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Traffic noise and risk of mortality from diabetes

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Dear Editor,

Research on traffic noise has focused on cardiovascular effects [1] and recently on respiratory effects [2]. Road traffic noise at normal urban levels can lead to stress and sleep disturbances, which may lead to higher risk for type-2 diabetes [3]. A recent cohort study long-term exposure to road traffic noise was associated with a higher risk of diabetes [4]. Otherwise, air pollution caused by traffic has also been associated with short-term mortality from diabetes [5]. However, the impact of traffic noise on diabetes-related deaths has not been investigated yet.

We examined the association between daily diabetes-related deaths (International Classification of Diseases, 10th Revision: E10–E14) and noise levels in the city of Madrid (Spain), from 2001 to 2009, using a time-stratified case-crossover design. We used overdispersed Poisson regression adjusting for time-trend and seasonality, temperature, and primary chemical air pollutants (PM_{2.5} and NO₂). We examined short-term effects up to 3 days lag. Daily mortality from diabetes was obtained from the Madrid Regional Inland Revenue Department and daily

average levels of diurnal equivalent noise (for the 8–22-h period, Leqd) and night-time equivalent noise (for the 22–8-h period, Leqn) from background stations of the Madrid's Municipal Automatic Air Pollution Monitoring Network that also measure real-time equivalent noise levels.

Diurnal average noise was 65.1 dB(A), and average night time was 60.5 dB(A). Daily death counts from diabetes ranged from 0 to 6, 95 % aged more than 65 years. Noise levels showed low-moderate correlation with chemical air pollutants ($r = 0.01$ and $r = 0.28$, for PM_{2.5} and NO₂, respectively). The strongest effect for diurnal noise was at lag 0 (Fig. 1) with a risk of mortality of 3.2 % (95 % confidence interval (CI) [−0.3, 6.9]) for a rise of 0.5 db(A) ($p = 0.078$). For night-time noise, the strongest effect was at lag 1, with a risk of 4.6 % (95 % CI [1.5, 7.8]) ($p = 0.004$). Noise effects did not change after adjusting for air pollutants.

The mechanisms of action about how long-term exposure to traffic noise might contribute to type-2 diabetes have already been stated [3, 4]. It is also known that diabetics are more susceptible to the effects of environmental risk factors [5]. However, although our finding could be consistent with short-term effects mainly produced by reduction in sleep quality and duration, since the strongest effect was found with night-time noise levels, further research is needed for a proper understanding of the mechanisms of action for short-term exposure to traffic noise on diabetes. Otherwise, the short-term effect of traffic noise on cardiovascular mortality has been reported in Madrid also showing an independent effect of chemical air pollutants [1]. However, PM_{2.5} and NO₂ were not associated with mortality from diabetes, suggesting that the effect of noise levels is self-sufficient.

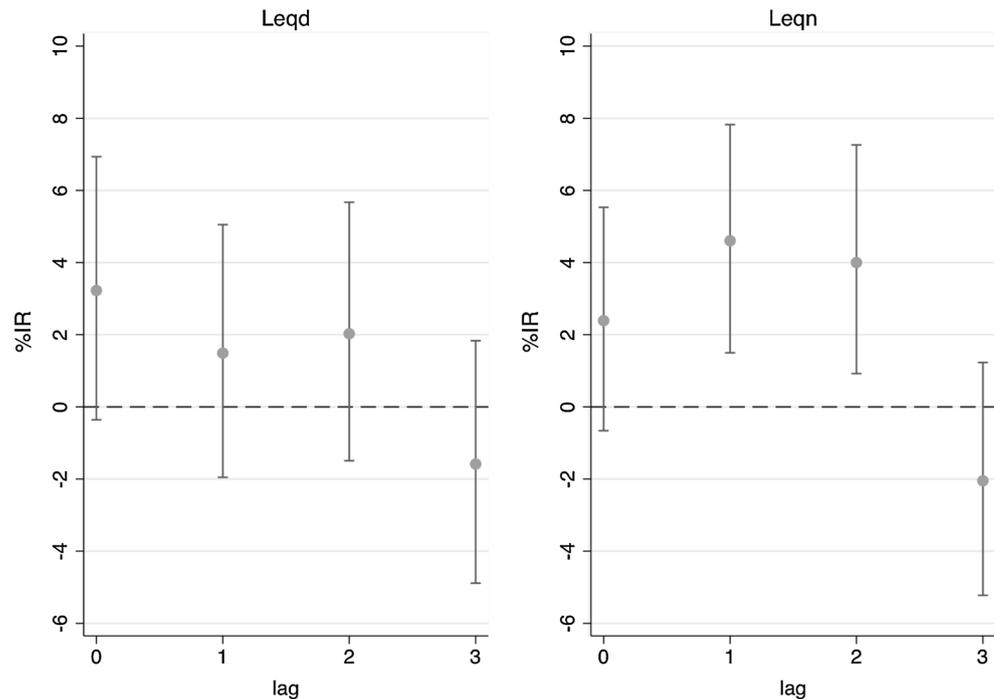
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Fig. 1 Percentage increase in risk (%IR) of mortality from diabetes by an increase of 0.5 db(A) of diurnal (Leqd) and night-time (Leqn) equivalent noise in Madrid for the study period 2001–2009



This association could apparently be surprising, but noise and diabetes incidence have also recently been related [4]. Furthermore, the strongest effect was found for night-time noise, which mainly leads to excess of stress hormones due to reduction in sleep quality and increasing the risk of diabetes. This suggests that the health-effects of exposure to high-noise levels are non-spurious.

Conflict of interest Aurelio Tobías, Julio Díaz, Alberto Recio and Cristina Linares declare that they have no conflict of interest.

Human and Animal Rights disclosure In this ecological study no experiments have been carried out on human subjects.

Informed consent disclosure Daily counts of deaths from diabetes were provided by Madrid Regional Inland Revenue Department according to privacy rules omitting all identifying details, and being lawfully stored and managed.

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