NOISE AND ACUTE MI MORTALITY IN AN SAWMILL COHORT

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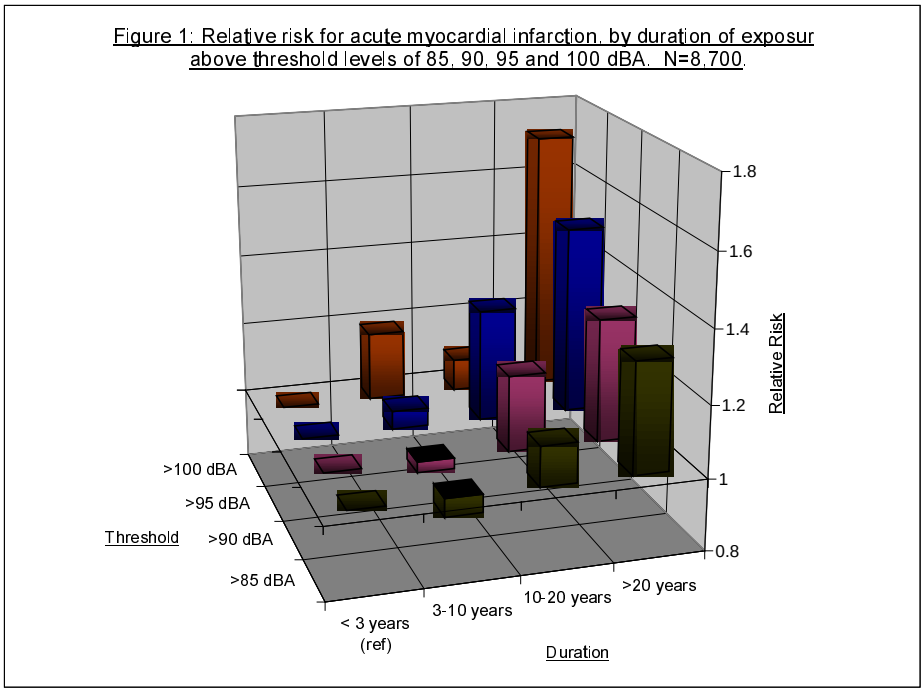
Introduction Noise is a powerful stressor that consistently produces physiological responses typical of a stress reaction in experimental settings. However, results of observational studies of a hypothesized link between noise and cardiovascular disease have been equivocal. This inconsistency might be due to limitations of study design, including study size, and misclassification of noise exposure. This retrospective study examined associations between occupational noise exposure and acute myocardial infarction (MI) in a large cohort of 27,499 lumber mill workers, using a quantitative exposure assessment technique.

Methods Subjects were males from 14 lumber mills employed at least one year between 1950 and 1995. Historical exposure levels were estimated by a determinants-of-exposure regression model, developed using 1,900 full-shift personal dosimetry measurements. The model was used to create an exposure data matrix of 3,809 unique mill/job-title/time period combinations (many of which had no exposure measurement data available). The matrix was then combined with work histories abstracted from mill personnel records to calculate various duration and cumulative noise exposure metrics. We examined acute MI (ICD9=410-410.9) and ischemic heart disease (ICD9 411-414.9, 429.2). SMR's were calculated with the regional general population as referents, using PC LTAS software[1]. Exposure-response relationships were examined using Poisson regression, adjusted for age, calendar year and ethnicity. STATA 7 was used for all analyses (STATA Corp, College Station TX). To reduce exposure misclassification resulting from hearing protector (HPD) use in the last 3 decades of follow up, a sub-cohort was created comprising only workers who terminated cohort employment before the widespread introduction of HPD in 1970 (N = 8,700).

Results In the full cohort there were 2,519 circulatory disease deaths, of which 910 were due to acute MI. SMR's for circulatory diseases were close to 1, although lower risks had been anticipated due to the "healthy worker" effect. SMR's for acute MI were elevated 30-40% for workers exposed more than 30 years over 95 dBA, or for 20 years over 100 dBA. Within the cohort, relative risk (RR) of acute MI was increased 30-60% for those exposed more than 30 years over 95 dBA. In the sub-cohort here were 1,633 circulatory disease deaths, of which 520 were due to acute MI. Sub-cohort RR's for those exposed more than 20 years above 85, 90, and 95 dBA ranged from 1.3-1.5 (P_{trend}'s, <0.05, see figure 1). RR's for cumulative exposure reached 1.6 in the highest exposure group (>115 dBA*yr, P_{trend} <0.001). RR's were greatest however when follow-up was limited to the period of employment, ranging from 2 to 4 (P_{trend}'s <0.01). RR's for ischemic heart disease were elevated but not as strongly or consistently as for acute MI.



Discussion This study found increased RR's for acute MI in individuals chronically exposed to noise at work when compared both to the general population, and to other lower exposed workers. RR's were higher in the sub-cohort that did not use HPD's. This supports the argument that misclassification of exposure assessment due to HPD use can result in attenuated risk estimates. The much higher risks evident for subjects still employed, compared to overall risk, may indicate that these noise-related effects are reversible. The study had certain limitations. Smoking is a strong risk factor for ischemic heart disease, but personal smoking data was unavailable. However, we were able to examine smoking rates in a subset of 1900 cohort subjects surveyed in 1997, and no major differences were found between cohort members and the general population, nor between different noise-exposed groups. Other individual risk factors could similarly not be measured on an individual basis. However for these factors to confound a relationship between noise and acute MI they would have to be correlated with noise exposure, and there was no reason to suspect such a relationship. Other potential cardio-toxic workplace exposures were considered. Of these, psychosocial job strain and physical stress had been previously investigated in this cohort and no association to CVD was found[2]. Other occupational risk factors (CO, temperature extremes, shift work) were considered unlikely to be associated with noise exposure in the work setting under study. Despite efforts to assess noise exposures as accurately as possible, some misclassification is certain. The majority of this can be assumed to be non-differential however, i.e. independent of health outcome status. Thus the risk estimates suggested by this research may in fact underestimate the true risk for acute MI in those exposed to noise.

Keywords: cohort, restrospective, acute MI, ischemic heart disease, exposure modeling

References

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