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American Chemistry Council Good Chemistry Makes It Possible

September 16, 2005

VIA HAND DELIVERY

TSCA Confidential Business Information Center (7407M)
EPA East - Room 6428 Attn: Section 8(e)
1201 Constitution Ave, NW
Washington DC 20004-3302

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Attention: TSCA 8(e) Coordinator

Contain NO CBI

RE: Toluene Diisocyanate (TDI) (CASRN not specified)

Dear TSCA 8(e) Coordinator:

The American Chemistry Council's Diisocyanates Panel (Panel) is submitting on behalf of its members[1] information contained in a report titled "Fatalities Linked to Diisocyanates"[2] (*see* attachment). The data provided herein are being submitted pursuant to Section 8(e) of the Toxic Substances Control Act (TSCA).

The attached document provides results of a search of historic information relative to fatalities associated with diisocyanates. This search identified three instances involving TDI which may not be present in information sources as defined in "information which need not be reported," Section VII(a) of EPA's TSCA Section 8(e) Policy Statement and Guidance, 68 Fed. Reg. 33129, 33139 (June 3, 2003).

A brief summary of each instance is provided below:

CCOHS (1990). [Joan Robins: verdict of Coroner's jury]. *Fatality Reports Record no. 2315*. Cause of death in 1989 of a woman with potential occupational exposure to TDI (CASRN not specified), given as respiratory failure secondary to combined effect of acute pneumonia, pulmonary oedema and acute tracheobronchitis with asthma.

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[1] The members of the Panel are BASF Corporation, Bayer MaterialScience, Dow Chemical Company, Huntsman Polyurethanes, and Lyondell Chemical Company.

[2] D. Pallapies and M.A. Collins, Fatalities linked to diisocyanates, III Report 2005/F, August 2005, International Isocyanate Institute Inc., Manchester, UK.

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Rohrer, R. (1976). Fears grow on chemical after worker dies. Sunday Times (London), 19 Sept. Newspaper item reports chemical worker fatality after exposure to TDI (CASRN not specified). Coroner reported death due to "acute bronchospasm due to bronchial asthma". Previous medical tests revealed worker was sensitized to TDI prior to death. The subject worked inspecting printing that used TDI in the ink, and reportedly should not have been exposed to TDI.

Frame, D. (1978). *Manchester Evening News*, 1 December 1978. Reported death of man 1 week after exposure to leaking containers of adhesive containing TDI (CASRN not specified) while transporting adhesive in his car. A post mortem was described to have found "severe damage" to the lungs. But the coroner could not positively say death was caused by the chemical exposure and the verdict was left open.

While being submitted in accordance with TSCA 8(e), the Panel has made no determination as to whether these reported fatalities were caused by TDI exposure.

If you have any questions, please contact me, the Diisocyanates Panel Manager, at 703-741-5635 or susan_lewis@americanchemistry.com.

Best regards,

Susan A. Lewis

Susan Anderson Lewis, Ph.D.
Manager, Diisocyanates Panel

Attachment
Cc: DII Panel

GIL Report

III ref. 24 1665

Fatalities linked to diisocyanates

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BASF

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Gilbert International Limited

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Note

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FATALITIES LINKED TO DIISOCYANATES

Introduction

The published literature relating to diisocyanates often remark that respiratory sensitisation caused by isocyanates may be fatal (NIOSH 1996). The literature has been searched for reports of fatalities, and the reports assessed for their content and the likelihood of the diisocyanate exposure being causal of the fatality.

Secondary references and press items are included so the totality of reports can be judged. One fatality may be reported in several items. In such cases cross reference is made. It is acknowledged that there may be additional reports as yet unidentified. A total of 25 separate reports are considered, the earliest dated 1953.

For convenience, the reports are categorised according to their completeness for assessing causality of the fatality. Thus the most complete reports have details of exposures, history, diagnostic and post-mortem details. The categories used are: A – fully documented reports, B – reports with some, but incomplete details, and C – poorly documented reports. For each report a judgment is made as to the likely causal relationship of the fatality with diisocyanate exposure*. Within category, reports are in date order.

Document Review

A. Fully documented reports.

A1.
Reinl 1953.
Several workers coming into contact with TDI developed between them a variety of symptoms including bronchitis, emphysema, headaches, vomiting, eye irritation, breathing difficulty, cough, dyspnoea, cyanosis, pharyngitis.

The fatality was a person who worked on an experimental project in which polyurethane foam was extruded from a screw-press. This person's job was to regulate the flow of TDI into the operation and clean the equipment. Soon after starting this work, respiratory symptoms appeared. The period between the appearance of the first symptoms and a severe attack of dyspnea (shortness of breath) is not clearly identified. It is also not known whether exposure to TDI was possible during this whole period. The worker was

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* ***Footnote:*** *"Possible" causal relationship with diisocyanates defined as: Activity involving isocyanates shortly before or during development of respiratory symptoms; death due to respiratory problems.*
"Probable" causal relationship with diisocyanates defined as having the features of "possible" association and, additionally, a previous positive isocyanate challenge, isocyanate exposure very likely and an acute asthma-like response.

hospitalized for three months after this attack and subsequently experienced severe difficulties in breathing, especially when performing physical labour and during periods of higher temperatures. Within four years of starting, the worker was issued a disability pension and within eight years he died.

The autopsy revealed well developed emphysema in both lungs, sheet-like pleural coalescence, well developed hypertrophy and dilation of the right ventricle, chronic congestion of the spleen, liver and kidney, ascites and pleural exudates, marked oedema of the extremities, marked general emaciation, slight arteriosclerosis of the aorta and coronary arteries, cortical adenoma of the left adrenal gland.

The death was considered a delayed consequence of the TDI over-exposure. Reinl did not actually study this person, but his reports are based on a review of workers' compensation insurer documents. It is of interest to note that Reinl indicates that at the time exposures to TDI first occurred preventive measures were not as refined or comprehensive as they are today. Apparently this is not a case of acute sensitisation, but lung function loss to which TDI might have contributed.

Conclusion: **Possible** causal relationship with diisocyanates

A2.
Linaweaver 1972.
This case report recounts the death of a diver who had become unconscious whilst assisting in the rescue of another diver who had been applying TDI foam in a partially submerged compartment of a grounded ship. Both men's air supply was interrupted resulting in loss of consciousness after breathing the atmosphere in the compartment. Both men were rescued after being comatose and one of them had been under water for an indeterminate time. Both were revived with artificial respiration and were hospitalized. One recovered uneventfully, but the other (who had been under water) did not. On admission he was noted to be covered with an oily aromatic material, and was in severe respiratory distress, but conscious. Despite treatment with oxygen, antibiotics, corticosteroids his condition worsened and he died four days after the accident. Autopsy revealed oedematous lungs with mucosal haemorrhage. Pathology showed alveolar invasion of inflammatory cells, areas of necrosis, and abscess formation, but no foreign organisms or debris. Death was attributed to anoxia secondary to acute pneumonitis and oedema. It was acknowledged that the concentration of TDI in the compartment was not known, and other materials such as blowing agents would have contributed to the initial unconsciousness, and possibly the lung effects. Immersion in sea water did not apparently play a role in the death, which was ascribed to the corrosive action of TDI and the hypoxic atmosphere.

Conclusion: **Possible** causal relationship with diisocyanates

A3.
Fabbri et al 1988.
A case report of a car painter who died within 1 hour of exposure to a polyurethane paint in the workplace. A diagnosis of asthma induced by TDI had been established six years before, but the subject continued to work with isocyanate containing paints, controlling symptoms with antiasthmatics. The fatal attack developed while he was mixing two components of a paint. Autopsy found over-inflation of lungs, mucous plugs in the airways, and histopathology revealed various changes such as epithelial desquamation, basement membrane thickening, inflammatory infiltration, mucosal oedema and hypertrophy/disarray of the smooth muscle. There were also changes in the alveoli with wall destruction and loss of spaces.

This case was also reported in a letter to an Italian journal (Carona and Mapp, 1987), and NIOSH (1996), **B3** and **C12** herein.

Conclusion: Very **Probable** causal relationship with diisocyanates

A4.
Carino et al 1997.
A case is described of a foundry worker involved in formation of moulds using MDI, who presented for assessment of occupational asthma. There were no isocyanate specific IgE antibodies, borderline bronchioconstriction by spirometry, but methacholine challenge revealed airway hyperresponsiveness. A positive inhalation challenge to MDI resulted in an asthmatic response requiring medication. Six years later he was admitted for several days because of bronchioconstriction. The patient admitted lesser, but continuing, exposure to MDI at work, and occasional asthmatic episodes. Three months later he developed a severe asthmatic attack at work and although medication was used he was dead on arrival at hospital. Post mortem findings revealed over-inflation of lungs, and histopathology revealed various changes such as epithelial desquamation, inflammatory infiltration, airway oedema and hypertrophy/disarray of the smooth muscle.

A table of deaths from isocyanate asthma reported in the literature is also given.

Conclusion: **Probable** causal relationship with diisocyanates

A5.
Haegy et al 2001.
This is a case report of fatal bronchospasm after TDI exposure. The subject was hospitalized for bronchospasm following exposure to TDI through non-use of PPE. The attack was resolved with medication and later testing revealed moderate obstructive airway disease, which was not responsive to beta-2 blockers. There were no isocyanate specific IgE antibodies. His job was changed to reduce exposure, but one year later, he intervened at an incident, which allowed an exposure to TDI. He complained of lack of

air and rapidly collapsed. Emergency services detected asystolic heart failure and cyanosis. At intubation glottal oedema was noted. Severe bronchospasm was treated without effect by intravenous adrenaline. An anti-cyanide treatment was administered. The resuscitation failed. Autopsy revealed signs of mechanical asphyxia by laryngeal oedema with mucoid congestion of trachea and bronchi, pulmonary emphysema. Blood was analysed and TDI determined as present (sic).

Conclusion: **Probable** causal relationship with diisocyanates

A6.
Chester et al 2005.
Full publication of the incident reported also in **B5** and **B6**. A small business car worker was alone whilst installing a spray-on liner to the inside of a van (an MDI formulation). A half mask supplied-air respirator was apparently used for the process, but all equipment was removed and switched off when he was found gasping for breath. He was taken to an urgent care clinic where he collapsed. CPR was carried out and he was shortly taken by ambulance to hospital where he was pronounced dead less than an hour later. Post mortem examination revealed mucus in the airways with eosinophilia. There was thickening of the basement membrane, and diffuse pulmonary emphysema, numerous macrophages in peribronchial alveoli and heavy anthracosis. It is thought possible that the previous asthma might have been chronic obstructive pulmonary disease. The authors suggest that the isocyanate exposure either aggravated the preexisting lung conditions, or that there was a respiratory sensitization in addition to the preexisting disease. The medical examiner's impression was an asthmatic reaction due to inhalation of chemicals.

The deceased had a 10 year history of asthma and had consulted physicians for shortness of breath. A record in the year prior to death reports he inhaled chemicals at work and once had been exposed for 10 minutes spraying a truck bed liner. Within a short period he had respiratory difficulties. He was prescribed steroid and an inhaler, the latter also for long term asthma management. Lung function tests were not done. Coworkers reported that he had complained of breathing difficulties after other spray operations.

Conclusion: **Possible** causal relationship with diisocyanates

B Reports with some, but incomplete, details

B1.
Schürmann 1955.
Two deaths are reported, but one of these is secondary to the report by Reinl 1953 (**A1**). The other death was that of a worker who had applied diisocyanate-containing floor-lacquer for about one year. He had repeated asthmatic attacks although he had not previously experienced asthma or other lung diseases. On renewed working with the lacquer he had an asthmatic attack. He developed an urticarial rash over the whole body, and then vomiting, swallowing difficulty, and finger cramp. Within 24 hrs he died during an asthma attack. The autopsy gave the cause of death as severe bronchitis with tissue eosinophilia, and lung inflammation with highly developed oedema of both lungs.

Conclusion: **Possible** causal relationship with diisocyanates

B2.
Gervais et al 1973.
As the result of an explosion a worker was knocked unconscious (the severity of the head trauma is not further characterized), and inhaled a massive amount of TDI. In hospital the observations included severe respiratory distress, acute oedema of the lungs, marked cyanosis, unconsciousness, and the chest appeared blocked. Even with intubation, positive pressure ventilation, and noradrenaline infusion, the patient died after 4 hours.

Conclusion: **Possible** causal relationship with diisocyanates

B3.
Corona 1987.
A letter to a journal more fully reported later by Fabbri et al (1988) see **A3**.

Conclusion: See **A3**, no additional death.

B4
CCOHS 1990.
This is a report of a coroner's verdict on a fatality dated April 1989. A never-smoking woman with a long history of asthma worked at a flame lamination machine. The report says that the process was known to release TDI. While working, the subject developed an acute asthmatic attack that failed to respond to salbutamol aerosol. She was taken to an industrial medicine clinic, received adrenaline, and was then taken to hospital, but suffered respiratory arrest while in transit. On arrival she was comatose and never regained consciousness, and died after 26 days. Cause of death determined at autopsy was given as respiratory failure secondary to combined effect of acute pneumonia, pulmonary oedema and acute tracheobronchitis with asthma.

Conclusion: **Possible** causal relationship with diisocyanates

B5.
MIFACE Investigation Report, 2003.
This report from MIFACE (Michigan Fatality and Control Evaluation), Occupational and Environmental Medicine, Michigan State University, reports the same incident as A6 and B5. There are practical details of the incident, but no pathology results.

Conclusion: See **A6**, no additional death.

B6.
Michigan State University 2003.
This is a secondary source of an incident in Michigan. The incident is also reported in **B5** and is fully written up and published in **A6**

Conclusion: See **A6**, no additional death.

C. Poorly Documented reports

C1.
Gerritsen, 1955.
Dutch language report includes mention of a painter working inside with Desmodur (TDI) based paint. On the fifth day of work he experienced considerable tightness of the chest and was taken to hospital; there, he was shortly dead after status asthmaticus.

Conclusion: No comment possible

C2.
Baader 1956.
Baader reports that he is generally aware of more than 100 more or less severe cases caused by isocyanates, of which four were fatalities. No specific information is given with regard to these four cases.

Conclusion: No comment possible

C3.
Elkins et al 1962.
Cites Baader (herein as **C2**) as knowing of four deaths from TDI and two further fatal cases in another report (Schürmann, herein as **B1**). The report does describe a case of a worker admitted to hospital in 1961 in a critical condition after TDI exposure. The previous seven days working with TDI had resulted in chest tightness and cough in the evening. Although his condition was critical for an hour, with supportive treatment he recovered. This information is also given in Brugsch and Elkins, 1963.

Conclusion: No additional death reported

C4.
Brugsch and Elkins, 1963.
The same information to that given in Elkins et al, 1962.

Conclusion: No additional death reported

C5.
Rohrer, Sunday Times (London), 19 September 1976.
Newspaper item reports that a chemical worker died after exposure to TDI. The coroner reported death was due to "acute bronchospasm due to bronchial asthma". Medical tests carried out before his death revealed that the subject was sensitised to TDI. The subject worked inspecting printing that used TDI in the ink, and reportedly should not have been exposed to TDI.

Conclusion: **Improbable** causal relationship with diisocyanates

C6.
Frame, Manchester Evening News, 1 December 1978.
A man was transporting samples of adhesives containing TDI in his car, when the lid blew off one and another leaked at the seam. He reported feeling unwell over the next day and then saw his physician reporting breathing difficulties after two days, and died a week after the incident. A post mortem was described to have found "severe damage" to the lungs. The coroner could not positively say death was caused by the chemical exposure, and the verdict was left open.

Conclusion: No comment possible

C7.
HSE 1985.
A brief report of a worker using a 2-pack paint in a garage without respiratory protection. Although PPE was available this was not used for short jobs. After spraying on one day,

and then again the next day the worker experienced chest tightness and breathing difficulties. Later following a bout of vomiting medical attention was sought, although paint spraying work was not revealed. His condition deteriorated further with coughing of blood and he was admitted to hospital. Despite treatment (undefined) he died after one week with pulmonary oedema, pulmonary infiltrates, bleeding and kidney failure.

Conclusion: **Possible** causal relationship with diisocyanates

C8.
Fabbri et al 1988.
In the introduction to Fabbri et al 1988 (**A3**), mention is made to a personal communication by Pauli to Fabbri of a death during inhalation challenge at a hospital.

Conclusion: No comment possible

C9.
Luo et al, 1990.
This is a report of an incident in which two police troopers were exposed to TDI at the site of an accidental spill. The paper says that both cases reported themselves to a medical centre following the exposure. The accident occurred in December 1981, and the two patients were monitored for seven years following. Although the symptoms had persisted, considerable improvement had occurred in both cases. No death was reported in this paper. This incident is also reported in other sources and an internet article by Peterson (2001) on www.firehouse.com states that the troopers were found unconscious in their car, which is at variance with the report of Luo et al. This item also reports that both troopers retired on disability and one died from complications a short time later". This incident is also reported in an article titled "Transportation of hazardous materials", available from the US Office of Technology Assessment (1986) at www.wws.princeton.edu/cgi-bin/byteserv.prl/ ~ota/disk2/1986/8636/863607.PDF, but this does not mention the later death. However, the latter item does cite a source reference which is a newspaper article, (Times Union, Albany NY, Apr 7, 1985, p1).

Conclusion: **Improbable** causal relationship with diisocyanates

C10.
NIOSH 1994.
In September 1991, the State of Alabama Department of Public Health requested technical assistance from the National Institute for Occupational Safety and Health (NIOSH) in investigating a possible health hazard at Distinctive Designs International, Inc., Russellville, Alabama. The request was prompted by the death of a former employee who initially became ill after working with diphenylmethane diisocyanate (MDI) at the plant. This individual had a respiratory illness which his physician felt was consistent with isocyanate-induced hypersensitivity pneumonitis. The facility used an

MDI-based polyurethane foam to fabricate decorative products. No further clinical details are given.

Also reported in a NIOSH Alert (1996), **C11** below.

Conclusion: No comment possible

C11.
NIOSH 1996.
This report lists two fatalities, one (Fabbri et al 1988) dealt with as **A3**, and the other (NIOSH 1994) as **C10** above. More information on the fatality in the latter report is given in the NIOSH alert than in the original 1994 report. The following text is quoted from the document.

"Isocyanate foam operation (One death). A maintenance worker became ill after repairing an MDI foaming system at a plant that manufactured artificial plants with polyurethane foam bases. The worker later suffered recurrent bouts of respiratory illness (diagnosed as isocyanate-induced HP). After showing further respiratory symptoms associated with isocyanate exposure, the worker quit his job but continued to experience coughing and progressive loss of lung function. His illness was eventually complicated by productive cough, weakness, sweats, muscle aches, and shortness of breath. Ultimately, he died. Worksite evaluations found detectable air concentrations of MDI and inadequate ventilation systems in the foaming areas. Vapors and aerosols were observed rising into the faces of employees working with the foam. Skin contact with the curing foam was also noted during the survey."

Conclusion: **Possible** causal relationship with diisocyanates

C12.
Liss et al 1999.
In a survey of mortality among workers compensated for asthma in Ontario, Canada, the authors identified one death from asthma in a person compensated for occupational asthma due to isocyanates. There are no details of the actual case.

Conclusion: No comment possible

C13.
Bakke, 2000.
This report states that in Finland, "In 1997 a sensitised person died after further exposure to MDI while working on a sports track in Helsinki." There are no other details.

Conclusion: No comment possible

Conclusion.

Overall, there have been twenty-two deaths reported in the literature as having some link to diisocyanate-exposure. Of these, eleven are judged to have probable or possible causal relationship to diisocyanate exposure. Many of the cases are poorly documented. Nevertheless, in three of the relatively well documented reports, diisocyanate exposure has to be considered as the most probable cause of death. In all of these three cases obvious asthmatic symptoms (after repeated exposures to probably relatively high concentrations) had been observed previously, and would have required avoidance of re-exposure of these probably sensitised individuals.

In most of the cases for which a causal relationship with diisocyanate exposure is judged probable or possible, re-exposure to diisocyanates of already sensitised individuals with previous high exposures seems to have occurred. In two of the cases, however, death due to sequelae of incidental very high exposure with loss of lung function and progressive respiratory symptoms has been discussed. The information given for these cases does not allow a clear judgment on the cause-effect relationship.

Overall it is clear that once an individual has been sensitised to diisocyanates, further re-exposures should be strictly avoided.

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