Thank you Ms. Witherspoon and good morning Madam Chairwoman and members of the Board. In today’s Health Update I will discuss the results of an epidemiologic study that for the first time shows gender-specific associations between fine particle air pollution and fatal heart attacks.
Study Design*

- Middle-aged Californians followed for 22 years (1977-1998) as part of the AHSMOG study (Adventist Health Study on the Health effects of Smog)
  - >3,000 healthy, non-Hispanic white participants
  - 10 years at initial residence in 1976


- Coronary Heart Disease (CHD) deaths were determined from death certificates

- Gender-specific and time dependent statistical analysis


The more than 3,000 healthy, middle-aged participants in this study were part of the Adventist Health Study on the Health effects of Smog or Adventist Study; a study initiated by EPA and ARB and partially supported by the Board from the 1970’s to the 1990’s. Study participants were non-smoking, non-Hispanic whites who also abstained from alcohol and lived at their enrollment addresses for greater than 10 years. Monthly residences and work histories were available for the years 1966-1998.

Estimates of monthly pollutant exposure for PM2.5, ozone, sulfur dioxide and nitrogen dioxide were assigned by zip code for each participant. PM10 concentrations prior to 1987 were estimated using season-specific analysis of total suspended particles. Measures of visibility collected from airports were used to estimate historic PM2.5 concentrations; therefore the majority of these Adventist Study participants lived near major airports in the South Coast, San Francisco or San Diego air basins.

Fatal heart attacks, and other deaths attributed to CHD were linked to the pollution concentrations in statistical analysis. The initial statistical model found that gender was significant to the outcome of fatal CHD; therefore all further analyses were gender-specific.
This slide shows the estimated monthly ambient concentrations of PM2.5 for participants in two of the areas that were studied, Ontario and San Diego; these are plotted against the averages for all of the Adventist participants in the study. As you can see, participants living in Ontario’s east basin were exposed to the highest concentrations of this pollutant while those living in the San Diego air basin were exposed to the lowest concentrations. Average estimated values for all of the Adventist participants in the study are between these two extremes. Also apparent in this figure is the decline in PM2.5 concentrations over time—with the steepest decline in the most polluted area. A calendar-time variable was added to the statistical model to account for this downward trend.
For females in the single pollutant PM2.5 models, there were significant associations between each fractional increase of PM and the risk of fatal coronary heart disease. The strongest association was for PM2.5 with a 42 percent increased risk of fatal CHD for each 10 microgram per cubic meter incremental increase in PM2.5 concentrations. In a two-pollutant model with PM2.5 and ozone, all of the associations between PM and risk of fatal CHD became significant—reaching nearly a 100 percent increased risk for females. No significant associations were found between any of the gaseous pollutants and CHD; and no associations between fatal CHD and PM were found for males.
Using a cumulative monthly average pollution estimate and dividing the female sub-cohort into three different groups: those exposed to low, median or high concentrations of PM2.5; the investigators found that the risk levels increased—with the greatest risk seen in the two-pollutant model at concentrations exceeding 38 micrograms per cubic meter. After adjusting for ozone in the two-pollutant model, the risk estimate for PM2.5 increased to nearly 5.5 times greater risk for those in the highest exposure classification. This enhanced risk for increased exposure or dose-response is further evidence of a causal association between fatal CHD and PM2.5.
The positive association between particle pollution and risk of death due to cardiopulmonary disease has been known for some time. Recent evidence indicates that heart disease is responsible for the largest portion of these deaths.

This is the first study to find a gender-specific effect between coronary heart disease deaths and PM. However, at least one other long-term prospective cohort study identified a larger but non-significant association between risk of cardiopulmonary death and PM for women (1). The results from this study are also consistent with the results of a study we presented to you in September, that found an increase in the risk for atherosclerosis among older women with increased PM2.5 exposure (2). Further studies will be needed to fully understand any differential risk experienced by women.

It has been demonstrated that fine particles deposit to a greater extent and in a more localized fashion in females versus males. Particles are known to have an inflammatory effect on the lung. This inflammatory process may effect blood viscosity—and blood viscosity has been associated with severity of heart disease. In addition, women have fewer circulating red blood cells, thereby making them more prone to these effects.

From a regulatory standpoint, given the chronic health effects of coarse PM observed in this study, USEPA’s proposal to forgo a coarse particle annual standard could be detrimental for Californians.
