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A single positive *Pseudomonas aeruginosa* sputum culture is not associated with increased long-term mortality in COPD http://ow.ly/x5wvD

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Does traffic noise influence respiratory mortality?

To the Editor:

Over the last decade, several studies have investigated the association between noise levels, primarily due to road traffic in large cities, and pathologies not related to either traditional hearing impairments or sleep disorders. These pathologies are mainly hypertension and cardiovascular, connective system and respiratory diseases [1]. Their impact on public health has been assessed in time series studies, indicating a traffic noise effect on both cardiovascular and respiratory hospital admissions rates similar to that attributed to air pollutants [2]. In addition, a recent study has shown an association of noise levels with cardiovascular mortality [3]. The description of the physiopathological mechanisms involved in this association reveal an actual impact of current road traffic noise levels on health [1]. Others question this because of the high correlation between traffic noise and air pollution [4], although a previous study in our setting showed an independent association between noise and cardiovascular mortality from the effect of the primary chemical air pollutants [5]. However, the effect of noise on respiratory mortality has not yet been investigated.

In this study, we examined the association between daily mortality due to respiratory causes (International Classification of Diseases, 9th revision, codes 460–519) and daytime noise levels in the city of Madrid

TABLE 1 Percentage increase in risk of respiratory mortality caused by an increase of 1 db(A) of diurnal noise in Madrid, Spain

	Age years	
	<65	⩾65
Diurnal noise at lag 0	-1.4 (-23.4–20.5)	4.8 (-0.7–10.4)#
Lag 1	-5.3 (-27.5–16.7)	6.5 (0.8–12.2)*
Lag 2	2.4 (-20.0–24.0)	-5.2 (-10.8–5.1)
Lag 3	-4.0 (-26.0–17.9)	-2.2 (-7.9–32.9)
Diurnal noise at lag 1		
Adjustment for $PM_{2.5}$	-6.4 (-29.3–16.5)	6.1 (0.2–12.0)*
Adjustment for NO_2	-5.1 (-27.2–17.1)	6.2 (0.6–11.9)*

Data are presented as percentage increase in risk (95% CI). $PM_{2.5}$: particulate matter with aerodynamic diameter <2.5 μm. #: $p<0.1$. *: $p<0.05$.

(Spain), from January 1, 2003 to December 31, 2005, using a time-stratified case–crossover design. We used overdispersed Poisson regression adjusting for temperature, with natural cubic splines with four degrees of freedom for averaged lags 0–1 and 2–6, dummy variables for public holidays and influenza epidemics, and the three-way interaction between day of the week, month and year to control for seasonality and time trends [6]. We examined short-term effects up to 3 days lag. Moreover, we provided evidence of an air pollution-independent association [7] after adjusting the results for primary chemical air pollutants (particulate matter with aerodynamic diameter <2.5 μm ($PM_{2.5}$) and nitrogen dioxide) at the same lag as noise levels. Daily mortality was obtained from the Madrid Regional Inland Revenue Department, which is the department responsible for mortality registry. Diurnal equivalent noise levels (during the 08:00–22:00 h period) were provided by six of the urban background stations of the Madrid Municipal Automatic Air Pollution Monitoring Network that also measures real-time noise pollution levels, while air pollution data were supplied by all 24 monitoring stations.

The average daytime noise level in Madrid was 65 dB(A), ranging from 62.1 to 71.0 dB(A). The average daily death rate due to respiratory causes was 9.5, ranging from 0 to 32, with >90% of deaths in those aged ⩾65 years. Noise levels showed low correlation with chemical air pollutants ($r=0.28$ and $r=0.01$ for nitrogen dioxide and $PM_{2.5}$, respectively). The strongest and statistically significant ($p<0.05$) effect was found in subjects >65 years at lag 1, with a risk increase of respiratory mortality of 6.5% (95% CI 0.8–12.2%) for an increased daytime noise interquartile range (IQR) of 1 dB(A) (table 1). No effect was found for subjects aged <65 years. For those aged >65 years, the noise effect did not change after adjusting for $PM_{2.5}$ (at lag 1) (6.1%, 95% CI 0.2–12.0%) or nitrogen dioxide (at lag 1) (6.2%, 95% CI 0.6–11.9%). In these multiple-exposure models adjusting for primary chemical air pollutants, an independent effect of noise was found for $PM_{2.5}$ (3.3% (95% CI 0.3–6.2%) for an IQR of 10 $\mu g \cdot m^{-3}$) and nitrogen dioxide (0.9% (95% CI 0.1–1.6%) for an IQR of 5 $\mu g \cdot m^{-3}$), both being statistically significant ($p<0.05$). However, to exclude a spurious relationship of this association we examined at same lag mortality in people over 65 years for circulatory causes (ICD9: 390-459), since it has also been related with noise, and more importantly for all natural causes excluding respiratory and circulatory causes (ICD9: 001-799 excluding ICD9: 460-459 and 390-459). We found a consistent noise effect for circulatory causes (4.4% (95% CI 0.3–8.5%) for an IQR of 1 db(A)) but not other causes (-1.3% (-4.7–2.0%) for an IQR of 1 db(A)), even after adjusting for chemical air pollutants and effects of $PM_{2.5}$ and nitrogen dioxide.

The novel finding in our study, regarding the impact of traffic noise on respiratory mortality, is consistent with those previously reported in Madrid relating traffic noise to respiratory hospital admissions [2]. Our result suggests that there are independent effects of noise and primary chemical air pollutants. This agrees with others who concluded that there is little confounding between traffic noise and air pollution [7]. The fact that noise is also related to cardiovascular mortality, as previously reported [4, 5], but not with other types of mortality that exclude respiratory and cardiovascular causes, as well as for both chemical air pollutants, reinforces the association between diurnal equivalent noise level and respiratory mortality. This association could appear surprising but it has also been reported in a recently published study [8], where the combined effects of chronic exposure to traffic-related air pollution and noise on the risk of skin and respiratory diseases in children and adults were studied. This suggests that the associations indicating the short-term effects of exposure to high noise levels are not spurious and are related to increased blood

cortisol levels. This hormone is released during stress reactions, and a relationship between elevated cortisol levels and increased risks of asthma, chronic bronchitis and neurodermitis, which in turn increase with increasing traffic volume [9], has been shown.

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First description of the novel association between traffic noise and respiratory mortality, independent of air pollution http://ow.ly/rFAjz

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CD14/Toll-like receptors interact with bacteria and regulatory T-cells in the development of childhood asthma

To the Editor:

The susceptibility to asthma development in childhood is influenced by genetic as well as environmental factors, and interactions between these factors [1–3]. However, at present, their exact role is still largely undetermined. Genetic variations in the innate immune system may lead to different adaptive immune responses to bacteria and may therefore vary the development of asthma [2, 4, 5]. We performed a prospective longitudinal study in preschool children, in which we determined polymorphisms in Toll-like receptors (*TLR*s) and *CD14*, the presence of bacteria, and the proportion of regulatory T-cells (T_{reg}) all in relation to an asthma diagnosis at 6 years of age. We hypothesise that specific genetic variants in genes that affect the innate immune system influence the response to bacteria and the recruitment of T_{reg} in preschool children, leading to an increased likelihood of asthma at 6 years of age.