

**Practical learnings from an epidemiology study on TDI-related occupational asthma. Part II - Exposure without respiratory protection to TWA-8 values indicative of peak events is a good indicator of risk.**

## **Supplemental Information - 2**

### **Application to TDI-consuming plants**

It is reasonable to ask if conclusions for TDI production plants could be applied to facilities consuming TDI. Whereas this may be the case for the general link with net exposure to high event concentrations, additional factors may have to be considered:

- Potential that the distribution of exposure concentrations is different (background *versus* events);
- Potential exposure to other irritant agents, e.g. amine-based polyurethane formation catalysts (Belin et al., 1983);
- Contribution of manual handling of (foamed) products (risk of dermal exposure);
- Contribution of other specific polyurethane processing operations not present in a TDI production plant (e.g., [hot-wire] cutting, flame lamination), whereby fumes containing particles, isocyanates and other compounds could be generated (Karlsson et al., 1998, 1999, 2001, 2002; Tinnerberg et al., 1996, 1997).

Unless placed close to the breathing zone of the workers, area monitors may not identify all potential exposures at the actual workstation. There is a remarkable observation made by Gui et al. (2014) that in a new and modern TDI-based foam plant, development of asthma symptoms seemed tendentially higher in the medium exposure group (fumes from foam cutting, vapors from laboratory or maintenance work) than in the high exposure group (workers in the foaming area, who generally used respiratory protection). Depending upon the location of the monitors, the regular peaks up to 5 ppb in 18-minute averaged area samples reported by Gui et al. (2014) may not have recorded the true corresponding peaks or the concentration at the workstation itself.

References in addition to those mentioned in the main paper

Belin DL, Wass U, Audunsson G, et al. (1983) Amines: possible causative agents in the development of bronchial hyperreactiveness in workers manufacturing polyurethanes from isocyanates. *British Journal of Industrial Medicine* 40(3): 251-257.

Gui W, Wisnewski AV, Neamtiu I, et al. (2014) Inception cohort study of workers exposed to toluene diisocyanate at a polyurethane foam factory: Initial one-year follow-up. *American Journal of Industrial Medicine* 57(11): 1207-1215.

Karlsson D, Dahlin J, Skarping G, et al. (2002) Determination of isocyanates, aminoisocyanates and amines in air formed during the thermal degradation of polyurethane. *Journal of Environmental Monitoring* 4(2): 216-222.

Karlsson D, Dalene M and Skarping G (1998) Determination of complex mixtures of airborne isocyanates and amines Part 5: Determination of low molecular weight aliphatic isocyanates as dibutylamine derivatives. *Analyst* 123: 1507-1512.

Karlsson D, Dalene M, Skarping G, et al. (1999) Determination of isocyanates in thermal degradation products of polyurethane coatings. In: *Scientific Program & Book of Abstracts, 6th European Meeting on Mass Spectrometry in Occupational and Environmental Health*, Stockholm, 1-3 Sept., 1999, paper P18, 119-120.

Karlsson D, Dalene M, Skarping G, et al. (2001) Determination of isocyanic acid in air. *Journal of Environmental Monitoring* 3(4): 432-436.

Tinnerberg H, Spanne M, Dalene M, et al. (1996) Determination of complex mixtures of airborne isocyanates and amines. Part 2: Toluene diisocyanate and toluenediamine after thermal degradation of a toluene diisocyanate-polyurethane. *Analyst* 121(8): 1101-1106.

Tinnerberg H, Spanne M, Dalene M, et al. (1997) Determination of complex mixtures of airborne isocyanates and amines. Part 3: Methylenediphenyl diisocyanate, methylenediphenylamino isocyanate and methylenediphenyldiamine and structural analogues after thermal degradation of polyurethane. *Analyst* 122: 275-278.