

Is it traffic-related air pollution or road traffic noise, or both? Key questions not yet settled!

Maria Foraster

Published online: 30 July 2013
© Swiss School of Public Health 2013

There is substantial evidence for the association of long-term exposure to both road traffic noise and to ambient and traffic-related air pollution on cardiovascular diseases (CVDs) (Brook et al. 2010; van Kempen and Babisch 2012). As traffic is the main common source of noise and air pollution, the question remains whether there is a mutual confounding effect of these stressors on common cardiovascular outcomes (Brook et al. 2010; Babisch 2011). Identifying whether air pollution and/or noise drive this association is relevant to implement adequate abatement policies.

In this issue, Tétreault et al. (2013) provide the first systematic review that assesses whether the cardiovascular effects of long-term exposure to road traffic noise or to air pollution are mutually confounded based on the existing literature. Interestingly, this review comes to the conclusion that there is not much confounding (i.e. a change below 10 % in effect estimates), although this confounding is higher in some of the 9 studies that met the inclusion criteria.

Although this finding could be interpreted as that there is no need to adjust for traffic-related air pollution or noise in studies, the authors acknowledge that, due to the scarce literature, this review cannot draw a final conclusion regarding the degree of mutual confounding effects of these environmental factors on their long-term association with any cardiovascular outcome. Moreover, while a low

confounding effect may well be true in several settings, findings may not be generalized to all study populations. Indeed, many factors may contribute to the confounding, such as the health outcome assessed, the study design, the exposure assessment, the population and urban characteristics, and the sample size.

The review points to several key questions. First, conceptually, the degree of confounding will depend on the magnitude of the relationships between the exposures (i.e. air pollution and noise) and between the exposures and the outcome. In turn, the confounding may change across study areas given that the spatial correlation between traffic-related air pollution and road traffic noise can vary with urban features (e.g. Allen et al. 2009; Foraster et al. 2011). However, Tétreault et al. (2013) do not see an influence of the correlation on the confounding effects across studies. Although authors propose that this agrees with a low confounding, it may indeed relate to the fact that not all studies observed associations between the main exposure (e.g. air pollution) and the outcome, and the same may apply to the confounding factor (e.g. noise). In any case, the observation by Tétreault et al. (2013) would call for the assessment of the confounding effect irrespective of the correlation observed in each study area.

Second, different noise and air pollution indicators were used in the literature, which may contribute to the heterogeneity in the confounding effect across studies. Besides, the air pollution indicators were not always specific of the traffic-related emissions, which would lead to an inadequate control for the potential confounding effect.

Third, the authors acknowledge that not all studies provided information on the quality of the exposure assessment, thus preventing the interpretation of the quality of the adjustments. In addition, it should be noted that the final treatment of the exposure variable (e.g. categorization,

This Editorial is part of the special issue: “Environment and Health Reviews”.

M. Foraster (✉)
Centre for Research in Environmental Epidemiology (CREAL),
Doctor Aiguader, 88, 08003 Barcelona, Catalonia, Spain
e-mail: mariafp@gmail.com

cut-offs for noise at low levels, etc.) may also play a role. These factors were reported as sources of heterogeneity in the association between long-term exposure to road traffic noise and prevalence of hypertension in a recent meta-analysis (Van Kempen and Babisch 2012) and they may also apply to air pollution. This may particularly happen in studies where information on the secondary exposure (i.e. the confounding factor) is insufficient, leading to residual confounding.

Finally, and most importantly, it is not yet clear how personal exposure may differ from the commonly used surrogate of outdoor residential exposure. Although this applies to both air pollution and noise, relevant differences are expected for road traffic noise and little is known about its consequences. In contrast to the usually non-perceptible air pollution, noise is a nuisance for many people. Thus, people may clearly try to cope with noise, e.g. by closing windows or using ear plugs. Moreover, the bedroom orientation, type of windows, and shielding elements in general will substantially modify noise levels indoors, and likely in a greater extent than air pollution levels. Exposure can be also affected by hearing impairments—a frequent problem in an aging population—which limits the perception of noise. As these factors may combine in complex manners, we should be cautious in accepting the notion that adjustment for outdoor road traffic noise levels at the postal address will truly capture the individuals' exposure and will disentangle the effects of traffic-related air pollution from those of road traffic noise. For these reasons, further studies should advocate for improvements in noise exposure assessment, moving from the outdoor road traffic noise levels to better markers of personal road traffic noise exposure. This assessment should be done in the bedroom and during the night, when noise may more likely impact cardiovascular health (World Health Organization 2009).

In summary, is it traffic-related air pollution or road traffic noise, and what should we do next? While there is no conclusive evidence whether or to what extent the

suggested cardiovascular effects of traffic-related air pollution and of road traffic noise have been disentangled, as suggested by Tétreault et al. (2013), the independent cardiovascular effects of the two environmental factors are likely. This is supported by the proposed biological mechanisms and the evidence on experimental studies for both noise and air pollution and cardiovascular endpoints (Brook et al. 2010; Babisch 2011). Thus, to properly estimate the magnitude of their independent effects, further studies are needed that address confounding with refined exposure assessments and using markers specific to traffic-related air pollution and noise. To settle these key questions, close collaboration of the noise and air pollution research communities is required.

References

- Allen RW, Davies H, Cohen MA et al (2009) The spatial relationship between traffic-generated air pollution and noise in 2 US cities. *Environ Res* 109:334–342. doi:10.1016/j.envres.2008.12.006
- Babisch W (2011) Cardiovascular effects of noise. *Noise Health* 13:201–204. doi:10.4103/1463-1741.80148
- Brook RD, Rajagopalan S, Pope CA 3rd et al (2010) Particulate matter air pollution and cardiovascular disease: an update to the scientific statement from the American Heart Association. *Circulation* 121:2331–2378. doi:10.1161/CIR.0b013e3181d8e3e1
- Foraster M, Deltell A, Basagaña X et al (2011) Local determinants of road traffic noise levels versus determinants of air pollution levels in a Mediterranean city. *Environ Res* 111:177–183. doi:10.1016/j.envres.2010.10.013
- Tétreault L, Perron S, Smargiassi A (2013) Cardiovascular health, traffic-related air pollution and noise: are associations mutually confounded? A systematic review. *Int J Public Health*. doi:10.1007/s00038-013-0489-7
- Van Kempen E, Babisch W (2012) The quantitative relationship between road traffic noise and hypertension: a meta-analysis. *J Hypertens* 30:1075–1086. doi:10.1097/HJH.0b013e328352ac54
- World Health Organization (2009) Night noise guidelines for Europe. World Health Organization Europe, Copenhagen