IIAC

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# Diseases with multiple known causes and rebuttal

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**Summary**

1. Claims for Industrial Injuries Disablement Benefit (IIDB) are tested, among other things, against the terms of prescription set out in Schedule 1 of the Social Security (Industrial Injuries) (Prescribed Diseases) Regulations 1985 whether the claimant has the prescribed disease in question (the 'diagnostic question') and the scheduled occupational exposure (the 'occupational question'). Additionally, the so-called 'causation' question must be decided: whether or not the disease arose from the scheduled exposure and so from work.

2. In adjudging this last question, decision-makers are not uncommonly faced with claimants who have the occupational risk factor defined by legislation but also one or more non-occupational risk factors for the same disease. In principle, (under Regulation 4 of the Social Security (Industrial Injuries) (Prescribed Diseases) Regulations 1985), claims can be rebutted (rejected) if proof exists that the disease was not caused by occupation but by some factor outside work. This paper considers how the 'causation' question should be answered and how to determine attribution to work when other known risk factors of the disease are also present.

3. Mistakes in causal reasoning can easily arise under these circumstances. Several key points are emphasised and illustrated that may at first sight appear counter-intuitive.

4. Firstly, a disease which is caused by an occupational risk factor, 'X', can also be caused in the same individual by a non-occupational risk factor, 'Y'. The presence of 'Y' does not mean that 'X' can be dismissed as a cause of the disease.

5. Secondly, even if 'Y' is a more potent risk factor for the disease in question than 'X', 'X' may still be causal in claimants on the balance of probabilities. Rejecting a claim on the basis that a non-occupational risk factor carries a higher relative risk of the prescribed disease than a scheduled occupational risk factor is potentially unsound.

6. Thirdly, causal probabilities can sum to more than 100%. It may seem reasonable to assume that, if ‘Y’ is known to cause 90% of cases of the disease, ‘X’ can at most account for only 10% of cases – meaning that ‘Y’ is more likely than ‘X’ to be the cause of disease in a claimant with both exposures. In fact this logic does not apply.

7. Because of these challenges, rebuttal should be reserved only for exceptional cases with clear evidence of non-entitlement. More generally, for the particular class of prescribed diseases on which this report focuses, the terms in Schedule 1 offer a fairer, more appropriate, simpler, basis for deciding whether a disease is due to the nature of employment. Rebuttal should rarely be used.

*This report contains some technical terms, the meanings of which are explained in a concluding glossary.*

**Background**

8. Under the Social Security Contributions and Benefits Act 1992 the Secretary of State may prescribe a disease where he/she is “satisfied that the disease:

   a) ought to be treated, having regard to its causes and incidence and any other relevant considerations, as a risk of the occupation and not as a risk common to all persons; and

   b) is such that, in the absence of special circumstances, the attribution of particular cases to the nature of the employment can be established or presumed with reasonable certainty.”

9. In other words, a disease may be prescribed if there is a recognised risk to workers in an occupation, and the link between disease and occupation can be established or *reasonably presumed* in individual cases.

10. For some diseases attribution to occupation flows from specific clinical features of the individual case. For example, the proof that an individual's asthma is caused by their occupation may lie in its improvement when they are on holiday and regression when they return to work, and in the demonstration that they are allergic to a specific substance which they encounter only at work. It can be that a particular disease only occurs as a result of an occupational hazard (e.g. coal workers' pneumoconiosis); or that cases of it rarely occur outside the occupational context (e.g. mesothelioma); or that the link between exposure and illness is fairly abrupt and clear-cut (e.g. several of the chemical poisonings and infections covered by the Scheme). In these circumstances attribution to work is fairly straightforward.

11. Increasingly, however, prescription has proved possible for diseases that are not only caused by occupation but are common in the population at large, and which, when caused by occupation, are *clinically indistinguishable* from the same disease occurring in someone who has not been exposed to the causal agent at work. Examples include lung cancer, chronic obstructive pulmonary disease and osteoarthritis of the knee. Other factors at play in the population

(e.g. smoking, recreational knee injury) account for a proportion of such cases and no clinical features in the claimant allow certain attribution to employment.

12. Early in the 20th century government advisors considered this an insuperable barrier to compensation for diseases like those described above (Samuel Committee, 1907). Since then the objection of the Samuel Committee, that the cause cannot be determined reliably in the individual claimant ("no-one can tell"), has been circumvented using probabilistic reasoning. Nowadays, prescription for such diseases involves identifying the occupational circumstances in which the average risk of disease is increased by a factor of two or more.

13. The requirement for at least a doubling of risk follows from the fact that if a hazardous exposure doubles risk, for every 50 cases that would normally occur in an unexposed population, an additional 50 would be expected if the population were exposed to the hazard. Thus, out of every 100 cases that occurred in an exposed population, 50 would do so only as a consequence of their exposure while the other 50 would have been expected to develop the disease, even in the absence of the exposure. Therefore, for any individual case occurring in the exposed population, there would be a 50% chance that the disease resulted from exposure to the hazard. Below the threshold of a doubling of risk only a minority of cases in an exposed population would be caused by the hazard; above it, a majority would be.

14. For diseases like Prescribed Disease (PD) A14 (knee osteoarthritis) PD D12 (chronic obstructive pulmonary disease) and numerous others, these causal probabilities are weighed by the Industrial Injuries Advisory Council (IIAC) before defining the prescription schedule. The Council includes among its membership appropriately qualified, experienced and professionally competent expert epidemiologists and researchers equipped to make statistical assessments and assess the strengths and weaknesses of biomedical reports. The aim is to spare medical assessors and other

stakeholders the challenge of assessing causal probabilities case by case in situations where this would be taxing and perhaps even unsound.

15. However, claimants often present for medical assessment with a health problem potentially covered by prescription and have also been exposed to a cause or risk factor of the disease in the population at large. For example, many claimants of PD D8A (primary lung cancer following high exposure to asbestos) may smoke cigarettes; claimants of knee osteoarthritis(PD A14) may have a previous history of non-occupational knee injury or knee surgery; and claimants of vascular Hand Arm Vibration Syndrome (Vibration White Finger) (PD A11) may have a family history of Raynaud's phenomenon. How should this affect their assessment?

16. This report addresses this matter from the perspective of the so-called *'causation' question* – i.e. whether the disease can be said to be due to the nature of the claimant's occupation: is it work-caused? (A separate report will give consideration to how the effects on disablement should be apportioned when arising from two causal factors, and how the percentage eligible for benefit may be affected; here it is stressed that the 'disablement' question is a distinctly different issue and not the focus of this report.)

**The causation question**

17. The correct approach to the 'causation' question, when two or more potential causes, occupational and non-occupational, coexist in individual claimants, can be illustrated using two hypothetical but roughly true examples – that of lung cancer in workers formerly exposed to asbestos (including workers who smoke), and that of knee osteoarthritis in ex- miners (including ex-miners with a previous history of non-occupational knee injury).

18. It should be noted that in these hypothetical examples, and in the arguments developed from them, levels of exposure are assumed that would be sufficient to cause the disease in question. For example, in comparing the effects of

smoking with those of occupational asbestos, a sufficient causal dose is assumed of each of the two risk factors. (In practice, where feasible, the concept of exposure-response is accommodated in the Scheme by setting the conditions of exposure under which occupations are prescribed, as for example by requiring a qualifying duration of employment.)

19. For simplicity, this report describes the treatment of causal probabilities for so-called 'stochastic' diseases (those which, like cancer, are either present or not, as compared with those like deafness and chronic obstructive pulmonary disease, where in theory functional loss may exist across a continuum.) In practice, non-stochastic diseases recognised by the Scheme are defined typically in terms of functional loss above a certain threshold (e.g. the prescribed loss of hearing (PD A10) or $FEV_1$ (Forced Expiratory Volume in 1 second) (PD D12), the extent and frequency of finger blanching (PD A11), or defined as present or not at the time of clinical diagnosis (e.g. PD A13, osteoarthritis of the hip), and in these circumstances the logic below applies similarly.

**Example 1: Occupational exposure to asbestos and risk of lung cancer**

20. Table 1 shows data from a hypothetical study of lung cancer in relation to exposure to asbestos. The study involved 2,000 workers substantially exposed to asbestos, among whom 40 developed lung cancer, a rate of 20 per 1,000; the comparison group comprised 2,000 people who had never been exposed to asbestos, among whom 10 developed lung cancer, a rate of 5/1,000. What is the probability that any individual case was caused by their exposure to asbestos? The rate of lung cancer was 20/1,000 in those exposed to asbestos and 5/1,000 in those who were not. All of these lung cancers would be indistinguishable by cause, so there is no way to tell clinically which arose from exposure to asbestos and which did not. All that is known for certain is that for every 20 cases in the asbestos-exposed workers, 5 would have happened even in the absence of their exposure (since the background rate of disease is 5/1,000) and so 15 additional cases were caused by asbestos. So for any individual case, the probability that the

disease was caused by their exposure to asbestos can be estimated to be 15/20 or 75%.

**Table 1: Risk and relative risk of lung cancer in relation to substantial exposure to asbestos**

| Asbestos exposure | Cases/Size of study group | Cases per 1,000 population |
|---|---|---|
| Yes | 40/2,000 | 20 per 1,000 |
| No | 10/2,000 | 5 per 1,000 |
| Relative risk | (Exposed vs. not) | 4.0 |

21. Since this attributable risk exceeds 50%, each individual case is likely on the balance of probabilities to have arisen from exposure to asbestos (corresponding to the greater than doubling of risk requirement for prescription outlined in paragraph 13). This logic can be applied to all 20 cases since none can be distinguished reliably by cause. A case can therefore be made for all claimants receiving benefit, assuming other qualifying conditions are met.

22. It should be noted that in Table 1 there was no information on whether the study participants were cigarette smokers. It might be argued that it is well known that smoking causes 90-95% of cases of lung cancer (i.e. 90-95% of cases of lung cancer would not have occurred if nobody had smoked), so for a worker who smokes, smoking would be a more likely cause of their lung cancer. However, this transpires to be incorrect.

23. To illustrate this, Table 2 splits the data from Table 1 into findings for smokers and non-smokers. Amongst smokers, the rate of lung cancer was 36/1,000 in those who were also substantially exposed to asbestos and 9/1,000 in those who were not. Thus, for every 36 cases of lung cancer in a population with both exposures, 9 would have happened anyway, and an extra 27 arose from exposure to asbestos. In any individual case, the probability of causation would be the same as that in Table 1 (27/36 or 75%). Amongst non-smokers, for every 4 cases of lung cancer in asbestos-exposed workers, 1 would have

happened anyway, and 3 were additional cases which arose from asbestos exposure, so the probability of causation was also unchanged (3/4 or 75%).

**Table 2: Risk and relative risk of lung cancer in relation to exposure to asbestos and smoking habits**

| Exposure | Cases/Size of study group | Cases per 1,000 population |
|---|---|---|
| *Smokers:* | | |
| Exposed to asbestos | 36/1,000 | 36 per 1,000 |
| Not exposed | 9/1,000 | 9 per 1,000 |
| Relative Risk | (Exposed vs. not) | 4.0 |
| *Non-smokers:* | | |
| Exposed to asbestos | 4/1,000 | 4 per 1,000 |
| Not exposed | 1/1,000 | 1 per 1,000 |
| Relative Risk | (Exposed vs. not) | 4.0 |
| ALL | 50/4,000 | 12.5 per 1,000 |

24. In this example, smoking habit would be irrelevant in deciding whether a case of lung cancer was attributable to exposure to asbestos. If a worker is a non-smoker, they have a low background risk of lung cancer (1/1,000) and asbestos exposure multiplies this 4-fold; the probability of causation is 75%. If a worker is a smoker, they have a higher background risk of lung cancer (9/1,000) but exposure to asbestos still multiplies this higher risk by a factor of 4; the probability of causation is (36-9)/36 or 75%.

25. Table 2 employs hypothetical data, but the figures are similar to results that have been found in real studies. Sometimes the relative risk (RR) estimated from exposure to asbestos has been a little lower in smokers than in non-smokers and the probability of causation would therefore be a little lower in smokers than in non-smokers (e.g. 70% vs. 80%). However, for both groups, the RRs are similar and both are greater than 2; the probabilities of causation are also similar and both are greater than 50% - i.e. the disease can be attributed to the occupational exposure on the balance of probabilities, despite another important non-occupational risk factor also being present.

**Example 2: Knee osteoarthritis in ex-miners with a past history of knee injury**

26. A second example considers the causation of knee osteoarthritis (OA) in underground coal miners.

27. The RR for knee OA in miners is likely to lie in a range from 2.9 to 5.4, as set out in paragraphs 33, 34, 36, 40 and 41 of Cm. 7440 (Kellgren *et al*., 1952; Lawrence, 1955; Greinemann, 1997; for kneeling/squatting and heavy lifting vs. neither of these – Coggon *et al*., 2000; Cooper *et al*., 1994). This evidence is summarized in Table 3 which again presents hypothetical data.

**Table 3: Risk and relative risk of osteoarthritis of the knee in relation to work in underground coal mining**

| Worked as a miner | Cases/size of the study group | Cases per 1,000 population |
|---|---|---|
| Yes | 50/2,000 | 25 per 1,000 |
| No | 10/2,000 | 5 per 1,000 |
| Relative Risk | (Exposed vs. not) | 5.0 |

28. This illustrative study involved 2,000 ex-miners, 50 of whom developed OA of the knee, a rate of 25 per 1,000; the comparison group comprised 2,000 people who had never worked in mining, of whom 10 developed OA of the knee, a rate of 5/1,000. What is the probability that any individual case of OA of the knee was attributable to mining? In this example, the rate of disease is 25/1,000 in those who worked as a miner and 5/1,000 in those without such a work history. As with the lung cancer example, there is no reliable way clinically to tell which cases were caused by mining and which were not. All that is known for certain is that for every 25 cases in ex-miners, 5 would have happened anyway (this is the background rate of disease – 5/1,000) and 20 happened because of work in mining. So for any individual case in an ex-miner, the probability of a causal relationship to mining is 20/25 or 80%. Since this is greater than 50%, each individual case is more likely than not to have arisen from mining. This logic can be applied to all 25 cases, since none can be distinguished reliably on clinical grounds by cause; a case exists, therefore,

that all should be awarded benefit (assuming that the Scheme's other qualifying criteria are met).

29. The RR for knee OA given a past history of joint injury probably lies in a range of 3.5 to 7, being even higher if unilateral (Davis *et al*., 1989; Gelber *et al*., 2000; Wilder *et al*., 2002; Toivanen *et al*., 2010).

30. Since these RRs are similar to, and somewhat greater than, those quoted in paragraph 27, on the face of it, an ex-miner with knee OA who also has a past history of knee injury is at least as likely, and perhaps more likely to have acquired his disease through injury than through coal mining. However, Table 4 illustrates that – as with smoking and asbestos – matters are more complicated than implied by simply comparing the size of RRs from two different causes of the disease.

31. In those with a history of knee injury, the probability that their OA was caused by mining is 78% ((42-9)/42), but in the absence of injury to the knee it is rather similar ((8-1)/8 or 88%). So the probability of causation, although slightly different in the two groups, is in both instances much greater than 50%. It follows that a history of injury to the knee is irrelevant when deciding if the disease can be attributed to coal mining on the balance of probabilities in an individual with OA of the knee and both exposures. The figures in Table 4 are illustrative, but they are not dissimilar to results that have been found in real studies involving OA of the knee, knee injury, and heavy knee-straining activities akin to those found in mining. There are grounds, therefore, for considering that these calculations are supported by available evidence.

**Table 4: Risk and relative risk of osteoarthritis of the knee in relation to work in mining and history of previous knee injury**

| Exposure | Cases/Size of study sample | Cases per 1,000 population |
|---|---|---|
| *History of knee injury:* | | |
| Worked as a miner | 42/1,000 | 42 per 1,000 |
| Did not work as a miner | 9/1,000 | 9 per 1,000 |
| Relative Risk | (Exposed vs. not) | 4.7 |

| | | |
|---|---|---|
| *No history of knee injury:* | | |
| Worked as a miner | 8/1,000 | 8 per 1,000 |
| Did not work as a miner | 1/1,000 | 1 per 1,000 |
| Relative Risk | (Exposed vs. not) | 8.0 |
| ALL | 60/4,000 | 15 per 1,000 |

**Are there exceptions?**

32. In the first of these examples, the effects of the two factors (asbestos-smoking) multiplied together, so that the RR for the occupational exposure (asbestos) was the same whether or not exposure to the other risk factor (smoking) had occurred. In the second example, the effects did not multiply together, so the RR from mining was different in those with a history of knee injury and those without such a history. However, the differences were not large and did not affect the decision about probability of causation. Are there exceptions to this? Exceptions probably exist, but only rarely to the extent that they would affect the final determination of eligibility for benefit. It can happen that the RR is different in different subgroups, but this would only affect decisions regarding the causation question within the IIDB Scheme if there was firm evidence that the RR was less than 2.0 in a particular subgroup. Considering the case of OA of the knee in a miner, for example, this would require that the RR from mining was less than 2.0 (e.g. 1.5) in those with a history of knee injury.

33. Situations of this kind may occur, but they appear to be rare, and to enable robust decision-making they would need to be observed consistently and be well-documented. In the absence of such firm evidence, any decisions about individual workers should be based on population averages, meaning that in most circumstances the concurrence of another non-occupational risk factor will not lessen the probability of causation by work activity, as defined in the Scheme's prescription schedule.

**The legal context**

34. Regulation 4 of the Social Security (Industrial Injuries) (Prescribed Diseases) Regulations 1985 identifies the diseases from Schedule 1 of those Regulations that can be presumed attributable to occupation. Regulation 4

spares decision makers and claimants the burden of proving causation case by case – it can be *presumed*.

35. Regulation 4 is nuanced, however. In particular, it grants decision makers the power to rebut (reject) a claim if they consider there is 'proof to the contrary' i.e. proof that the disease was *not* caused by the occupational exposure set out in prescription but by something else.

36. It may be seen that for certain diseases covered by the Scheme (identified in further detail in the Council's report Cm 9030, *Presumption that a disease is due to the nature of employment: the role of rebuttal in claims assessment*) proof to the contrary when based on alternative sources of risk can be very challenging.

**Conclusions**

37. This report on the 'causation' question has sought to explain how erroneous causal reasoning can easily arise when two or more risk factors operate together. It is tempting to think of a disease as having one single cause, which is to be identified, and therefore to treat risk factors as if *competing* as causes of disease ('X' cannot cause the disease if 'Y' does). In reality, they often act *independently* and *in concert*.

38. It is also tempting to compare the size of RRs of two risk factors: if 'X' (which is occupational) only increases risks 3-fold and 'Y' (which is non-occupational) increases risks 5-fold, then 'Y' is a more likely cause of disease in the claimant than 'X' because it is a more potent risk factor. As shown in Tables 2 and 4, this reasoning is flawed.

39. Another counter-intuitive point is that causal probabilities can sum to more than 100%. For example, while the calculations based on Table 2 conclude that 75% of cases of lung cancer were caused by exposure to asbestos, 50 cases in total, 90% (45) were caused by smoking: a common error would be to assume that only a maximum of 10% is 'left over' for other causes. This error of reasoning arises from a failure to consider that some cases are

caused by both risk factors acting together in some individuals. (This issue is explored more formally in the concluding Appendix.)

40. More generally, for diseases of the ‘no-one can tell’ variety identified by the Samuel Committee, where Regulation 4 accords the benefit of presumption, the terms in Schedule 1 should offer a fairer, more appropriate, simpler, basis for deciding whether a disease is due to the nature of employment. Rebuttal in these circumstances should be reserved only for exceptional cases with clear evidence of non-entitlement, care being taken to avoid the pitfalls of faulty probabilistic reasoning.

**Diversity and equality**

41. IIAC seeks to promote equality and diversity as part of its values. The Council has resolved to seek to avoid unjustified discrimination on equality grounds, including age, disability, gender reassignment, marriage and civil partnership, pregnancy and maternity, race, religion or belief, gender and sexual orientation. During the course of the review of diseases with multiple causes no diversity and equality issues were apparent.

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## Appendix: The proportion of cases arising from a combination of exposures

1. What is the risk of lung cancer arising from the combination of smoking and exposure to asbestos? Hypothetically, in Table 2 (in main body of report) there were 36 such cases per 1,000 people with the combination of exposures (those in the top left cell of the table). Their makeup can be deduced from the values in the other cells of Table 2.

2. One case (3%) occurred in the absence of known causal exposures (U). This is indicated by the rate in the bottom right cell of Table 2, of 1 per 1,000 in workers with neither exposure.

3. Three cases (8%) arose through mechanisms involving asbestos exposure alone (and not smoking), together with other unknown background exposures (U'). This can be deduced from the difference between the rate in workers exposed only to asbestos and that in workers exposed to neither risk factor (4 per 1,000 minus 1 per 1,000 = 3 per 1,000).

4. Eight cases (22%) occurred through mechanisms involving smoking alone (and not asbestos), together with a potentially different combination of unknown background exposures (U''). This can be deduced from the difference between the rate in workers exposed only to smoking and that in workers exposed to neither risk factor (9 per 1,000 minus 1 per 1,000 = 8 per 1,000).

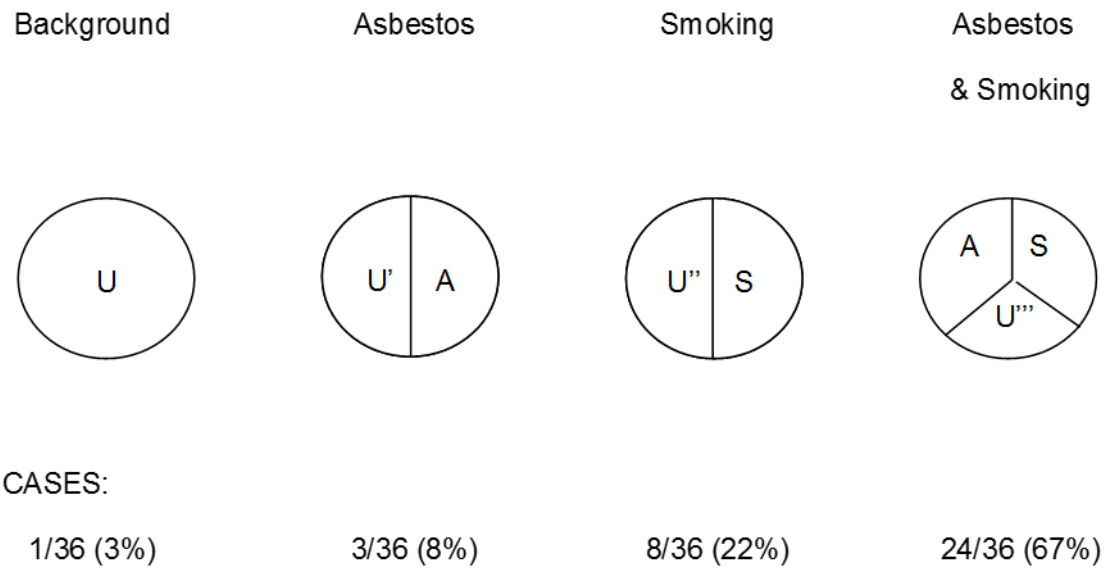
5. Since 36 per 1,000 cases arose from the combination of exposures and 12 are now accounted for (1+3+8), 24 cases (67%) must have occurred through mechanisms involving *both* factors, acting together with unknown background exposures (U''').

6. Figure 1 illustrates the various patterns of exposure and their relative frequency. It follows that 89% of the cases (22% + 67%) could have been prevented by avoidance of smoking, while 75% (8% + 67%) could have been prevented by avoidance of exposure to asbestos.

7. In this example the attributable risks for the individual risk factors of smoking (89%) and asbestos (75%) sum to more than 100%. This is possible because 24 of the cases occurred through mechanisms that involved *both* exposures, and which consequently could be prevented by preventing *either* exposure.

8. A person without this perspective might conclude that, since 89% of cases of lung cancer are attributable to smoking, only 11% of cases can be due to other factors, including exposure to asbestos, and, further, that exposure to asbestos was an unlikely cause of lung cancer in a patient who smoked. However, as can be seen from this example, this is not the case.

**Figure 1: Numbers and proportions of lung cancer cases occurring through background factors, exposure to asbestos, cigarette smoking, and their combination**




*Key: A – exposure to asbestos; S – cigarette smoking; U, U', U'', U''' – unknown background risk factors that either act on their own to cause lung cancer (U), or with asbestos (U'), or with cigarette smoking (U''), or with both asbestos and cigarette smoking (U''')*

## Glossary

**Risk:** The probability that an event will occur (e.g., that an individual will develop disease or die within a stated period of time or by a certain age).

**Attributable risk**: The proportion of the rate (or risk) of a disease that can be attributed to the exposure. This can be assessed for the whole population or within exposed individuals. (*In this report the term is used in reference to claimants of IIDB who satisfy the criteria for diagnosis and exposure set out in Schedule 1. Among this group, it represents the fraction of disease that can be attributed to the scheduled exposure*.)

**Relative risk (RR)**: The ratio of the risk of an event among the exposed to the risk in the unexposed.

Taken from Porta M (ed). A Dictionary of Epidemiology. 5th Edition. Oxford: Oxford University Press, 2008.

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