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Risk estimate of ischaemic heart disease in workers exposed to beryllium

Mary Schubauer-Berigan and coworkers[1] presented quantitative beryllium exposure measurements and an increased risk of cor pulmonale (standardised mortality ratio (SMR) 1.17; 95% CI 1.08 to 1.26) in a well-conducted cohort study. However, no risk estimate of ischaemic heart disease (IHD) was reported.

In the mid-1990s, a general hypothesis was launched linking inhalation of particles to the occurrence of IHD in urban as well as working environments by an inflammatory pathway associated with increased blood coagulation. In 2010, the American Heart Association concluded strong epidemiological evidence of a relation between short-term (days) or long-term exposure (months to years) to $PM_{2.5}$ (fine particulate matter) urban air pollutants and the occurrence of IHD. Today, studies indicate strong overall mechanistic evidence for a systemic inflammatory response as an intermediary pathway between inhalation of particles and IHD.[2]

Chronic beryllium exposure may stimulate the acquired immune response to release mediators of chronic inflammation in the lung involving cellular and molecular components of innate immunity, and it is this vicious cycle driven by beryllium that results in progressive impairment of lung function, granuloma formation and progression to lung fibrosis.[3] An association has been suggested between exposure to other agents known to cause pulmonary fibrosis such as silica[4] and asbestos[5] and an increased incidence of IHD. Furthermore, one previous US cohort study of beryllium workers observed an increased risk of IHD.[6]

With quantitative exposure estimates being available in the latest cohort of US beryllium workers, Schubauer-Berigan and coworkers have an excellent opportunity to study potential dose–response relations between beryllium exposure and IHD.

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CORRECTIONS

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P Mikkonen, Eira Viikari-Juntura, Jouko Remes *et al*. Physical workload and risk of low back pain in adolescence. *Occup Environ Med* 2012;**69**:4 284–290. The authors noticed errors concerning the rates of incidence and persistence of LBP on page 3. Some of the numbers in this section are incorrect. The following sentences are incorrect: In adolescents without LBP at baseline, the incidence of "Reporting LBP" was 29% in girls and 19% in boys and that of "Consultation for LBP" was 2% in both genders. The persistence of "Reporting LBP" was 53% in girls and 46% in boys and that of "Consultation for LBP" 19% in girls and 17% in boys. The correct version of these sentences is as follows: In adolescents without LBP at baseline, the incidence of "Reporting LBP" was 46% in girls and 31% in boys and that of "Consultation for LBP" was 5% in girls and 4% in boys. The persistence of "Reporting LBP" was 69% in girls and 62% in boys and that of "Consultation for LBP" 23% in girls and 21% in boys.

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R Hampel, S Breitner, W Zareba *et al*. Immediate ozone effects on heart rate and repolarisation parameters in potentially susceptible individuals. *Occup Environ Med* 2012;**69**:6 428–436. The authors of this paper noticed some errors in the numbers of the Tamp section of table 2. In the [95%–CI] column for Outside and summer, the first 5 numbers were missing a minus sign. Please see the correct version of the table below.

Table 2

		Whole study period		Inside or winter		Outside and summer	
		% of Ozone		% of Ozone		% of Ozone	
	Confounder	Measurements*	% Change (95% CI)	Measurements*	% Change (95% CI)	Measurements*	% Change (95% CI)
HR	Main model	100	0.62% (0.02% to 1.22%)	100	0.32% (−0.33% to 0.97%)	100	1.13% (0.47% to 1.80%)
	+ Sulfate†	96	0.74% (0.13% to 1.35%)	96	0.45% (−0.21% to 1.11%)	95	1.17% (0.49% to 1.85%)
	+ UFP†	97	0.81% (0.15% to 1.46%)	96	0.44% (−0.27% to 1.16%)	99	1.25% (0.55% to 1.95%)
	+ $PM_{2.5}$†	100	0.82% (0.21% to 1.46%)	100	0.56% (−0.12% to 1.24%)	100	1.29% (0.61% to 1.97%)
	+ PM_{10}†	100	0.85% (0.22% to 1.48%)	100	0.56% (−0.13% to 1.24%)	100	1.28% (0.60% to 1.96%)
	+ Time of day	100	0.30% (−0.30% to 0.90%)	100	0.06% (−0.58% to 0.70%)	100	0.80% (0.13% to 1.47%)
Tamp	Main model	100	−1.32% (−2.19% to −0.45%)	100	−1.27% (−2.22% to −0.33%)	100	−1.48% (−2.44% to −0.52%)
	+ Sulfate†	96	−1.40% (−2.29% to −0.51%)	96	−1.37% (−2.35% to −0.40%)	95	−1.52% (−2.50% to −0.53%)
	+ UFP†	97	−1.18% (−2.13% to −0.24%)	95	−1.06% (−2.10% to −0.02%)	99	−1.40% (−2.42% to −0.39%)
	+ $PM_{2.5}$†	100	−1.50% (−2.40% to −0.59%)	100	−1.50% (−2.50% to −0.50%)	100	−1.63% (−2.61% to −0.65%)
	+ PM_{10}†	100	−1.54% (−2.47% to −0.63%)	100	−1.54% (−2.54% to −0.54%)	100	−1.65% (−2.64% to −0.67%)
	+ Time of day	100	−0.85% (−1.71% to 0.02%)	100	−0.89% (−1.79% to 0.08%)	100	−0.95% (−1.91% to 0.01%)
Tcomp	Main model	100	2.16% (0.81% to 3.52%)	100	2.13% (0.68% to 3.60%)	100	2.42% (0.94% to 3.92%)
	+ Sulfate†	96	2.27% (0.89% to 3.67%)	96	2.24% (0.84% to 3.86%)	95	2.37% (0.85% to 3.91%)
	+ UFP†	97	2.14% (0.69% to 3.60%)	96	2.08% (0.50% to 3.67%)	99	2.40% (0.85% to 3.97%)
	+ $PM_{2.5}$†	100	2.30% (0.90% to 3.72%)	100	2.33% (0.81% to 3.88%)	100	2.54% (1.04% to 4.09%)
	+ PM_{10}†	100	2.53% (1.12% to 3.95%)	100	2.59% (1.06% to 4.15%)	100	2.72% (1.21% to 4.26%)
	+ Time of day	100	1.54% (0.21% to 2.90%)	100	1.60% (0.16% to 3.06%)	100	1.77% (0.30% to 3.36%)

Main model: time-trend, air temperature, and relative humidity.
Winter: October–March, summer: April–September.
*Observations with missing values in outcome or confounder variables were removed.
†Air pollutants were included with the same lag as the analysed ozone lag.
CI, confidence interval; HR, heart rate; Tamp, T-wave amplitude; Tcomp, T-wave complexity; UFP, ultrafine particles; PM_{10}: particulate matter with an aerodynamic diameter below 10 μm; $PM_{2.5}$: particulate matter with an aerodynamic diameter below 2.5 μm.

Correction

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Notes