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Myocardial infarction in Swedish subway drivers

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High levels of airborne particulates have been detected on underground platforms in the subway system of Stockholm (1), as well as in London (2, 3), New York (4), and Rome (5). The particles originate mainly from brakes, wheels, and rails and contain a high proportion of iron. The particles are mainly in the size range of 1–10 µm (3, 6, 7) and are thus larger than particles generated by motor vehicles. The level of particulate matter with an aerodynamic diameter of <10 µm (PM10) in the air of an underground platform in Stockholm was found to be 470 µg/m³ (measured during 2 weeks in the year 2000, average level during weekdays between 0700 and 1900), which is 4–5 times higher than the levels of PM10 found in one of the busiest streets in Stockholm (1). The subway system is thus an important source of exposure to particles, for both commuters and subway staff. This situation has led to concern for negative health effects in both groups.

The association between particles in urban air and cardiovascular morbidity and mortality in the general population is well documented (8–10). Inhaled particles are deposited in the small airways and may cause an inflammatory response and, therefore, lead to an increased risk of myocardial infarction (11–13). The PM10 from the Stockholm subway has been found to induce oxidative stress in cultured human lung cells, and the effect of subway particles was stronger than that of street-level particles, when compared on a weight basis (14, 15). Subway particles can also induce inflammatory cytokines in human macrophages (15). Particles from the London underground have been shown to be cytotoxic and to have inflammatory properties (3). However, there...
have been no previous studies on the risk of myocardial infarction among persons exposed to airborne subway particles.

The aim of this study was to investigate whether there is an increased incidence of myocardial infarction for subway drivers in Stockholm. Due to the small number of cases of myocardial infarction among female subway drivers (3 cases), this study was restricted to men.

**Study population and methods**

The study population and methods for identifying the cases and controls have been described in detail previously (16), but are summarized here. The study population consisted of all men 40 to 69 years of age residing in Stockholm County during the period 1976–1996. We identified incident cases of acute myocardial infarction in the study population by using registers of hospital discharges and deaths (17–19). The method of case ascertainment has been validated and found to have a high sensitivity for identifying new cases of myocardial infarction in a defined population and also a high positive predictive value for Sweden (20) and Stockholm County (19). We identified a total of 24 315 incident first myocardial infarction cases. We selected the controls randomly from the general population using registers of the total population of Stockholm County on 31 December of each calendar year. For the period 1976–1984, the sampling of controls was frequency-matched for gender, age (5-year age groups), and calendar year, and we selected two controls for each case. For the period 1985–1996, we selected 1500 controls per age (5-year age groups) and calendar year stratum. Persons with a previous history of myocardial infarction were excluded. We selected a total of 138 484 controls.

We obtained information about occupation, branch of industry, and socioeconomic group for the cases and controls using the national censuses of 1970, 1975, 1980, 1985, and 1990. We identified subway drivers by combining the occupational code for “locomotive driver” (code 631) with the branch of industry code for “bus and tram traffic” (code 7112 in census 1970, 1975 and 1980; code 71120 in census 1985 and 1990) (21). A participant was classified as a subway driver if he had reported working as such in any census preceding the year of inclusion in the study. Fifty-four cases of myocardial infarction and 250 controls had worked as subway drivers. The analyses were restricted to people holding the same type of job (occupation and branch of industry) in two censuses (job duration ≥5 years) preceding inclusion in the study as a case or a control in order to investigate whether subway drivers with a longer duration of employment had a higher risk. To study the importance of latency time (ie, whether there was a delay in the effect), we performed analyses restricted to persons first holding an occupation ≥10 years before inclusion. To study the importance of recent exposure (ie, whether there was a short duration of the effect), we performed analyses restricted to persons who were still in the occupation or had stopped no more than 5 years before inclusion.

In the Swedish system for the classification of socioeconomic groups (22), people are subdivided into manual workers, nonmanual employees, and self-employed persons. We classified the socioeconomic group for the cases and controls from the census preceding the year of inclusion. If the participant was not employed at that census, we used information from the next previous census back in time until all available censuses had been used. People with no employment in any census were excluded. The number of cases and controls included in the study (after the exclusion of controls with previous myocardial infarction or people for whom there was no information on occupation) are presented in table 1. The study was evaluated and approved by the Regional Ethics Committee in Stockholm, Sweden.

**Statistical methods**

The association between work as a subway driver and the first acute myocardial infarction was evaluated by computing odds ratios by stratified analysis using the

| Table 1. Number of male cases and controls 40 to 69 years of age according to occupation. |
|---------------------------------------------------------------|
| **Group** | Occupation in any census before inclusion | Job duration ≥5 years before inclusion | Start of employment ≥10 years before inclusion | End of employment ≤5 years before inclusion |
|-----------|------------------------------------------|---------------------------------------|-----------------------------------------------|------------------------------------------|
| Subway drivers | 54 | 250 | 23 | 120 | 25 | 154 | 19 | 96 |
| Others gainfully employed | 22 257 | 131 246 | 9 773 | 68 387 | 16 893 | 112 209 | 12 741 | 93 941 |
| Other manual workers | 8 694 | 43 432 | 2 993 | 17 043 | 6 598 | 36 483 | 4 363 | 27 741 |
| Total (subway drivers and others gainfully employed) | 22 311 | 131 496 | 9 796 | 68 507 | 16 918 | 112 363 | 12 760 | 94 037 |

*Subcategory of others gainfully employed includes manual workers who never worked as a subway driver.*
Results

Altogether, there were 54 incident cases of myocardial infarction among the male subway drivers in Stockholm during the study period (1976–1996) (table 1). The incidence of myocardial infarction was not elevated among the subway drivers, neither when compared with other manual workers (RR 0.92, 95% CI 0.68–1.25) nor when compared with those gainfully employed as something other than a subway driver (RR 1.06, 95% CI 0.78–1.43) (table 2). Subgroups were formed to investigate whether the risk was higher among those employed long-term (job duration ≥5 years), after a long latency (≥10 years since the start of employment) or if the risk was associated especially with current or recent exposure (≤5 years since the end of employment). In all, there were 23 cases of myocardial infarction among the subway drivers with a duration of employment of ≥5 years. No increased risk was observed in this subgroup, and the relative risks were close to those reported for all of the included subway drivers (table 2). The analyses of latency time did not show any elevated risk for subway drivers with a long latency time since the start of employment or for current or recent drivers (table 2). We performed additional analyses with more clearcut restricted groups for the analysis of the importance of latency time (≥10 years since the start of employment but no recent exposure), and recent exposure (≤5 years since the end of employment but <10 years since the start of employment), but it gave similar results. We also calculated the relative risk for myocardial infarction among subway drivers for the time periods 1976–1984 and 1985–1996 separately in order to evaluate the potential effect modification according to calendar time. The incidence of myocardial infarction was not elevated for the subway drivers in 1976–1984 (RR 1.04, 95% CI 0.70–1.54 versus other manual workers and RR 1.15, 95% CI 0.78–1.70 versus those gainfully employed as something other than a subway driver) or in 1985–1996 (RR 0.79, 95% CI 0.49–1.27 versus other manual workers and RR 0.94, 95% CI 0.59–1.52 versus those gainfully employed as something other than a subway driver). The number of myocardial infarction cases among the subway drivers was small in the separate age strata, and it was hard to judge whether there was an effect modification by age.

Discussion

Subway drivers exposed to airborne particulates in the Stockholm underground did not show an increased incidence of myocardial infarction when compared with other manual workers. Several factors should be taken into account in the interpretation of these findings.

The study was very large and included the entire population of Stockholm County over a period of 20 years. There are no other Swedish cities with a subway system. It is possible that a slightly increased incidence of myocardial infarction among the subway drivers could pass undetected as a result of fluctuations in incidence due to chance. However, the confidence intervals were reasonably narrow, and a true excess risk greater than about 30% seems very unlikely. The method of identifying cases has been evaluated previously and found to have a high agreement with established diagnostic criteria for myocardial infarction (17–19).

Table 2. Relative risk (RR) of myocardial infarction for subway drivers in any census and for subway drivers according to the timing or duration of employment. The subgroups were formed to investigate whether the risk was higher for long-term employed (job duration ≥5 years), after a long latency (start of employment ≥10 years previously) or whether the risk was associated especially with current or recent exposure (end of employment ≤5 years previously). The RR, with 95% confidence intervals (95% CI), were adjusted for age group and calendar year.

| Control group                         | Subway drivers in any census before inclusion | Job duration ≥5 years before inclusion | Start of employment ≥10 years before inclusion | End of employment ≤5 years before inclusion |
|---------------------------------------|-----------------------------------------------|---------------------------------------|-----------------------------------------------|--------------------------------------------|
|                                       | RR 95% CI                                    | RR 95% CI                            | RR 95% CI                                    | RR 95% CI                                  |
| Other manual workers a                 | 0.92 0.68–1.25                               | 0.84 0.53–1.33                       | 0.73 0.49–1.13                               | 0.99 0.59–1.65                             |
| Others gainfully employed             | 1.06 0.78–1.43                               | 0.96 0.61–1.52                       | 0.86 0.56–1.32                               | 1.10 0.66–1.84                             |

* Subcategory of others gainfully employed includes manual workers who never worked as a subway driver.
Very high levels of particulates in the size range 1–10 µm were found in the air of the underground platforms, but drivers spend only part of their worktime at the platforms. Measurements in the driver’s cabins at Stockholm’s three subway lines in 2002 have shown average PM$_{10}$ levels of 62 µg/m$^3$, 108 µg/m$^3$, and 125 µg/m$^3$, whereas the average PM$_{10}$ on the underground platforms have varied between 52 and 418 µg/m$^3$ during the day (23). An investigation with the use of personal sampling during three workshifts for 13 subway drivers in Stockholm during the period 2004–2005 showed an average PM$_{1.5}$ of 19 µg/m$^3$ (24). There is support for an association between the coarse fraction of PM$_{10}$ (particles >2.5 µm) and cardiovascular disease, but the evidence is stronger for fine particles (PM$_{2.5}$) (25).

However, negative health effects on people who stay on platforms for longer periods cannot be excluded. No excess risk was found when the subway drivers were compared with other gainfully employed persons or when they were compared with manual workers who had never worked as subway drivers. Still, it could be argued that subway drivers have a healthier lifestyle than others gainfully employed in general and that a true effect of the particulate exposure is hidden by a low baseline rate of myocardial infarction. However, no higher risk was noted for those employed >5 years than for the subway drivers in general; this finding speaks against such an effect. Nevertheless, the lack of data on major risk factors of myocardial infarction, such as smoking, hypertension, and metabolic diseases, is a weakness of this study. Since the subway drivers were compared with other workers, a healthy worker effect would not be expected to be a major problem of this study. The classification of occupation was based on census information with self-reported data available every fifth year, and the occupation may have been miscoded for some of the men. The risk of misclassification of occupation was lower in the analysis of persons with a job duration of ≥5 years, but the risk of myocardial infarction for subway drivers with a longer duration of employment did not differ from the main results, which indicate that the negative findings were not caused by a misclassification of occupation.

The analyses restricted to people who started to work ≥10 years before being included in the study showed similar results to those for everyone included; this finding indicates that the negative finding was not caused by a short follow-up period. Furthermore, a very short duration of the effect cannot explain the negative findings, since the analyses restricted to people with current or recent employment showed no increased risk.

In conclusion, we found no increased incidence of myocardial infarction for subway drivers in Stockholm. The negative findings do not appear to be explained by chance fluctuations or an insufficient follow-up time.

The findings suggest that exposure to airborne particles in the Stockholm subway did not cause an elevated risk of myocardial infarction, either because the average exposure to particles was too low or because there was no such effect. The negative findings of this study do not rule out negative health effects for subway staff who spend a longer time on the platforms, such as cleaners and ticket controllers.

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References

1. Johansson C, Johansson P-Å. Particulate matter in the underground of Stockholm. Atmos Environ. 2003 Oct;37:3–9.
2. Adams HS, Nieuwenhuijsen MJ, Colville RN, McMullen MA, Khandelwal P. Fine particle (PM$_{2.5}$) personal exposure levels in transport microenvironments, London, UK. Sci Total Environ. 2001;279(1–3):29–44.
3. Seaton A, Cherrie J, Dennekamp M, Donaldson K, Hurley JF, Tran CL. The London underground: dust and hazards to health. Occup Environ Med. 2005;62(6):355–62.
4. Chillrud SN, Grass D, Ross JM, Coulthab D, Slavkovich V, Epstein D, et al. Steel dust in the New York City subway system as a source of manganese, chromium, and iron exposures for transit workers. J Urban Health. 2005;82(1):33–42.
5. Ripanucci G, Grana M, Vicentini L, Magrini A, Bergamaschi A. Dust in the underground railway tunnels of an Italian town. J Occup Environ Hyg. 2006;3(1):16–25.
6. Hurley F, Cherrie J, Donaldsson K, Seaton A, Tran L. Assessment of health effects of long-term occupational exposure to tunnel dust in the London underground. Aberdeen (Scotland): Institute of Occupational Medicine, University of Aberdeen; 2003. Research Report TM/03/02.
7. Johansson C. Källor till partiklar i Stockholms tunnelbana [Particle sources in the underground of Stockholm]. Stockholm: SLB-analys; 2005 [cited 27 June 2007]. Available from: http://www.slb.nu/slb/rapporter/pdf/partikelhalter_kallor_t_bana_6_2005.pdf.
8. Le Tertre A, Medina S, Samoli E, Forsberg B, Michelozzi P, Boumghar A, et al. Short-term effects of particulate air pollution on cardiovascular diseases in eight European cities. J Epidemiol Community Health. 2002;56(10):773–9.
9. Pekkanen J, Brunner EJ, Anderson HR, Tiittanen P, Atkinson RW. Daily concentrations of air pollution and plasma fibrinogen in London. Occup Environ Med. 2000;57(12):818–22.
10. Pope C, Dockery D. Epidemiology of particle effects. In: Holgate S, Koren H, Maynard R, Samet J, editors. Air pollution and health. London: Academic Press; 1999. p 673–705.
11. Iballd-Mulli A, Wichmann HE, Kreyling W, Peters A. Epidemiological evidence on health effects of ultrafine particles. J
12. Ross R. Atherosclerosis—an inflammatory disease. N Engl J Med. 1999;340(2):115–26.
13. Seaton A, MacNee W, Donaldson K, Godden D. Particulate air pollution and acute health effects. Lancet. 1995;345(8943):176–8.
14. Karlsson HL, Ljungman AG, Lindbom J, Moller L. Comparison of genotoxic and inflammatory effects of particles generated by wood combustion, a road simulator and collected from street and subway. Toxicol Lett. 2006;165(3):203–11.
15. Karlsson HL, Nilsson L, Moller L. Subway particles are more genotoxic than street particles and induce oxidative stress in cultured human lung cells. Chem Res Toxicol. 2005;18(1):19–23.
16. Bigert C, Klerdal K, Hammar N, Hallqvist J, Gustavsson P. Time trends in the incidence of myocardial infarction among professional drivers in Stockholm 1977–96. Occup Environ Med. 2004;61(12):987–91.
17. Ahlbom A. Acute myocardial infarction in Stockholm—a medical information system as an epidemiological tool. Int J Epidemiol. 1978;7(3):271–6.
18. Hammar N, Nerbrand C, Ahlmark G, Tibblin G, Tsipogianni A, Johansson S, et al. Identification of cases of myocardial infarction: hospital discharge data and mortality data compared to myocardial infarction community registers. Int J Epidemiol. 1991;20(1):114–20.
19. Linnersjo A, Hammar N, Gustavsson A, Reuterwall C. Recent time trends in acute myocardial infarction in Stockholm, Sweden. Int J Cardiol. 2000;76(1):17–21.
20. Hammar N, Alfredsson L, Rosen M, Spetz CL, Kahan T, Ysberg AS. A national record linkage to study acute myocardial infarction incidence and case fatality in Sweden. Int J Epidemiol. 2001;30 suppl 1:S30–4.
21. Statistiska centralbyrån [Statistics Sweden]. Folk- och bostadsräkningen 1980, yrke (del 9, bilaga 6–7) [National censuses 1980, occupation (pt 9, suppl 6–7)]. Stockholm: Statistics Sweden; 1984.
22. Statistiska centralbyrån [Statistics Sweden]. Socioekonomisk indelning (SEI) [Swedish socioeconomic classification]. Stockholm: Statistics Sweden; 1982. Reports on statistical Co-ordination 1982:4. (English summary)
23. Wisell T. Halter av partiklar i Stockholms tunnelbaneluft: mätning av PM10 och PM1 [Particulate matter in the underground of Stockholm: measurements of PM10 and PM1; internal report]. Stockholm: SL Infrateknik AB; 2002 [cited 27 June 2007]. Available from: http://www.sl.se/Upload/rapporter/uploads/000006959/Halter_partiklar%20i%20Sthlm%20tunnelluft.pdf.
24. Plato N, Bigert C, Larsson B-M, Svartengren M, Gustavsson P. Luftföroreningar i Stockholms tunnelbana: exponering för partiklar och kvävedioxid bland tunnelbanepersonal [Particulate matter in the underground of Stockholm: exposure to particles and nitrogen dioxide among subway staff]. Stockholm: Department of Occupational and Environmental Health, Stockholm Centre for Public Health; 2006 [cited 27 June 2007]. Internal Report 2006:2. Available from: http://www.folkhalsoguiden.se/upload/Arbetsliv/Arbetsliv%20-%20rapporter/Luftf%3C%3Breoeringar%20Stockholms%20tunnelbana_2006_2.pdf.
25. Brunekreef B, Forsberg B. Epidemiological evidence of effects of coarse airborne particles on health. Eur Respir J. 2005;26(2):309–18.

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