



IIAC

THE INDUSTRIAL INJURIES ADVISORY COUNCIL

POSITION PAPER 38

**Noise, occupational deafness and
Industrial Injuries Disablement Benefit**

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Noise, occupational deafness and Industrial Injuries Disablement Benefit – a Position Paper

Among the 70 or more ‘prescribed’ diseases (PDs) for purposes of Industrial Injuries Disablement Benefit, occupational deafness (PD A10) poses a particular challenge in terms of extending the field of eligibility for benefit. Paradoxically, while much research has been conducted and a great deal is known about the injurious effects of noise at work, with strict standards set in legislation to protect workers’ hearing, the terms of PD A10 are formulated, not (as one might imagine) in terms of a stipulated amount of noise exposure at work, but in terms of a selected list of occupations, tools and exposure circumstances, evidence on which is assessed case by case.

This position paper documents the history of PD A10, the current terms of the prescription, the scientific issues which have helped to shape but also limit those terms, the criteria presently used to extend the prescription list, and some of the alternative approaches that have been explored in recent times.

A glossary explains the meaning of technical terms used in the report.

Hearing loss

1. Hearing loss is the amount (measured in decibels (dB)) of change for the worse as a result of some hearing insult. It exists in two broad forms: (a) sensorineural hearing loss, which is caused by damage to the sensitive hair cells inside the inner ear or damage to the auditory nerve; and (b) conductive hearing loss, which arises because blockage prevents the passage of sound from the outer ear to the inner ear. Noise-induced hearing loss (NIHL) is sensorineural in character, but this pattern of hearing loss is also seen in the absence of noise, especially in older people, and may have a variety of other causes (genetic, infective, traumatic, etc.).
2. Hearing loss is common. Estimates suggest that there may be about 8.8 million people in the United Kingdom with an average of 25 or more dBs of hearing loss in the better ear, men being more commonly affected than women. In the National Survey of Hearing (Davis, 1989), over 48,000 questionnaires were mailed to randomly selected subjects from four British cities. The prevalence of 'severe' hearing difficulty (great difficulty or inability to hear at all in the better ear) ranged from 0.2% in 17-30 year olds to 4.1% in those aged 61-70 years, while more than 3% of respondents reported at least moderate bilateral difficulty in hearing in the quiet. A subset of people underwent comprehensive audiological assessment, among whom, in the 51-60 year age band, 8% had bilateral hearing loss of 35-45 dB and 5% had a greater deficit.

The Industrial Injuries Disablement Benefit Scheme

3. The Industrial Injuries Disablement Benefit (IIDB) Scheme provides a benefit that can be paid to employed earners because of an occupational accident or 'prescribed' disease. The benefit is no-fault, tax-free, non-contributory and administered by the Department for Work and Pensions. It is paid in addition to other incapacity and disability benefits but is taken into account when determining the level of payment for income-related benefits.
4. The 'prescribed' diseases for purposes of IIDB are those listed in Schedule 1 of the Social Security (Industrial Injuries) (Prescribed Diseases) Regulations 1985. This schedule defines each prescribed disease in one column and sets out alongside it, in a second column, the qualifying circumstances of exposure.

5. Benefit under the Scheme is paid for *disablement* resulting from *loss of faculty*. Loss of faculty has been held in the courts to be broadly equivalent to “impairment of the proper functioning of mind or body”. ‘Disability’, which is the basis of some compensation schemes and civil settlements, is a more subjective concept, defined by the WHO in 1980 as “any restriction or lack of ability to perform an activity in the manner considered normal for a human being” and in British equalities legislation in terms of a “substantial” and “long-term” impairment adversely affecting “day-to-day activities”.
6. Benefit is payable under some circumstances for loss of the faculty of hearing caused by noise at work. PD A10 (‘occupational deafness’) is one of the four most frequently claimed prescribed diseases.

Current terms of PD A10

7. The terms of prescription, as they appear in Schedule 1, are reproduced in Appendix 1. ‘Occupational deafness’ is defined clinically in terms of a minimum level of hearing loss: ‘sensorineural hearing loss amounting to at least 50 dB in each ear, being the average of hearing losses at 1, 2 and 3 kHz frequencies’. To be eligible, a claimant with such hearing loss must also have been an employed earner (employed rather than self-employed) in one of several noisy occupations stipulated in the schedule – for example, ones involving work with powered saws, pneumatic percussive tools, textile machinery, or a list of other defined equipment.
8. The 1985 Regulations also provide various other clarifications, definitions, and conditions for qualification and assessment in respect of the disease. These need not be entered into here, save for two of note: 1) the qualifying noisy employment must have lasted at least 10 years in aggregate by the time of claim; and 2) that claim must be made no later than 5 years after leaving that employment. In other words, the qualifying exposure such be sufficiently prolonged and the claim for occupational deafness should not be unduly delayed.

9. IIAC is an independent statutory body established in 1946 to advise the Secretary of State for Social Security on matters relating to the IIDB Scheme. IIAC advises on the prescription of occupational diseases; matters referred by the Secretary of State; draft regulations or proposals concerning the Scheme; and any other matter relating to the Scheme or its administration.
10. IIAC is a non-departmental public body and has no power or authority to become involved in individual cases or in their decision making processes.

Prescribed Disease provisions of the IIDB Scheme

11. The Social Security Contributions and Benefits Act 1992 states that the Secretary of State may prescribe a disease where he or she is satisfied that the disease:
 - a) “Ought to be treated, having regard to its causes and incidence and any other 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 employment can be established or presumed with reasonable certainty.”
12. In other words, a disease may only 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. This is the framework in which the Council must work when considering the prescription of occupational diseases.
13. Some occupational diseases are relatively simple to verify, as the link with occupation is clear-cut. Some only occur due to particular work (e.g. pneumoconiosis in coal miners); or are almost always associated with work (e.g. mesothelioma in the UK); or have specific medical tests that prove their link with work (e.g. occupational asthma); or have a rapid link to exposure or other clinical features that make it easy to confirm the work connection (e.g. certain infections and chemical poisonings). Thus, for example, the proof that an individual’s dermatitis 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 with which they come into contact only at work.

14. However, many other diseases are not uniquely occupational and, when caused by occupation, are indistinguishable from the same disease occurring in someone who has not been exposed to a hazard at work. In these circumstances, attribution to occupation depends on epidemiological evidence that work in the prescribed job or with the prescribed occupational exposures causes the disease on the balance of probabilities (previous reports of the Council give further detail¹). In turn the Council looks for evidence that a particular occupational exposure or circumstance increases the risk of developing the disease by a factor of two or more.
15. The requirement for, at least, a doubling of risk follows from the fact that if a hazardous material 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 only do so 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 an individual case occurring in the exposed population, there would be a 50% chance that it would have occurred even without the exposure. Below the threshold of a doubling of risk only a minority of cases in an exposed population would be caused by the hazard and individual cases therefore could not be attributed to exposure on the balance of probabilities; above it, they may be. The epidemiological evidence required should ideally be drawn from several independent studies, and be sufficiently robust that further research at a later date would be unlikely to overturn it.
16. In considering the terms of PD A10, the Council has considered both of the potential routes to prescription – that which is based on doubling of risks (paragraphs 14-15) and that which is based on unique clinical features (paragraph 13).
17. The history of PD A10 is convoluted, however, pre-dating the time when IIAC first began using epidemiological reasoning and the “doubling of risk” approach to tackle

¹ See also: <https://www.gov.uk/government/publications/how-decisions-are-made-about-which-diseases-iidb-covers>

the prescription of diseases with the characteristics outlined in paragraph 14. Since first being prescribed in 1975, PD A10 has been the subject of six further reports by the Council, which have referred to the unsatisfactory compromise reached over its terms and the shortfall in fundamental scientific evidence underpinning prescription. The history of the prescription is given below. Some features of NIHL, and of administration within the Scheme, are also highlighted which have shaped the terms that appear in Appendix 1.

History of the current prescription

18. Historically, prescription for occupational deafness did not rest on the unique diagnostic features of individual cases or a doubling of risk in groups with defined exposures. Rather, in a complex field where progress proved surprisingly difficult, it was decided pragmatically to prescribe in the most clear cut circumstances, where noise levels had been sufficient to deafen a high proportion of workers. This provided an initial list, with the option to extend coverage later on.
19. The original list seems not to have been accompanied by data specifying typical levels of noise exposure in the qualifying occupations. Also, the originally selected duration of exposure (20 years) was not grounded in empirical data but a sense from experts that this was a reasonable and suitable choice. Later the qualifying duration was shortened to 10 years, there being some evidence that noise damage happened early on and then slowed in rate of progression, and some measurements of noise were made at the Council's request.
20. Numerous problems in extending the terms of prescription have been identified in successive Council reports since 1965. These include:
 - i. The existence of other indistinguishable causes of sensorineural hearing loss in the general population, and other causes of non-sensorineural hearing loss.
 - ii. The wide biological variation in hearing capacity in the general population, which seems to exist independent of noise, age and well known otological pathologies.
 - iii. Notwithstanding (ii), a wide variation in individual susceptibility to noise.
 - iv. Widespread exposure in the general population to non-occupational sources of noise.

- v. The insidious nature of deafness, with no clear definitions of disease and disease onset.
- vi. Difficulty in accurately verifying individuals' lifetime occupational exposures to noise (level, duration, frequency, continuous vs. impulsive, etc.).
- vii. Uncertainty about exposure-response relationships, with various models proposed on the back of sophisticated statistical analyses of selected cross-sectional populations, and with differing assumptions.
- viii. Uncertainty about how to handle the impact of age, which is the other known main cause of sensorineural hearing loss.
- ix. Disagreement about the functional impact arising from different degrees of hearing loss, whether measured by pure tone audiometry (PTA) or by alternative methods.
- x. Technical problems in PTA, including (a) a lack of repeatability and (b) concern about the impact of temporary threshold shift (in temporarily biasing assessment of hearing thresholds).
- xi. At one stage, insufficient specialist resource to deliver assessments on the expected scale.

21. Some of these barriers are substantial. Large biological variation in hearing capacity in the general population (issue ii) has been noted in many reports. For example:

- Robinson found there to be a 30dB dispersion of hearing threshold levels among “thoroughly screened young persons”.
- In the British National Survey of Hearing (Lutman *et al*, 1994), the median hearing thresholds at 4 KHz ranged, in an unscreened population of 18-30 year olds, from -3.0 dB at the 5th percentile to 32.0 dB at the 95th percentile, a span of 35 dB. The respective percentile figures for a screened population (after excluding obvious alternative ear, nose or throat pathology) were -3.0 and 20.5 dB, still a span of 23.5 dB.
- A similar range to the Robinson report was found in a NIOSH study of 2,066 workers across various US industries (Prince, 2002).
- In the US Framingham Study, a wide variation in hearing thresholds was found in 57-89 year olds, especially at higher frequencies (Figure 1 from Mościcki *et al*, 1985).
- Lutman *et al* (1991) have reported that hearing tends to be worse by about 2-6 dB in manual workers, seemingly independent of noise.

22. Variation in individual susceptibility to noise has been shown, for example, in a very homogeneous workforce of weavers with well characterised noise exposures, who had a 60dB spread in hearing loss at 3 KHz (Taylor *et al*, 1965). Similarly, wide variability in temporary and permanent threshold shift have been demonstrated in animals exposed to noise experimentally.
23. Crucially, biological variability between individuals appears to be large relative to the impact of noise. Lutman *et al* (1991) described the effect of noise as 'modest' and put it at <12dB at 4 KHz for 50 years of exposure at 90-100 dB. If so, this creates a significant problem in that *natural variation tends to swamp the signal being measured*.
24. The threshold of hearing loss in the IIDB Scheme was chosen to represent a disabling loss of hearing, but in a way which reflected these uncertainties and made some necessary compromises.
25. Initially, advice was received from the British Association of Otolaryngologists (BOAL) that hearing loss would usually exceed 30dBHL before affected individuals took their problem to a healthcare professional. It was also suggested that (i) 40dBHL at 1, 2, 3 KHz in the better ear was the lower threshold for loss of faculty (1% disablement), and that (ii) total deafness (100%) occurred at >90dBHL. The scale of % disablement was set such that each 5dBHL above 40dBHL equated to a further 10% loss of faculty on a linear scale (1973 report). In 1973 the Council decided to compensate only 'severe' degrees of deafness – defined by the BAOL as 50dBHL at 1, 2, 3 KHz in the better ear, which corresponded to 20% disablement. This decision was based in part on the precedent of state compensation schemes from overseas (which focussed only on severe disease); in part on the perception that milder degrees of occupational deafness would be very common; in part on data indicating insufficient expert resource to assess and process lesser scales of deafness (problem xi above); and in part because the assessment method is somewhat generous and pragmatic in the sense of applying no offset for conductive hearing loss where present (issue i) and in attributing all of the hearing loss to noise at work without qualification or restriction.
26. It was also influenced and reinforced by three more of the concerns enumerated above: setting a threshold some way above 40dBHL was deemed appropriately

conservative as: (a) the average effect of otological disease in the population amounted to about 5-8 dBHL (issue i); (b) temporary threshold shift could raise hearing thresholds by 5-7dB for up to 48 hours after noise exposure (issue x(ii)), and it was deemed impractical to insist on a noise-free holiday as long as this before assessment to eliminate the problem; (c) PTA was said to have an error of ± 5 dB, while by 1982 it became apparent that on repeat testing at 5 years some 31% of results improved by 10dB and 13% no longer met the prescription criteria (issue x(i)).

27. The choices and methods need to be viewed in the context of wide international variation in measuring and assessing disablement – for example, deciding where it starts (“low fence”), what its upper limit (“high fence”) is, what scale to use in-between, and how to weigh the better ear relative to the worse (Melnick *et al*, 1991) (issue ix).

28. The terms of PD A10 require substantial hearing loss in *both* ears, with disablement defined by reference to the better ear. Underlying this may be an assumption that NIHL from occupational sources would normally be roughly symmetrical, arising from ambient noise spread around the worker, whereas hearing loss in only one ear would be more likely to have a non-occupational cause. (The different position of acute acoustic trauma is described below.) Another reason for restricting prescription to bilateral loss might be if hearing in the better ear compensated for that in the worse ear and the disability were markedly less for unilateral loss.

29. It was recognised early on, and has been since in several Council reports, that the threshold of substantial hearing loss in both ears is a high one; also, that the list of qualifying occupations was restrictive and narrow. However, it was hoped that as more otolaryngological resource became available, as technical issues of diagnosis were solved, and as more evidence was obtained on dose-response relationships, the field of compensation could be extended with loosening of the qualifying terms. In 1982, for example, the Council wrote that the 50dBHL criterion “should be kept under review, with a view to relaxing it as soon as possible”; and several reports alluded to the desirability of being able to prescribe by *exposure level* rather than by *job title*.

30. With time some new knowledge did emerge and a few changes were made to the assessment protocol. Firstly, the hearing loss corresponding to total deafness was

revised upwards to 110dB and a revised (sigmoid) scale of disablement was introduced under which each dBHL from 30 to 50, and from 90 to 110, increased the assessment by 1%, and each dBHL from 51 to 89 by 1.5%. (This still sets the qualifying threshold of 50dBHL at 20%, and not 14% as elsewhere in the Scheme.) Secondly, an offset of -0.5% of assessed disablement per year after 65 years had originally been applied to allow for ageing (issue viii), as in civil claims (Cmnd 5461); but experience showed that this rarely affected assessments that were rounded and so the offset was dropped (Cmnd 8749). Age was also accounted for already within the Scheme, to the extent that claims had to be assessed within five years of leaving the noisy occupation. (Beyond this period it was deemed difficult to distinguish the hearing decline from noise from that of presbycusis – i.e. hearing loss of normal ageing).

31. More significantly, the work of Burns and Robinson (1970), the later review by Robinson (1988), and the ISO 1999 set out formulae to quantify the purported relation between noise and hearing loss by percentile of the population affected. This last development on exposure-response relations (issue vii) raised the possibility of prescribing in terms of exposure level.
32. The Burns-Robinson data, with their various potential limitations (Appendix 2), were founded on statistical and theoretical modelling of cross-sectional field observations. However, there have been continuing disagreements about the relationship since (e.g. Robinson has suggested a modification of his own formula), with various proposals and counter-proposals.
33. The model was influential in Council deliberations, in that searches were made for industries with 'high' exposures as defined by the model and in some cases this resulted in additions to the prescription list.
34. However, when the Council reviewed the issue in 2002, it was less convinced that the modelled data were sufficiently robust to accept the dose-response relationship as well established.
35. The Council also explored approaching the prescription of occupational deafness as it had chronic obstructive pulmonary disease (COPD) in miners (another condition existing across a biological continuum), by seeking epidemiological evidence about the

exposure levels needed to double the risk of *a disabling loss of function*. 50dB bilateral hearing loss (the current threshold) and 44dB bilateral hearing loss (equating to 14% disablement) were considered as thresholds. However, the Council could not find peer review evidence on the dose of noise required to double risks at these or any other thresholds.

The challenge in context

36. The 11 problems listed in paragraph 20 need to be viewed in the context of established PDs. Most of the challenges are not without precedent, and should not therefore be taken as absolute bars to prescription. For example:

- It is common for there to be other non-occupational risk factors for disease (including age) – hence the doubling of risk approach commonly adopted by the Council (paragraph 14-15).
- For some outcomes (e.g. chronic obstructive pulmonary disease, PD D12) there is biological variation: these are of the “how much” kind rather than the “all of none” (e.g. cancer).
- Many occupational diseases have an insidious onset, the lack of a clear cut-point being a particular feature of the “how much” disorders.
- There is likely to be individual susceptibility to many occupational hazards.
- There is usually a difficulty in reconstructing the historic exposure level, except crudely (e.g. as years served in a job).
- There is often a lack of good exposure-response information.
- There may be difficulties in diagnosis for the “how much” disorders, and also in assessing the extent of disablement – transient decrement of function (ix(ii)) is a concern in assessing hearing loss, but also in measuring lung function.

37. Table 1 compares some of the characteristics of hearing loss with loss of FEV₁, which has proved compensable. By analogy there are many similarities. If not unique then, how might the barriers be to overcome?

Table 1: Common issues arising in the compensation of a loss defined on a continuous scale: hearing loss vs. loss of FEV₁

	FEV ₁ loss	Hearing loss
Has non-occupational as well as occupational causes	Yes	Yes
Exists across a biological spectrum, with no clear dividing line between health and disease	Yes	Yes
Definition of a disabling loss is not straight forward, and to a degree arbitrary	Yes	Yes
Is innately variable within the population	Yes	Yes
Is assessed by reference to tables of expected values, which are based on a regression equation that allows for some (e.g. age, height) but not all (e.g. smoking) other factors	Yes	Might be?
(i) By convention, expected value is defined relative to the population mean (if normally distributed, by 50 th percentile) (ii) actual overall % loss in an individual is unknown	Yes (to both)	(i) No, defined relative to perfect hearing; (ii) Yes
There is disagreement about which reference table to use and which non-occupational co-factors to allow for	Yes	Yes

Prescription based on evidence of a doubling of risks?

38. A cornerstone of modern prescription, for diseases that are clinically indistinguishable when occupationally caused from their non-occupational counterparts, has been to follow the doubling of risks approach (paragraph 14-15). In principle a doubling of risk of, say, median hearing loss above a certain threshold might be demonstrable in one occupational group compared with a suitable reference population. No such data were found in 2002, there being doubts about the qualifying level of exposure; and no such data have come to light so far.

39. However, even if such data could be found, the margin of uncertainty in any individual claimant would seem wide – perhaps more so than elsewhere in the Scheme. The small impact of noise relative to biological variability in ‘normal’ hearing seems a serious impediment since *it makes both the attribution of moderate noise-induced decrements of hearing loss, and the assessment of the ensuing disablement, problematic*. The decision to compensate only severe hearing loss is understandable in these terms alone (although it should be noted that some experts regard a 44dBHL as almost as disabling as a 50 dBHL; Professor Luxon, personal communication).

Prescription based on unique clinical features?

40. Classically, NIHL causes a notch to appear on a person's audiogram in the high frequency range (3, 6 and particularly at 4 KHz). In principle, therefore, the audiogram might be used as a means to identify claimants of IIDB with occupational deafness, the notch potentially providing a means to make the occupational attribution on the basis of clinical features alone (paragraph 13) and without the requirement to demonstrate a doubling of risks (paragraphs 14-15).
41. However, the audiometric notch is not a wholly reliable diagnostic feature of NIHL. In more advanced cases the deficit may extend across all of the higher frequencies, disguising the notch and making the pattern difficult to distinguish from that due to ageing; noisy sources vary in their predominant frequencies, so the notch sometimes occurs at a frequency higher or lower than 4 KHz; and there are case reports of 4 KHz notches following viral infection or use of ototoxic drugs. Thus, a 4 KHz notch is not found in every case of occupational deafness (i.e. the test is imperfectly sensitive) and can sometimes be found in the absence of noise (i.e. it is imperfectly specific).
42. How significant are these limitations? A review of 28 relevant research reports highlights several major concerns.
43. High frequency notching is very common in the general population:
- In 1,662 elderly subjects from the Framingham Study, 66% of men and 40% of women had notches at 4 or 6 kHz (Gates *et al*, 1999).
 - In 2,159 people aged 58-100 years from the Beavers Dam Wisconsin Epidemiology of Hearing Loss Study, notches were present in 12- 47% of individuals (varying by case definition) – 20-70% of men and 5-30% of women (Nondahl *et al*, 2009).
 - Of 3,349 consecutive patients to an audiology clinic in Tennessee (age 20 -89, mean 62 years), 40.6% had a 4 kHz notch in at least one ear; even among people in their 20s, the prevalence was 11% (Wilson, 2011).
 - Of 149 consecutive patients to an audiology clinic in Surrey (age 19-91, mean age 45 years), 40% had a high frequency notch (Osei-Lah *et al*, 2010).
44. Notching is also common in noise-exposed populations. For example:

- In a Taiwanese survey of workers exposed to 85-90 dBA and under audiological surveillance, notches (at 3, 4 or 6 KHz) were present in one-third of 4,598 ears (Chang *et al*, 2012).
- "Coles" notches were found in 63% of 12,055 railway workers from Norway and 4 kHz notches in 31% of the workers (Arve *et al*, 2015).
- In a survey of electrical transmission workers from New Zealand, which the audiograms being read by three independent assessors, at least two out of three assessors reported a high frequency notch in 49% of the study population (McBride *et al*, 2001).

45. Of particular concern, notching is comparatively common *even in those who appear never to have been exposed to noise occupationally*:

- In the Beavers Dam study (Gates *et al*, 1999), one-third of those with notching were not exposed to occupational noise and 11% appeared never to have been exposed to any noise at work or in leisure.
- In the Norwegian rail workers' study (Arve *et al*, 2015), "Coles" quote notches were found in 53% of non-noise exposed traffic controllers, among whom 21% had a notch at 4 kHz.
- In the New Zealand study of electrical transmission workers (McBride *et al*, 2001), a 4 kHz notch was found in 103 subjects described as exposed to noise only at low levels.
- In the audiology outpatient study from Surrey (Osei-Lah *et al* 2010), only three of 62 patients with a high frequency notch had a previous history of occupational exposure to noise.

46. There is disagreement among experts about how notches should be defined and when they are present. For example:

- In the Beavers Dam study (Gates *et al*, 1999), four different algorithms for diagnosis were applied and these generated markedly different prevalences of notching (varying from 12% to 47%).
- In the New Zealand study of electrical transmission workers (McBride *et al*, 2001), agreement between the three assessors was poor: when faced with the same audiograms, raters 1, 2 and 3 identified 26%, 49% and 68% of the individuals as

having a notched audiogram respectively, intra class correlations between the ratings of pairs of raters ranging from 0.14 to 0.52.

47. An expert consultee has previously highlighted that, while the first indications of NIHL occur at 4 KHz, the disability mainly arises from losses at lower frequencies. As the audiometric 4 KHz notch tends to be subsumed by lower frequency losses with disease progression, this raises the possibility that the notch and disabling loss tend not to be seen together.
48. The Council considers that the various clinical disagreements over diagnosing notches might be overcome with further work; also, that the imperfect sensitivity of the test is only a relative problem. It is widely thought that the current terms of PD A10 under-compensate cases of NIHL, but even if a minority of genuine cases exhibited a 4 KHz notch, then the terms of prescription could be extended at least to these individuals.
49. Another concern, however, arises from the risk of over-compensation. Had 4 KHz notches been uncommon in the absence of noise, had non-occupational causes of notching been uncommon, and provided that associations with occupational exposure to noise were much stronger than for sensorineural deafness as a whole, attribution to work could be made with improved confidence. However, the weak specificity of high-frequency notching (the fact that it is common even in the absence of noise) would appear to be a major impediment to the use of the notch in making occupational attribution in claimants of IIDB. The weak correlation with disability would further limit its utility in practice.

Ensuring consistency

50. In its 2002 Command Paper (Cm 5672), the Council succeeded in extending the terms of prescription for occupational deafness under several circumstances, notwithstanding the many challenges highlighted above.
51. It did so on the basis of measured noise levels in the job, assessed as in the 1988 report (Cm 817), namely as Leq (8 hr) (the equivalent continuous sound level over a working day normalised to an 8 hour exposure) (para 92). *“Occupations and processes studied were those where best information indicated they involved high noise levels, i.e.*

98dB(A) and above and with regular sustained exposure". Exposures of this magnitude were deemed compatible with those in occupations already prescribed. Although the original list did not have an accompanying list of exposure measurements, additions to the list were made for occupations with exposures of this magnitude in the 1970s on the strength of the Burns-Robinson model.

52. The Council therefore accepted a case for ensuring consistency with previous reasonable decisions taken within the Scheme and *avoiding the anomaly that would arise if occupations proven to have similar exposures to those already prescribed were denied benefit*.

53. In principle this has provided a straight-forward basis by which the prescription coverage could be extended over time. Simply, the Council seeks evidence of sufficient exposure to noise in the occupation in question. Following this model, the Council calls for evidence that workers using specific tools or in specific occupations have been exposed to noise levels over 98 dB(A) averaged over an 8-hour working day. In practice, prescription is often limited by the availability of such exposure data; but the criteria for prescription can be clearly specified and have sometimes enabled the eligible pool of workers to be enlarged, albeit in a piecemeal fashion.

54. It is recognised that this threshold for compensation lies well above that requiring actions to protect workers' hearing under the Control of Noise at Work Regulations, 2005.

Acoustic trauma

55. Acute acoustic trauma (from sudden loud explosions) is compensable under the Scheme's accident provisions, rather than the terms of PD A10. Damage of this kind differs importantly from NIHL in that it may be unilateral, or far more severe in one ear, while the loss sustained at work occurs abruptly, and not over a prolonged interval of time during which the individual ages and adjusts. Attribution to work is more straight-forward than for NIHL gradually acquired from sustained exposure to noise.

56. The Council has been informed that no threshold akin to the case definition for PD A10 (at least 50dB of loss in both ears over certain frequencies) is applied in determining

entitlement to benefit. Assessment of percentage disablement is made – as for other occupational injuries – using Schedule 2 from the Social Security (General Benefit) Regulations 1982 (table of Statutory Scheduled Assessments), as interpreted by the courts and tribunals, as a guiding framework.

Summary and conclusions

57. As well as summarising the history of PD A10, this review has sought to share thinking on two broad questions relating to its prescription: (i) Can the exposure terms of PD A10 be framed in terms of a qualifying *level of noise*? (ii) Can the *disease* terms of PD A10 be framed in terms of a diagnostically specific clinical feature, such as an audiometric notch?
58. On present evidence, neither of the options seems possible. In lieu of this, the present approach, used to extent the terms of this difficult prescription, have been outlined (paragraphs 50-53).
59. The Council welcomes further evidence relevant to this prescription and suggestions for alternative approaches to prescription that are compatible with the science, the legislation and are feasible to implement. Evidence and comments can be sent at any time to the Council's secretariat by emailing iiac@dwp.gsi.gov.uk.

Prevention

60. The Control of Noise at Work Regulations 2005 requires employers to prevent or reduce risks to health and safety from exposure to noise at work. The HSE has produced practical guidance to help employers identify whether they have a noise problem in the workplace, and if so, how to carry out a risk assessments to decide what action is needed to protect their employees.
61. Risk assessments should identify where in the workplace there may be a risk from noise and who is likely to be affected. They should contain estimates of employees' noise exposure and identify appropriate noise-control measures and hearing protection. Consideration should be given to whether any employees need to be provided with

health surveillance, and whether any are at particular risk. Employers should provide employees with appropriate information and training. In turn, employees are required to co-operate with employers in implementing measures to control exposure.

62. Further information about controlling noise exposure at work is available on the HSE website at: <http://www.hse.gov.uk/noise/index.htm>.

GLOSSARY OF TERMS

Acoustic trauma

Deafness caused by a sudden loud noise in the ear, e.g. firearms, rifles. Usually one ear is very much more affected than the other. The hearing loss is greatest at 4 kHz and often temporary, improving even to normal with time, but it can be permanent.

Conductive hearing loss

A form of hearing impairment where there is an abnormality of the external or middle ear which prevents the normal transmission of sound to the inner ear.

Deafness

Total hearing loss.

Decibel (dB)

The unit used for expressing the physical magnitude of sounds.

Disability

A disability is any restriction or lack (resulting from an impairment) of ability to perform an activity in the manner or within the range considered normal for a human being. Disability represents a departure from the norm in terms of performance of the individual, as opposed to that of the organ or mechanism.

Hearing Level

For a specified frequency of pure-tone and testing system, the sound pressure level (essentially the physical magnitude of the sound in the case of air conduction audiometers) or vibratory force level (essentially the physical magnitude of vibration in the case of bone conduction audiometers) of the tone relative to that of a reference zero (as defined by an International or National Standard). It is the dial setting of an audiometer at which sound is heard if the instrument has been properly calibrated. Expressed in decibels, i.e., as dB HL.

Hearing Loss

An impairment of hearing that exceeds a specified level. No units, but may be qualified in

terms of severity as mild, severe etc.

Hearing threshold level (HTL)

For a particular ear, and a given frequency and test system, it is an individual's threshold of hearing (i.e., the quietest sound that he can hear) as determined in a stated manner and expressed by the system's indicated hearing level value. Expressed in decibels, i.e., as dB HTL.

Hearing threshold shift

A change in the threshold of hearing for a given frequency (or group of frequencies) over a particular period of time: expressed in decibels.

Hertz (Hz)

Unit of frequency (formerly cycles per second)

Impairment

Any loss or abnormality of psychological, physiological, or anatomical structure or function.

Noise-induced sensorineural hearing loss

Sensorineural hearing loss due to chronic exposure to noise, whether leisure, social or occupational. The loss is greatest in the highest frequencies, particularly at 4-6 kHz and is usually bilateral and symmetrical in both ears. Initially, loss is temporary, then as exposure continues it becomes permanent.

Non-organic hearing loss (also known as exaggerated or over-estimated hearing loss)

Here the patient complains of hearing loss or a degree of deafness which cannot be identified or accounted for objectively.

Occupational noise-induced hearing loss

Noise-induced sensorineural hearing loss caused by exposure to loud noise at work over an extended time period.

Sensorineural hearing loss

A form of hearing impairment in which the abnormality is in the inner ear (cochlea), the auditory nerve or in the brain itself. There are various causes, including constitution, age and noise.

Tinnitus

A subjective noise sensation, not associated with any external acoustic, electrical or mechanical stimulus. Often described as ringing, heard in one or both ears. It may relate to acoustic trauma, Meniere's Disease or otosclerosis, or reflect age or noise-related hearing loss, or occur for no apparent reason.

Sensitivity and specificity (of a test)

In medical diagnosis or screening, test **sensitivity** measures the ability of a test to correctly identify those with the disease (true positive rate), whereas test **specificity** measures the ability of the test to correctly identify those without the disease (true negative rate). Both of these measures can be expressed as probabilities, ranging from 0 to 1, or as a percentage.

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Appendix 1: The current prescription of PD A10

Prescribed disease	Any occupation involving:
<p>A.10. Sensorineural hearing loss amounting to at least 50 dB in each ear, being the average of hearing losses at 1, 2 and 3 kHz frequencies, and being due in the case of at least one ear to occupational noise (occupational deafness).</p>	<p>The use of, or work wholly or mainly in the immediate vicinity of the use of, a –</p> <p>(a) band saw, circular saw or cutting disc to cut metal in the metal founding or forging industries, circular saw to cut products in the manufacture of steel, powered (other than hand powered) grinding tool on metal (other than sheet metal or plate metal), pneumatic percussive tool on metal, pressurised air arc tool to gouge metal, burner or torch to cut or dress steel based products, skid transfer bank, knock out and shake out grid in a foundry, machine (other than a power press machine) to forge metal including a machine used to drop stamp metal by means of closed or open dies or drop hammers, machine to cut or shape or clean metal nails, or plasma spray gun to spray molten metal;</p> <p>(b) pneumatic percussive tool to drill rock in a quarry, on stone in a quarry works, underground, for mining coal, for sinking a shaft, or for tunnelling in civil engineering works;</p> <p>(c) vibrating metal moulding box in the concrete products industry, or circular saw to cut concrete masonry blocks;</p> <p>(d) machine in the manufacture of textiles for:- weaving man-made or natural fibres (including mineral fibres), high speed false twisting of fibres, or the mechanical cleaning of bobbins;</p> <p>(e) multi-cutter moulding machine on wood, planing machine on wood, automatic or semi-automatic lathe on wood, multiple cross-cut machine on wood, automatic shaping machine on wood, double-end tenoning machine on wood, vertical spindle moulding machine (including a high speed routing machine) on wood, edge banding machine on wood, bandsawing machine (with a blade width of not less than 75 millimetres) on wood, circular sawing machine on wood including one operated by moving the blade towards the material being cut, or chain saw on wood;</p> <p>(f) jet of water (or a mixture of water and abrasive material) at a pressure above 680 bar, or jet channelling process to burn stone in a quarry;</p> <p>(g) machine in a ship's engine room, or gas turbine for performance testing on a test bed, installation testing of a replacement engine in an aircraft, or acceptance testing of an Armed Service fixed wing combat aircraft;</p> <p>(h) machine in the manufacture of glass containers or hollow ware for automatic moulding, automatic blow moulding, or automatic glass pressing and forming;</p> <p>(i) spinning machine using compressed air to produce glass wool or mineral wool;</p> <p>(j) continuous glass toughening furnace;</p> <p>(k) firearm by a police firearms training officer; or</p> <p>(l) shot-blaster to carry abrasives in air for cleaning.</p>

Appendix 2: The Burns Robinson report

The work of Burns and Robinson, a joint study between the MRC and National Physical Laboratory, is reported in a book published by the DHSS.

Some 32 factories from 239 initially considered were studied during 1963-7. Factors underlying selection included reasonable numbers of workers exposed to 'noise of sufficient intensity through the working day', and a reasonable number of previously unexposed school leaver entrants. Processes covered included: sugar packaging, caulking, chipping, riveting, machining ball bearings, pressing cans and metal boxes, sparking plug assembly, fettling, spinning & weaving, and printing.

Within participating companies, managers helped the investigators to screen their personnel to exclude workers (i) whose noise exposures were 'not amenable to quantitative description'; (ii) who had served in the armed forces, or had been exposed to gunfire, or had previous noise exposure from a different occupation; (iv) who were known to have pre-existing ear disease; or (v) had language difficulty. Further exclusions were applied following an otological examination (11% of those reaching this stage of consideration were deemed to have another ear pathology).

Analyses were finally based on 759 workers (400 and 581 in preliminary analyses). Response rates were not mentioned, although the study was described as one of volunteers.

Participants underwent PTA – about 4,000 audiograms in all, and so involving repeated measurements over time. No details were given of the number of workers with 1, 2, 3, 4, or 5 records, but 91 visits were made to the 32 factories over the 5 years of data collection, about 3 per factory: the handling of serial observations within individuals is not described.

Finally, measurements of noise exposure were made in the various workplaces. The technical details of field measurement were reported in detail, but the number of measurements and their locations, the duration of sampling, and the method of relating location measurements to individuals' work histories were not stated. Noise exposures ranged from 75 to 120dB (80 to 110 dB in dose-response analyses) but no details were given of the distribution. In

constructing an individual's lifetime exposure to noise, account was taken of their questionnaire responses, their duration of employment in calendar months, and noise levels considering the detail of "the factory layout, work methods, any changes to plant and machinery, and employers' records, obtained in consultation with factory managers and personnel departments". No detail is given of the method and few on the underlying assumptions. Presumably, however, these included an assumption about the representativeness of noise measurements over time and place, and the applicability of contemporaneous area-based measures to historic personal exposures. (Effort had been made to assemble subjects only working in the same occupation and no other, with no other sources of noise exposure).

It was also assumed that the effects of age and noise on hearing were additive, although a 'less than additive at older ages' model has since been favoured by some, including Professor Robinson.

Thus, exposure-response relationships were modelled on largely cross-sectional data. Despite a remarkable amount of careful fieldwork, the estimation of historic personal exposures remained a major challenge.

