Status report

The mortality of DuPont workers in relation to exposure to PFOA (C8)

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The C8 Science Panel has conducted a study of the 5793 workers at the DuPont chemical plant in Parkersburg, West Virginia to determine the rates of death from different causes. The population included workers employed at the DuPont Washington Works plant at any time between 1948 and 2002. The mortality of these workers was tracked through 2008, extending an earlier reported follow-up by 6 years. Information on death and cause of death was obtained by matching to the National Death Index. We compared their patterns of death to the US population, and also compared the patterns between workers with high- and low-exposure to PFOA at the plant.

To make comparisons between low- and high-exposed workers, we estimated the blood PFOA levels over time. To do this we used over 2000 measurements taken by DuPont of blood levels of their workers in different jobs in the period between 1979 and 2004. The measurements enabled us to estimate the PFOA blood levels of workers at any point in time in different job categories.

On average, workers were employed for 19 years, with an average first employment in 1976 and an average last employment in 1995. The average year of birth was 1948, and the average length of follow-up was 30 years. Women made up 19% of the workers, and 5% were non-whites. The average blood level of PFOA for all workers was 350 ng/mL, compared to about 80 ng/mL among community residents in the 2005-2006 C8 Health Project. Most workers in the plant were not directly exposed to PFOA because they did not work in the area where Teflon was produced. Among all the jobs 8% had direct PFOA exposure, 10% had indirect PFOA exposure, 1% were in Teflon maintenance, 15% were in non-Teflon maintenance, and 66% were non-exposed.

During the follow-up period, 18% of the workers died (1085/5793). Overall mortality was low, as is common when comparing worker populations to the US population. This is because the US

population includes many people who are sick, while worker populations, during the period when they are working, do not. This is called the 'healthy worker effect'. The ratio of mortality from all causes of workers compared to the US population was 0.70, which means the DuPont workers had 30% lower mortality than the general US population. In this comparison we took into account the age, sex, and racial distribution of the workers when comparing them to the US. Similarly, worker deaths from cancer were 26% less than expected based on the US population.

We looked at 92 specific causes of death. The only one which was significantly increased compared to the US population was mesothelioma, a rare cancer which is only caused by asbestos exposure, and is unlikely to be related to PFOA. Six workers died of mesothelioma during the period 1999-2008, compared to only 1.2 expected deaths.

It is important to look not only at overall numbers of deaths observed versus deaths expected for different causes, but also to consider whether highly exposed workers had more deaths than expected, compared to low-exposed workers. We divided the workers into 4 groups by the total exposure to PFOA in their blood over time. We found significantly increased rates of death among the more highly exposed workers, compared to the low-exposed workers, for three causes: 1) kidney cancer, 2) non-malignant kidney disease (chronic kidney disease), and 3) mesothelioma. While the trends of increased rates with more exposure were statistically significant for kidney disease, they were based on small numbers (12 kidney cancers, 13 chronic kidney disease cases), and there was no overall excess risk of these disease for all workers combined.

We believe the increased risk with higher PFOA exposure for mesothelioma was not due to PFOA but more likely to asbestos, because individuals with higher PFOA also happened to have had higher past exposure to asbestos, for example, as maintenance workers. However, the increased risk for the more highly exposed in relation to malignant kidney and non-malignant kidney disease could possibly be due to PFOA; the kidney is a site in the body where PFOA is found.

It is important to note that mortality studies, such as this one, are not the best way to study many diseases which may not be fatal (such a non-malignant kidney disease). For these health

outcomes, future C8 Science Panel studies based on disease occurrence, rather than deaths, will provide better evidence about whether there is a link between PFOA and specific diseases. We have conducted interviews regarding past diseases with about 4,500 workers and about 30,000 community residents, and matched them with cancer registries and other sources of medical records. Results from these studies will be available in the first half of 2012.