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EDITORIAL

Mechanistic data support protecting non-smokers from the lethal effects of second-hand smoke

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In 1998 when California became the first major jurisdiction to ban smoking in bars, Paul Blanc and colleagues took advantage of this natural (governmental) experiment to advance our understanding of the health impact of second-hand smoke (SHS) on non-smokers. Anticipating the reduction in exposure, they studied 53 bar tenders before and 2 months after the ban, demonstrating clear improvements in pulmonary function (Eisner et al. 1998). Although previous studies had demonstrated reduced symptoms following exclusion of smoking from workplaces, this was the first study to employ objective biomarkers of dysfunction. Sixteen years later, data are still being marshaled to clarify the health implications of eliminating smoking from workplaces, with an important contribution by Rajkumar et al. in this issue of the International Journal of Public Health (Rajkumar et al. 2014).

Taking advantage of the non-uniform distribution of recent smoking bans in Swiss hospitality workplaces, they measured biomarkers of cardiovascular effects, before and after the bans, in 55 non-smoking hospitality workers who worked in venues targeted for a smoking ban, compared to 23 controls who had continued exposure to SHS. A major strength of this study was the use of nicotine badges to quantify smoke exposure as cigarette-equivalents-per-day (CE/d) (Rajkumar et al. 2013). Over 12 months, pulse wave velocity (PWV) and heart rate variability (HRV) changed in a dose-dependent manner such that a one CE/d decrease was associated with a 2.3 % higher RMSSD (root mean squared of successive differences in the R–R interval), a 5.7 % higher HF (high frequency) HRV, and a 15.1 % lower PWV. These findings add support for two distinct mechanisms, vascular dysfunction and autonomic dysfunction, to the strong and consistent epidemiologic associations between reductions in SHS and decreased risk for acute adverse cardiovascular events.

What does this study add to the evidence basis for public health actions to regulate smoking as a danger to non-smokers? Almost 50 years ago, Sir Austin Bradford Hill put forth his now famous guidelines, notably not criteria, to be considered in support of making a determination of causality between an (occupational) exposure and an outcome (Hill 1965). Beyond robust epidemiological associations from multiple studies, arguably the most important benchmark is biological plausibility or mechanistic data, often from animal models, but increasingly recognized to come from experimental or quasi-experimental human studies with biomarkers.

Since Blanc's ground-breaking 1998 investigation, epidemiologic data on adverse cardiovascular events have accumulated to clearly link smoking restrictions in multiple populations to decreased cardiovascular health events (particularly, acute myocardial infarction) in subsequent months (Tan and Glantz 2012). Parallel data on likely mechanistic underpinnings for these epidemiologic observations have been recently reviewed for both SHS and ambient air pollution (Barnoya and Glantz 2005; Brook et al. 2010). In particular, there are substantial data that link smoking to chronic changes in arterial stiffness (Kubozono et al. 2011; Tomiyama et al. 2010) and arterial stiffness to changes in risk for cardiovascular events (Vlachopoulos et al. 2010). Moreover these, as well as other biomarkers of coagulation and endothelial function reflect change over an acute to subacute to subchronic time frame, allowing conduct of panel studies and quasi-experiments such as the

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partial Swiss hospitality smoking ban considered here (Peters et al. 1997; Rich et al. 2012; Tan et al. 2000).

Acute effects of exposure to SHS on HRV, a marker of autonomic tone, have been shown in previous studies using cross-sectional approaches (Pope 2001; Wilson et al. 2010; Zhang et al. 2013), but Rajkumar et al. present results of the first prospective study to examine the effects of reducing SHS exposure on HRV, with addition of a dose–response in a real-world setting. Alterations in HRV have been linked to changes in particulate matter air pollution in a number of studies, but the results have not always been consistent, perhaps due to differences in methods and host factors (age, sex, comorbidities, etc.) between studies (Pieters et al. 2012). Although lower HRV has been linked to increased cardiovascular mortality in different at-risk patient groups (Bilchick et al. 2002; Tapanainen et al. 2002), the relatively short-term changes in HRV that have been associated with environmental exposures have not been directly linked to health outcomes. More work is needed to understand how SHS, as well as ambient air pollution, affects HRV, and how changes in autonomic function may mediate adverse cardiovascular outcomes.

We have seen how the power of complementary epidemiologic and mechanistic data, from both human biomarker and animal studies, has led to strong conclusions about causal relationships between ambient air pollution and adverse cardiovascular effects, providing evidence for successive reductions in permissible levels for criteria air pollutants in Europe and the USA. In setting health-protective standards for ambient air pollution, we confront the challenges of clearly defining dose–response relationships, including identification of possible thresholds. For SHS, there is no technical barrier and also no economic barrier to banning smoking completely from workplaces, practically eliminating this source of exposure (Schulz et al. 2012). It's time to do just that.

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