Summary of BPA submissions on the new Hearing Loss EEG

1. The guidelines demand a level of causal connection more stringent than the law requires. They do not allow for entitlement based on a significant, but less than primary, contribution from service noise exposure and/or early service-related acoustic trauma.
    - *Cole v. Canada,* 2015 FCA 119

2. Such restrictive guidelines are not supported by the available medical evidence. On the contrary, the evidence demonstrates that non-disabling noise exposure from service may significantly contribute to the development of future hearing loss disability in many, if not most, cases. This is because:

    a. Noise damage is progressive and cumulative in nature.
        - Exhibit 1: Liberman, M. C., & Kujawa, S. G. (2017). Cochlear synaptopathy in acquired sensorineural hearing loss: Manifestations and mechanisms. *Hearing Research*, *349*, 138–147
        - Exhibit 2: Dr. J. Rutka, VRAB Discussion Paper on Hearing Loss, updated December 2011, (excerpts) pp. 14, 32
        - Exhibit 3: Noise and sound: Hearing loss and tinnitus, Health Canada, 2024-05-29, p. 7, 8
        - Exhibit 4: Merck Manual Professional Version: Hearing Loss, p. 11
        - Exhibit 5: Yong, Wang. Impact of noise on hearing in the military. *Military Medical Research,* 2015, p. 2-3
        - Exhibit 6: Kujawa SG, Liberman MC. Adding insult to injury: cochlear nerve degeneration after "temporary" noise-induced hearing loss. J Neurosci. 2009 Nov 11;29(45):14077-85, p. 1, 8
        - Exhibit 7: Liberman MC. Noise-induced and age-related hearing loss: new perspectives and potential therapies. *F1000Research* 2017, 6 (F1000 Faculty Rev):927 p. 1, 3, 7
        - Exhibit 17: Gates et. al. (2000). Longitudinal threshold changes in older men with audiometric notches. *Hearing Research 141,* 220-228, p. 1

    b. Early noise exposure contributes to and accelerates age-related hearing loss.
        - EEGs Hearing Loss, p. 9
        - Exhibit 2: Dr. J. Rutka, VRAB Discussion Paper on Hearing Loss, updated December 2011, (excerpts), p. 17, 32
        - Exhibit 3: Noise and sound: Hearing loss and tinnitus, Health Canada, 2024-05-29, p. 7, 8
        - Exhibit 4: Merck Manual Professional Version: Hearing Loss, p. 11
        - Exhibit 7: Liberman MC. Noise-induced and age-related hearing loss: new perspectives and potential therapies. F1000Research 2017, 6 (F1000 Faculty Rev):927, p. 3
        - Exhibit 8: Kujawa, S. G., & Liberman, M. C. (2006). Acceleration of Age-Related Hearing Loss by Early Noise Exposure: Evidence of a Misspent Youth. *The Journal of Neuroscience*, *26*(7), 2115–2123, p. 2

- Exhibit 9: Fernandez, Jeffers, Lall, Liberman and Kujawa, “Aging after Noise Exposure: Acceleration of Cochlear Synaptopathy in “Recovered” Ears”, 2015, *The Journal of Neuroscience*, 35(19): 7509.
- Exhibit 10, Yamasoba T, Lin FR, Someya S, Kashio A, Sakamoto T, Kondo K. Current concepts in age-related hearing loss: epidemiology and mechanistic pathways. Hear Res. 2013 Sep;303:30-8. doi: 10.1016/j.heares.2013.01.021. Epub 2013 Feb 16. PMID: 23422312; PMCID: PMC3723756, p. 1-2
- Exhibit 11: Fink, D. “What is the safe noise exposure level to prevent noise-induced hearing loss?” Journal of Exposure Science & Environmental Epidemiology, 2025, p. 2
- Exhibit 12: Kohrman, et. al. “Hidden Hearing Loss: A Disorder with Multiple Etiologies and Mechanisms”, Cold Spring Harbour Perspectives in Medicine, 2020, p. 4
- Exhibit 17: Gates et. al. (2000). Longitudinal threshold changes in older men with audiometric notches. *Hearing Research 141,* 220-228, p. 1
- Exhibit 18: World Health Organization. World Report on Hearing (2021) (excerpts, p. 7)

3. The guideline’s reliance on threshold audiograms is inconsistent with:

   a. the military environment where members are trained to adopt a mission-first attitude and where audiograms are not routinely performed after high-noise events likely to have caused a temporary threshold shift (TTS). Many TTS are likely to occur but remain undetected and therefore absent from service health records.
      - VRAB Decisions 100003937933; 100004407577

   and with

   b. current medical evidence on the impact of “hidden hearing loss”, which demonstrates that losses not visible on a threshold audiogram are nevertheless capable of causing both immediate and future hearing dysfunction.
      - Exhibit 1: Liberman, M. C., & Kujawa, S. G. (2017). Cochlear synaptopathy in acquired sensorineural hearing loss: Manifestations and mechanisms. *Hearing Research*, *349,* 138–147, p. 1-2
      - Exhibit 2: Dr. J. Rutka, VRAB Discussion Paper on Hearing Loss, updated December 2011, (excerpts), p. 11, 32
      - Exhibit 4: Merck Manual Professional Version: Hearing Loss, p. 10
      - Exhibit 6: Kujawa SG, Liberman MC. Adding insult to injury: cochlear nerve degeneration after "temporary" noise-induced hearing loss. J Neurosci. 2009 Nov 11;29(45):14077-85, p. 1, 7-8
      - Exhibit 7: Liberman MC. Noise-induced and age-related hearing loss: new perspectives and potential therapies. *F1000Research* 2017, 6 (F1000 Faculty Rev):927 p. 8
      - Exhibit 11: Fink, D. “What is the safe noise exposure level to prevent noise-induced hearing loss?” Journal of Exposure Science & Environmental Epidemiology, 2025, p. 1
      - Exhibit 12: Kohrman, et. al. “Hidden Hearing Loss: A Disorder with Multiple Etiologies and Mechanisms”, Cold Spring Harbour Perspectives in Medicine, 2020, p. 3

- Exhibit 13: Bramhall et al. Auditory Brainstem Response Altered in Humans With Noise Exposure Despite Normal Outer Hair Cell Function. U.S. Department of Veterans Affairs. Ear Hear. 2017; 38(1): e1 –e12. https://pubmed.ncbi.nlm.nih.gov/27992391/
- Exhibit 14: Waddell K, Wu N, Demaio P, Bain T, Bhuiya A, Wilson MG. Rapid evidence profile #71: Examining the association between noise exposure and hearing loss. Hamilton: McMaster Health Forum, 10 May 2024
- Exhibit 18: World Health Organization. World Report on Hearing (2021) (excerpts, p. 7)

4. The available evidence supports a reasonable inference that military noise exposure in particular is likely to cause significant hearing damage within extremely short exposure periods.
    - Entitlement Eligibility Guidelines: Hearing Loss, p. 7
    - Exhibit 4: Merck Manual Professional Version: Hearing Loss, p. 12
    - Exhibit 5: Yong, Wang. "Impact of noise on hearing in the military". *Military Medical Research,* 2015, p. 2-3) *Military Medical Research,* 2015, p. 2-3
    - Exhibit 15: CCOHS Noise – Occupational Exposure Limits in Canada, Table 1.A, extension added.
    - Exhibit 16: Yankaskas, K. (2013). Prelude: Noise-induced tinnitus and hearing loss in the military. *Hearing Research*, *295*, 3– 8

CONCLUSION:

We submit that the 2025 guideline criteria for hearing loss fail to apply the causal connection standard set out in the legislation; that is, the significant causal factor standard. The guidelines fail to follow the legislative requirements to draw every reasonable inference from all available evidence and to respect the benefit of the doubt principle.

We submit that the evidence noted here supports two key reasonable inferences that support entitlement in hearing loss disability claims:

(1) a reasonable inference that the nature and levels of noise exposure common in the military environment likely cause significant hearing damage, whether or not decibel losses were captured on audiograms during service.

(2) a reasonable inference that such service-related damage is likely a significant contributor to the later onset of a hearing loss disability.

BPA's submission on the new Hearing Loss EEGs

The new Entitlement Eligibility Guideline for Hearing Loss now requires audiometric evidence that service noise exposure, other than acoustic trauma, directly caused a *disabling* level of hearing loss during or just prior to the clinical onset or aggravation of that disability before Veterans Affairs will recognize that the disability is sufficiently connected with military or RCMP service for entitlement purposes.

The criterion for acoustic trauma requires evidence that the trauma occurred "just prior" to the clinical onset or aggravation of sensorineural hearing loss.

With respect, we submit that the new guideline demands a causal connection standard more stringent than the law requires. Moreover, neither criterion is supported by current medical evidence, which demonstrates that non-disabling noise-induced hearing damage, including even a single episode of acoustic trauma, significantly contributes to and/or accelerates the development of hearing disability later in life.

The guideline is inconsistent with the legislation

The most authoritative jurisprudential statement of the causal connection standard required by Veteran's legislation is *Cole v. Canada (Attorney General)*, 2015 FCA 119. The Federal Court of Appeal ruled that a disability is "directly connected with" service when "service was a significant factor in bringing about that claimed condition." The Court firmly rejected the notion that service factors must be *more significant* than other contributing factors in order to justify entitlement to veterans' disability benefits. (*Cole,* paras. 89-92, 97)

We submit that the new guideline for noise-induced hearing loss and acoustic trauma is inconsistent with the Federal Court of Appeal's ruling. By requiring evidence that service noise exposure was "of sufficient intensity and duration" to cause *disabling* hearing loss "during or just prior" to the onset of disability, the criterion demands much more than a significant contribution from service. It demands evidence sufficient to establish that service noise exposure was likely the *primary cause* of the hearing loss disability. As a result, the new guideline requires a level of causal connection to service that the Federal Court of Appeal has definitively rejected as contrary to the purpose and scope of the benefits scheme.

Pursuant to the "significant factor" causal connection requirement, we submit that the new guideline cannot be justified unless it is supported by medical evidence demonstrating that non-disabling noise damage and/or early acoustic trauma are *insignificant* in the later development of a hearing loss disability. (*Cole,* paras. 98-99, 121) The current medical evidence, including medical literature referenced by the new guideline as well as evidence from Health Canada and others, does not support that conclusion.

Evidence that non-disabling noise damage significantly contributes to later-onset hearing loss disability

(a) <u>Noise damage is cumulative:</u>

The new guideline relies on the assertion that "any SNHL [sensorineural hearing loss] related to noise exposure does not progress after the noise exposure has stopped." However, research published in 2017 found that "[c]ontrary to existing dogma, results demonstrated that noise can cause ongoing changes in cochlear structure and function <u>long after it has ceased</u>." (Exhibit 1: Liberman, M. C., & Kujawa, S. G. (2017). Cochlear synaptopathy in acquired sensorineural hearing loss: Manifestations and mechanisms. *Hearing Research*, *349*, 138–147; Exhibit 17: Gates et. al. (2000). Longitudinal threshold changes in older men with audiometric notches. *Hearing Research 141,* 220-228, p. 1)

Importantly, the guideline's narrow view of the impact of noise exposure also fails to acknowledge the *cumulative* nature of noise-induced hearing damage: the addition of more noise exposure on noise-damaged ears results in increased damage and eventually hearing loss disability. The original noise-induced damage thus contributes to the total accumulation of noise damage resulting in disability. (Exhibit 2: Dr. J. Rutka, VRAB Discussion Paper on Hearing Loss, updated December 2011, pp. 14, 32; Exhibit 3: "Noise and sound: Hearing loss and tinnitus", Health Canada, 2024-05-29, p. 7, 8)

Moreover, in an apparent internal contradiction, even the new guideline acknowledges that noise-induced hearing damage progressively worsens with additional noise exposure. The guideline states that "[w]ith continued noise exposure, the HL becomes permanent because of irreparable damage to the cochlea's hair cells." (EEG, p. 7). Obviously, based on this acknowledgement, a veteran with non-disabling hearing

damage from military noise exposures is more likely to suffer disabling hearing loss later in life than someone without previous noise damage if both individuals later experience the same additional noise exposure.

The cumulative nature of noise-induced hearing loss is explained in part by the fact that we are born with a finite number of hair cells in the cochlea. “Repeated exposure to loud noise ultimately results in loss of hair cells...” (Exhibit 4: Merck Manual Professional Version: Hearing Loss, p. 11) Once those cells are destroyed by noise, they cannot be replaced. (Exhibit 3: “Noise and sound: Hearing loss and tinnitus”, Health Canada, 2024-05-29, p. 6) When additional noise exposure is layered over early noise damage, more hair cells are destroyed and the resulting accumulated loss causes hearing disability. In this way, noise-induced hearing damage that is initially non-disabling significantly contributes to the later onset of hearing disability.

To borrow a mathematical example from *Cole,* if 49% of the hair cells required to avoid a disabling level of hearing loss are damaged by service noise exposure, and the remaining 51% are damaged by post-service noise exposure and aging, the new guideline would not permit entitlement to be granted for the resulting hearing loss disability because it would not have met the audiometric definition of disability prior to release. Nevertheless, we submit that the service noise exposure would clearly have significantly contributed to the onset of the claimed hearing loss disability, thus meeting the statutory requirements for entitlement.

Understanding the full, cumulative impact of early noise exposure is complicated by the fact that hearing threshold levels can “recover” after a temporary threshold shift (“TTS”) caused by excessive noise. This has led to the theory that noise-induced hearing damage is “reversible” because observed decibel losses no longer remain *clinically apparent* on audiometric testing. This does not mean, however, that there has been no significant damage capable of contributing to later-onset hearing disability. As Dr. Rutka acknowledges in his discussion paper originally written in 2010:

> . . . not all hair cells possibly recover following a TTS but enough do so as to prevent hearing loss. Continued exposure to excessive noise will therefore result in <u>further</u> hearing loss.

> In other words, when an ear sustains a TTS, it is conceivable that there may be some permanent injury to some of the inner/outer hair cells at the frequencies tested that is not reversible. Thankfully there is a significant amount of redundancy in the inner ear. It is only when a quantum number of hair cells are injured irreparably that we then begin to clinically notice a permanent threshold shift (PTS) in hearing.
>
> (Exhibit 2: Dr. J. Rutka, VRAB Discussion Paper on Hearing Loss, updated December 2011, p. 32, emphasis added)

Health Canada recognizes that when an individual experiences temporary hearing loss or tinnitus after a loud sound exposure (i.e. a TTS), "it means there was permanent damage done to some of [their] thousands of hair cells. They may have died off or become very broken. That damage will add up to permanent problems over time." (Exhibit 3: "Noise and sound: Hearing loss and tinnitus", Health Canada, 2024-05-29, p. 7, emphasis added)

Moreover, noise-induced hearing loss is not related to hair cell loss alone. Research has shown that it also involves auditory nerve degeneration and synaptic impairment. A 2015 article on the "Impact of Noise on Hearing in the Military" explains that during a TTS, "[e]ven though hair cells recover normal function, there is rapid extensive and irreversible loss of synapses and delayed and progressive loss of cochlear neurons over many months." (Exhibit 5: Yong, Wang. "Impact of noise on hearing in the military". *Military Medical Research,* 2015, p. 2-3, emphasis added)

Dr. Liberman's research from 2009 on hair cell synapses and cochlear nerve terminals indicated that "noise-induced damage to the ear has progressive consequences that are considerably more widespread than are revealed by conventional threshold testing." His research demonstrated "that reversibility of noise-induced threshold shifts mask progressive underlying neuropathology that likely has profound long-term consequences on auditory processing." (Exhibit 6: Kujawa SG, Liberman MC. Adding insult to injury: cochlear nerve degeneration after "temporary" noise-induced hearing loss. J Neurosci. 2009 Nov 11;29(45):14077-85, p. 1, 8)

Additional research conducted in 2017 demonstrated that noise exposures "causing only reversible threshold shifts (and no hair cell loss) nevertheless cause permanent loss of >50% of the synaptic connections between hair cells and the auditory nerve." This destruction contributes to hearing disability, such as difficulty understanding speech with background noise, but it "does not affect tests of threshold detection so long as the loss is not complete." (Exhibit 7: Liberman MC. Noise-induced and age-related hearing loss: new perspectives and potential therapies. *F1000Research* 2017, 6 (F1000 Faculty Rev):927 p. 1, 3, 7, emphasis added)

We submit that this evidence is sufficient to support a reasonable inference that non-disabling hearing damage from service noise exposures, even after threshold "recovery", nevertheless significantly contributes to the later onset of hearing disability.

(b) Noise damage accelerates age-related hearing loss:

In addition to the accumulation of noise damage explained above, recent research has also demonstrated that noise-exposed ears age differently than ears without a history of significant noise exposure. According to the research, "previous noise exposure has significant, deleterious effects on the nature and progression of an age-related hearing loss." (Exhibit 8: Kujawa, S. G., & Liberman, M. C. (2006). Acceleration of Age-Related Hearing Loss by Early Noise Exposure: Evidence of a Misspent Youth. *The Journal of Neuroscience*, *26*(7), 2115–2123, p. 2; Exhibit 17: Gates et. al. (2000). Longitudinal threshold changes in older men with audiometric notches. *Hearing Research 141,* 220-228, p. 8)

Additional studies completed in 2015 presented "clear evidence that prior noise dramatically exacerbates synaptic and neural losses that otherwise occur with aging." Even "apparently reversible noise damage can have dramatic long-term consequences in amplifying age-related sensorineural hearing loss…" (Exhibit 9: Fernandez, Jeffers, Lall, Liiberman and Kujawa, "Aging after Noise Exposure: Acceleration of Cochlear Synaptopathy in "Recovered" Ears", 2015, The Journal of Neuroscience, 35(19): 7509.)

This research has been accepted and cited within the Merck Manual Professional Version, a medical text that is readily available to the public and medical practitioners.

The Merck Manual states: “Research also strongly suggests that early noise exposure accelerates age-related hearing loss.” (Exhibit 4: *Merck Manual Professional Version: Hearing Loss,* p. 11)

Health Canada has come to the same conclusion. After acknowledging that “[t]he effect that loud sounds have on your hearing is cumulative”, they explain: “Repeated exposure to loud sounds and noise in earlier years could speed up age-related hearing loss in later life, more than what would be expected due to the natural process of aging.” (Exhibit 3: Noise and sound: Hearing loss and tinnitus, Health Canada, 2024-05-29, p. 8)

Moreover, it is now generally accepted that age-related hearing loss is not, in fact, merely age-related. Rather it involves *the combined effect* of noise exposure and deterioration due to the aging process. Even the new guidelines acknowledge that age-related hearing loss is “multifactorial” and “includes hearing loss due to significant noise exposure.” (EEGs, p. 9; See also: Exhibit 10, Yamasoba T, Lin FR, Someya S, Kashio A, Sakamoto T, Kondo K. Current concepts in age-related hearing loss: epidemiology and mechanistic pathways. Hear Res. 2013 Sep;303:30-8. doi: 10.1016/j.heares. 2013.01.021. Epub 2013 Feb 16. PMID: 23422312; PMCID: PMC3723756, p. 1-2)

Although the proportional contribution of aging vs. noise damage is impossible to ascertain with any rigour, there is evidence that noise exposure plays a substantial role. Dr. Rutka admits that “not everyone as they age will experience age-related presbycutic change.” (Exhibit 2, p. 17) Dr. Liberman explains: “The two types of hearing loss are likely interrelated, as people in minimally industrialized areas (e.g. the Sudanese desert) do not show the inexorable age-related deterioration of hearing seen in the developed world.” (Exhibit 7: Liberman MC. Noise-induced and age-related hearing loss: new perspectives and potential therapies. F1000Research 2017, 6 (F1000 Faculty Rev):927, p. 3) An article published in 2025 states: “What is often called age-related hearing loss or presbycusis largely represents the effects of cumulative lifetime noise exposure on the ears.” (Exhibit 11: Fink, D. “What is the safe noise exposure level to prevent noise-induced hearing loss?” Journal of Exposure Science & Environmental Epidemiology, 2025, p. 2)

This evidence establishes that hearing loss, absent significant noise exposure, is not an inevitable part of growing older. Rather, age-related hearing loss involves a significant contribution from accumulated noise exposure, including in this context early service noise exposure.

Finally, there is some evidence “that a single exposure to moderate noise levels (TTS type) early in life predisposes to an accelerated, progressive hearing loss across the life span (Kujawa and Liberman 2009; Fernandez et al. 2015).” (Exhibit 12: Kohrman, et. al. “Hidden Hearing Loss: A Disorder with Multiple Etiologies and Mechanisms”, Cold Spring Harbour Perspectives in Medicine, 2020, p. 4)

Accordingly, we submit that the current medical evidence does not support a negative inference that aging operates independently to cause the onset of hearing disability to the *exclusion* of a significant contribution from damaging levels of service noise exposure. Since both age-related and noise-induced hearing loss are multifactorial, no reduction in entitlement for the effects of age is warranted.

Limitations of threshold audiograms

The new guideline is also problematic because it relies heavily on threshold audiograms to demonstrate the necessary causal connection to service. Such a requirement ignores both the realities of the military environment and the known progression of hearing disability described above.

Periodic service audiograms likely miss temporary threshold shifts:

While RCMP and CAF members may undergo periodic audiograms during their service, these tests are generally performed as part of routine, pre-scheduled medical examinations, often years apart, with no connection to periods of intense noise exposure. Members are not rushed to an audiologist simply because they have been exposed to loud and potentially damaging noises. This is why several VRAB panels have noted that it is “entirely plausible that a temporary threshold shift can occur but never be detected”. (See for example: VRAB Decisions 100003937933; 100004407577)

Nor can we reasonably expect that non-audiometric evidence of temporary threshold shifts, such as complaints of temporary tinnitus, ear "fullness", or depressed hearing, would be captured in the service health records. Serving members are expected to continue in their duties and are trained to adopt a "mission first" attitude. Symptoms that are temporary, intermittent, primarily annoying rather than painful, and don't prevent a member from getting on with the job are unlikely to be reported. The likelihood drops further for those whose positions could be jeopardized by admitting problems with hearing acuity. Moreover, as no treatment is available for such symptoms, no purpose is served by reporting to the MIR.

We submit that when the realities of the military environment and training are taken into account, the absence of demonstrable temporary threshold shifts on service audiograms or hinted at in service health records cannot reasonably support the conclusion that no such shifts likely occurred.

Significant hearing damage may exist despite "normal" audiograms

Substantial research has been conducted in recent years on the phenomenon of "hidden hearing loss", a term describing significant hearing damage not visible on threshold audiograms.

First, the VRAB Discussion Paper written by Dr. Rutka acknowledges that members and veterans may have experienced irreparable damage to their hearing despite the absence of "an appreciable change in pure tone threshold hearing." (Exhibit 2: p. 11, emphasis added) Dr. Rutka explains: "Although an individual may have apparently normal hearing it does not necessarily mean the cochlear nerve is undamaged." (Exhibit 2: p. 11, emphasis added) Even hair cell loss from noise exposure does not become visible on an audiogram until "a certain critical point is breeched." (Exhibit 2: p. 31, emphasis added)

Dr. Liberman's 2017 research demonstrated that the same is true for cochlear neural damage caused by excessive noise:

> Existing data from humans and animals make it clear that significant cochlear neural damage can occur without hair cell damage and thus can **hide behind a normal audiogram**. This neural damage is likely to be a

handicap in difficult listening situations, especially as overt hearing loss (i.e. threshold elevation and hair cell damage) is added to the mix. (Exhibit 7: Liberman MC. Noise-induced and age-related hearing loss: new perspectives and potential therapies F1000Research 2017, 6(F1000 Faculty Rev):927*,* p. 8, emphasis added.)

Dr. Liberman explained that this form of neural damage is called "hidden hearing loss" because "the damage is not visible in routine cochlear histopathology and because primary neural degeneration **does not significantly affect the threshold audiogram until it exceeds ~80%.**" (Exhibit 7: Liberman MC. Noise-induced and age-related hearing loss: new perspectives and potential therapies F1000Research 2017, 6 (F1000 Faculty Rev):927*,* p. 8, emphasis added.)

Although this research has been primarily based on animal models in controlled laboratory studies because detection methods are invasive, there has been some "recent confirmation of the same effects in post-mortem studies of human temporal bone specimens." (Exhibit 11: Fink, D. "What is the safe noise exposure level to prevent noise-induced hearing loss?" Journal of Exposure Science & Environmental Epidemiology, 2025, p. 1)

Another study published in 2017 revealed results "similar to the decreased ABR wave I amplitudes observed in animal models of noise-induced cochlear synaptopathy" for veterans and non-veterans with a history of firearms use. (Exhibit 13: Bramhall et al. Auditory Brainstem Response Altered in Humans With Noise Exposure Despite Normal Outer Hair Cell Function. U.S. Department of Veterans Affairs. Ear Hear. 2017; 38(1): e1 –e12. https://pubmed.ncbi.nlm.nih.gov/27992391/)

In addition, a recent review by McMaster University identified eight relevant non-animal studies. Five of these reported "biomarkers consistent with cochlear synaptopathy among military personnel and Veterans with a history of impulsive noise exposure." A sixth study, described as a "low-quality evidence synthesis" identified a weak (but nevertheless present) association between noise exposure history and auditory nerve responses. Two additional studies also found evidence consistent with a significant contribution from prior noise exposure to new-onset hearing loss:

One study re-analyzed data from three published studies on the effects of noise exposure on the progression of hearing loss and **found that noise exposure can accelerate the progression of hearing loss where the hearing loss is absent or mild at the end of military service** (i.e., threshold levels up to 50 db HL).(fn 7: Moore BCJ. The effect of exposure to noise during military service on the subsequent progression of hearing loss. *Int J Environ Res Public Health* 2021; 18(5): 2436)

The final study found that in a significant sample of U.S. military members (n=48,000), 7.5% reported new-onset hearing loss during follow-up surveys administered three years after the baseline reporting. New-onset hearing loss was associated with a history of combat deployment, being male, and older age. Among deployed military members, new-onset hearing loss was associated with reported proximity to improvised explosive devices and having experienced a combat-related head injury. (fn 8: Wells TS, Seelig AD, Ryan MA, Jones JM, Hooper TI, Jacobson IG, Boyko EJ. Hearing loss associated with US military combat deployment. *Noise Health* 2015; 17(74): 34-42)

(Exhibit 14: Waddell K, Wu N, Demaio P, Bain T, Bhuiya A, Wilson MG. Rapid evidence profile #71: Examining the association between noise exposure and hearing loss. Hamilton: McMaster Health Forum, 10 May 2024)

Additional research has indicated that “a single blast exposure may also result in HHL [hidden hearing loss] both in animals (Niwa et al. 2016; Hickman et al. 2018) and in humans (Bressler et al. 2017).” (Exhibit 12: Kohrman, et. al. “Hidden Hearing Loss: A Disorder with Multiple Etiologies and Mechanisms”, Cold Spring Harbour Perspectives in Medicine, 2020, p. 3) Another study demonstrated that “a single episode of synaptopathic noise early in life can exaggerate dramatically the loss of cochlear synapses and cochlear neurons that otherwise occurs with age and can produce delayed loss of threshold sensitivity and outer hair cells (OHCs). (Exhibit 9: Fernandez, Jeffers, Lall, Liberman and Kujawa, “Aging after Noise Exposure: Acceleration of Cochlear Synaptopathy in “Recovered” Ears”, 2015, *The Journal of Neuroscience*, 35(19): 7509, p. 2)

The World Health Organization also recognizes hidden hearing loss “attributed to the destruction of synaptic connections between hair cells and cochlear neurons (cochlear synaptopathy) which occurs well before the hair cells are damaged and as a

result of exposure to noise." (Exhibit 18: World Health Organization. World Report on Hearing (2021) (excerpts, p. 7))

As with noise-induced acceleration of age-related loss, these research conclusions have been accepted and re-published in generally accessible contemporary medical literature designed for both the public and medical professionals such as the Merck Manual:

> Even before hearing loss can be documented, noise exposure can damage auditory neurons and their synapses on hair cells; this damage is referred to as "hidden hearing loss" or "synaptopathy," and patients may notice difficulty hearing in noisy environments and have accelerated age-related hearing loss. …
> (Exhibit 4: Merck Manual Professional Version: Hearing Loss, p. 10)

We submit that the preponderance of the evidence cited here is sufficient to support a reasonable inference that service noise exposure likely causes damage to the cochlear neurons, synapses, hair cells or all three. This damage is irreversible. Although it may not immediately become apparent on a threshold audiogram, it is nevertheless significant, and it initiates a process of progressive deterioration that ultimately results in permanent hearing loss disability.

Evidence supporting a reasonable inference that military noise exposure causes significant hearing damage:

We submit further that there is medical evidence within the guideline for hearing loss itself that supports a reasonable inference that military noise exposure likely causes significant hearing damage. The guideline recognizes that gunfire and explosions from grenades and other artillery are capable of causing both acoustic trauma and hearing damage, such as a TTS. The guideline acknowledges the following facts at page 7:

- "The ear is particularly vulnerable to frequencies in the range of 2000 to 4000 Hz, or even 6000 Hz. These frequencies are likely to be generated … by gunfire, explosions, and some types of aircraft noise";
- "A single, intense exposure above 140 db can cause immediate hearing damage";

- The approximate decibel levels produced by rifles (163 dB), handguns (166 dB), shotguns (170 dB), artillery fire at 500 feet (150 dB) as well as military aircraft at take-off (140 – 150 dB) are mostly well in excess of the levels known to cause immediate hearing damage.

The Merck Manual lists “gun muzzle blast” and “jet engine” at 140 dB, and states that “even brief exposure injures unprotected ears; injury may occur even with hearing protectors.” (Exhibit 4: Merck Manual Professional Version, p. 12, emphasis added)

The previous version of the hearing loss guideline explicitly acknowledged that ““The maximum exposure time” for avoiding hearing loss on a single episode at only 110 decibels is one minute and 29 seconds.” The newest guideline refers readers to the *Canadian Centre for Occupational Health and Safety* website for occupational exposure limits. Following the “3 dB rule” noted there to be endorsed by “most experts”, the maximum daily exposure time for avoiding hearing loss at only 112 dB is less than one minute. At 121 dB it is 7 seconds, and at 130 dB it is less than one second. (Exhibit 15: CCOHS Noise – Occupational Exposure Limits in Canada, Table 1.A, extension added)

Research from 2015 that specifically examined the impact of noise on hearing in the military provides additional evidence of noise decibel levels common in the land, sea, and air environments. This evidence demonstrates that typical exposures in military environments are at levels likely to cause hearing damage either immediately, within minutes, or within at most two hours, according to the 3dB rule favoured by CCOHS. (Exhibit 5: Yong, Wang. “Impact of noise on hearing in the military”. *Military Medical Research,* 2015, p. 2-3) *Military Medical Research,* 2015, p. 2-3. See also Exhibit 16: Yankaskas, K. (2013). Prelude: Noise-induced tinnitus and hearing loss in the military. *Hearing Research*, *295*, 3– 8, which also describes hazardous noise environments in military settings)

On this evidence, and particularly with the benefit of s. 39 of the *VRAB Act,* we submit that it is reasonable to infer that military noise exposure causes significant hearing damage. These exposures often occur at frequencies to which the ear is known to be

"*particularly vulnerable*" and at decibel levels known to cause hearing damage immediately or within exceedingly short exposure periods.

As explained above, the absence of recorded threshold shifts on periodic service audiograms is not sufficient evidence to infer that no significant noise damage occurred.

CONCLUSION:

The new guideline repeats elements of the current Veterans Affairs policy on hearing loss, acknowledging that "[w]here it is determined that hearing loss was documented during service *or* at the time of discharge *and/or* service is reasonably found to be the initiating factor causing the current hearing loss disability, then entitlement to disability benefits may be granted."

However, the criteria set out in the guidelines for entitlement based on service noise exposure and acoustic trauma demand much more than this and are therefore of little to no assistance to veterans.

We submit this apparent contradiction is rooted in the department's failure to apply the causal connection standard set out in the legislation and explained in *Cole* as well as their failure to follow the legislative requirements to draw every reasonable inference from all available evidence and to respect the benefit of the doubt principle. Regrettably, the new guideline appears to take no notice of the legislation under which disability decisions must be made. We submit legislation must prevail.

Pursuant to the legislation, the questions that must be asked in every hearing loss claim are (1) Whether the evidence supports a reasonable inference that the member's service noise exposure likely caused hearing damage; and (2) Whether the evidence supports a reasonable inference that the hearing damage was likely a significant factor contributing to the development of the hearing disability.

We submit that the evidence discussed above, which includes extensive medical literature deemed current and credible by the department's own guideline, supports a reasonable inference that the nature and levels of noise exposure common in the military environment likely cause significant hearing damage for many, if not most, military

members, whether or not decibel losses were captured on audiograms during service. The totality of the evidence and current state of medical research also supports a strong probability that such service-related damage is likely to be a significant contributor to the later onset of a hearing loss disability.

All of which is respectfully submitted,

Bureau of Pensions Advocates