

# Urban Noise Levels are High Enough to Damage Auditory Sensorineural Health

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**Abstract:** This commentary outlines the preventable auditory sensorineural health risk of cumulative episodic high level indoor and outdoor non-occupational urban noise. Irreversible auditory sensorineural damage includes hidden hearing loss (cochlear synaptopathy), tinnitus, signal-to-noise ratio loss, noise-induced hearing impairment, and accelerated age-related hearing loss (presbycusis). There is evidence of ubiquitous high level urban noise exceeding public health limits, and increased prevalence of sensorineural auditory damage in all age groups internationally. Sensorineural components differ greatly between late-onset sensory presbycusis in people with no hazardous exposure and early-onset neural presbycusis in people with a history of high noise exposure. Public health auditory safe listening limits are more stringent than occupational limits in order to fully protect the general population from permanent sensorineural noise damage. Speech interference noise limits prevent social isolation of individuals with noise-induced auditory system damage or hearing impairment from any cause. Quieter urban noise levels meeting public health limits are urgently needed to protect auditory sensorineural health, lower healthcare costs, and permit communication access in public spaces for people with auditory disabilities.

**Keywords:** hearing impairment, tinnitus, noise-adverse effects, speech interference, residential segregation, health equity

Hearing involves processing sound and speech through the sensorineural auditory system (inner ear cochleas and hearing nerves). Ubiquitous high level indoor and outdoor urban noise levels capable of damaging auditory sensorineural health are a serious public health issue. World Health Organization (2018) critical health outcomes include hearing impairment or noise-induced hearing loss and tinnitus (perception of sound in the absence of an external sound source). Both noise-induced hearing loss and tinnitus are preventable irreversible conditions that negatively impact quality of life.

## Cumulative Episodic Noise Exposure

Urban noise is typically episodic with quiet periods in between. Flamme et al. (2012, p. S10) state, “overall exposure is dominated by a small number of high-level exposures.” Auditory damage is cumulative with indoor or outdoor noise exposure over time (Williams et al., 2010; Flamme et al., 2012; Neitzel et al., 2014).

Fink (2017, p. 44) identifies high level intermittent outdoor noise sources including “yard equipment, construction, vehicles, and aircraft” and high level indoor noise sources including “restaurants, movie theaters, gyms, concerts, sports events, and other places.” Other hazardous urban noise sources include transit, stadiums, bars, and nightclubs (Gershon et al., 2006; Hodgetts and Liu, 2006; Neitzel et al., 2009; Williams et al., 2010; CAOHC, 2015). Neitzel et al. (2014, p. 277) found, “The average individual received 57.5% of their weekly exposure from nonoccupational activities.”

Different sources use different hearing loss definitions. WHO (2014) defines disabling hearing impairment in children and adults as hearing threshold speech frequency pure tone average (0.5-1-2-4-kHz) >35 dB in the better ear. Based on 1999-2010 data, NIH (2016) estimated disabling hearing loss prevalence in US adults at 2% age 45-54, 8.5% age 55-64, 25% age 65-74, and 50% age 75 and older.

Seidman and Standring (2010) reported noise-induced hearing loss in 250 million people worldwide, with 30 million more exposed to high level noise. Hearing loss prevalence has significantly increased in US adolescents, with noise-induced hearing loss estimated in 17% of US teens aged 12 to 17 (Shargorodsky et al., 2010; Henderson et al., 2011). Goman et al. (2017) estimate by 2020 15% of US adults aged 20 and older will have hearing loss (44.11 million) increasing to 55.4% of US adults aged 70 and older. High level episodic urban noise cannot be ruled out as a contributing factor across age groups.

WHO (2011) estimated over 17 million cases of non-occupational noise-induced tinnitus in Europe. Disability Adjusted Life Years (DALYs) represent years lost due to disability and premature mortality. WHO (2018) estimates DALYs equivalent to 22,000 years of healthy life lost among people with tinnitus.

## **Mechanisms of Auditory System Damage**

Depending on the cause, auditory system damage impacts cochlea sensory cells, synapses, and neural pathways (auditory nerves). Cochlear synaptopathy is defined as “the loss of connections between sensory cells of the cochlea and the neural elements that carry their information to the brain” (Guo and Kujawa, 2018, p. 1).

Synaptopathy results in zero sound transmission for the frequency/intensity processed by that neural pathway. The brain receives incomplete auditory information.

The most common causes of acquired sensorineural hearing loss are presbycusis (age-related) and high level noise damage (WHO, 2014). Although sensorineural hearing loss patterns look similar audiometrically, underlying mechanisms of auditory damage can be sensory and/or neural.

### **Primary Sensory Pathology**

Presbycusis is primarily a loss of cochlear sensory cells caused by mechanical and metabolic changes (Fernandez et al., 2015; Vaden et al., 2017). Onset occurs at age 55 and older in 75% of people (Arvin et al., 2013).

Nuttall et al. (2018) found sound and speech are coded differently within the cochlea, with specialized speech coding occurring before synaptic transmission to the auditory nerves. This suggests sensory pathology has a greater impact on speech understanding than previously understood.

Cochlear synaptopathy develops gradually with presbycusis; loss of connections between sensory cells and neural pathways are diffuse across frequencies (Fernandez et al., 2015). Secondary neural degeneration develops gradually over time.

Ability to understand speech depends on hearing impairment and signal-to-noise ratio: target speech level versus environmental noise levels. Speech-to-noise ratio loss is defined as degraded speech-in-noise understanding compared to normal hearing listeners (Killion et al., 2004). Speech-to-noise ratio loss occurs in people with normal or impaired hearing.

Presbycusis is associated with worse speech-in-noise understanding than normal (Peelle et al., 2011; Fernandez et al., 2015). This is likely a primary sensory speech-to-noise ratio loss.

## **Primary Neural Pathology**

High level urban noise causes primary neural pathology after cochlear synaptopathy. Fernandez et al. (2015) describe immediate and permanent loss of synapses, with single synaptopathic events resulting in long term progressive auditory nerve degeneration post noise exposure.

Cochlear synaptopathy is called hidden hearing loss because there are no symptoms and hearing is normal audiometrically; hidden hearing loss is identified post-mortem (Song et al., 2017; Guo and Kujawa, 2018). Cochlear synaptopathy and related nerve degeneration primarily impact high frequencies needed to understand speech (Fernandez et al., 2015).

Missing synaptic connections and auditory nerve degeneration result in significant distortion from incomplete sound and speech signals processed through the auditory system (Fernandez et al., 2015; Liberman et al., 2016; Shi et al. 2016; Guo and Kujawa, 2018). This significantly impacts interpersonal communication and quality of life, even with normal hearing thresholds audiometrically.

Chasin (2017, p.1) states hidden hearing loss points “to sometimes subtle neural pathologies that may result in future communication degradation.” This is likely primary neural speech-to-noise ratio loss.

## **Tinnitus**

Damage from high level urban noise is associated with persistent tinnitus defined as lasting longer than 6 months (Tunkel et al., 2014). Sensorineural tinnitus is incurable. Available treatment includes audiology tinnitus management counselling and services. Sound generators, amplification, or amplification with built-in sound generators are prescribed as needed. Psychology services may be needed depending on tinnitus distress severity (Schlee et al., 2018).

Noise-induced cochlear synaptopathy is a risk factor for tinnitus in people with normal hearing (Hickox and Liberman, 2014; Fernandez et al., 2015; Guo and Kujawa, 2018).

Young adults with normal hearing and tinnitus need greater listening effort to understand speech (Degeest et al., 2017). Those with additional history of non-occupational noise exposure do significantly poorer understanding speech-in-noise (Gilles et al., 2016). This is likely primary neural speech-to-noise ratio loss. People with bothersome noise-induced tinnitus have worse quality of life than people with no tinnitus (Joo et al., 2015; Ribiera Rocha et al., 2017). Severe tinnitus is associated with anxiety, insomnia, depression, and concentration problems (Schlee et al., 2018).

Van Kamp and Davies (2013) found people with tinnitus are higher risk for adverse non-auditory health impacts from urban noise. Fink (2017, p. 44) describes these effects including “increases in stress hormones, hypertension, obesity, cardiac disease, and mortality—at average daily exposures of only 55 decibels.”

## **Temporary Threshold Shift**

The first measurable sign of noise damage is temporary threshold shift: decreased hearing thresholds after high level noise that resolve back to pre-exposure levels within 16-18 hours after the exposure ends (Chasin, 2017). Historically, temporary threshold shift was not considered a significant auditory health risk. Although a minority of researchers deny hidden hearing loss post temporary threshold shift (e.g. Grinn et al., 2014), post-mortem histological evidence demonstrates cochlear synaptopathy after temporary threshold shift

and after noise exposure in the absence of temporary threshold shift (Kujawa and Liberman, 2009; Fernandez et al., 2015; Song et al., 2016). Any temporary noise-related effects (e.g. tinnitus, temporary threshold shift) indicate permanent neural damage has occurred.

Cumulative episodes of cochlear synaptopathy and temporary threshold shift cause greater temporary threshold shift with subsequent exposures, neural speech-to-noise ratio loss, and increased risk of permanent sensorineural noise-induced hearing loss (Flamme et al., 2012; Fernandez et al., 2015; Liberman et al., 2016).

The international evidence-based guideline to prevent temporary threshold shift is maximum unprotected non-occupational noise exposure equivalent to 100 dB for 15 minutes (NIOSH, 1998; WHO, 2018). Countries with the least protective temporary threshold shift prevention occupational regulations limit unprotected exposure equivalent to 100 dB for 1 hour (US Occupational Safety and Health Administration).

Moshhammer et al. (2014) used temporary threshold shift-producing noise of 100 dB for 20 minutes on unprotected human subjects. In a related commentary, NIOSH reminded the noise science community of ethics against causing permanent auditory neural health damage to human subjects by exceeding recommended temporary threshold shift prevention limits (Themann, 2014).

## **Permanent Threshold Shift or Noise-Induced Hearing Loss**

Noise-induced hearing loss is a permanent sensorineural threshold shift from exposure to high level noise. Cumulative episodic noise exposure over time is associated with an increased risk of noise-induced hearing loss (NIOSH, 1998; Neitzel et al., 2014; Fink, 2017). The average individual is expected to develop noise-induced hearing loss after 1 to 20 years duration of non-occupational exposure (Lewis et al., 2013).

Flamme et al. (2012) found approximately 50% of urban dwellers were exposed to high level noise sufficient to cause noise-induced hearing loss. McAlexander

et al. (2015) predicted risk of noise-induced hearing loss in nearly everyone exposed to urban street level noise in New York City.

Amplification is the only treatment for noise-induced hearing loss. Devices include costly hearing aids and less costly personal sound amplification products. Cochlear implants are not a treatment for noise-induced hearing loss. Noise alone never causes the profound hearing loss severity required for cochlear implant candidacy (Clark, 2000).

Amplifiers receive and transmit amplified sound through the outer-middle ear systems before signals enter the sensorineural system. Amplification has limited ability to correct auditory processing distortion introduced after amplified sound or speech leaves the hearing aid (Lesica, 2018).

A majority of people do not get hearing aids. McCormack and Fortnum (2013) report hearing aid use in only 4.3% of adults aged 50-59 years rising to 22.1% for adults aged 80 years and older. Reasons people reject hearing aids include hearing loss denial, stigma, cost, and distorted sound quality (Lesica, 2018; Reed et al., 2018; Wallhagen, 2010).

People with untreated hearing loss are higher risk for social isolation, more frequent falls, diabetes, stroke, anxiety, major depression, cognitive decline, and dementia (Archbold et al., 2014; Goman et al., 2017; Reed et al., 2018). Reed et al. (2018) estimated 38 million Americans have untreated hearing loss resulting in 46% higher total healthcare costs over a 10 year period.

Preventing noise-induced hearing loss and tinnitus would significantly reduce healthcare costs by eliminating the need for treatment and eliminating the burden of untreated noise-induced hearing loss on healthcare systems.

## **Accelerated Presbycusis**

Presbycusis is accelerated by neural degeneration in people with prior high level noise exposure causing hidden hearing loss, temporary threshold shift, or noise-induced hearing loss (Kujawa and Liberman, 2009; Sergeyenko et al., 2013; Fernandez et al., 2015; Viana et al., 2015). Fernandez et al. (2015, p. 7518) state,

“noise damage can have dramatic long-term consequences in amplifying age-related sensorineural hearing loss [which] is of significance in the consideration of noise-risk assessment for human populations.” This is a serious public health concern, particularly with high urban noise levels impacting the general population, and noise-induced hearing loss increasing in younger age groups.

## **Speech Interference**

Ability to understand speech depends on hearing acuity, speech-to-noise ratio loss, and signal-to-noise ratio. The worse the neural speech-to-noise ratio loss and/or noise-induced hearing loss, the higher the speech-to-noise ratio needed for communication (Robinson and Casali, 2003).

Zelaya et al., (2015) estimated 21% of US adults aged 18-69 years have difficulty with speech-in-noise, and adults aged 70 and older have over three times greater difficulty. Zahnert (2011, p. 433) states,

“Even mild hearing loss can be a major disadvantage in a world of ever-faster information exchange. People who cannot hear spoken language well enough to process it quickly may find themselves cut off from others at work, at home, or in social situations.” Zahnert (2011)

Holt-Lunstad et al. (2015) identify actual or perceived social isolation as a significant risk factor for early mortality.

The International Communication Project identifies communication as a fundamental human right for people to achieve their full potential. Fink (2017) raises the important issue of disability rights since urban noise causing speech interference prevents equal communication access in public spaces for all people with auditory disabilities.

Public health noise limits preventing speech interference reduce the negative communication impact for everyone with auditory disabilities by improving the speech-to-noise ratio (Kardous et al., 2016; WHO 2018).

**Table 1:  
Sensorineural Components of Presbycusis versus  
Noise Damage + Presbycusis**

<b>Presbycusis</b>	<b>Noise Damage + Presbycusis</b>
Permanent progressive sensorineural hearing loss	Permanent progressive sensorineural hearing loss
Late onset	Early onset at age of first hidden hearing loss or temporary threshold shift
Primary cause = mechanical and metabolic changes	Primary cause = cochlear synaptopathy
Primary sensory pathology = cochleas	Primary neural pathology = auditory nerves
	Neural speech-to-noise ratio loss (hearing tests normal)
	Tinnitus (normal or impaired hearing)
	Noise-induced hearing loss
Presbycusis onset (age 55+)	Presbycusis onset (age 55+)
Primary sensory presbycusis	Primary neural presbycusis greater than sensory presbycusis alone
Primary sensory speech-to-noise ratio loss	Primary neural speech-to-noise ratio loss greater than sensory speech-to-noise ratio loss alone

speech-to-noise ratio loss = degraded communication in indoor/outdoor noise exceeding public health speech interference limits.

## **Safe Listening Limits**

High urban indoor and outdoor noise exceeding public health guidelines increases risk of auditory damage in all age populations. To prevent noise-induced hearing loss and tinnitus, public health science (WHO, 2018) recommends noise levels  $\leq 70$  dB L Aeq(24) (daily average exposure over a 24-hour period). Average noise includes all sources present over the measurement time including continuous, intermittent, and impulse noise, e.g. horns, sirens, construction activities, motorcycles, vehicles, transit.

Urban noise levels worldwide are consistently higher than recommended limits. Neitzel et al. (2014) found international noise exposures ranging from 74.9 to 79 dB L Aeq(24) with a mean of 73.6 dB L Aeq(24). Exposures consistently exceeded limits with 88% exceedance in Sweden, 70% exceedance in US, 84% exceedance in Spain, and 85% exceedance in China. In Europe, over 5 million people inside urban areas are at risk of auditory damage from road traffic noise alone (EEA 2014).

In men and women aged 20 to 68 years old, Flamme et al. (2012, p. S10) found average exposures exceeded recommended limits “for 70% of men and 65% of women”. McAlexander et al. (2015) found more than 90% of urban dwellers exceeded 70 dB L Aeq(24), with the most hazardous noise coming from sidewalk or street level noise from sirens, construction, large water fountains, and high levels of pedestrian traffic. Due to problems with site location and design, urban pocket parks intended as quiet refuges were associated with higher noise levels than surrounding streets and public areas.

The safe public noise limit to prevent noise-induced hearing loss is significantly lower and listening time is significantly less than occupational noise exposure limits which do not protect the entire noise-exposed worker population. Workers exposed at or above 80 dBA L Aeq(8) over a working lifetime are at risk of noise-induced hearing loss (NIOSH, 1998; WHO, 2018). Depending on the jurisdiction, 8% and 25% of workers internationally are unprotected by occupational limits of 85 dBA and 90 dBA L Aeq(8) respectively (NIOSH 1998; Arenas and Suter, 2014).

Non-occupational limits allow “no risk of hearing impairment due to environmental noise” (WHO, 2018, p. 23). The 70 dB L Aeq(24) limit is equivalent to an 8 hour average noise level at 75 dB L Aeq(8) (Kardous et al. 2016; WHO 2011, 2018).

**Table 2: Auditory Safe Listening Limit by Average Noise Level**

<b>Auditory Safe Listening Limit</b>	<b>Average Noise Level</b>
15 minutes	90 dB Leq
30 minutes	87 dB Leq
1 hour	84 dB Leq
2 hours	81 dB Leq
4 hours	78 dB Leq
<b>8 hours</b>	<b>75 dB Leq</b>

Auditory Safe Listening Limit (WHO, 2011, 2018). dB Leq = equivalent continuous noise level

## **Conclusion**

Ambient urban noise levels in many cities are high enough to cause irreversible sensorineural noise damage. Preventable permanent hidden hearing loss, speech-to-noise ratio loss, tinnitus, noise-induced hearing loss, and accelerated presbycusis, whether treated or untreated, have negative impacts on people’s quality of life and increased healthcare costs. With rising noise-induced hearing loss prevalence in all age populations internationally, quieter urban noise levels meeting public health guidelines are urgently needed to prevent auditory damage and speech interference. Cumulative hazardous exposures exceed limits for disturbing sleep, disrupting communication, and causing non-auditory health impacts. Meeting public health noise limits for preventing adverse non-auditory effects would lower urban noise to levels so they no longer represent an auditory sensorineural health risk.

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