issuing a warning. There was no suggestion from the Government that smoking should be banned or that cigarettes should be withdrawn from sale. There was no evidence that the Royal College of Physicians were recommending prohibition of the sale of cigarettes.*

(7.137) *A further consideration which was relevant, counsel submitted, was that ITL were, and remained, a substantial company upon which many people relied for their livelihood. Given that the Government then and now regarded smoking as a legitimate choice for individuals to make, it was not incumbent on ITL in all the circumstances unilaterally in effect to cease trading.*

ITL went on to say that the risk in smoking was widely known having been heavily promoted by Government and others. The TV advertising ban came into effect in 1965 and was heavily publicised. ITL went on to describe in some detail newspaper articles about smoking and health. As regards advertising and promotion, ITL said that it was hard to see what the court was being asked to decide. Tobacco was a lawful product and promotion took place in the normal way. Government could have banned it but didn't. The public were aware of the risks and therefore there was no duty for tobacco companies to write warnings on the packets – there was no 'hidden' danger. In any case: did ITL have a 'duty of care' to Mr McTear personally? ITL cited a case which has interesting implications:

(7.154) *Counsel referred to McWilliams v Sir William Arrol & Co 1962 S.C. (H.L.) 70. In this case, a workman was killed when he fell a distance of seventy feet from a steel lattice tower which was in the course of erection at a shipyard. After evidence had been led, it was found proved that both his employers, who were independent contractors in charge of the operation of erecting the tower, and the occupiers of the shipyard were in breach of their respective duties to provide a safety belt. It was also found proved that, even if a safety belt had been provided, the deceased would not have worn one.*

That means that, although the employer was negligent in not providing a safety belt, the lack of safety belt was not the reason that the workman fell. It was that he was not wearing one, and that he would not have worn one if one had been provided. That is, the negligence did not bring about the injury. As regards Mr McTear, it is very unlikely that he would have taken notice of warning messages on cig packs had they been there.

(7.159) *Counsel submitted that the next question for consideration was whether it was proved that Mr McTear smoked a sufficient quantity of ITL's products to have caused or materially contributed to his lung cancer. The starting point for consideration of this question was whether Mrs McTear had proved how much of ITL's products Mr McTear consumed.*

This was quickly dealt with by the lack of evidence regarding which products he smoked and the lack evidence re causation.

ITL then considered the matter of addiction. They suggested that Mr McTear could have stopped smoking had he wanted to – personal responsibility. In fact, when told that he had lung cancer, he did stop. It was suggested that his attempts in the past to quit had been 'half-hearted'.

Finally, on 'personal responsibility':

(7.166) *In answer to a question by me, Mr Jones submitted that there was no distinction between the civil law and the criminal law where it came to the general proposition that individuals were responsible for their own actions. Counsel referred to Galbraith v HM Advocate (No.2) 2002 J.C. 1, in which the law of diminished responsibility was reconsidered by a court of five judges, of whom I was one. In delivering the Opinion of the Court, the Lord Justice-General (Lord Rodger of Earlsferry), said at p.17, para.44:*

"It is, of course, impossible to attempt to describe the ambit of the doctrine of diminished responsibility without even attempting to describe the operation of the doctrine itself. A common theme in the cases where judges have left the issue to the jury is that they involve some abnormality of the accused's mind. While philosophers continue to debate the timeless questions – about the nature of the relationship between mind and body and about the extent to which individuals have control over their actions – our law proceeds on the basis that an adult person of sound mind has sufficient control over his acts, and over his omissions to act, as to be responsible for them in law. Criminal acts and omissions are punished accordingly."

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Discussion (1): Negligence.

MCTEAR had claimed that Mr McTear's lung cancer occurred because he was not warned about the dangers. But they did not claim 'negligence' on these grounds. Nimmo said that this was not surprising, *given the state of the evidence* (7.168).

MCTEAR claimed negligence because ITL did not stop making cigarettes.

(7.172) *Assuming for present purposes that cigarette smoking can cause lung cancer, and that tobacco can therefore be described as a dangerous product, I can find no support in the authorities for the proposition that as soon as they became aware of this ITL had a duty effectively to cease manufacture. They could only have had such a duty if the law held that a manufacturer must ensure the safety of the consumer. The cases do not support this approach. In Holmes v Ashford, referred to at paras.[7.69] to [7.70], the Court of Appeal went no further than to hold that every person who put on the market a dangerous article must take reasonable steps in all the circumstances.*

Nimmo cited a number of cases to illustrate the point in ensuing paras, but the fate of MCTEAR's assertion was effectively sealed there.

(7.177*) I reject the insidious suggestion which, if not expressly advanced, was at least implicit in aspects of the pursuer's case based on the evidence of Professor Hastings, to the effect that because he was in a lower socio-economic class Mr McTear was somehow to be regarded as more a victim of circumstances and as having less than full responsibility for his own choices and actions.*

I like that quote. It is a complete rejection of the Nanny State, ASH ET AL, and the rest. A person takes his own decisions and is responsible for them.

Finally:

(7.178) *The policy of the law in a society such as ours seems to me to be entirely clear. Adults of full age and not suffering from legal incapacity are equal in the eyes of the law. They have equal rights and duties, freedoms and responsibilities. Each of them is presumed to be reasonable, and to have the responsibility of making reasonable choices, not least in matters affecting his or her safety, health and welfare. This approach is fundamental to the workings of our society. It is reflected, for example, in the equality of individuals as members of the electorate, and in the approach of the criminal law, which is to treat all individuals as having full criminal responsibility in the absence of special circumstances (see Galbraith v HM Advocate (No.2), referred to at para.[7.166]). Individuals are assumed to be reasonably well informed and reasonably responsible members of society.*

(7.181) *For these reasons in my opinion there was no lack of reasonable care on the part of ITL at any point at which Mr McTear consumed their products, and the pursuer's negligence case would accordingly fail.*

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2) Fault causation.

Mr McTear knew about the risks. Written warnings would not have stopped him from starting smoking when he did in 1964, nor did they stop him when they did appear in 1971. If ITL had ceased manufacturing cigs, there was no evidence that he would not have used another company's products. Even if the Government had banned tobacco, it could still have been brought in from abroad. The Government would have had to employ massive force to stop it. *Attempts at prohibition of substances which people enjoy using and strongly desire to use are notoriously ineffective.* (7.183)

"Volenti non fit injuria"

Which means that a person who 'voluntarily' acts cannot claim 'injury'.

We discussed this matter in the early part of this post. The judge said that, had McTear proved injury, then he (Nimmo) would not have allowed ITL to claim that Mr McTear brought the harm upon himself.

(7.208) *For these reasons I prefer to take the view that, as can be seen from my summary of counsel's submissions, the issues I have been asked to consider under this heading are the same as those which arise and of which I have already taken account in the context of the question whether ITL were negligent; and if I had found that they were, I would not have been disposed to sustain the defenders' fourth plea-in-law.*

We can therefore skip the whole of this section which is a detailed discussion of the law relating to "Volenti non fit injuria"

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PART 8: DAMAGES.

Nimmo describes the law relating to damages. He says what damages he *would have* awarded had McTear succeeded. This matter is not relevant to us. Nevertheless, just for the sake of 'the record':

(8.23) *My assessment of damages, with interest to 31 May 2005, is therefore as follows:*

Section 1(4) claim	£25,000.00
Interest thereon	9,141.78
Section 2(1) claim for solatium	45,000.00
Interest thereon	43,880.55
Section 8 claim for services	8,000.00
Interest thereon	7,800.99_____
Total damages	**£138,823.32**

PART 9: CONCLUSION AND RESULT.

This section is quite brief. I quote it all. I hope that the analysis above will help everyone to understand clearly what Lord Nimmo Smith is saying:

Conclusions

[9.1] I now set out my main conclusions, which should be read in conjunction with the passages of discussion to which cross-references are given.

[9.2] It is not in dispute that Mr McTear died of lung cancer (para.[1.4]). I accept that he smoked the John Player brand or brands of cigarettes manufactured by ITL for many years, as part of his consumption of cigarettes. I am not, however, prepared to hold it proved that it was ITL's products that Mr McTear smoked at any time prior to 1971. I do not accept that he smoked John Player brand cigarettes exclusively from the early 1970s onwards until the last few years of his life. I conclude that he smoked a significant quantity of roll-ups made from Old Holborn tobacco along with his smoking of John Player brand cigarettes for many years, perhaps as many as twenty years, but I am not able to decide in what proportion he divided his smoking between John Player brand cigarettes and roll-ups. They both made a material contribution to his total consumption from about 1971 onwards (para.[4.228]).

[9.3] Mr McTear started smoking no earlier than 1964. I am satisfied that advertising had nothing to do with his reasons for starting to smoke. He started smoking because it was socially acceptable and most young people started smoking as part of becoming adults (para.[4.226]). I am prepared to accept that Mr McTear found it difficult to wean himself off his habit once he had started smoking and in that sense could be described as addicted. I do not accept that he was for this reason unable to stop smoking (paras.[4.229] and [6.202] to [6.208]). The averment that tobacco is more addictive than cocaine is not proved.

[9.4] I am satisfied that at all material times, and in particular by 1964, the general public in the United Kingdom, including smokers and potential smokers, were well aware of the health risks associated with smoking, and in particular of the view that smoking could cause lung cancer (para.[3.1] and Part III generally). I am also

satisfied that Mr McTear was aware, in common with the general public, well before 1971 of the publicity about the health risks associated with smoking, and in particular the risk of lung cancer. Therefore by the time he is shown by acceptable evidence to have started smoking the John Player brand of cigarettes he was already aware of the publicity about the health risks. As with many other aspects of his life, he chose to ignore it (para.[4.230]).

[9.5] The pursuer can succeed in this case only if she proves all of the following (paras.[1.5] and [6.29]):

(1) That cigarette smoking can cause lung cancer, in the sense that both in the general population and in any individual case it can be said that but for the smoking of cigarettes lung cancer would probably not have been contracted (general causation).

(2) That cigarette smoking caused Mr McTear's lung cancer, in the sense that but for his having smoked cigarettes he would probably not have contracted lung cancer (individual causation).

(3) That Mr McTear smoked cigarettes manufactured by ITL for long enough and in sufficient quantity for his smoking of their products to have caused or materially contributed to the development of his lung cancer.

(4) That Mr McTear smoked cigarettes manufactured by ITL because ITL were in breach of a duty of care owed by them to him.

(5) That such breach caused or materially contributed to Mr McTear's lung cancer either by making at least a material contribution to the exposure which caused his lung cancer or by materially increasing the risk of his contracting lung cancer (fault causation).

[9.6] There is no direct evidence that ITL, as a company, have ever accepted that there was a causal connection between smoking and disease, and the evidence before me does not satisfy me that this is the inference which should be drawn (para.[2.76]). The fact that they have never sought to challenge the public health message, that cigarette smoking does cause lung cancer, does not in my opinion constitute such an admission (para.[2.78]). Accordingly, in my opinion, ITL are entitled to put the pursuer to proof of her averment that cigarette smoking can cause lung cancer (para.[2.80]).

[9.7] I must base my decisions about questions of fact on the evidence, and that alone (para.[1.8]). It is not open to me to take account of any passage in any document, the terms of which were not agreed, and to which reference was not made in the course of the evidence of any witness (para.[1.37]). It is not within judicial knowledge that cigarette smoking can cause lung cancer: this is an issue which I am duty-bound to approach with an open mind and to decide on the basis of the evidence led before me; and the burden of proving it is on the pursuer (para.[1.12]).

[9.8] The law relating to expert witnesses is as discussed at para.[5.17]. Above all, the purpose of leading the evidence of any expert witness should have been to impart

to me special knowledge of the subject-matter, including published material, lying within the witness's field of expertise, so as to enable me to form my own judgment about that subject-matter and the conclusions to be drawn from it.

[9.9] The pursuer relies on epidemiology to prove general causation. I have not been sufficiently instructed by the expert evidence relating to this discipline to be able to form my own judgment as to whether or not this averment is proved. Special knowledge of this subject-matter was not imparted to me, so as to enable me to form my own judgment about it. The pursuer has accordingly failed to prove this averment (paras.[6.149] to [6.171]).

[9.10] In any event, the pursuer has failed to prove individual causation. Epidemiology cannot be used to establish causation in any individual case, and the use of statistics applicable to the general population to determine the likelihood of causation in an individual is fallacious. Given that there are possible causes of lung cancer other than cigarette smoking, and given that lung cancer can occur in a non-smoker, it is not possible to determine in any individual case whether but for an individual's cigarette smoking he probably would not have contracted lung cancer (paras.[6.172] to [6.185]).

[9.11] In any event there was no lack of reasonable care on the part of ITL at any point at which Mr McTear consumed their products, and the pursuer's negligence case fails. There is no breach of a duty of care on the part of a manufacturer, if a consumer of the manufacturer's product is harmed by the product, but the consumer knew of the product's potential for causing harm prior to consumption of it. The individual is well enough served if he is given such information as a normally intelligent person would include in his assessment of how he wishes to conduct his life, thus putting him in the position of making an informed choice (paras.[7.167] to [7.181]).

[9.12] In any event, there is no basis upon which I could hold it established that, if ITL had not manufactured cigarettes at any material time, so that Mr McTear did not smoke their products and accordingly their products could not have made a material contribution to his contracting lung cancer, it would have made any difference. On the contrary, all the evidence is that Mr McTear would have started smoking when he did, and would have continued to smoke, for the same length of time and in the same quantities, as he in fact did. Fault causation would therefore not in any event be established (paras.[7.182] to [7.183]).

[9.13] On my interpretation of the law relating to the maxim volenti non fit iniuria, and in the circumstances of this case, I would not have been disposed to sustain the fourth plea-in-law for ITL, if the pursuer had otherwise succeeded on the foregoing issues (paras.[7.204] to [7.208]).

[9.14] The damages which I would have awarded, had the pursuer succeeded, would have been £25,000 for her claim for compensation under section 1(4) of the Damages (Scotland) Act 1976 (as amended), £45,000 for her claim under section 2(1) of the Act as Mr McTear's executrix for solatium for the pain, suffering and loss of the amenities of life experienced by him, and £8,000 for her claim under section 8(1) of the Act for services rendered by her to him during his final illness (paras.[8.20] to

[8.22]). With interest to 31 May 2005 the total award of damages would have been £138,823.32 (para.[8.23]).

Result

[9.15] In my opinion therefore, for all the foregoing reasons, the pursuer's case fails on every issue on which I would have needed to find in her favour were I to hold the defenders liable to her in damages. I accordingly sustain the second and third pleas-in-law for the defenders and assoilzie them from the conclusions of the summons.

THE END.

By Junican.