**Acta Acustica united with Acustica**
**Vol. 103 (2017) 531 – 534**

**Short Communications**
DOI 10.3813/AAA.919081

# Kinetic Molecular Theory of Sound Exposure in Personal Listening Devices

R. P. Oates[1], Stephen Ambrose[2], David M. Klein[1]
[1] Institute of Environmental and Human Health, Texas Tech University, 1207 S Gilbert Drive, Lubbock, TX 79416. rp.oates@ttu.edu
[2] Asius Technologies LLC, 1257 Whitehall Drive, Longmont, CO 80504

**Summary**
Noise-induced hearing loss associated with earbud use is an increasing trend in modern society. All smartphones are coupled with earbuds that expose the human auditory system to unsafe sound pressures. Acoustic waves enclosed in the ear canal force the tympanic membrane to move 100–1000 times greater than acoustic waves of equal amplitude in open air. Classical acoustics has not fully explained this biophysical phenomenon. Maxwell's kinetic molecular theory was used, in conjunction with Special relativity, to quantify sound pressure exposures in personal listening devices more accurately. A non-linear dependence of sound intensity on speaker excursion is predicted, demonstrating that earbuds sealed in the ear canal can exceed the National Institute for Occupational Safety and Health's ceiling limit of 140 dB. Sound intensities predicted from molecular mass and velocity produce similar results to Beranek's model of acoustic waves in a closed, rigid cylinder and support previous observations of trapped volume insertion gain in personal listening devices.



**PACS no. 43.80.Gx**

## 1. Introduction

An assessment by The World Health Organization estimates that up to 1.1 billion young people worldwide are at risk of hearing loss due to unsafe listening practices [1]. In middle and high-income countries, nearly fifty percent of young adults (ages 12–35) are exposed to unsafe levels of sound from personal audio devices [1]. This risk of noise-induced hearing loss (NIHL) has been associated with the recreational use of personal audio devices [2, 3, 4]. These risks are arguably underestimated, due to the increasingly pervasive coupling of mobile devices with earbuds. In 2015, 1.4 billion smartphones were sold to end users [5]. Digital music exposure at elevated sound pressures has been shown to impair hearing function [6].

Commercial earbuds seal the ear canal and are often used to drown out environmental noise, which can result in acoustic pressures that exceed occupational noise limits in open air. Workers with occupational noise exposure showed personal exposure and earphone output were 87.9 dB and 87.6 dB, respectively. Earphone output exceeded 85 dB for two-thirds of the subjects, which is above the occupational limit of noise exposure. Nearly all the subjects lacked hearing protection devices (HPDs) on their earbuds [7]. It is clear that earbuds have enabled behaviors that vastly increase the risk of NIHL [8, 9]. The National Institute of Occupational Safety and Health (NIOSH) suggests lowering the volume at the source as a mode of hearing protection. Lowering the source volume has not been shown to prevent exposure of harmful pneumatic pressures generated from earbuds that enclose the ear canal.

Basic auditory research has advanced the understanding of NIHL, through the identification of underlying biochemical mechanisms [10, 11, 12]. A biophysical basis of how acoustic pressure from earbuds directly impacts hearing loss in the human auditory system is not fully understood. It has been demonstrated that acoustic waves enclosed in the ear canal force the tympanic membrane to move 100–1000 times greater than acoustic waves of equal amplitude in open air [13]. This observation has led to the authors' application of kinetic molecular theory (KMT) to acoustics, to develop a biophysical model of this phenomenon that has not yet been fully explained by classical acoustics.

## 2. Methods

Oscillating movements of a solid boundary is an example mechanism for sound generation described in classical acoustics [14]. Sounds of this nature adopt a complex range of pressure variations, yet have not been shown to raise the barometric mean in the surrounding medium. Sounds are complex mixtures of pressure variations that vary with respect to phase, frequency, and amplitude but have not been completely described on a molecular basis [15].

The propagation of acoustic pressure is described by successive collisions of molecules that propagate sound from a source vibration. This phenomenon has similar characteristics to Maxwell's Kinetic Theory, which describes the behavior of ideal gases and is associated with changes in pressure. In KMT, pressure is due to the rate at which gas molecules at different velocities collide with the walls of a container [15].

Personal listening devices typically seal a speaker inside the ear canal, creating a trapped volume of air molecules that transfer pressure from the diaphragm of an oscillating speaker diaphragm to the tympanic membrane. Although the displacement of an individual molecule (mean free path) cannot be measured, the velocity of larger groups of molecules can be calculated using KMT. Relative velocities of molecules are used to describe pressure contributions attributed to enclosing speakers in the ear canal that are not present during open air sound propagation. The purpose of this paper is to model contributions of acoustic and pneumatic pressure from personal listening devices to the acoustico-mechanics of the ear.

In Figure 1, compressions are defined as moments where gas molecules are made more dense by a speaker excursion compared to the resting density. A rarefaction is defined as a decrease in density below resting density. Excursion will be used here to define the speaker cone's travel from rest. Molecules on the surface of the cone of the speaker are compressed towards the tympanic membrane, parallel to the propagation of sound.

The compression volume $V$ is approximated by the volume of a cylinder in Equation (1), with height $h$ (equal to an excursion of the speaker) and radius $r$ of the speaker diaphragm.

$$V = \pi r^2 h. \tag{1}$$

Speaker diaphragms in personal listening devices have wide ranges and excursion lengths vary based on acoustic frequency.

Received 21 September 2016,
accepted 24 February 2017.

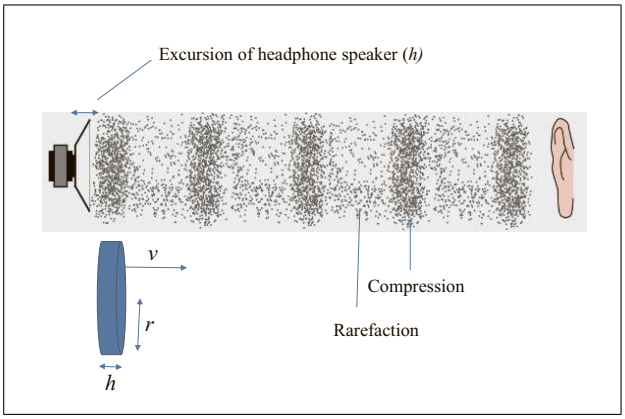


Figure 1. Molecular compressions and rarefactions generated by an earbud speaker.

The ideal gas density of air $\rho_{air}$ was approximated by equation (2), where $M$ is the molar mass of air, $P$ is ambient atmospheric pressure, $R$ is the molar gas constant, and $T$ is temperature. The mass of air associated with a compression event $m_{air}$ (3) was approximated by the compression volume from (1) and the density of air in (2).

$$\rho_{air} = \frac{MP}{RT}, \tag{2}$$

$$m_{air} = \rho_{air} V. \tag{3}$$

In the KMT model, individual molecules are defined as having ideal properties that conserve momentum through perfectly elastic collisions with the walls of a container and one another [15]. This contrasts with Kirchhoff's theory of sound propagation in a tube, which incorporated thermal and viscous losses based on the texture of the tube walls [16]. More general expressions for acoustic loss in fluid mechanics are discussed elsewhere and will not be incorporated into this model.

Individual molecules have 'resting' velocity at a given temperature with an associated energy. Macroscopically, sound propagation relates to the bulk gas as it alternatively compresses and rarifies with the motion of a sound source. Molecules that comprise the bulk gas retain their individual average velocities, caused by the heat content of the gas. In KMT, local particle resting velocities exceed the speed of sound in air and are temperature dependent.

$$v = \sqrt{\frac{\omega RT}{M}}, \tag{4}$$

$$c = \sqrt{\frac{3RT}{M}}. \tag{5}$$

For an ideal gas, the speed of sound $v$ is given by equation (4), where the average molecular weight $M$ of air is 0.029 kg/mol, the gas constant $R$ is 8.314 J/mol-K, temperature $T$ is 310 K (average body temperature in the ear canal), and the adiabatic constant $\omega = 1.4$ for a diatomic gas.

Average velocities of molecules $c$ in a closed system before a sound wave is introduced is defined by equation (5), where $M$ of air is 0.029 kg/mol, the gas constant $R$ is 8.314 J/mol-K, temperature $T$ is 310 K (average body temperature in the ear canal). Substituting all values into equations (4) and (5) gives $v$ as 353 m/s and $c$ as 516 m/s.

It is generally considered that the non-relativistic kinetic energy of a sound wave moves at the speed of sound, as the bulk gas alternatively compresses and rarifies, but local molecular velocities are not considered to contribute to the overall pressure of the system as sound propagates through it. Macroscopic acoustic theory is limited in its ability to estimate molecular contributions of pressure that occur simultaneously with sound propagation from an oscillating boundary in sealed conditions.

A relativistic approach can be used to determine the amount of kinetic energy associated with changing the molecular velocity of a closed system, as sound passes through it. For low velocities, the kinetic energy $KE$ is classically written as

$$KE = \frac{1}{2} m v^2, \tag{6}$$

where $m$ is the mass and $v$ is the velocity. However, this equation does not describe the additional kinetic energy imparted on a closed system of molecules that possess a resting velocity greater than the speed of sound through the medium.

One postulate of special relativity states that the laws of physics are invariant in all inertial systems [17]. Special relativity applied to acoustics is necessary because classical approaches have yet to adequately define the total energy associated with additional momentum imparted on a medium by a sound wave, while the individual molecules that comprise the medium are in motion. The speed of sound is classically associated with inertia, as seen in the Newton-Laplace equation for elasticity of ideal gases. The analogy of molecular acoustics to Special relativity is found in Mach's work "...a particle's inertia is due to some interaction of that particle with all the other masses in the universe; the local standards of non-acceleration are determined by some average of the motions of all the masses in the universe, and all that matters in mechanics is the relative motion of all the masses." [17].

In Special relativity, the Einstein relationship for total energy $E$ in equation (7) includes both the kinetic energy $KE$ and rest mass energy $E_0$ for a particle or group of air particles. Equation (7) can be viewed as the additional 'boost' from a sound event given to molecules of a given resting velocity.

$$E = E_0 + KE. \tag{7}$$

Equation (7) can be rearranged to give the kinetic energy $KE$ that is imparted on the system by a compression event (Equation 8).

$$KE = E - E_0. \tag{8}$$

Substituting the Einstein relationship of mass and energy for $E_0$ and $E$ gives equation (9):

$$KE = \gamma m_0 c^2 - m_0 c^2, \tag{9}$$

where $m_0$ is defined as the resting mass. A Lorentz factor $\gamma$ is defined in Equation (10) and was applied to determine the total energy kinetic energy of molecules at relative velocities.

$$\gamma = \frac{1}{\sqrt{1 - \frac{v^2}{c^2}}}. \tag{10}$$

Substituting $\gamma$ into equation (9) gives equation (11) and its reduced form is shown in equation (12).

$$KE = \frac{m_0 c^2}{\sqrt{1 - \frac{v^2}{c^2}}} - m_0 c^2, \tag{11}$$

$$KE = m_0 c^2 \left( \frac{1}{\sqrt{1 - \frac{v^2}{c^2}}} - 1 \right). \tag{12}$$

Substituting the relative molecular velocity of air $v$ (speed of sound) and the root mean square velocity $c$ into equation (12) gives the reduced form (13).

$$KE = (0.371)m_0c^2. \tag{13}$$

Equation (13) can be used to determine the kinetic energy in Joules (J) imparted on molecules in a closed acoustic system, with resting molecular velocities relative to a compression event.

A speaker diaphragm oscillates and transfers kinetic energy to the tympanic membrane by molecular compression. In this model, speaker compressions cause temporary increases in the number of molecular collisions, which increases the total pressure of the system.

In earbuds, the number of compression events over a given time period can be monitored by a song's tempo. The tempo $t$ of a song in beats per minute can be converted to the number of compression cycles per second $f$ in Hertz by equation (14).

$$t\left(\frac{\text{beats}}{\text{min}}\right)\left(\frac{\text{min}}{60\,\text{s}}\right) = f, \tag{14}$$

Sound power $\Phi$ is defined as the rate at which sound energy is emitted per unit time and is measured in Watts (W). The amount of sound power $\Phi$ is given by equation (15).

Substituting equation (13) into (15) gives the peak sound power associated with each molecular compression from an earbud speaker diaphragm (Equation 16).

$$(KE)f = \Phi, \tag{15}$$

$$(0.371)m_0c^2f = \Phi. \tag{16}$$

Sound intensity level $I$ is the sound power per unit area $A$ in W/m$^2$ and is defined in equation (17).

$$I = \frac{\Phi}{A}. \tag{17}$$

The sound intensity level $I$ at the ear drum can be calculated by substituting equation (16) into (17), giving equation (18).

$$I = \frac{(0.371)m_0c^2f}{A}. \tag{18}$$

An acceptable surface area $A$ of the adult eardrum is approximately $5.5 \cdot 10^{-5}$ m$^2$. The sound intensity level $B$ at the eardrum in dB is given by equation (19), using the standard reference intensity of the eardrum $I_0$ ($10^{-12}$ W/m$^2$).

$$B = 10\log_{10}\frac{I}{I_0}. \tag{19}$$

## 3. Results

Most earbud speaker drivers are between 7 and 15 mm in diameter, with excursions ranging from nanometers to millimeters. Earbud diaphragm diameters of 0.5 to 15 mm were chosen as a representative population of earbud speakers. Lengths of 0.1 µm (microns) to 1 mm were used as common earbud speaker excursions, which are associated with various frequencies in popular music. The number of driver compressions per unit time was modeled by a tempo of 120 bpm. Figure 2 shows that sound intensity increases non-linearly with speaker excursion size, independent of speaker diameter.

Based on the increase in molecular kinetic energy from a speaker excursion, this model predicts that even small excursions and speaker diameters are capable of producing excessive sound intensity levels when enclosed in the ear canal. Criterion for the permissible time for safe listening limits have been set forth by The National Institute of Occupational Safety and Health (NIOSH) in 1998 (Figure 3).

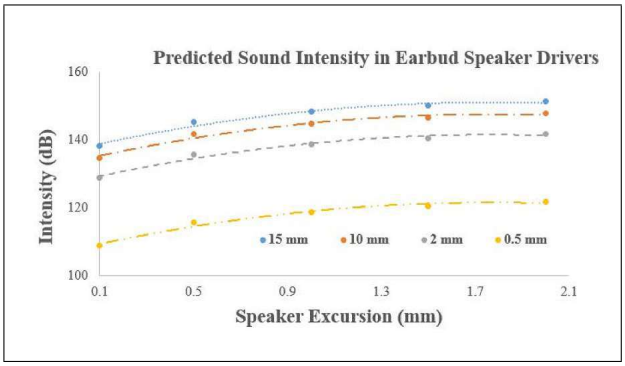


Figure 2. Predicted sound intensity level in decibels vs speaker excursion size at 120 bpm for speaker diaphragm diameters of 15, 10, 2, and 0.5 mm.

| Noise Level (dBA) | Maximum Exposure Time per 24 Hours |
|---|---|
| 85 | 8 hours |
| 88 | 4 hours |
| 91 | 2 hours |
| 94 | 1 hour |
| 97 | 30 minutes |
| 100 | 15 minutes |
| 103 | 7.5 minutes |
| 106 | 3.7 minutes |
| 109 | 112 seconds |
| 112 | 56 seconds |
| 115 | 28 seconds |
| 118 | 14 seconds |
| 121 | 7 seconds |
| 124 | 3 seconds |
| 127 | 1 second |
| 130–140 | less than 1 second |
| 140 | NO EXPOSURE |

Figure 3. Maximum Recommended Noise Dose Exposure Levels (NIOSH 2016).

NIOSH criteria for recommended occupational noise-level exposure shows that as sound levels increase, the time allowed for safe listening decreases. It has been previously reported that the output of personal music players ranges from 110–125 dB and can damage hearing after 15 minutes of exposure per day [18]. This model contains manufacturer settings that confirms these ranges of personal music players within an order of magnitude. This KMT model predicts that earbuds sealed in the ear canal have an expanded dB range and can exceed the NIOSH ceiling limit of 140 dB. The non-linear dependence of sound intensity on speaker excursion in this model indicates that the level of sound exposure may vary widely, based on manufacturer speaker diameter and tempo of music played by the consumer.

## 4. Discussion

This model is a first estimation of acoustic energy imparted on air molecules in sealed conditions. KMT describes ideal gas behavior and the density of air is temperature dependent (Equation 2). This model does not account for local temperature fluctuations and only accounts for even dispersion of gas molecules throughout the medium of the ear canal. Gas composition of the ear canal takes into account the percentages of nitrogen and oxygen, 79% and 21% respectively. It is included in the average molecular weight of air in relevant equations, but excludes other gases found in air which may affect sound propagation. The model considers sound energy to be transferred to the tympanic membrane at a rate based on tempo. This approximation does not include energetic contributions from sound waves based on complex sinusoidal motions and does not consider viscous or thermal loss in sound propagation through a fluid. The KMT model is dependent on relative molecular velocity of a finite mass of molecules from special relativity. It allows for the conservation of momentum, in that all sound energy is transferred to the ear drum by molecular collision parallel to sound propagation. Adult tympanic membranes are approximately $5.5{\cdot}10^{-5}$ m$^2$ and this model can account for various sizes ear drums, to determine individual sound level exposures.

## 5. Conclusion

It was previously observed that when a sound producing device is sealed in the ear canal, the trapped volume of air in the ear canal acts like a pneumatic piston [13]. This pneumatic piston transmits an oscillating static pressure to the tympanic membrane that can greatly boosts the sound pressure level in the ear canal [13]. This KMT model of sound exposure in personal listening devices supports this conclusion on a molecular basis and is capable of modeling sound pressure levels in closed air conditions that produce similar results to Beranek's model of acoustical waves in a closed, rigid cylinder [19]. It is the authors' intention that this analysis will provide a foundation for more thorough investigations at the interface between pneumatic and acoustic pressures in enclosed conditions to prevent future hearing loss associated with earbuds, in-ear monitors, and hearing aids.

## References

[1] E. Krug, M. Alarcos Cieza, S. Chadha, L. Sminkey, T. Morata, D. Swanepoel, A. Fuente, W. Williams, J. Cerquone, R. Martinez, G. Stevens, M. Peden, S. Rao, P. Agarwal, E. Zeeck, A. Bladey, M. Arunda, A. Ncube: Hearing loss due to recreational exposure to loud sounds: A review. World Health Organization. Government Document No. 9789241508513, 2015.

[2] P. Jian-Hua, T. Ze-Zhang, H. Z. Wu: Risk of damage to hearing from personal listening devices in young adults. J. Otolaryngol. **36** (2007) 181–185.

[3] J. Shargorodsky, S. G. Curhan, G. C. Curhan, R. Eavey: Change in prevalence of hearing loss in US Adolescents. JAMA **304** (2010) 772–778.

[4] S. Ahmed, S. Fallah, B. Garrido, A. Gross: Use of portable audio devices by university students. J. Canadian Acoustical Association **35** (2007) 35–54.

[5] Gartne: Number of smartphones sold to end users worldwide from 2007 to 2015. http://www.statista.com/statistics/263437/global-smartphone-sales-to-end-users-since-2007, Accessed July 2016.

[6] C. G. Le Prell, S. Dell, B. Hensley, J. W. Hall III, K. C. M. Campbell, P. J. Antonelli, G. E. Green, J. M. Miller, K. Guire: Digital music exposure reliably induces temporary threshold shift in normal hearing human subjects. Ear Hear. **33** (2012) 44–58.

[7] T. Nakao, M. Kakei, I. Araki, T. Tsutsui, N. Satoh, J. Inoue, S. Horie: Assessment of exposure to voices and noise via earphones in manufacturing industry workers in Japan. J. Occup. Health **56** (2014) 285–291.

[8] I. Vogel, P. M. van de Looij-Jansen, C. L. Mieloo, A. Burdorf, F. de Waart: Risky music listening behaviors and associated health-risk behaviors. Pediatrics **129** (2012) 1097.103.

[9] C. D. Portnuff, B. J. Fligor, K. H. Arehart: Teenage use of portable listening devices: a hazard to hearing? J. Am. Acad. Audiol. **22** (2011) 663.77.

[10] Y. Song, A. Xia, H. Y. Lee, R. Wang, A. J. Ricci, J. S. Oghalai: Activity-dependent regulation of prestin expression in mouse outer hair cells. J. Neurophysiology **113** (2015) 3531–3542.

[11] B. Mazurek, H. Haupt, N. Amarjargal, Y. M. Yarin, A. Machulik, J. Gross: Up-regulation of prestin mRNA expression in the organs of Corti of guinea pigs and rats following unilateral impulse noise exposure. Hearing Research **231** (2007) 73–83.

[12] S. Delmaghani, J. Defourny, A. Aghaie, M. Beurg, D. Dulon, N. Thelen, I. Perfettini, T. Zelles, M. Aller, A. Meyer, A. Emptoz, F. Giraudet, M. Leibovici, S. Dartevelle, G. Soubigou, M. Thiry, E. S. Vizi, S. Safieddinem, J. P. Hardelin, P. Avan, C. Petit: Hypervulnerability to sound exposure through impaired adaptive proliferation of peroxisomes. Cell **163** (2015) 894–906.

[13] S. Gido, R. B. Schulein, S. D. Ambrose: Sound reproduction within a closed ear canal: Acoustical and physiological effects. Presented at the 130th Convention in May 13–16, 2011, London, UK.

[14] S. W. Rienstra, Hirschberg: An introduction to acoustics. Eindhoven, Netherlands, Eindhoven University of Technology, 2016.

[15] J. Maxwell: Illustrations of the dynamical theory of gases. Phil. Mag. **19, 20** (1860) 19–32, 21–37.

[16] R. H. Withnell, L. E. Gowdy: An analysis of the acoustic input impedance of the ear. JARO **14** (2013) 611–622.

[17] W. Rindler: Relativity: Special, general, and cosmological. 2nd edition. Oxford University Press, New York, 2006.

[18] E. Daniel: Noise and hearing loss: a review. J. Sch. Health **77** (2007) 225–231.

[19] L. L. Beranek: Acoustics. McGraw-Hill, New York, 1954. Section 2.4, pp. 28-35.