To: Julio Prieto and Juan Saenz

From: Nicholas Moodie

Date: Monday 2nd February 2009

RE: The standard of proof in US common-law toxic tort negligence claims

ASSIGNMENT:
This memorandum provides examples of the plaintiff's burden of proof taken from common-law jurisdictions. There is then discussion of how the evidence against Texaco might be used to meet the required burden of proof.

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SECTION 1- Standards from case law

It is clear that, despite what Texaco claims, the standard of proof required is not one of scientific causation. Case law from various jurisdictions makes it clear that legal causation is quite different.

In this section I have outlined some formulations of the standard.

Juan/Julio, I'm unable to discern any practical difference between them. However, I've set them out separately in this section, in case you are able to see significant practical differences.

A) Substantial factor (Californian case law)

Reasonable medical probability that D's conduct was a substantial factor in contributing to the aggregate dose of toxic substance and hence the risk of developing the disease. 5

ELEMENTS
- Reasonable medical probability
  o More likely than not 2
  o Greater than 50% chance (ie if the injury would have likely resulted in any event, it cannot be legally attributed to D) 3
  o Requires more than showing a mere possibility 4
- Substantial factor
  o Relatively broad standard- cause must be more than negligible or theoretical (a force that only plays an ‘infinitesimal’ part in bringing about the injury is not a substantial factor) 5
- In contributing to
  o No need to prove that it was toxic chemical’s from D’s conduct that actually produced the malignant growth (due to the ‘irreducible uncertainty of which particular fibre or fibres actually caused the cancer to begin forming’ 6
- Aggregate dose (and therefore risk)

1 This standard is taken from the decision of the California Supreme Court in Rutherford v. Owens-Illinois, Inc. (1997) 16 Cal.4th , and analysed favourably by the California Court of Appeals in Whitley v Phillip Morris, Inc., et al.
2 Whitley v Phillip Morris, Inc., et al., 78.
3 This is the ‘balance of probabilities’ standard generally in US and Australian negligence claims. Some sources suggest there is a difference between the ‘balance of probabilities’ and ‘reasonable medical probability’, and that the latter is more strict. However I couldn’t find proof of this distinction in the case law- they were treated as analogous, for example in Whitley v Phillip Morris, Inc., et al., 78.
4 Whitley v Phillip Morris, Inc., et al., 75.
5 Whitley v Phillip Morris, Inc., et al., 75
B) **Probable Contributing Cause** (US and Australian case law)

*Reasonable medical probability that D’s conduct contributed to P’s injury.*

**ELEMENTS**
- **Reasonable medical probability**
  - Possibility is not sufficient
  - Australia: balance of probabilities (>50%)
  - Australia: Causation can be established by a process of inference that combines primary facts, even if each piece of evidence alone does not rise above the level of possibility
- **D’s conduct contributed to P’s injury**
  - Need not require that the toxic agent be the sole cause: suffices if it is a contributing cause
  - Australia: ’Contribution’ means D’s conduct was more than a minimal, trivial or insignificant factor

C) **Reasonably exclusive factual connection test** (US case law)

*P has a burden to demonstrate the existence of substantial, appropriate, persuasive and connecting factors between their injuries and D’s conduct.*

**ELEMENTS**

P must prove:
- D’s conduct significantly increased the risk of injury. Factors include:
  - Probability that P was exposed to substance
  - P’s injury is biologically/statistically consistent with those known to be caused by exposure to petroleum products
  - Time and extent of exposure
  - Sensitivity factors such as age or special sensitivities of afflicted organ/tissue
  - Retroactive internal/external dose estimation
  - Latency period consistent with a petroleum aetiology
  - Observed statistical incidence of alleged injury greater than expected incidence in same population
- If this is proven, it can then be inferred that D’s conduct was a substantial factor contributing to P’s injury

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7 Articulated by the California Supreme Court in *Boekrath v. Aldrich Chemical Co.* (1999) 21 Cal.4th 71, see discussion in *Whitley v Phillip Morris, Inc., et al.* It is also the standard used in Alabama case law. *Ex parte Valdez* 636 So. 2d 401 (Ala. 1994); *Ex parte Vongsouvanh* 795 So. 2d 625 (Ala. 2000); and generally in Australian negligence case law
9 *Seltsam Pty Ltd v McGuinness.* (2000) 49 NSWLR 262 at paras 91 and 98.
10 *Ex parte Valdez* 636 So. 2d 401 (Ala. 1994) at 402-3
11 *Allen et al v US* 588 F Supp 247 (1984); reversed on other grounds, 816 F 2d 1417 (10th Cir 1988); certiorari denied, 484 US 1004 (1988)
SECTION 2 - Establishing causation in the Texaco case

In this section I have set out the elements of the various standards, and applied them to the facts of the Texaco case. The first two standards are combined, as they are almost identical.

A) Substantial factor/Probable contributing cause

When these two standards of causation are abstracted, they are obviously similar:

<table>
<thead>
<tr>
<th>Substantial factor: More than 50% chance that D’s conduct was a more than negligible or theoretical factor in contributing to aggregate dose and hence risk of developing disease</th>
</tr>
</thead>
<tbody>
<tr>
<td>Probable contributing cause: More than 50% chance that D’s conduct was more than a minimal, insignificant or trivial factor leading to P’s injury</td>
</tr>
</tbody>
</table>

This standard contains four elements: (1) More than 50% chance that (2) D’s conduct was a substantial factor in (3) contributing to (4) P’s injury (or risk of P’s injury, depending on which standard is used).

It is a bit difficult to pinpoint exactly what the first two elements require- this makes it hard to say exactly which elements the evidence we have would help satisfy. Perhaps one way to make sense of them is to see element (2) as the general causation requirement (can exposure to petroleum waste be a substantial factor in the development of cancer/leukemia/birth defects?) and to see element (1) as the specific causation requirement (did such exposure cause the plaintiffs’ injuries?). It seems that the evidence we have might overlap in making out these two elements.

Julio/Juan, you may not agree with this analysis of what the individual elements actually entail. It is certainly not obviously the correct way of interpreting the first two elements- maybe thinking in terms of general and specific causation only confuses things. It would be good to get your feedback on this point.
1) Reasonable medical probability (specific causation)

This element has been articulated in a number of ways in the case law. For example:

- Greater than 50% chance
- More likely than not
- If the injury would have likely resulted in any event, it cannot be legally attributed to D

In any case, a common observation is that it requires more than showing a mere possibility that P’s injury is causally linked to D’s conduct. The important point here is that epidemiological evidence can only establish the possibility of causation in a particular individual. Courts have often viewed epidemiological evidence with “discomfort and suspicion” when plaintiffs have attempted to prove causation.\(^{12}\)

There are some possible responses to this argument. The first uses the ‘relative risk’ value in an epidemiological study. A relative risk of 1.0 means an agent (i.e., petroleum) has no effect on the incidence of disease. A relative risk of 2.0 means that the agent is responsible for an equal number of cases of disease as all other background causes. Therefore if a relative risk value of more than 2.0 were found in a methodologically sound epidemiological study, it could be argued that the ‘reasonable medical probability’ threshold has been met.\(^{13}\)

- The study of childhood leukemia by Hurtig AK. and San Sebastian M. found RR values above 2.0.\(^{14}\)
- The study comparing cancer rates in populations in relation to residence near oil fields found RR values significantly higher than 2.0 for stomach, rectum, skin, soft tissue and kidney cancers in men and cervix and lymph node cancers in women, as well as for haematopoietic cancers in children under 10 years.\(^{15}\)

In Australian case law, one approach called the ‘strands of a cable’ approach has been adopted, in which individual pieces of evidence can be combined to meet the ‘reasonable medical probability’ standard, even if by themselves those pieces of evidence don’t establish a probable link.\(^{16-17}\) On this reading, our epidemiological evidence alone is not enough to make out this element, but nonetheless is an extremely important ‘strand’ in the causal story.

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\(^{13}\) To see a list of toxic tort cases in which this reasoning has been accepted by courts, see Reference Guide on Epidemiology p384 citation


\(^{16}\) Seltsam Pty Ltd v McGuinness. (2000) 49 NSWLR 262 at paras 91 and 98.

\(^{17}\) An analysis of how this evidential burden was met in the case Sharp v Port Kembla RSL CLUB can be found at http://www.mja.com.au/public/issues/176_03_040202/ste10611_fin.html
The case law indicates that, on the whole, further scientific evidence beyond epidemiology is required to satisfy the specific causation threshold. In *Allen v United States*, Judge Jenkins J articulated a list of factors that might be relevant in determining whether D's conduct actually contributed to P's injury. A number of these factors have been used successfully in making out this element in toxic tort litigation. Apart from epidemiological evidence, the judge listed these factors (possible examples of evidence we have are listed under each factor):

- **Probability that P was exposed to the toxic substance**
  - Evidence of elevated TPH, BTEX, PAH and metal levels from judicial inspections\(^\text{16}\)
  - Data from the U.S. show that the waste from drilling can contain significant quantities of a wide variety of contaminants such as antimony, arsenic, cadmium, chrome, lead, magnesium, zinc, benzene and other hydrocarbons as well as toxic levels of sodium and chloride\(^\text{19}\)
  - Chemicals associated with crude oil can be ingested, inhaled or absorbed through skin\(^\text{20}\)
  - Is there concrete evidence that Texaco discharged *untreated* produced water into the Oriente? (Texaco claims it was treated and therefore harmless- San Sebastian’s studies claim it was untreated). If water was untreated, this increases the probability that communities were exposed to toxic chemicals
  - Note: The following evidence may be too vague to count towards specific causation. We would have to ensure that the sites tested were Texaco’s responsibility, and that the people included in the disease studies are those represented in this case
  - Evidence of exposure to TPH in certain communities is outlined in epidemiological studies\(^\text{21}\)
  - Evidence of elevated TPH levels from a study by the *Laboratorio de Aguas y Suelos*\(^\text{22}\)
  - Evidence of elevated TPH levels from the *Instituto de Epidemiología y Salud Comunitaria “Manuel Amunáriz”*\(^\text{23}\)
  - Evidence of oil contamination from studies by Ecuadorian government\(^\text{24}\)
  - Evidence of elevated PAH levels in a 1994 study by the Centre for Economic and Social Rights\(^\text{25}\)

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\(^{16}\) A crucial summary of this evidence is contained in Cabrera’s report


\(^{20}\) Cabrera’s report p29


- **P's injury is biologically/statistically consistent with those known to be caused by exposure to petroleum products**
  - **Note:** In a US court the nature of the injuries suffered by the plaintiffs would have to be documented with great specificity. The following effects of oil contamination relate to varied alleged diseases found in the Oriente.
  - Exposure to **crude oil** in humans can irritate the skin, cause itchiness or irritation of the eyes from accidental contact or exposure to the vapours, and can cause nausea, vertigo, headache, or dizziness from prolonged or repeated exposure to low concentrations of its volatile components. Inhalation of mineral oils can cause lipoid pneumonia and death.
  - Exposure to high concentrations of benzene causes leukemia and neurotoxic symptoms; prolonged exposure can cause lesions of the bone marrow.
  - Chromium VI is a known human carcinogen.
  - **Mercury** poisoning can cause disordered thinking, difficulty walking, talking, chewing and swallowing, and tunnel vision.
  - Elevated consumption of cadmium produces nausea, vomiting, abdominal pain, diarrhea, and renal disease. Studies have suggested an increase in lung cancer mortality in workers exposed to cadmium - other studies have also indicated an association between the level of cadmium in drinking water and prostate cancer.
  - Studies show the adverse health effects of air contamination associated with oil drilling.
  - One study conducted a year after the 1989 Exxon Valdez oil spill off the coast of Alaska suggested an increase in anxiety attacks, post-traumatic stress disorder, and depressive symptoms in the affected communities after the spill. The populations most at risk for these conditions were women and Indians.
  - Laboratory studies on animals show.

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33 Summarised in the “Yani Curi” report p22.
35 Summarised in the “Yani Curi” report p17.
- Skin tumours in rats following the application of crude oil (similar effects have been observed using mice\textsuperscript{36})
- Reduction of red and white blood cells in primary lymphatic organs of birds
- Functional changes in hepatic cells of rats
- Diminished survival and growth of the marine fish *Menidia beryllina*
- Adverse effects on reproduction in rats and birds
  - Massive deaths of marine birds following oil spills, haemolytic anemia in ducks following ingestion of crude oil; significant differences in weight and blood hemoglobin levels in otters inhabiting contaminated and non-contaminated areas following the 1989 ExxonValdez oil spill\textsuperscript{37}
  - Evidence of molecular mechanisms by which the disease develops would be extremely useful, but it is unclear whether the medical science has reached this stage

- **Retroactive internal/external dose estimation**
  - Documented medical evidence of high levels of toxins in individual plaintiffs would be strong evidence of specific causation

- **Latency period consistent with a petroleum waste aetiology**

- **Sensitivity factors such as age or special sensitivities of afflicted organ/tissue**


\textsuperscript{37} Summarised in the “Yani Curi” report p18
2) **Substantial factor (general causation)**

The concept of a ‘substantial factor’ has been interpreted broadly. In the Californian case law it was defined as ‘more than negligible or theoretical' (for example a force that only plays an ‘infinitesimal’ part in bringing about the injury is not a substantial factor)\(^{38}\). In Australia it has been described as ‘more than a minimal, trivial or insignificant factor’.

I feel that satisfying this element involves showing the court that exposure to waste from oil operations is physically capable of causing the alleged injuries. This is because if we can’t show a solid link between petroleum waste and disease, it will be impossible to convince the court that Texaco’s waste was more than a ‘negligible or theoretical’ factor. This may not be the best way of interpreting this element.

Just because this element has been interpreted broadly does not mean it is easy to prove. In a recent Scottish case the plaintiff was unsuccessful in convincing the court of a causal connection between his smoking and cancer, despite the existence of overwhelming medical evidence that such a link is extremely probable in long-term smokers\(^{39}\).

The task of meeting this burden is made even more difficult in the case against Texaco because the scientific evidence is underdeveloped. Plaintiffs in tobacco litigation have decades of research from various sources at their disposal - in comparison, our arsenal of evidence is much more limited. History shows that toxic tort litigation is generally unsuccessful until the scientific evidence is sufficiently developed to support causal inferences - for example, early asbestos and tobacco claims were often unsuccessful\(^{40}\).

Again, epidemiological evidence will be crucial in showing this ‘general causation’ element. It is widely accepted that epidemiological studies by themselves only show associations and not causal links. However, epidemiologists often use certain criteria that are commonly thought to justify the inference from association to causation. A well-known formulation is the Bradford-Hill criteria, used by epidemiologists and lawyers to make causal inferences. While each individual criterion met strengthens the case for causation, epidemiologists acknowledge that not every criterion need be satisfied. Hill himself said “None of my nine viewpoints can bring indubitable evidence for or against the cause-and-effect hypothesis and none can be required sine qua non.”

Note: It is likely that in a US court, these nine criteria would have to be applied to the association between oil contamination and each alleged disease - for example, the causal connection of each individual type of cancer would be analysed. In the following analysis I have not done so, as the evidence I have at hand is so haphazard. Thus this analysis is about the association between oil contamination and disease in general (which would likely be unsatisfactory in a US case).

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\(^{38}\) Discussed by California Court of Appeals in Whitley v Phillip Morris et al p75

\(^{39}\) For an analysis of this case see [http://tobaccocontrol.bmj.com/cgi/content/abstract/16/5/e4](http://tobaccocontrol.bmj.com/cgi/content/abstract/16/5/e4)

\(^{40}\) [www.beasleyallen.com/webfiles/Litigating%20Toxic%20Torts.pdf](http://www.beasleyallen.com/webfiles/Litigating%20Toxic%20Torts.pdf) p4
1 The strength of the association
   - Dr San Sebastian's studies have shown strong associations, in the form of high RR values

2 The consistency of observed associations
   - The more epidemiological/observational studies are undertaken (by different people, different samples) the stronger this criteria will be
   - Dr San Sebastian has conducted numerous studies, which strengthens our case for consistency
   - It has been difficult to find other observational studies linking oil contamination to disease. One such example is Sathiakumar N, Delzell E, Cole P, Brill I, Frisch J, Spivey G. A casecontrol study. American Journal of Industrial Medicine 1987; 11(6): 615-625 which found an association between oil and gas field workers and acute myeloid leukemia. Other studies concerning air contamination, crude oil, benzene, cadmium and mercury were cited in the previous section on 'specific causation'

3 The specificity of the association
   - Causation is likely if a very specific population at a specific site and disease with no other likely explanation. The more specific an association between a factor and an effect is, the bigger the probability of a causal relationship

4 The temporal relationship of the association
   - The effect has to occur after the cause (and if there is an expected delay between the cause and expected effect, then the effect must occur after that delay)

5 Whether a biological gradient was present
   - Greater exposure should generally lead to greater incidence of the effect. However, in some cases, the mere presence of the factor can trigger the effect.

6 Whether the suspected causation was biologically plausible
   - A plausible mechanism between cause and effect is helpful (but Hill noted that knowledge of the mechanism is limited by current knowledge)

7 Whether the cause and effect interpretation of the data was coherent with the generally known natural history and biology of the disease
   - Coherence between epidemiological and laboratory findings increases the likelihood of an effect. However, Hill noted that "...lack of such [laboratory] evidence cannot nullify the epidemiological affect on associations"
   - The studies on laboratory animals and observational studies of wild animals mentioned in the previous section on 'specific causation' give strength to this criterion

8 Whether experimental experience could be drawn upon
   - Occasionally it is possible to appeal to experimental evidence

9 Whether it was possible to interpret the data by analogy to similar studies
   - The effect of similar factors may be considered
3) Contribution

The idea of 'contribution' is emphasised in the case law in order to make it clear that disease often has multiple causes, and plaintiffs are often exposed to more than one brand of a particular toxic product. Therefore the plaintiff need not establish:

- It was a particular toxic substance from D’s conduct that actually produced the disease (in an asbestos case, P doesn’t have to connect the particular fibre that caused the cancer with D’s conduct). This may be a way of arguing that even though Petroecuador may have a role in the prevalence of disease in the Oriente, this does not absolve Texaco. Even though it might be impossible to show whether it was waste from Texaco or Petroecuador that caused a particular instance of disease (because of the “irreducible uncertainty” involved in disease causation), evidence that Texaco contributed to the prevalence of disease is enough. Thus this may be a further argument, supplementing the theory of joint and separate liability, that Texaco cannot hide behind Petroecuador.

- D’s toxic agent was the sole cause of disease (emphasised in US and Australian case law)

4a) Aggregate dose (and therefore risk) – Rutherford; Allen

4b) P’s injury - Bockrath, Australian and Alabama case-law

In some of the cases the plaintiff has had to prove that D contributed to the risk of injury, and in other cases it is simply that D contributed to the injury. At first I thought that the first was evidence of a lower standard- after all, showing an increased risk is easier than showing an actual causal connection. However, the first standard of increased risk, at least in Rutherford, is clarified later in the judicial reasoning. must be read the context of the rest of the commentary. It is explicitly stated that “[i]ncreased risk alone is insufficient…plaintiff cannot prevail on a negligence cause of action unless he or she demonstrates a reasonable probability (i.e., a greater-than-50% chance) defendant actually caused plaintiff’s injury. If the injury would have likely resulted in any event, it cannot be legally attributed to defendant”\textsuperscript{41}. This means that it would be wrong to argue that all we need to prove a reasonable medical probability of increased risk- instead, the standard seems to remain a reasonable medical probability that D’s conduct caused P’s injury. The concept of 'risk' seems to be relevant only in recognising that D’s conduct need only contribute to the disease, not the sole cause.

(Juan/Julio, maybe you are ale to extract a more lenient formulation of the standard of proof)

\textsuperscript{41} Cited in Whitley v Phillip Morris, Inc., et al p78
B) **Reasonably exclusive factual connection test**

The test outlined by Judge Jenkins in *Allen v United States*\(^2\) case does not appear to make the general/specific causation distinction. Perhaps this is sensible, given the amount of overlap in the previous discussion of the 'substantial factor' test.

Using this standard, it would seem that general causation is just a component of the required 'preponderance' of evidence that D's conduct increased the risk of injury to P, and that the increased risk has manifested itself in a biologically consistent injury.

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 SECTION 3 – Miscellaneous

Accumulation of Scientific Data

I think the main problem we have is that the evidence is all over the place, and while there are observed associations between oil contamination and many different forms of disease, the evidence concerning the causal link of any one particular disease is sparse. This seems to be because the medical science still has to catch up. For example, I can’t find biological models that explain the way in which crude oil chemicals cause malignant growths in humans. While some of these chemicals such as benzene are known carcinogens, we have been accused of not having direct evidence that the plaintiffs have been exposed in sufficiently high doses (at least according to Texaco’s website). There is also the accusation that Texaco’s produced water was treated before being released into the Oriente, and that crude oil contamination has not been found in quantities that can harm humans. These factors weaken the case both of general and specific causation.

Incriminating Industry Documents

Tobacco litigation has become more successful following the release of industry documents which prove that tobacco companies knew of the relationship between tobacco smoke and cancer for decades, while they publicly denied the link. For example in the US Department of Justice litigation “Judge Kessler was presented with and gave credence to millions of pages of internal tobacco industry documents that she found to be utterly convincing evidence that the tobacco industry not only knew that its products were harmful and addictive but also strove to make them more addictive and marketed them aggressively at the cost of its customers’ health.”

I doubt that we would be lucky enough to get out hands on similar documentation from Texaco, because it is very unlikely that oil companies would have bothered with scientific investigation in the same way the tobacco companies did. A lack of public interest in the effects of oil contamination means that oil companies have not had to set up their own scientific laboratories in order to create the façade of diligent inquiry for public relations purposes (as far as I know, maybe I’m wrong?).

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43 Dr San Sebastian’s studies acknowledge that there is inadequate literature on the health effects of oil contamination in laboratory animals and humans. For a summary of the scientific literature see San Sebastian M., Armstrong B., Cordoba JA, and Stephens C., Exposures and cancer incidence near oil fields in the Amazon basin of Ecuador. OCCUPATIONAL & ENVIRONMENTAL MEDICINE, 58(8):517-22 (2001)

44 http://tobaccocontrol.bmj.com/cgi/content/abstract/16/5/e4