Comments to the Science Advisory Board on Chemicals by Robert Sussman, Counsel to Safer Chemicals Healthy Families

I'm pleased to be here today to share the perspective of Safer Chemicals Healthy Families (SCHF) on EPA's draft risk evaluation for Perchloroethylene (PCE) under the Toxic Substances Control Act (TSCA). SCHF is committed to assuring the safety of chemicals used in our homes, workplaces, and the many products to which our families and children are exposed each day.

Along with other groups, we've submitted detailed comments on the draft evaluation which I hope SAAC has had an opportunity to review. Today, I'd like to focus on one issue which is a significant concern for PCE and for other chemicals like TCE.

The draft evaluation only estimates risks to consumers from acute exposure to PCE. EPA's position is that consumers have limited and intermittent exposure to PCE and so there is no risk of chronic health effects like cancer and developmental toxicity. We believe EPA is wrong and that multiple lines of evidence demonstrate that consumers have long-term PCE exposure. SACC should highlight this flaw in EPA's approach because it has significant implications for the health of millions of consumers.

As we show in our comments, there is extensive data showing the presence of PCE in indoor air. The draft evaluation identifies 19 valid studies with a median detection frequency of 95 percent (pp. 200-201). The PCE concentrations vary but some are quite high; one study reported PCE levels of 78 µg/m3 and another reported 171 µg/m3. According to the 2012 IRIS assessment, over a lifetime, these levels would present cancer risks greater than 1 in 100,000.

Another line of evidence is the presence of PCE in human blood, urine and breath samples in multiple studies described in the draft risk evaluation (p. 107). These samples show a high frequency of PCE detection, typically 35 percent or greater for blood and over 50 percent for breathing zones. The draft evaluation notes the consistency of the blood levels across studies and over time: "PCE concentrations in blood were similar between the NHANES, SHIELD, and NHEXAS surveys conducted between 1995 and 2016."

Although the amount of data is smaller, PCE has also been found in human breast milk.

The consistent detection of PCE in human blood, urine, breath, and breast milk is incompatible with the assumption that consumer exposure is short-term and episodic. Instead, it provides strong evidence of continuous exposure to PCE by consumers. Reinforcing this conclusion is the relatively short elimination half-life of PCE, also noted in the draft evaluation.

There are probably multiple sources that account for the long-term PCE body burden in the population. One is contaminated drinking water in many different areas in the US. Another is the high volume of PCE air emissions, particularly in areas near vapor degreasing and other unenclosed industrial operations. A third is exposure pathways related to dry cleaners, including air emissions, co-location of dry cleaners with businesses and apartment buildings and the wearing of clothing dry cleaned with PCE. A fourth is vapor intrusion of PCE in buildings near contaminated waste sites or legacy industrial facilities like large dry cleaners.

However, EPA's exclusive focus in the draft evaluation is on a single source of exposure -- consumer product use. This is surely an important contributor to overall exposure, but it should not be assessed in isolation from other known pathways of consumer exposure and without considering evidence of the long-term body burden of TCE in consumers.

EPA is likely following this approach based on its position that it has no obligation under TSCA to consider environmental releases and other PCE sources that impact risk to the general population. But this just underscores the arbitrariness of picking one set of exposure scenarios (consumer products) and ignoring others (environmental releases) when it is the combination of all sources that likely results in long-term exposure to PCE by consumers and the risk of chronic health effects.

Even looking just at consumer products, we disagree with EPA that "it is unlikely that the expected use patterns would cumulatively" result in repeated exposure p. 136). There are clearly significant subpopulations that engage in repeated use of PCE-containing consumer products, such as hobbyists, household cleaners,

home renovators, artists, and do-it-yourself vehicle mechanics. Moreover, while EPA's draft assumes use of a single product type during a day, many consumers likely use different PCE-containing products on the same day or over time. Thus, intensive users of PCE-containing consumer products are plainly exposed to PCE on a chronic basis. These users would be a Potentially Exposed or Susceptible Subpopulation (PESS) under TSCA and EPA must directly address whether they are at risk of chronic health effects.

We are troubled by EPA's statement (p. 386) that it cannot account for these risks because of the "uncertainty regarding the extrapolation from continuous studies in animals to the case of repeated, intermittent human exposures." This is a strange position. Risk assessors typically use repeated dose toxicity studies to estimate the long-term health risks of similar exposure scenarios. For example, PCE industrial and commercial use scenarios likely involve fluctuations in exposure over time based on worker practices and job responsibilities. Why EPA can determine risks to workers using repeat dose animal studies but not to consumers is baffling.

EPA could construct chronic exposure scenarios for PCE-exposed consumers on the basis of central tendency and upper bound PCE concentrations in indoor air and personal breathing zones. It could also undertake PBPK modeling using biomonitoring studies showing PCE levels in blood and urine. These methods would allow for a calculation of steady-state PCE exposures that account for day-to-day variations in exposure, much as EPA does in estimating worker exposures and risks.

EPA would be remiss if it does not estimate risks to PCE-exposed consumers from chronic health endpoints. Failure to address these risks would be a gap in public health protection because a major exposure and risk scenario would be excluded from the evaluation.