

Residential Noise Exposure and Chronic Stress in Children

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ABSTRACT

One of the suspected impacts of chronic noise exposure on children is elevated stress. Airport studies and a few road traffic studies show associations with ambient noise exposure and children's blood pressure. Few studies have examined these linkages in terms of interior noise levels or used neuroendocrine assessments of chronic stress. Nine year old children living in noisier homes have higher levels of chronic physiological stress. Overnight, urinary (12 hour) levels of cortisol, epinephrine, and norepinephrine, are each elevated. Two, 2 hour assessments of noise exposure (Leq, dBA) were completed on two different days in the home. Poverty, duration of residence in the home, gender, family composition, and maternal education were incorporated as statistical controls given the reliable association of socioeconomic status with both noise exposure and chronic stress. The potential health implications on children of noise levels well below those necessary to produce hearing damage warrant further attention by scientists and policy makers alike.

INTRODUCTION

Several studies reveal elevated physiological stress from noise exposures primarily from aircraft and road traffic (Babisch, 2006; Evans, 2006; Ising & Krupa, 2007; Paunovic, Stansfeld, Clark & Belojevic, 2011). Most of these studies assess physiological stress with blood pressure and a few also mark chronic stress with neuroendocrine stress hormones such as cortisol. In this paper we examine residential interior noise levels and chronic physiological stress among 9 year old children. We assess chronic physiological stress with overnight cortisol, epinephrine, and norepinephrine. Because the data are part of a larger study on poverty and risk exposure among children, half of the sample is from low-income households and half from middle-income households.

An important contribution this study makes is to investigate typical, everyday noise levels within the residential environments of families with children. Because we have oversampled low-income households, these data also reflect a larger range in household noise levels relative to most surveys that consist of predominantly middle socioeconomic status (SES) households. Another interesting thing about the present sample is the examination of rural households. This means that most if not all of the ambient noise is generated internally. None of the sample herein lives proximate to airports or major highways.

By examining overnight neuroendocrine hormones we also limit our focus to chronic stress. The hormonal measures taken overnight indicate basal, resting levels. This is of interest because these measures are collected during the child's sleep and thus reflect chronic alterations in basic stress physiology rather than reactivity to acute noise exposure.

METHOD

Participants

Two hundred and eleven children ($M = 9.18$, $SD = 1.17$ years, 51% male) with data on overnight neuroendocrine

hormones and noise exposure were included in the sample. These children were part of a study on child development, risk exposure, and rural poverty. Children from low-income households were oversampled with approximately half of the sample below the US poverty line and the other half from middle-income families. Forty two percent of the children resided with a single parent and seven percent of the mothers in the study had not completed high school.

Procedure

Ambient noise levels were measured for two hours inside the main social space of the residence while the family was at home. Families were asked to engage in whatever normal activities they typically would do. The decibel meter was placed above ground where a child could not speak directly into the microphone. Adults were asked to not speak directly into the microphone. These two hour sessions of noise assessment were conducted on two different days with a Bruel & Kjaer noise level meter (Model 2239 A). LeqA was calculated for each of the two hour periods and averaged as an estimate for noise exposure.

Twelve hour, overnight urine was collected from 8 PM to 8 AM in the child's home then processed and deep frozen until subsequent biochemical assays by technicians blind to the child's noise exposure or family background. Epinephrine and norepinephrine were assayed with high performance liquid chromatography with electrochemical detection (Riggin & Kissinger, 1977), and free cortisol was measured by radioimmuno assay (Baxter Travenol Diagnostics, 1987). Creatinine was also assayed in order to control for differences in body mass and incomplete voidings (Tietz, 1976). Epinephrine and norepinephrine are stress hormones secreted by the adrenal medulla gland indicative of sympathetic nervous system activity. Cortisol from the adrenal cortex is a marker of the hypothalamic pituitary adrenal axis. In concert overnight measures of these stress hormones provide a reliable indicator of chronic physiological stress. Overnight measures are indicative of basal, resting levels of these hormones. It is important to understand that the stress hormonal levels were assessed overnight during periods of relative

quiet and thus do not reflect acute noise exposure. They are a marker of chronic stress associated with overall, ambient noise level exposures.

RESULTS

Mean LeqA across the entire sample was 62.84 (SD = 6.65). Table 1 provides descriptive information on mean levels of each of the stress hormones depicted at ambient noise level tertiles (< 60, 60-65.6, > 65.5). As can be seen all of the stress hormones differ in the expected direction. The noise exposure levels were trichotomized for descriptive purposes only. The inferential analyses of noise on chronic stress described below all maintained the Leq as a continuous metric.

Table 1. Mean Stress Hormone Levels and Noise Exposure

	Noise Tertiles		
	Low	Medium	High
Cortisol _{log}	-1.71	-1.63	-1.57
Epinephrine _{log}	.45	.47	.62
Norepinephrine _{log}	1.39	1.46	1.47

We used ordinary least squares regression to examine the relation between residential, ambient noise exposure and basal or resting neuroendocrine indices of chronic stress. Gender, poverty status (above or below the US Census poverty line), family structure (single parent or two parents), maternal education (high school completion or drop out) were all included as covariates in the regression analyses given the association of socioeconomic status (SES) with both noise levels and with physiological stress (Evans, 2004). Because of our interest in chronic noise exposure and the large variability in residential tenure ($M = 60.38$ months, $SD = 66.76$), we also included a statistical covariate for the number of months the child had lived in the home where the noise assessments and neuroendocrine measures were conducted.

This statistical technique provides a way to examine the relation between ambient noise levels and stress hormones statistically controlling for potential confounding variables. The hormonal measures are all skewed so log transformations are typically used for this type of data. We report raw b weights for residential noise exposure along with the ΔR^2 as an indication of effect size for noise exposure. These statistics are all after inclusion of the five covariates described above (gender, poverty status, family structure, maternal education, and housing tenure). The unstandardized b is a measure of the slope of the line in the original units of measurement for each of the stress hormones. The b divided by its *Standard Error (SE)* yields a t -test comparing the b to the null hypothesis that the slope is 0. The probability values on the right (p) are two tailed tests. The ΔR^2 value reflects the proportion of variance in each stress hormone, after statistically controlling for the five covariates. It can be considered an estimate of effect size and can be interpreted similarly to a squared correlation coefficient. Thus in Table 2 residential noise levels are more strongly associated with norepinephrine.

As can be seen in Table 2, noise levels were significantly related to all three overnight stress hormones.

Table 2. Noise Effects on Chronic Stress

Hormone	b (SE)	ΔR^2	p
Cortisol	.008 (.003)	.03	.014
Epinephrine	.010 (.003)	.05	.009
Norepinephrine	.007 (.003)	.03	.009

DISCUSSION

Many studies of transportation noise have uncovered evidence of elevated physiological stress, principally blood pressure, among both adults and children living near airports and major roads. In this study we show that interior, ambient household noise may also be capable of elevating chronic physiological stress. Nine year old children living in noisier rural households have elevated, overnight stress hormones.

These results are from a cross sectional comparison and thus causal conclusions are not warranted. Although we have statistically controlled for several variables likely associated with both noise exposure and chronic stress, we cannot be sure that some other variable might be driving the associations uncovered. Ideally one would track the same children over time as household noise levels changed.

The levels of stress hormones herein are within the normal range for pre-pubescent children and are unlikely to be clinically significant. However it is also the case that elevated physiological stress levels early in life tend to track throughout the life span, eventually becoming risk factors for morbidity.

Although considerable important work on the nonauditory, stress effects of noise has been conducted, we need to examine more mundane, everyday noise exposures in addition to focusing on transportation sources such as airports and roadways. Even though the physiological impacts of ambient, household noise are likely less than those found proximate to airports and major roadways, the numbers of children affected by modest elevations in household noise far surpass those living in high noise impact zones.

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