

Evaluation of Exposure to Crystalline Silica, Welding Fume, and Isocyanates During Water Heater Manufacturing

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The employer is required to post a copy of this report for 30 days at or near the workplace(s) of affected employees. The employer must take steps to ensure that the posted report is not altered, defaced, or covered by other material.

The cover photo is a close-up image of sorbent tubes, which are used by the HHE Program to measure airborne exposures. This photo is an artistic representation that may not be related to this Health Hazard Evaluation. Photo by NIOSH.

Highlights of this Evaluation

The Health Hazard Evaluation Program received a union request about employees' exposures to silica, welding emissions, and methylene diphenyl diisocyanate during water heater manufacturing.

What We Did

- We observed the process of making water heaters and interviewed employees from the research and development, tank fabrication and finishing, and maintenance departments.
- We measured exposures to silica, welding fume, and methylene diphenyl diisocyanate in air.
- We administered medical questionnaires and took blood samples from employees who worked in foam booths or performed maintenance work on booths. The blood was tested to see if employees showed evidence of exposure and sensitization to methylene diphenyl diisocyanate.

What We Found

- Some local exhaust ventilation systems for welding fume in the tank fabrication area were not working.
- Some ventilated exhaust enclosures in the tank finishing department allowed spray mist to escape.
- The local exhaust ventilation on the mill room hopper was not effective.
- Some employees in foam booths cut slits in their Tyvek® suit to aid in cooling, creating a potential for skin exposure to methylene diphenyl diisocyanate.
- Of 91 employees interviewed, 22 reported symptoms related to work.
- Nine of 10 employees' exposure to crystalline silica exceeded an occupational exposure limit.
- Air sampling results for methylene diphenyl diisocyanate and welding emissions were below occupational exposure limits.
- Six of 29 employees had work-related asthma symptoms, but some had not been seen by a physician.
- Two of 28 employees had blood tests that showed they were sensitized to methylene diphenyl diisocyanate. This puts them at higher risk for asthma due to methylene diphenyl diisocyanate exposure.

We measured exposure to crystalline silica, welding fume, and methylene diphenyl diisocyanate during water heater manufacturing. We held medical interviews and collected blood for biomarkers of isocyanate exposure and sensitization. We found overexposure to crystalline silica during brushing, tank and flue spraying, and mill room operations. Questionnaires and blood tests indicate some employees have developed isocyanate asthma. Some ventilation controls were either not working or were ineffective in containing airborne contaminants. Improvements in ventilation system design, use, and maintenance is needed.

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- Five of 28 employees had blood tests that showed evidence of exposure to methylene diphenyl diisocyanate. This indicates that exposure is occurring despite the use of engineering controls and personal protective equipment.

What the Employer Can Do

- Develop a more effective system for transferring silica to the mixing tank.
- Redesign the local exhaust ventilation for the mill room hopper to eliminate release of powder.
- Add vinyl strip curtains or other controls to the ventilated exhaust enclosures used during tank finishing operations to improve the containment of overspray.
- Provide training on the hazards of working with both silica and diisocyanates.
- Begin a medical surveillance program for employees exposed to methylene diphenyl diisocyanate and silica.
- Promptly refer employees who report asthma-like symptoms to an occupational medicine physician to determine whether or not they can continue to work around these chemicals. Remove any employee with isocyanate-induced asthma from the work environment where isocyanate exposure occurs.
- Create a tracking system that allows employees to submit requests for repairs or equipment maintenance, especially for exposure control systems like local exhaust ventilation.

What Employees Can Do

- Do not cut your Tyvek suit.
- Learn about the risks of working with silica and diisocyanates.
- Report work-related symptoms to the appropriate managers so they can look for ways to reduce your exposures and refer you to an occupational medicine physician for evaluation.

Abbreviations

$\mu\text{g}/\text{m}^3$	Micrograms per cubic meter
ACGIH®	American Conference of Governmental Industrial Hygienists
CFR	Code of Federal Regulations
IgE	Immunoglobulin E
IgG	Immunoglobulin G
MDI	Methylene diphenyl diisocyanate
MIG	Metal-inert-gas
NIOSH	National Institute for Occupational Safety and Health
OEL	Occupational exposure limit
OSHA	Occupational Safety and Health Administration
PEL	Permissible exposure limit
REL	Recommended exposure limit
TLV®	Threshold limit value
TWA	Time-weighted average

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Introduction

In February and July 2016, the Health Hazard Evaluation Program visited a water heater manufacturer. The United Automobile, Aerospace and Agricultural Implement Workers of America were concerned about employees' exposures to silica during tank finishing operations, welding fume during tank fabrication, and methylene diphenyl diisocyanate (MDI) during tank insulation using a spray foam application.

In February 2016, we met with union and employer representatives, toured the plant, observed production processes and work practices, and interviewed employees. We returned to the plant in July 2016 to conduct personal air sampling for silica, welding fume, and MDI. We also collected blood samples from employees who handled MDI to look for indicators of exposure and sensitization. We individually notified participants in writing of their blood and air test results and what these results meant.

Process Description

The plant, which began operations in the 1930s, manufactures gas-fired and electric water heaters, and operates 24 hours/7 days per week. Residential and commercial water heaters are produced at this plant. The assembly lines operate two shifts of approximately 10–12 hours each while other departments (subassembly, preparation, welding, fabrication, and finishing) operate over three shifts of 8 hours each. There are 980 production workers in the plant with a capacity of making 14,000 water heaters per day.

Each water heater tank and outer cover is made from mild steel. Sheet steel is rolled into a cylinder and the joint welded using an automated metal-inert-gas (MIG) machine referred to as a Carando welder. The wire used by the machine to produce the weld consists mostly of manganese and iron. Other automated MIG welding operations secure the top and bottom of each tank as well as the steel flue in the center of each tank. A resistance spot welder attaches threaded spuds and washers to the top of each tank.

Once the tank is completely welded, it is suspended on a trolley hook and moved along a conveyor line to the tank finishing department. The inside of each tank and flue is coated with a silica-based enamel using an automated spray head inside a ventilated enclosure. To produce the spray enamel, a mill room operator loads a hopper with a mixture of powdered bentonite, silica, and porcelain enamel frit. The hopper is equipped with a three-sided hood to control emissions during the dumping process. Once the hopper is full, the powder is released via gravity feed to a mixing tank below. The tank contains water, boric acid, potassium nitrate, and sodium nitrate, which are mixed with the powder to form a slurry. After 10–15 minutes of mixing, the contents are pumped via a network of conduit lines to each spraying apparatus. Once sprayed, the tank enters an oven that bakes on the finish and forms a lining on the inside of the tank. After the enamel has cooled, the tank moves along the line to a brushing station where an employee uses a pneumatically driven rotating wire brush to clean the threads of the plumbing fittings (spuds and bungs). After this step, the tanks are moved to another department where they are fitted with wiring, thermostats, and other controls before being inserted into an outer shell.

Prior to completing the assembly of the water heater, the unit moves via a conveyor belt to a ventilated foam shooting booth where an insulating foam is injected into the void between the inner tank and outer shell. The insulating foam is created through a chemical reaction between MDI monomer and a resin compound. These two substances (Part A and B) are carried to the booth through two hoses. The two hoses terminate at a nozzle that serves as a mixing point for Part A and B. An employee inserts the nozzle into a hole in the top of the outer shell and deposits the mixture under pressure into the void where the foam expands as it reacts and cures. Once this step is completed, the water heater then leaves the booth and moves to another department for final inspection and testing.

Personal Protective Equipment

Anyone entering the production area is required to wear hearing protection (soft foam ear plugs) and eye protection. Personnel in the welding area are not required to wear respiratory protection; however, personnel performing tasks in the tank finishing area (mill room, tank/flue sprayers, brushers) are required to wear either an N95 filtering facepiece respirator (3M model 8210) or a half-mask air purifying respirator equipped with organic vapor cartridges and N95 prefilters (3M model 6001). During MDI spray foam applications, employees are required to wear a full-body Tyvek suit, nitrile gloves, and a face shield. They do not wear respiratory protection. The plant has a written respiratory protection program.

Engineering Controls

In the mill room of the tank finishing department, a three-sided, ventilated hopper is used to control the release of powdered material when loading the mixing tank. In the tank spraying area, each tank is fed into the automated spraying apparatus via a conveyor line. Each tank enters a ventilated enclosure that is open on opposite sides. The overspray is exhausted at the top of the ventilated enclosure through an opening in the roof. In the flue spraying area, each flue is moved into a three-sided ventilated booth where an automated spraying apparatus applies the paint while the flue assembly rotates on a pedestal. Overspray is exhausted at the back of the enclosure and discharged via a duct to the roof.

In the welding department, local exhaust ventilation is used at each MIG or spot welding machine to control fume generated during the welding process. The inlet of a 6-inch flexible duct is attached to the machine at a position as close as possible to the welding arc. The duct is routed upward to an inline fan that discharges exhaust through the roof.

At the MDI spray foam application area, each water heater assembly is moved into and out of a downdraft-style ventilated booth via a conveyor line. Supply air is drawn from the plant via an in-line fan and delivered to the inside of the booth by a 6-inch duct positioned overhead of the operator. Air is exhausted from the bottom of the booth and discharged outside via ductwork through the roof.

Methods

The primary objectives of this evaluation were to:

- Measure employees' exposures to airborne contaminants during tank fabrication, tank finishing, and spray foam application.
- Determine if employees had adverse health effects from work exposures, especially asthma from MDI exposure.
- Determine if employees performing spray foam application or maintenance on the foam booths had biological indicators of exposure or sensitization to MDI (presence of Immunoglobulin E [IgE] and Immunoglobulin G [IgG] antibodies in blood).

Air Sampling

We collected 10 full-shift personal air samples for respirable particulate in the tank finishing area for the following employees over 2 days:

1. Mill room operator
2. Flue sprayer
3. Tank sprayer
4. Brusher

Each sample was analyzed for respirable particulate using National Institute for Occupational Safety and Health (NIOSH) Method 0600 and silica content by x-ray diffraction using NIOSH Method 7500 [NIOSH 2017].

We collected 10 full-shift personal air samples on welders for manganese and iron oxide, the primary metal and metal oxide contaminants expected in the fume on the basis of the safety data sheet for the wire used during MIG welding. Sampling was done on 2 days using NIOSH Method 7303 [NIOSH 2017] during the following welding operations:

1. Resistance welding
2. MIG tank seam welding using a Carando welding machine
3. Tank base welding using a MIG welding machine
4. Flue welding using an automated MIG welding machine

We collected full-shift personal air samples for MDI on nine employees performing spray foam applications using the sampling and analytical method described in NIOSH Method 5525 [NIOSH 2017]. We also collected bulk samples of the chemicals used to create the MDI foam to help identify the monomer and oligomer fractions.

Medical Assessment

During our first site visit, from a personnel roster, we selected and interviewed a subset of serially selected employees working in the following departments: research and development,

tank fabrication and finishing, and maintenance.

On the second site visit, we administered a questionnaire to all current or former foam booth employees who were present during the evaluation, and to maintenance personnel who regularly serviced the foam booths and were present during the evaluation. The questionnaire asked about their job duties, medical history, and current respiratory symptoms. The respiratory questions, including validated questions on asthma symptoms from the European Community Respiratory Health Survey [Grassi et al. 2003], were as follows:

1. Have you been woken up with a feeling of tightness in your chest at any time in the last 12 months?
2. Have you had an attack of asthma in the last 12 months?
3. Are you currently taking any medicine (including inhalers or pumps, aerosols, or tablets) for breathing problems or asthma?
4. Have you had wheezing or whistling in your chest at any time in the last 12 months?
 - a. Have you been at all breathless when the wheezing or whistling noise was present?
 - b. Have you had this wheezing or whistling when you did not have a cold?

A positive response on any of these questions has a sensitivity of 75% and a specificity of 80% for asthma symptoms on the basis of a clinical examination with IgE testing against common allergens, spirometry, and methacholine challenge testing. We modified these questions by adding “or since beginning your current position if in that position less than 12 months,” because some participants had not been in their current position for 12 months. If participants responded positively to any of these questions, they were classified as having asthma symptoms. In addition, we added questions about changes in symptoms or medication use on days off work or on vacation. If participants responded that symptoms improved on days off work or on vacation, or that medication use or asthma attacks were less frequent on days off or on vacation, then their symptoms were classified as work related.

We took blood samples from employees who completed the questionnaire and consented to testing of their blood for the antibodies IgG and IgE to MDI. Blood samples were analyzed at the Wisnewski-Redlich laboratory at Yale University. The presence of these MDI specific antibodies demonstrates evidence of recent (months) exposure (IgG) or sensitization to MDI (IgE). We followed universal (standard) precautions for working with blood and blood products [Siegel et al. 2007; 29 CFR 1910.1030]. We individually notified participants in writing of their blood test results and what these results meant.

Results and Discussion

Air Sampling

Results of the air samples collected for respirable dust containing silica are shown in Table 1. We compared the air sample results to the permissible exposure limit (PEL) set by the

Occupational Safety and Health Administration (OSHA). This limit is calculated based on the percent of crystalline silica measured in each respirable dust air sample (Appendix A). We also compared the results to other occupational exposure limits (OELs) such as those published by NIOSH and the American Conference of Governmental Industrial Hygienists (ACGIH). The NIOSH and ACGIH OELs are compared directly to the crystalline silica concentration in the sample. The values in bold indicate exposures above any OEL.

Table 1. Full-shift personal air sample results for respirable dust containing crystalline silica

Job category	Sampling time (minutes)	Percent crystalline silica in sample	Respirable dust concentration ($\mu\text{g}/\text{m}^3$)	OSHA PEL ($\mu\text{g}/\text{m}^3$)	Crystalline silica concentration ($\mu\text{g}/\text{m}^3$)
Mill room operator (day 1)	432	36	245	263	89
Mill room operator (day 2)	403	30	346	313	104
Flue sprayer (day 1)	335	15	349	588	52
Flue sprayer (day 2)	389	13	590	667	76
Tank sprayer (day 1)	215*	9	462	909	41
Tank sprayer (day 2)	467	11	338	769	39
Brusher (residential) (day 1)	401	11	306	769	34
Brusher (residential) (day 2)	425	17	63	526	11
Brusher (commercial) (day 1)	429	15	190	588	28
Brusher (commercial) (day 2)	556	21	243	435	51
NIOSH REL					50
ACGIH TLV					25

$\mu\text{g}/\text{m}^3$ = Micrograms per cubic meter of air

REL = Recommended exposure limit

TLV = Threshold limit value

*This employee worked for half of a shift and then changed to a non-tank spraying job.

Note: Values in bold indicate the concentration exceeds the OEL

The mill room operator's level of respirable dust containing crystalline silica exceeded the OSHA PEL on the second day of our visit. In addition, 50% of the samples collected across the five job categories exceeded the NIOSH REL for crystalline silica of $50 \mu\text{g}/\text{m}^3$, and 90% of these samples exceeded the ACGIH TLV of $25 \mu\text{g}/\text{m}^3$ (Table 1). All of the crystalline silica was present as quartz; no cristobalite or tridymite was found on any air sample. To improve worker health and safety, and reduce the adverse health effects of silica exposure, OSHA instituted an updated silica PEL on June 23, 2016. The silica PEL ($50 \mu\text{g}/\text{m}^3$ as an 8-hour time-weighted average [TWA]), is the same as the NIOSH REL, and is scheduled to be enforced beginning 2 years after the effective date of June 23, 2018. Therefore, if the silica exposure data collected during this evaluation was compared to the new OSHA PEL, half of the exposures measured would exceed the PEL. A detailed discussion of the health effects from silica exposure is in Appendix A.

We observed some release of silica powder when the operator dumped large sacks of the powder into the ventilated hopper during mixing tank loading. Some bag labels had adhered to the exhaust grates attached to the hopper and blocked the airflow into the exhaust. This interference resulted in reduced effectiveness of the ventilation control and contributed to the migration of the powder to other areas of the mill room. We also saw surfaces outside of the mill room contaminated with silica powder or silica-based overspray, potentially resulting in others in the plant being exposed unnecessarily. We noticed that some ventilated enclosures at the automatic tank spraying operation did not effectively capture the silica-based enamel spray mist, allowing the mist to escape the enclosure and migrate out to the general plant. Better spray mist control could be achieved by installing vinyl strip curtains on the open sides of the enclosure, increasing the exhaust airflow, or a combination of the two.

Manganese concentrations on welders ranged from 5.5 µg/m³ to 88 µg/m³, while iron oxide concentrations (as iron) ranged from 19 µg/m³ to 240 µg/m³ (Table 2). All sample results were below any applicable OEL. We saw some automatic welding machines emitting uncontrolled fume during welding. Upon closer examination, we noticed the following: one welding machine had an inoperable exhaust fan, another had inadequate exhaust airflow, and another had the exhaust inlet positioned too far from the source of the fume. A detailed discussion of the health effects from welding exposure is in Appendix A.

Table 2. Full-shift personal air sample results for manganese and iron oxide

Welding type	Sampling time (minutes)	Manganese concentration (µg/m ³)	Iron oxide concentration (µg/m ³)
Resistance (day 1)	425	6.6	44
Resistance (day 2)	431	5.6	65
Carando (day 1)	433	88	45
Carando (day 1)	426	7.6	33
Carando (day 2)	427	13	42
Base (day 1)	430	8.6	48
Base (day 1)	419	5.5	19
Base (day 2)	418	30	100
Base (day 2)	421	9.7	39
Auto flue (day 2)	227*	13	240
OSHA PEL		5,000†	10,000
NIOSH REL		1,000	5,000
ACGIH TLV		100‡	5,000

*This employee worked for half a shift and then changed to a non-welding job.

†A ceiling limit that should not be exceeded at any time during a shift.

‡A limit based on the inhalable fraction of manganese particulate.

Each of the nine full-shift air samples on employees spraying MDI insulating foam inside a ventilated booth was analyzed for the MDI monomer and oligomer components. Both monomer and oligomer are sensitizers, therefore, if we analyze for only one form, we could potentially miss relevant exposure. The monomer air concentrations ranged from not detected (minimum detectable concentration was 0.037 $\mu\text{g}/\text{m}^3$) to 0.79 $\mu\text{g}/\text{m}^3$; no air samples measured oligomer concentrations above the minimum detectable concentration of 0.037 $\mu\text{g}/\text{m}^3$. No MDI results exceeded the OELs (Table 3). However, we did notice some employees cut slits in the back of their Tyvek suit to help with cooling. This practice reduces the effectiveness of the suit and could lead to MDI skin exposure. In addition, at times the conveyor line abruptly stopped and started and resulted in the MDI spray nozzle being pulled out of the water heater and releasing MDI foam into the booth. Such an event can also lead to skin or respiratory exposure. A detailed discussion of the health effects from isocyanate exposure is available in Appendix A.

Table 3. Full-shift personal air sample results for MDI monomer and oligomer

	Sampling time (minutes)	Monomer concentration ($\mu\text{g}/\text{m}^3$)	Oligomer concentration ($\mu\text{g}/\text{m}^3$)
Day 1	591	ND*	ND
	541	0.48	ND
	529	0.51	ND
	496	0.79	ND
	585	0.24	ND
Day 2	593	0.73	ND
	592	0.41	ND
	429	0.64	ND
	461	ND	ND
OSHA PEL		200†	None
NIOSH REL		50‡	None
ACGIH TLV		50§	None

*Not detected, the concentration was below the minimum detectable concentration of 0.037 $\mu\text{g}/\text{m}^3$ (monomer and oligomer).

†This PEL is a ceiling limit that should not be exceeded at any time during a work shift.

‡An OEL for a TWA up to 10 hours and a ceiling value of 200 $\mu\text{g}/\text{m}^3$ not to be exceeded during a 10-minute period during a shift.

§An 8-hour TWA

Medical Assessment

During our first visit, we held confidential interviews with 91 out of over 250 employees from research and development, tank fabrication and finishing, and maintenance. Seventy-six percent (69/91) of employees reported no symptoms related to work. Twenty-two employees reported symptoms they related to work. These included cough (five); shortness of breath (four); sore throat (three); wheezing, bloody nose, headache, runny or stuffy nose, and sinus problems (two each); and eye irritation, post-nasal drip, and sneezing (one

each). Five employees reported a diagnosis of asthma or other allergic breathing problems since beginning work at the plant. Two of the employees who reported asthma had onset of symptoms when working in the foam booths. We requested medical records from employees who had symptoms or medical conditions that we determined to be consistent with workplace exposures and who had seen a physician. Records were reviewed for all five employees who reported asthma. A diagnosis of asthma or prescription for an inhaler was found in all records; however, a relationship to work was only documented in one record, that of an employee who was working with MDI foam.

During our second visit, 29 employees were asked to complete the questionnaire; 29 did the questionnaire and 28 had their blood drawn. We were unable to obtain a blood sample from one employee. Four of these employees were maintenance employees, and the rest were either current (19) or former (6) MDI foam booth employees. Eight of the current foam booth employees were full-time in the booths, while the other 10 were back-up in the booth. Six of these employees answered yes to at least one question indicative of asthma, and all six reported that symptoms improved on days off work or on vacation, or that medication use or asthma attacks were less frequent on days off or on vacation; therefore, we classified their symptoms as work related. One of these six was a maintenance employee; the rest were either current or former MDI foam booth employees. Three reported they had not been diagnosed with asthma by a healthcare provider, although one had been prescribed an inhaler. The other three had been diagnosed with asthma, one as a child. One additional employee answered yes to at least one question indicative of asthma, but the symptoms were not work related according to our definition.

MDI-specific IgG and IgE test results are listed in Table 4. Two employees had both positive MDI-specific IgE tests and IgG tests. One had symptoms of work-related asthma based upon the questionnaire; the other did not. Three other employees had positive MDI-specific IgG tests. One of these three employees had been removed from work in the foam spraying booth several months prior to the blood test because of symptoms of work-related asthma. One employee had an uninterpretable test, and the rest had normal IgG tests.

Table 4. MDI-specific IgG and IgE results by job category

Test result	Job category (number of participants)			
	Primary foam booth (8)*	Back-up foam booth (10)†	Former foam booth (6)	Maintenance (4)
Positive IgG and IgE	1	0	1	0
Positive IgE and normal IgG	0	0	0	0
Positive IgG and normal IgE	1	1	1	0
Normal IgG and IgE	5	8	4	4

*One employee had a slightly positive IgE due to elevated total IgE; employee is not included in the table.

†One employee had an uninterpretable IgG; employee is not included in the table.

A positive MDI-specific IgE test means a person is “sensitized” to MDI and is strongly suggestive of isocyanate asthma. Being sensitized can make an individual more at risk for an allergic reaction when exposed to MDI. Allergic reactions include stuffy nose and congestion, shortness of breath, wheezing, cough, and asthma. Employees with a positive MDI-specific IgE test need to be evaluated by an occupational medicine physician or other physician who is familiar with the health effects of isocyanate exposure. However, not everyone who develops allergic problems from exposure to MDI has IgE antibodies in their blood. According to the laboratory who analyzed the blood samples for IgE, it is estimated that up to 50% of people with isocyanate asthma have normal isocyanate-specific IgE tests. A positive MDI-specific IgG test means a person has recently been exposed to MDI, but does not mean they have isocyanate-induced asthma. Not all people who are exposed to MDI develop IgG antibodies, so not having a positive test does not mean a person was never exposed to MDI. This may explain the lack of MDI-specific IgG among some employees. It may also reflect an absence of exposure in those employees, or the passage of too much time between exposure and testing.

The findings from this evaluation indicate that employees who work in the foam booths can be exposed to MDI despite the use of engineering controls and personal protective equipment, and that some of these employees have developed asthma as a result of this exposure.

We measured overexposure to silica and historical silica exposure measurements made by industrial hygiene consultants at this plant have documented repeated overexposures at the current PEL. Silica exposure is known to cause disabling lung conditions such as silicosis, as well as lung cancer. Silica exposure is also associated with autoimmune diseases, kidney disease, and an increased risk of tuberculosis. Despite measuring overexposures to silica, relatively few employees reported respiratory symptoms during the interviews. It can take years for effects from silica in the lungs to become evident, as well as for silicosis and lung cancer to develop. The fact that we did not find symptoms or evidence of health effects does not mean that exposure to silica does not cause harm. What it means is that it is too early in the process to measure the health effects. In June 2016, OSHA promulgated a new PEL for silica (50 µg/m³ as an 8-hour TWA) and in the rulemaking comments noted that even at the new PEL, cases of silicosis and cancer will occur. That is why we believe it is important to protect employees from silica exposure.

Observations and Document Reviews

We noticed a lack of maintenance of the engineering controls (inoperable fans, inadequate airflow, and exhaust inlet too far from source) as well as poor air contaminant containment (ventilated enclosures did not fully capture silica overspray) throughout the plant. This observation was also confirmed by reports from some employees that ventilation systems were inoperable or did not fully capture air contaminants such as silica or welding fume. Some employees were unsure how to report the need for repairs of equipment. No formal reporting system was in place that would allow the tracking of maintenance requests.

The plant’s written respiratory protection program was created by a third party respirator program administrator and the program is managed by the safety manager. The document

addresses all OSHA required elements such as program administration, medical clearance, fit testing, hazard evaluations, equipment selection criteria, training, cleaning, and maintenance. However, some sections in the written program needed additional detail, specifically those sections dealing with the frequency of training, medical clearance evaluation(s), and respirator fit testing. The document also did not provide guidance on cartridge change-out schedules.

Conclusions

A health hazard existed at this plant from exposure to crystalline silica and MDI. Employees reported symptoms consistent with work-related asthma, and two employees had MDI-specific IgE, consistent with MDI asthma, in their blood. Five employees had MDI-specific IgG in their blood, indicating recent exposure to MDI despite the use of engineering controls and personal protective equipment. Personal exposure to manganese and iron (welding fumes) did not exceed any of the applicable OELs.

Recommendations

On the basis of our findings, we recommend the actions listed below. We encourage this water heater manufacturing plant to use a labor-management health and safety committee or working group to discuss our recommendations and develop an action plan. Those involved in the work can best set priorities and assess the feasibility of our recommendations for the specific situation at this plant.

Our recommendations are based on an approach known as the hierarchy of controls (Appendix A). This approach groups actions by their likely effectiveness in reducing or removing hazards. In most cases, the preferred approach is to eliminate hazardous materials or processes and install engineering controls to reduce exposure or shield employees. Until such controls are in place, or if they are not effective or feasible, administrative measures and personal protective equipment may be needed.

Engineering Controls

Engineering controls reduce employees' exposures by removing the hazard from the process or by placing a barrier between the hazard and the employee. Engineering controls protect employees effectively without placing primary responsibility of implementation on the employee.

1. Develop a more effective system for transferring bulk silica to the mixing tank in the mill room. The system should prevent the release of silica powder when the transfer occurs. One possible system to consider using would involve a vacuum source to move powder from the bulk container to the mixing tank. Such a system is commonly used in the pharmaceutical industry.
2. Improve capture of overspray in the tank and flue spraying areas to prevent contaminating adjacent areas. Installing vinyl curtains on the openings of the ventilated enclosures is one possible solution.

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3. Ensure all local exhaust ventilation systems installed on the tank fabrication department welding machines are operating and providing effective capture of welding fume.
 4. Investigate the feasibility of introducing conditioned air into the foam shooting booths during hot weather to address the thermal comfort problem encountered by employees wearing Tyvek suits.

Administrative Controls

The term administrative controls refers to employer-dictated work practices and policies to reduce or prevent hazardous exposures. Their effectiveness depends on employer commitment and employee acceptance. Regular monitoring and reinforcement are necessary to ensure that policies and procedures are followed consistently.

1. Encourage employees to report work-related skin and respiratory symptoms and the results of the blood tests we performed to the safety manager, their supervisor, and the contract healthcare provider. Employees who report work-related symptoms should be evaluated by a physician experienced in occupational medicine or allergy, whether or not they have a positive test for MDI-specific IgE or IgG.
2. Start a medical surveillance program for employees who are exposed to MDI and crystalline silica. Work with an occupational medicine physician to design and implement this program. These physicians can be located through a variety of sources, including the Association of Occupational and Environmental Clinics at <http://www.aoec.org/> and the American College of Occupational and Environmental Medicine at <http://www.acoem.org/>. One program near the plant is at the Michigan State University Department of Medicine.

Our recommendations for medical surveillance regarding silica-exposed employees are listed in Appendix A. Some general recommendations for medical monitoring for asthma are as follows:

- a. Provide preplacement, annual, and exit general medical examinations with the following:
 - i. Special emphasis on the respiratory system
 - ii. Medical history including an extensive work history, history of pre-existing respiratory conditions such as asthma, and a smoking history
 - iii. Spirometry—Information for employers and employees can be found on the Spirometry Information sheet at <http://www.osha.gov/Publications/osh3415.html> and Spirometry Worker Information sheet at <http://www.osha.gov/Publications/osh3418.html>.
 - b. Inform employees with a history of respiratory conditions of the potential for increased health risks associated with exposure to isocyanates.
3. Remove any employee with isocyanate-induced asthma or rhinitis from the work environment where isocyanate exposure occurs and evaluate those jobs to determine

what you can do to lower exposures to prevent other employees from becoming sensitized. The only effective intervention for employees with isocyanate-induced asthma or rhinitis is cessation of all isocyanate exposure. Place them in a job without MDI exposure while maintaining their earnings, seniority, and other rights and benefits. Explain to employees the workplace policies, workers compensation, pay, and benefits that are available to them.

4. Update the written respiratory protection program to include more details specific to each job that requires the use of a respirator. Emphasize proper respirator use, training, cleaning, maintenance, and storage. Ensure medical clearance and respirator fit testing are conducted on a time-schedule as outlined in the OSHA respiratory protection program guidelines.
5. Check employees periodically for proper respirator wear.
6. Develop a respirator cartridge changeout schedule and a system to track employee compliance.
7. Train employees on the hazards associated with silica and diisocyanates.
8. Improve housekeeping (e.g., the use of wet methods to clean surfaces) to reduce bystander exposure to employees in departments where silica is present.
9. Create an equipment maintenance request and tracking system for employees to use when equipment is not performing correctly or needs repair.

Personal Protective Equipment

Personal protective equipment is the least effective means for controlling hazardous exposures. Proper use of personal protective equipment requires a comprehensive program and a high level of employee involvement and commitment. The right personal protective equipment must be chosen for each hazard. Supporting programs such as training, change-out schedules, and medical assessment may be needed. Personal protective equipment should not be the sole method for controlling hazardous exposures. Rather, personal protective equipment should be used until effective engineering and administrative controls are in place.

1. Train employees on the danger of cutting slits in their Tyvek suits. Sensitization can occur from skin exposure.
2. Develop, track, and enforce a respirator cartridge changeout schedule for all employees required to wear a respirator.

Appendix A: Occupational Exposure Limits and Health Effects

NIOSH investigators refer to mandatory (legally enforceable) and recommended OELs for chemical, physical, and biological agents when evaluating workplace hazards. OELs have been developed by federal agencies and safety and health organizations to prevent adverse health effects from workplace exposures. Generally, OELs suggest levels of exposure that most employees may be exposed to for up to 10 hours per day, 40 hours per week, for a working lifetime, without experiencing adverse health effects. However, not all employees will be protected if their exposures are maintained below these levels. Some may have adverse health effects because of individual susceptibility, a pre-existing medical condition, or a hypersensitivity (allergy). In addition, some hazardous substances act in combination with other exposures, with the general environment, or with medications or personal habits of the employee to produce adverse health effects. Most OELs address airborne exposures, but some substances can be absorbed directly through the skin and mucous membranes.

Most OELs are expressed as a TWA exposure. A TWA refers to the average exposure during a normal 8- to 10-hour workday. Some chemical substances and physical agents have recommended short-term exposure limit or ceiling values. Unless otherwise noted, the short-term exposure limit is a 15-minute TWA exposure. It should not be exceeded at any time during a workday. The ceiling limit should not be exceeded at any time.

In the United States, OELs have been established by federal agencies, professional organizations, state and local governments, and other entities. Some OELs are legally enforceable limits; others are recommendations.

- The U.S. Department of Labor OSHA PELs [29 CFR 1910 (general industry); 29 CFR 1926 (construction industry); and 29 CFR 1917 (maritime industry)] are legal limits. These limits are enforceable in workplaces covered under the Occupational Safety and Health Act of 1970.
- NIOSH RELs are recommendations based on a critical review of the scientific and technical information and the adequacy of methods to identify and control the hazard. NIOSH RELs are published in the *NIOSH Pocket Guide to Chemical Hazards* [NIOSH 2010]. NIOSH also recommends risk management practices (e.g., engineering controls, safe work practices, employee education/training, personal protective equipment, and exposure and medical monitoring) to minimize the risk of exposure and adverse health effects.
- Another set of OELs commonly used and cited in the United States is the ACGIH TLVs. The TLVs are developed by committee members of this professional organization from a review of the published, peer-reviewed literature. TLVs are not consensus standards. They are considered voluntary exposure guidelines for use by industrial hygienists and others trained in this discipline “to assist in the control of health hazards” [ACGIH 2017].

Outside the United States, OELs have been established by various agencies and organizations and include legal and recommended limits. The Institut für Arbeitsschutz der Deutschen

Gesetzlichen Unfallversicherung (Institute for Occupational Safety and Health of the German Social Accident Insurance) maintains a database of international OELs from European Union member states, Canada (Québec), Japan, Switzerland, and the United States. The database, available at <http://www.dguv.de/ifa/GESTIS/GESTIS-Internationale-Grenzwerte-für-chemische-Substanzen-limit-values-for-chemical-agents/index-2.jsp>, contains international limits for more than 2,000 hazardous substances and is updated periodically.

OSHA requires an employer to furnish employees a place of employment free from recognized hazards that cause or are likely to cause death or serious physical harm [Occupational Safety and Health Act of 1970 (Public Law 91–596, sec. 5(a)(1))]. This is true in the absence of a specific OEL. It also is important to keep in mind that OELs may not reflect current health-based information.

When multiple OELs exist for a substance or agent, NIOSH investigators generally encourage employers to use the lowest OEL when making risk assessment and risk management decisions. NIOSH investigators also encourage use of the hierarchy of controls approach to eliminate or minimize workplace hazards. This includes, in order of preference, the use of (1) substitution or elimination of the hazardous agent, (2) engineering controls (e.g., local exhaust ventilation, process enclosure, dilution ventilation), (3) administrative controls (e.g., limiting time of exposure, employee training, work practice changes, medical surveillance), and (4) personal protective equipment (e.g., respiratory protection, gloves, eye protection, hearing protection). Control banding, a qualitative risk assessment and risk management tool, is a complementary approach to protecting employee health. Control banding focuses on how broad categories of risk should be managed. Information on control banding is available at <http://www.cdc.gov/niosh/topics/ctrlbanding/>. This approach can be applied in situations where OELs have not been established or can be used to supplement existing OELs.

Respirable Crystalline Silica

Silica, or silicon dioxide, occurs in a crystalline or noncrystalline (amorphous) form. In crystalline silica, the silicon dioxide molecules are oriented in a fixed pattern versus the random arrangement of the amorphous form. The more common crystalline forms in workplace environments are quartz and cristobalite, and to a lesser extent, tridymite. Occupational exposures to respirable crystalline silica have been associated with silicosis, lung cancer, pulmonary tuberculosis, and airway diseases. Several serious nonrespiratory diseases are associated with occupational exposure to crystalline silica. These include immunologic disorders and autoimmune diseases (including systemic sclerosis, rheumatoid arthritis, and systemic lupus erythematosus) and renal diseases.

Silicosis is a fibrotic disease of the lung caused by the deposition of fine crystalline silica particles in the lungs. It is the disease most often associated with exposure to respirable crystalline silica. This lung disease is caused by the inhalation and deposition of crystalline silica particles that are 10 micrometers or less in diameter. Particles 10 micrometers and below are considered respirable particles and classified as having the potential to reach the lower portions of the human lung (alveolar region). Although particle sizes 10 micrometers

and below are considered respirable, some of these particles can be deposited before they reach the alveolar region [Hinds 2012]. Symptoms of silicosis usually develop insidiously, with cough, shortness of breath, chest pain, weakness, wheezing, and nonspecific chest illnesses. Silicosis usually occurs after years of exposure (chronic), but may appear in a shorter period of time (acute) if exposure concentrations are very high. Acute silicosis is typically associated with a history of high exposures from tasks that produce small particles of airborne dust with a high silica content [NIOSH 2002]. Chronic silicosis can develop or progress even if exposure to silica ends [NIOSH 2002].

The International Agency for Research on Cancer [IARC 2012] and NIOSH [NIOSH 2002] have classified inhaled crystalline silica in the form of quartz or cristobalite as carcinogenic to humans in reference to lung cancer. While individuals with silicosis clearly are at risk of lung cancer, exposure to silica in the absence of silicosis also increases the risk for lung cancer [Liu et al. 2013].

Several forms of nonmalignant respiratory disease are associated with exposure to silica [NIOSH 2002]. These include chronic obstructive pulmonary disease (emphysema and chronic bronchitis) and asthma. Silica exposure is also related to other abnormalities noted on pulmonary function tests.

Exposure to silica increases the risk of developing tuberculosis even in the absence of silicosis [NIOSH 2002]. This increase is due to impaired macrophage function from silica. This risk for individuals with silicosis is even higher. The odds of an individual with silicosis dying with tuberculosis are 19 to 40 times higher than for individuals without silicosis [Calvert et al. 2003; Chen et al. 2007].

Exposure to crystalline silica is also associated with development of several autoimmune diseases [Cooper et al. 2002; Lee et al. 2014]. The strongest evidence exists for an association with systemic sclerosis, rheumatoid arthritis, systemic lupus erythematosus, and antineutrophil cytoplasmic autoantibody related vasculitis [Cooper et al. 2002; Lee et al. 2014].

Silica exposure is related to an increased risk of end-stage kidney disease [Ghahramani 2010; NIOSH 2002]. Kidney disease is associated with the effect of silica deposited in the kidneys and with an autoimmune process with activated macrophages. A wide range of kidney pathology is associated with silica exposure.

When proper practices are not followed or controls are not maintained, respirable crystalline silica exposures can exceed the OSHA PEL, NIOSH REL, or the ACGIH TLV. For general industry, the OSHA PEL for respirable dust containing 1% or more of quartz is calculated by dividing 10 milligrams per cubic meter (mg/m^3) by the percent quartz in the sample, plus two [OSHA 2017]. OSHA instituted an updated silica PEL on June 23, 2016. The updated silica PEL ($50 \mu\text{g}/\text{m}^3$ as an 8-hour TWA), is the same as the NIOSH REL (which is applied as a TWA up to 10 hours), and is scheduled to be enforced for general industry and the maritime industry beginning 2 years after the effective date (June 23, 2018). The NIOSH REL is intended to reduce the risk of developing silicosis, lung cancer, and other adverse health effects [NIOSH 2010]. The ACGIH TLV for quartz is $25 \mu\text{g}/\text{m}^3$, as an 8-hour TWA [ACGIH 2017].

We recommend medical surveillance for any employee who is exposed above the action level of 25 µg/m³ for 30 or more days per year. Our recommendations are identical to the medical surveillance requirements mandated by OSHA. This includes an initial examination within 30 days of initial assignment to the job. This examination must include the following:

- A medical and work history with emphasis on past, present, and anticipated exposure to respirable crystalline silica, dust, and other agents affecting the respiratory system; any history of respiratory system dysfunction, including signs and symptoms of respiratory disease (e.g., shortness of breath, cough, wheezing); history of tuberculosis; and smoking status and history
- A physical examination with special emphasis on the respiratory system
- A chest x-ray interpreted and classified according to the International Labour Office International Classification of Radiographs of Pneumoconioses by a NIOSH-certified B Reader
- A pulmonary function test to include forced vital capacity and forced expiratory volume in one second administered by a spirometry technician with a current certificate from a NIOSH-approved spirometry course
- Testing for latent tuberculosis infection
- Any other tests deemed appropriate by the physician or licensed healthcare provider. Periodic examinations including the same elements must be offered at least once every 3 years or more often if recommended by the physician or licensed healthcare provider.

Isocyanates

Diisocyanates and polyisocyanates (isocyanates) are a group of highly reactive, low-molecular-weight aromatic and aliphatic compounds [Lockey et al. 2015]. The most common isocyanates include the aliphatic compounds hexamethylene diisocyanate and isophorone diisocyanate, and the aromatic compounds toluene diisocyanate and MDI. Isocyanates are widely used in the production of polyurethane materials such as foams, adhesives, resins, elastomers, binders, and coatings.

Exposure to isocyanates can be irritating to the skin, mucous membranes, eyes, and respiratory tract [Lockey et al. 2015; NIOSH 1978, 2006]. The most frequent respiratory effect associated with isocyanate exposure is asthma due to sensitization [Lockey et al. 2015; Markowitz 2005]. Sensitization can occur from inhalation and from skin exposure [Arrandale et al. 2012; Heederick et al. 2012; Lummus et al. 2011; Redlich 2010; Wisnewski 2007]. Skin exposure might even be more effective at causing sensitization than inhalation [Heederick et al. 2012; Redlich 2010]. Less common health effects of isocyanate exposure include contact dermatitis, rhinitis, and hypersensitivity pneumonitis [Lockey et al. 2015].

Isocyanates are the most common cause of occupational asthma in many industrialized countries [Tarlo and Lemiere 2014]. The level of exposure influences sensitization rates, with lower levels of exposure leading to lower asthma rates [Heederick et al. 2012]. An employee with isocyanate-induced asthma exhibits the traditional symptoms of acute airway

obstruction such as coughing, wheezing, shortness of breath, tightness in the chest, and nocturnal awakening [NIOSH 1978, 1986]. Isocyanate-induced asthma occurs with variable latency following the initial exposure, although characteristically the asthma develops within 2 years of exposure [Markowitz 2005]. The asthmatic reaction may occur minutes after exposure (immediate phase), several hours after exposure (late phase), or both (dual phase) [Lumms et al. 2011]. After sensitization, any exposure, even to levels below OELs or below the level of detection, can produce an asthmatic response that may be life threatening [NIOSH 1978, 1996, 2006; Redlich 2010]. The only effective intervention for employees with isocyanate-induced asthma is cessation of all isocyanate exposure. This intervention can be accomplished by removing the employee from the work environment where isocyanate exposure occurs.

Isocyanate asthma is clinically indistinguishable from common allergic asthma [Wisnewski 2007; Wisnewski and Jones 2010]. Common allergic asthma is mediated by allergen-specific IgE and isocyanate-specific IgE is found in up to 50% of people with isocyanate asthma [Wisnewski 2007]. While isocyanate specific-IgE is not always found in people with isocyanate asthma, its detection is strongly predictive of asthma [Budnick et al. 2013; Wisnewski 2007]. IgE, which is a marker of sensitization, has a very short half-life of about 2 days so that it may disappear after short periods away from work [Wisnewski 2007]. Immunoglobulin G (IgG), which is a marker of exposure, has a half-life of about 30 days [Wisnewski 2007]. Isocyanate-albumin conjugate specific IgG is rarely observed in people without exposure to isocyanates [Wisnewski 2007], but is prevalent among exposed workers [Wisnewski et al. 2012].

The OSHA PEL for MDI monomer is a ceiling limit of 200 $\mu\text{g}/\text{m}^3$ that should not be exceeded at any time during a work shift. NIOSH has an REL of 50 $\mu\text{g}/\text{m}^3$ that is a TWA for up to 10 hours, while ACGIH has a TLV of 50 $\mu\text{g}/\text{m}^3$ as an 8-hour TWA. None of the three organizations (NIOSH, ACGIH, OSHA) publishes an OEL for MDI oligomer.

Welding Fumes

The effect of welding fumes on an individual's health can vary depending on the length and intensity of the exposure and the specific metals involved. Of particular concern are welding processes involving stainless steel, cadmium or lead-coated steel and metals such as manganese, iron, nickel, chrome, zinc, and copper. Fumes from these metals are considerably more toxic than those encountered when welding iron or mild steel. Epidemiologic studies and case reports of employees exposed to welding emissions have shown an excessive incidence of acute and chronic respiratory diseases [NIOSH 1988]. These illnesses include metal fume fever, pneumonitis, pulmonary edema, and lung cancer. Exposure to manganese has been associated with Parkinson-like health effects such as poor hand-eye coordination, motor slowing, increased tremor, reduced response speed, mood disturbance, and possible memory and intellectual loss [Antonini et al. 2006; Bowler et al. 2006; Lundin et al. 2014; Racette et al. 2012; Welch et al. 2004].

The content of welding fumes depends on the base metal being welded, the welding process and parameters such as voltage and amperage, the composition of the consumable welding

electrode or wire, the shielding gas, and any surface coatings or contaminants on the base metal. The flux coating (or core) of the electrode/wire may contain up to 30 organic and inorganic compounds. In general, welding fume constituents may include minerals, such as silica and fluorides, and metals, such as arsenic, beryllium, cadmium, chromium, cobalt, nickel, copper, iron, lead, magnesium, manganese, molybdenum, tin, vanadium, and zinc [NIOSH 1988; Welding Institute 1976]. OSHA has not established a PEL for total welding fumes; however, PELs have been set for individual welding fume constituents (e.g., iron, manganese) [29 CFR 1910.1000]. NIOSH has concluded that it is not possible to establish an exposure limit for total welding emissions because the composition of welding fumes and gases varies greatly, and the welding constituents may interact to produce adverse health effects. Therefore, NIOSH recommends controlling total welding fume to the lowest feasible concentration and meeting the exposure limit for each welding fume constituent [NIOSH 2010]. In addition to welding fume, many other potential health hazards exist for welders. Welding operations can produce gaseous emissions such as carbon monoxide, ozone, nitrogen dioxide, and phosgene (formed from chlorinated solvent decomposition) [NIOSH 1988; Welding Institute 1976]. Welders can also be exposed to hazardous levels of ultraviolet radiation from the welding arc if welding curtains or other precautions are not used.

References

ACGIH [2017]. 2017 TLVs® and BEIs®: threshold limit values for chemical substances and physical agents and biological exposure indices. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.

Antonini JM, Santamaria AB, Jenkins NT, Albini E, Lucchini R [2006]. Fate of manganese associated with the inhalation of welding fumes: potential neurological effects. *Neurotoxicology* 27(3):304–310, <http://doi.org/10.1016/j.neuro.2005.09.001>.

Arrandale VH, Kudla I, Kraut AG, Scott JA, Tarlo SM, Redlich CA, Holness DL [2012]. Skin and respiratory symptoms among workers with suspected work-related disease. *Occup Med (Lond)* 62(6):420–426, <https://dx.doi.org/10.1093/occmed/kqs133>.

Bowler RM, Gysens S, Diamond E, Nakagawa S, Drezgic M, Roels HA [2006]. Manganese exposure: neuropsychological and neurological symptoms and effects in welders. *Neurotoxicology* 27(3):315–326, <https://dx.doi.org/10.1016/j.neuro.2005.10.007>.

Budnick LT, Preisser AM, Permentier H, Baur X [2013]. Is specific IgE antibody analysis feasible for the diagnosis of methylene diphenyl diisocyanate-induced occupational asthma? *Int Arch Occup Environ Health* 86(4):417–430, <https://dx.doi.org/10.1007/s00420-012-0772-6>.

CFR. Code of Federal Regulations. Washington, DC: U.S. Government Printing Office, Office of the Federal Register.

Cooper GS, Miller FW, Germolec DR [2002]. Occupational exposures and autoimmune diseases. *Int Immunopharmacol* 2(2-3):303–313.

Ghahramani N [2010]. Silica nephropathy. *Int J Occup Environ Med* 1(3):108–115.

Grassi M, Rezzani C, Biino G, Marinoni A [2003]. Asthma-like symptoms assessment through ECRHS screening questionnaire scoring. *J Clin Epidemiol* 56(3):238–247.

Heederick D, Henneberger PK, Redlich CA [2012]. ERS Task Force on the management of work-related asthma. Primary prevention: exposure reduction, skin exposure and respiratory protection. *Eur Respir Rev* 21(124):112–124, <https://dx.doi.org/10.1183/09059180.00005111>.

Hinds WC [2012]. *Aerosol technology: properties, behavior, and measurement of airborne particles*. John Wiley & Sons, New York, New York.

IARC [2012]. *IARC monographs on the evaluation of the carcinogenic risks to humans: a review of human carcinogens: arsenic, metals, fibres, and dusts*. Vol. 100C. Lyon, France: World Health Organization, International Agency for Research on Cancer, pp. 355–397.

Liu Y, Steenland K, Rong Y, Hnizdo E, Huang X, Zhang H, Shi T, Sun Y, Wu T, Chen W [2013]. Exposure-response analysis and risk assessment for lung cancer in relationship to silica exposure: a 44-year cohort study of 34,018 workers. *Am J Epidemiol* 178(9):1424–1433, <https://dx.doi.org/10.1093/aje/kwt139>.

Lee S, Matsuzaki H, Kumagai-Takei N, Yoshitome K, Maeda M, Chen Y, Kusaka M, Urakami K, Hayashi H, Fujimoto W, Nishimura Y, Otsuki T [2014]. Silica exposure and altered regulation of autoimmunity. *Environ Health Prev Med* 19(5):322–329, <https://dx.doi.org/10.1007/s12199-014-0403-9>.

Lockey JE, Redlich CA, Streicher R, Pfahles-Hutchens A, Hakkinen PB, Ellison GL, Harber P, Utell M, Holland J, Comai A, White M. [2015]. Isocyanates and human health: multistakeholder information needs and research priorities. *J Occup Environ Med* 57(1):44–51, <https://dx.doi.org/10.1097/JOM.0000000000000278>.

Lummus ZL, Wisnewski AV, Bernstein DI [2011]. Pathogenesis and disease mechanisms of occupational asthma. *Immunol Allergy Clin North Am* 31(4):699–716, <https://dx.doi.org/10.1016/j.iac.2011.07.008>.

Lundin JI, Checkoway H, Criswell SR, Hobson AJ, Harris RC, Swisher LM, Evanoff BA, Racette BA [2014]. Screening for early detection of parkinsonism using a self-administered questionnaire: a cross-sectional epidemiologic study. *Neurotoxicology* 45:232–237, <https://dx.doi.org/10.1016/j.neuro.2013.08.010>.

Markowitz S [2005]. Chemicals in the plastics, synthetic textiles, and rubber industries. In: Rosenstock L, Cullen M, Brodtkin C, Redlich C, eds. *Textbook of clinical occupational and environmental medicine*, 2nd ed. Philadelphia, PA: Elsevier Saunders Publishers, pp. 1021–1022.

NIOSH [1978]. Criteria for a recommended standard: occupational exposure to diisocyanates. By Soucek S. Cincinnati, OH: U.S. Department of Health, Education, and Welfare, Center for Disease Control, National Institute for Occupational Safety and Health, DHEW (NIOSH) Publication No. 78-215.

NIOSH [1986]. Occupational respiratory diseases. By Merchant JA, Boehlecke BA, Taylor G, Pickett-Harner M. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 86-102.

NIOSH [1988]. Criteria for a recommended standard: welding, brazing and thermal cutting. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 88-110.

NIOSH [1996]. Preventing asthma and death from diisocyanates exposure. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 96-111.

NIOSH [2002]. Health effects of occupational exposure to respirable crystalline silica. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 2002-129.

NIOSH [2006]. NIOSH alert: preventing asthma and death from MDI exposure during spray-on truck bed liner and related applications. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for

Occupational Safety and Health, DHHS (NIOSH) Publication No. 2006-149.

NIOSH [2010]. NIOSH pocket guide to chemical hazards. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 2010-168c, <http://www.cdc.gov/niosh/npg/>.

NIOSH [2017]. NIOSH manual of analytical methods (NMAM). 5th ed. O'Connor PF, Ashley K, eds. Cincinnati, OH: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, DHHS (NIOSH) Publication No. 2014-151, <http://www.cdc.gov/niosh/nmam>.

OSHA [2017]. OSHA sampling and analytical methods. Hendricks W, ed. Salt Lake City, UT: U.S. Department of Labor, Occupational Safety and Health Administration, <http://www.osha.gov/dts/slhc/methods/index.html>.

Racette BA, Criswell SR, Lundin JI, Hobson A, Seixas N, Kotzbauer PT, Evanoff BA, Perlmutter JS, Zhang J, Sheppard L, Checkoway H [2012]. Increased risk of parkinsonism associated with welding exposure. *Neurotoxicology* 33(5):1356–1361, <https://dx.doi.org/10.1016/j.neuro.2012.08.011>.

Redlich CA [2010]. Skin exposure and asthma: is there a connection? *Proc Am Thorac Soc* 7(2):134–137, <https://dx.doi.org/10.1513/pats.201002-025RM>.

Siegel JD, Rhinehart E, Jackson M, Chiarello L [2007]. Healthcare Infection Control Practices Advisory Committee. Guideline for isolation precautions: preventing transmission of infectious agents in healthcare settings, <https://dx.doi.org/10.1016/j.ajic.2007.10.007>.

Tarlo SM, Lemiere C [2014]. Occupational asthma. *N Engl J Med* 370(7):640–649, <https://dx.doi.org/10.1056/NEJMra1301758>.

Welch LS, Rappaport MS, Susi P [2004]. Construction welding exposures to manganese likely to exceed proposed TLV. *J Occup Environ Hyg* 1(6):D63–D65, <https://dx.doi.org/10.1080/15459620490447929>.

Welding Institute [1976]. The facts about fume – a welding engineer's handbook. Abington, Cambridge, England: The Welding Institute.

Wisnewski AV [2007]. Developments in laboratory diagnostics for isocyanate asthma. *Curr Opin Allergy Clin Immunol* 7(2):138–145, <https://dx.doi.org/10.1097/ACI.0b013e3280895d22>.

Wisnewski AV, Jones M [2010]. Pro/Con debate: is occupational asthma induced by isocyanates an immunoglobulin E-mediated disease? *Clin Exp Allergy* 40(8):1155–1162, <https://dx.doi.org/10.1111/j.1365-2222.2010.03550.x>.

Wisnewski AV, Stowe MH, Nerlinger A, Opare-Addo P, Decamp D, Kleinsmith CR, Redlich CA [2012]. Biomonitoring hexamethylene diisocyanate (HDI) exposure based on serum levels of HDI-specific IgG. *Ann Occup Hyg* 56(8):901–910, <https://dx.doi.org/10.1093/annhyg/mes024>.

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