

Ischemic heart disease among cooks, cold buffet managers, kitchen assistants, and wait staff

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Objective The objective of our study was to investigate the occurrence of ischemic heart disease (IHD) among cooks and other kitchen workers.

Methods Male and female cooks, cold buffet managers, kitchen assistants, and wait staff were identified in the Swedish National Census of 1970. The cohorts were followed from 1970 until 31 December 1995 and linked to the Cause of Death Register. The referent group comprised all gainfully employed men or women, respectively, identified in the same census. In a second approach, cohorts comprising cooks, cold buffet managers, kitchen assistants, and wait staff – who held the same occupation in both 1970 and 1980 according to the Swedish National Censuses of those years – were followed from 1980 until 31 December 1995. The referent group for these cohorts comprised all gainfully employed men and women (regardless of profession) who held the same occupation in 1970 and 1980 according to the censuses. The standardized mortality ratio (SMR) was calculated as the ratio between observed and expected numbers of deaths.

Result An increased risk due to IHD mortality was observed among cooks, cold buffet managers, and kitchen assistants.

Conclusion This result indicates a possible relation between air pollutants in kitchen environments and IHD. However, the key message is that better dose estimates and confounding control is needed to study the possible relation between inhalation of these air pollutants and IHD.

Key terms air pollutant; inflammation; IHD; kitchen worker.

Food is prepared under high temperatures when grilled and fried. This process degrades sugar, protein, and fat and generates new compounds. Some of these new chemicals are transferred to the environment in aerosols and may contain irritating properties, for example, formaldehyde, acetaldehyde, and acrolein (1, 2). The mean concentrations of these aldehydes vary between 10–46 $\mu\text{g}/\text{m}^3$ in restaurant kitchens (2). However, higher levels have been measured. The highest concentration of formaldehyde was 750 $\mu\text{g}/\text{m}^3$, found in grill kitchens, and the highest concentration of acrolein was 590 $\mu\text{g}/\text{m}^3$ (1). During the pan-frying of beefsteaks, higher aldehydes have also been detected. Frying in margarine increased the level of total particles from

around 1 mg/m^3 (when frying in rapeseed, soybean, or olive oil) to about 12 mg/m^3 (3). Meat-frying is also associated with an increase of small particles; around 100 000 particles per cm^3 have been detected in the range 0.3–0.5 μm (3, 4). Inhalation of these air pollutants in the lungs will hypothetically create a low grade inflammation associated with an increase in plasma fibrinogen and inflammatory markers. The high concentration of fibrinogen will increase the likelihood of blood clotting and thereby raise the risk for myocardial infarction and ischemic heart disease (IHD) (5, 6). Some previous studies have observed an increased risk of IHD among cooks and kitchen workers (see section on previous studies).

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Objective

The aim of this study was to investigate the occurrence of IHD among kitchen workers potentially exposed to air pollutants. The study compared cooks, cold buffet managers, kitchen assistants, and wait staff with gainfully employed men and women in Sweden in order to investigate the occurrence of fatal IHD.

Methods

Male and female cooks, cold buffet managers, and kitchen assistants were identified in the Swedish National Census of 1970. Wait staff and head waiters were identified as a similar socioeconomic group exposed to low levels of kitchen-generated air pollutants. The three cohorts (ie, cooks and cold buffet managers; kitchen assistants; and wait staff) were followed from 1970 until 31 December 1995. The workers were identified by their 10-digit identification numbers and linked to the Cause of Death Register during the period of follow-up. The referent group comprised all gainfully employed men (N=2 047 861) and women (N=1 260 583) identified in the same census. The age of the exposed workers as well as the referents was 20–64 years at the time of entry in 1970.

In a second approach, cohorts comprising cooks, cold buffet managers, kitchen assistants, and wait staff – who held the same occupation in both 1970 and 1980 according to the Swedish National Censuses of those years – were followed from 1980 until 31 December 1995. The referent group for these cohorts comprised all gainfully employed men and women [regardless of profession (N=645 393 and N=370 903, respectively)] identified in these two censuses as having the same occupation. The age of the exposed workers and referents was 20–64 years at the time of entry in 1980.

Table 1. Observed and expected numbers of deaths and standardized mortality ratios (SMR) of ischemic heart disease in different cohorts of *females* exposed to kitchen and restaurant air pollutants, identified in the 1970 Census and followed until the end of 1995. (95% CI = 95% confidence interval)

Cohort	Number of workers	Observed deaths	Expected deaths	SMR	95% CI
Cooks and cold buffet managers	12 348	852	662.9	1.29	1.20–1.37
Kitchen assistants	34 609	1905	1577.6	1.21	1.15–1.26
Waitresses and head waitresses	27 802	1343	1333.8	1.01	0.95–1.06

Ischemic heart disease (IHD) was defined as code 410–414 of the International Classification of Diseases (ICD-7 and ICD-8).

We calculated the expected number of deaths in each age stratum by multiplying the death rate (number of deaths/number of person-years) for all gainfully employed people in this age stratum by the number of person-years among the selected occupational group in the same age stratum. The total number of expected deaths was the sum of expected deaths in all age strata. We calculated the standardized mortality ratio (SMR) as the ratio between observed and expected numbers of deaths.

Result

We observed an increased risk due to IHD among females exposed to air pollutants in kitchens, but not waitresses (table 1). Male kitchen workers had also an increased risk of IHD (table 2) as did – unlike the women – waiters. An increased risk of IHD among male and female kitchen workers with the same occupation in both 1970 and 1980 was also observed (tables 3 and 4).

Discussion

The discussion has been divided into four sections in order to focus on methodological and hypothetical aspects.

Validity and confounding

Cohort studies of occupational groups often use reference rates of incidence or mortality from the total population. Such a comparison is likely to result in an

Table 2. Observed and expected numbers of deaths and standardized mortality ratios (SMR) of ischemic heart disease in different cohorts of *males* exposed to kitchen and restaurant air pollutants, identified in the 1970 Census and followed until the end of 1995. (95% CI = 95% confidence interval)

Cohort	Number of workers	Observed deaths	Expected deaths	SMR	95% CI
Cooks and cold buffet managers	3 051	142	107.0	1.33	1.12–1.56
Kitchen assistants	924	96	61.8	1.55	1.26–1.90
Waiters and head waiters	3 745	243	198.0	1.23	1.08–1.39

underestimation of the true risk as the general population includes sick and disabled people unable to work. This underestimation is known as the healthy-worker effect (7). Thus the present comparison of kitchen workers with gainfully employed men and women is more valid than cohort studies comprising the total population. Furthermore, a possible random misclassification between the index workers and the referents can decrease the observed risk estimates. A weakness of this study was the lack of information regarding exposure intensity and duration, and possible confounding exposures.

Smoking is strongly associated with IHD; male, smoking doctors in the UK had a doubled risk compared with non-smoking doctors (8). This estimate was based on the smoking of ≥ 25 cigarettes/day compared with lifelong non-smokers. Smoking habits were surveyed in a Swedish population aged 18–69 years of age in 1963 (9). The total sample comprised 26 469 women and 25 450 men. The proportion of current smokers was 24% among female workers in the service sector and 19% in the general female population. The corresponding proportions for males were 57% current smokers among workers in the service sector and 49% in the general male population. Based on these smoking habits, the estimated calculated relative risk was 1.04 regarding IHD when female workers in the service sector were compared with the general female population. The corresponding relative risk for males in the service sector was 1.05. These calculations were performed according to the recommendation of Axelson & Steenland (10). The survey on smoking habits was carried out in 1963; certainly smoking habits have changed and decreased during the time of follow-up. Today the prevalence of smokers among women and men is 15% and 13%, respectively (11). However, it can be assumed that the relation between smoking habits among workers in the service sector and the general population is rather constant and that

the impact of smoking in the service sector increases the IHD risk by approximately 4–5% compared with the general population.

In a study of occupation and cancer in Sweden, wait staff were identified in the Swedish National Census of 1970 and followed until 1989. Male waiters had an increased risk of lung cancer [standardized incidence ratio (SIR) 2.5, 95% confidence interval (95% CI) 1.8–3.4] (12). If this increased risk of lung cancer was only caused by smoking, practically all the male waiters must have been smokers (13). If all the waiters were smokers this could hypothetically explain the increased risk of IHD found among male waiters.

During 1997–1999, a Swedish work environment survey was performed based on a sample of approximately 10 000 employees. It is not surprising that kitchen and restaurant workers more often have working hours that are not characterized as day work [28% versus 22% in the general population (14)]. Shift work has been associated with an approximately 40% increased risk of cardiovascular disease (15, 16). Shift work is slightly more common among workers in this sector, but there is probably no difference on a group level between kitchen workers and wait staff.

Psychological strain (reflected by job control and demand) probably does not differ very much between kitchen workers and wait staff. In the previously mentioned survey, 60% of all those employed agreed fully or partly that they had too much to do at work. This number is close to the number reported by cooks (65%) and kitchen and restaurant assistants (61%). Three out of four cooks and kitchen and restaurant assistants could – at most – set their own work pace 50% of the time, compared to half of the total working population (14). Symptoms associated with stress and psychological demands were not more common among cooks, kitchen assistants, or wait staff compared to other workers (17).

Table 3. Observed and expected numbers of deaths and standardized mortality ratios (SMR) of ischemic heart disease in different cohorts of *females* exposed to kitchen and restaurant air pollutants, identified in the 1970 and 1980 Censuses and followed until the end of 1995. (95% CI = 95% confidence interval)

Cohort	Number of workers	Observed deaths	Expected deaths	SMR	95% CI
Cooks and cold buffet managers	3 441	79	52.9	1.49	1.18–1.86
Kitchen assistants	6 621	142	103.0	1.38	1.16–1.63
Waitresses and head waitresses	6 462	105	118.1	0.89	0.73–1.08

Table 4. Observed and expected numbers of deaths and standardized mortality ratios (SMR) of ischemic heart disease in different cohorts of *males* exposed to kitchen and restaurant air pollutants, identified in the 1970 and 1980 Censuses and followed until the end of 1995. (95% CI = 95% confidence interval)

Cohort	Number of workers	Observed deaths	Expected deaths	SMR	95% CI
Cooks and cold buffet managers	870	31	16.6	1.86	1.27–2.65
Kitchen assistants	88	7	4.0	1.74	0.70–3.57
Waiters and head waiters	1 116	35	31.4	1.11	0.78–1.55

Previous studies

In a Finnish study (18), female cooks and kitchen workers were identified in 1970 and followed until 1991. The reference population consisted of all economically active persons. Fatal myocardial infarction was more common among female kitchen assistants (SMR 1.17, 95% CI 1.06–1.31). Mortality from IHD, other than acute myocardial infarction, was more common among female cooks and other kitchen staff (SMR 1.30, 95% CI 1.11–1.54) and among kitchen assistants (SMR 1.40, 95% CI 1.18–1.65).

An increased risk of IHD has also been found among female cooks in England and Wales (19) and male British army cooks (20).

A Swedish case-referent study comprised 12 304 women with myocardial infarctions; for each case, two referents were selected from the study base by random sampling, stratified by age, year of hospital admission, or death of the case. Kitchen assistants were found to be one of ten occupations with an increased risk of myocardial infarction. Waitresses were one of seven occupations found to have a decreased risk of myocardial infarction (21).

An increased risk of myocardial infarction was observed among male food processors in Germany (relative risk 2.0, $P < 0.001$) (22).

Thus, some previous studies have observed a relation between kitchen staff and the occurrence of IHD, however no studies have investigated a possible dose-response relationship.

A hypothesis on inflammation

During the last decade, increasing evidence has linked inflammation to the occurrence of atherosclerosis and thrombosis (23–25). Inflammation markers, such as fibrinogen, C-reactive protein, serum amyloid A protein, and leukocyte cell count, have become established risk factors for IHD (26–28).

A general hypothesis linking exposure to inhaled particles to the occurrence of IHD can be expressed in the following way. Long term inhalation of particles retained in the lungs will create a low grade inflammation associated with an increase in plasma fibrinogen and possibly other coagulants. The raised concentration of fibrinogen will increase the likelihood of blood clotting and thereby the risk for myocardial infarction and IHD (5, 6). Today, research findings provide persuasive evidence that exposure to fine particulate air pollution in the general environment has adverse effects on cardiovascular disease. The biological mechanisms behind this relation are complex and have not been adequately explored (29, 30).

A previous Norwegian study found an increase of respiratory symptoms in relation to kitchen work (31). Another study conducted in Singapore on 1282 non-smoking women found that chronic bronchitis was more common among those who reported their kitchens to be frequently filled with heavy cooking fumes compared to women whose kitchens were rarely filled with such fumes (OR 4.0, 95% CI 1.4–11.3). The same study found that women who cooked more at home also had a significantly lower forced expiratory volume in one second (FEV_1) compared with those who cooked less often (32).

Kitchen workers preparing hot meals have been found to have higher numbers of alveolar macrophages in their expectorate compared with non-exposed persons. This increase of alveolar macrophages is a biomarker of pulmonary irritation (33).

In an experimental study, two groups each comprising 12 healthy volunteers were exposed to cooking fumes in a model kitchen for two periods of two and four hours. The mean exposure of particles for each group was 20 mg/m^3 and 43 mg/m^3 , respectively. Six hours after exposure commenced, the study observed an increase of 15.7% regarding forced expiratory time, compared to a decrease of 3.2% after non-exposure. This was interpreted as a minor short-term spirometric effect after exposure to cooking fumes (34).

The occurrence of chronic bronchitis has been associated with IHD after adjustment for several confounders (35, 36). A relationship between a low FEV_1 and an increased risk regarding IHD has also been observed (37). Thus, if inhalation of cooking-generated air pollutants can cause chronic bronchitis and decrease FEV_1 then the hypothesis of a causal relationship between inhalation of kitchen air pollutants and IHD is strengthened.

Concluding remarks

Kitchen workers are exposed to air pollutants. The increased risk regarding IHD among kitchen workers may be explained by inhalation of these pollutants. The difference between kitchen and non-kitchen workers was more marked among women than men. There are several risk indicators associated with IHD that have not been taken into account in this study (eg, heat, noise, elevated cholesterol levels, hypertension, diabetes, obesity, heavy physical work load, and psychological stressors). The indicative results in this study must be further explored in future investigations with better estimates of exposure and more effective control for confounding factors such as smoking habits.

References

- Vainiotalo S, Matveinen K. Cooking fumes as a hygienic problem in the food and catering industries. *Am Ind Hyg Assoc J*. 1993;54:376–82.
- Svendsen K, Jensen HN, Sivertsen I, Sjaastad K. Exposure to cooking fumes in restaurant kitchens in Norway. *Ann Occup Hyg*. 2002;46:395–400.
- Sjaastad K, Svendsen K. Exposure to mutagenic aldehydes and particulate matter during panfrying of beefsteak with margarine, rapeseed oil, olive oil or soybean oil. *Ann Occup Hyg*. 2008;52:739–45.
- Afshari A, Matson U, Ekberg LE. Characterization of indoor sources of fine and ultrafine particles: a study conducted in a full-scale chamber. *Indoor Air*. 2005;15:141–50.
- Seaton A, MacNee W, Donaldson K, Goddon D. Particulate air pollution and acute health effects. *Lancet*. 1995;345:176–8.
- Sjögren B. Occupational exposure to dust: inflammation and ischaemic heart disease. *Occup Environ Med*. 1997;54:466–9.
- McMichael AJ. Standardized mortality ratios and the “healthy worker effect”: scratching beneath the surface. *J Occup Med*. 1976;18:165–8.
- Doll R, Peto R, Wheatley K, Gray R, Sutherland I. Mortality in relation to smoking: 40 years’ observations on male British doctors. *BMJ*. 1994;309:901–11.
- Central Bureau of Statistics. Smoking habits in Sweden: a mail survey – Spring 1963. Stockholm: Survey Research Center of the Central Bureau of Statistics; 1965.
- Axelson O, Steenland K. Indirect methods of assessing the effects of tobacco use in occupational studies. *Am J Ind Med*. 1988;13:105–18.
- Swedish National Institute of Public Health. Minskat bruk av tobak – var står vi idag? [Reduced use of tobacco – where are we today?] [Internet]. Östersund (Sweden): Swedish National Institute of Public Health; 2007 [cited 21 October 2009]. Report 14. Available from: http://www.fhi.se/PageFiles/3376/R2007_14%20Minskat_bruk_tobak_dec07.pdf
- Pollan M, Gustavsson P. Cancer and occupation in Sweden 1971–1989. Socialstyrelsen rapport 1999:1. Stockholm: Epidemiologisk Centrum, Socialstyrelsen; 1999. [Cited 21 October 2009]. Available from: http://www.socialstyrelsen.se/Lists/Artikelkatalog/Attachments/12191/1999-18-1_1999181.pdf
- Axelson O. Aspects on confounding in occupational health epidemiology [letter to the editor]. *Scand J Work Environ Health*. 1978;4:98–102.
- Swedish National Board of Occupational Safety and Health, Statistics Sweden. Arbetsmiljön 1999 [The work environment 1999; summary in English]. Solna (Sweden): Swedish National Board of Occupational Safety and Health, Statistics Sweden; 2000. AM 68 SM 0001.
- Bøggild H, Knutsson A. Shift work, risk factors and cardiovascular disease [review]. *Scand J Work Environ Health* 1999;25(2):85–99.
- Härmä M. Shift work among women – a century-old health issue in occupational health [editorial]. *Scand J Work Environ Health* 2008;34(1):1–3.
- Swedish National Board of Occupational Safety, Health and Statistics Sweden. Arbetsorsakade besvär 2001 [Work related health problems 2001; summary in English]. Solna (Sweden): Swedish National Board of Occupational Safety, Health and Statistics Sweden; 2001. AM 43 SM 0101.
- Notkola V, Pajunen A, Leino-Arjas P. Occupational mortality by cause in Finland 1971–91 and occupational mobility. Helsinki: Statistics Finland; 1997. Health 1997:1.
- Moser KA, Goldblatt PO. Occupational mortality of women aged 15–59 years at death in England and Wales. *J Epidemiol Comm Health*. 1991;45:117–24.
- Coggon D, Wield G. Mortality of army cooks. *Scand J Work Environ Health*. 1993;19:85–8.
- Hammar N, Alfredsson L, Smedberg M, Ahlbom A. Differences in the incidence of myocardial infarction among occupational groups. *Scand J Work Environ Health*. 1992;18:178–85.
- Bolm-Audorff U, Siegrist J. Occupational morbidity data in myocardial infarction: a case-referent study in West Germany. *J Occup Med*. 1983;25:367–71.
- Epstein SE, Zhou YF, Zhu J. Infection and atherosclerosis: emerging mechanistic paradigms. *Circulation*. 1999;100:e20–e28.
- Ridker PM. Inflammation, atherosclerosis, and cardiovascular risk: an epidemiologic review. *Blood Coagul Fibrinolysis*. 1999;10 suppl 1: S9–S12.
- Ross R. Atherosclerosis – an inflammatory disease. *N Engl J Med*. 1999;340:115–26.
- Danesh J, Collins R, Appleby P, Peto R. Association of fibrinogen, C-reactive protein, albumin, or leukocyte count with coronary heart disease. *JAMA*. 1998;279:1477–82.
- Danesh J, Whincup P, Walker M, Lennon L, Thomson A, Appleby P, et al. Low grade inflammation and coronary heart disease: prospective study and updated meta-analyses. *BMJ*. 2000;321:199–204.
- Danesh J, Lewington S, Thompson SG, Lowe GD, Collins R, Kostis JB, et al. Plasma fibrinogen level and the risk of major cardiovascular diseases and nonvascular mortality: an individual participant meta-analysis. *JAMA*. 2005;294:1799–809.
- Brook RD, Franklin B, Cascio W, Hong Y, Howard G, Lipsett M, et al. Air pollution and cardiovascular disease: a statement for healthcare professionals from the expert panel on population and prevention science of the American Heart Association. *Circulation*. 2004;109:2655–71.
- Pope CA III, Dockery DW. Health effects of fine particulate air pollution: lines that connect. *J Air Waste Manage Assoc*. 2006;56:709–42.
- Svendsen K, Sjaastad AK, Sivertsen I. Respiratory symptoms in kitchen workers. *Am J Ind Med*. 2003;43:436–9.
- Ng TP, Hui KP, Tan WC. Respiratory symptoms and lung function effects of domestic exposure to tobacco smoke and cooking by gas in non-smoking women in Singapore. *J Epidemiol Community Health*. 1993;47:454–8.

33. Sivertsen I, Sjaastad K, Svendsen K, Krökje Å. Alveolar macrophages as biomarkers of pulmonary irritation in kitchen workers. *Ann Occup Hyg.* 2002;46:713–7.
34. Svedahl S, Svendsen K, Qvenild T, Sjaastad AK, Hilt B. Short term exposure to cooking fumes and pulmonary function. *J Occup Med Toxicol.* 2009;4:9. Available online: www.occup-med.com/content/4/1/9.
35. Haider AW, Larson MG, O'Donnell CJ, Evans JC, Wilson PWF, Levy D. The association of chronic cough with the risk of myocardial infarction: The Framingham Heart Study. *Am J Med.* 1999;106:279–84.
36. Jousilahti P, Vartiainen E, Tuomilehto J, Puska P. Symptoms of chronic bronchitis and the risk of coronary disease. *Lancet.* 1996;348:567–72.
37. Hole DJ, Watt GCM, Davey-Smith G, Hart CL, Gillis CR, Hawthorne VM. Impaired lung function and mortality risk in men and women: findings from the Renfrew and Paisley prospective population study. *BMJ.* 1996;313:711–5.

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