How Welding Fumes Affect the Welder

A study helps reveal how certain welding fumes affect our lungs

BY J. M. ANTONINI, G. G. KRISHNA MURTHY, R. A. ROGERS, R. ALBERT, T. W. EAGAR, G. D. ULRICH AND J. D. BRIAN

It has been estimated that worldwide there are more than one million full-time welders, with even more workers welding intermittently. For 15 years there has been increasing interest in the effects of welding fumes on the health of these workers. Medical studies in epidemiology have reported a higher incidence of respiratory illness among welders. Reported effects range from bronchitis, airway irritation, metal fume fever and chemical pneumonitis to lung function changes and lung cancer (Ref. 1). Most welders know to reduce welding fume exposure whenever possible. Nevertheless, there is relatively little specific, quantitative data revealing how welding fumes affect the lungs.

Based on results from epidemiological research, many have speculated as to the cause of these respiratory problems and whether they pose a long-term health threat to welders. Although it is well known that iron oxide, the primary constituent in steel welding fume, is nontoxic, steels contain other alloying elements that, in their pure forms (as used in other industries), have been found to have serious health consequences. Since the 1800s, it has been known that ingestion of huge quantities of nearly pure manganese oxide can cause neurological disorders. It was proven several decades ago that soluble hexavalent chromium is a carcinogen; nickel has been found, in certain circumstances, to also promote cancer. Since manganese is in all steels, and chromium and nickel are in some steels, especially the stainless steels, there has been increasing interest in determining whether these elements, as present in welding fume, pose any health threat to welders.

It is generally accepted that the primary method of ingestion of welding fumes occurs during breathing of very fine, respirable particles. A respirable particle is considered to be one which is less than 10 μm in size, as larger particles have too much inertia to follow the airflow pathway through the nostrils and respiratory tract. Larger particles become trapped by the mucous on the walls of the respiratory tract. Particles finer than 10 μm will be carried with the airflow, turning the corners of the respiratory tract. Some researchers maintain that, though particles 0.1–1.0 μm will be inhaled deep into the lungs, most will also be exhaled. Particles finer than 0.1 μm impact the walls of the finest recesses of the lungs (the alveoli) (Ref. 2).

The fume produced during arc welding is composed of 1) metal evaporated primarily from the electrode due to the intense heat of the arc, 2) hydrocarbons evaporated from the surface of the hot base metal and 3) microspatter. Generally, the only respirable components come from evaporated metal or evaporated hydrocarbons, as microspatter is generally too large to enter deep into the lungs.

Note that respirable fume, deposited in the lungs, is only part of the total fume — a distinction often not recognized. Total fume consists of respirable particles (some of which are ultrafine and may subsequently be exhaled), larger particles, which would be trapped in the mucous membranes, and spatter. Since respirable fume forms by a different mechanism than other fume, its chemical composition may vary from the average composition of the total fume.

J. M. ANTONINI is with the Center for Disease Control and Prevention for Occupational Safety and Health, Health Effects Laboratory Division, Morgantown, W.Va. G. G. KRISHNA MURTHY, R. A. ROGERS and J. D. BRIAN are with the Physiology Program of the Department of Environmental Health, Harvard University School of Public Health, Boston, Mass. R. ALBERT and G. D. ULRICH are with the Department of Chemical Engineering, University of New Hampshire, Durham, N.H. T. W. EAGAR is with the Department of Materials Science and Engineering, Massachusetts Institute of Technology, Cambridge, Mass.

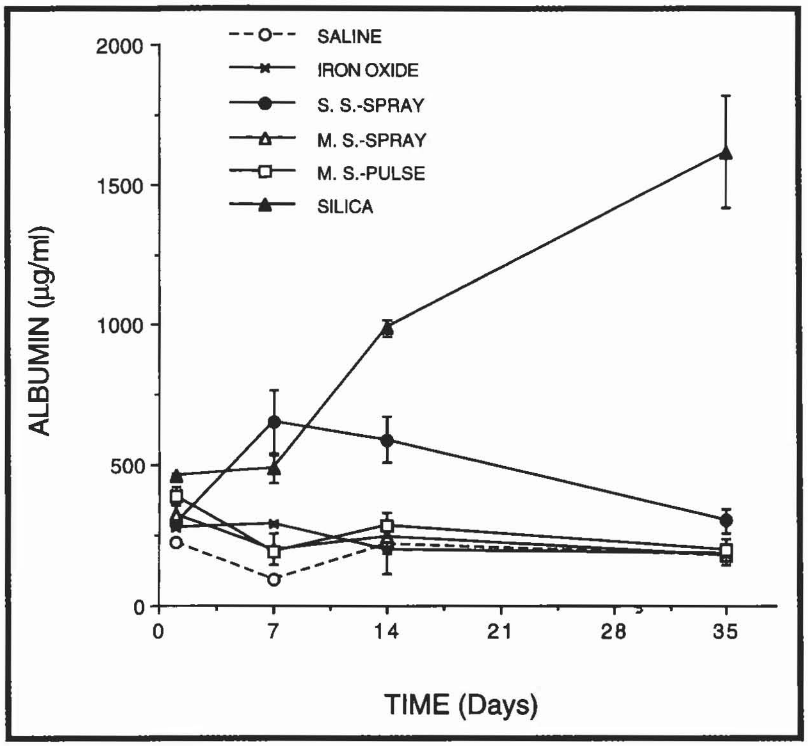

Fig. 1 — Albumin in the cell-free bronchoalveolar lavage fluid from the lungs of rats 1, 7, 14 and 35 days after the intratracheal instillation of different welding fumes: stainless steel spray transfer (SS-Spray), mild steel spray transfer (MS-Spray) and mild steel pulsed current (MS-Pulse).

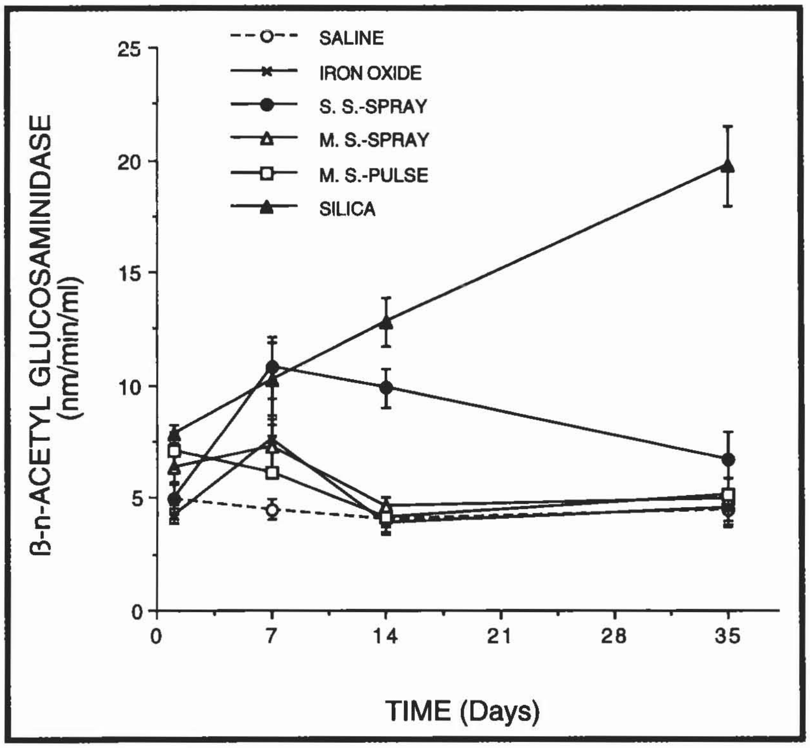

Fig. 2 — ß-n-acetyl glucosaminidase activity of the cell-free bronchoalveolar lavage fluid from the lungs of rats 1, 7, 14 and 35 days after the intratracheal instillation of different welding fumes: stainless steel spray, mild steel spray transfer and mild steel pulsed current.

The nature of respirable fume depends on the composition of the consumable electrode, the cleanliness of the base metal plate and the welding process used. The majority of fume is composed of oxides from electrode metals. Stainless steel and mild steel electrodes, two of the most common welding electrodes, have different elemental compositions and thus produce welding fumes with different chemical constituents.

It is important to distinguish between nontoxic and toxic fume. As noted above, iron oxide is considered a nontoxic fume. Nevertheless, a large amount of iron oxide entering the lungs causes a condition called siderosis, which may cause bronchitis, coughing or other respiratory irritation. On the other hand, elements in their pure form, such as manganese, chromium and nickel, may create toxic reactions under certain circumstances. Unfortunately, the specific circumstances by which these toxic reactions occur are not fully understood. In their pure form, these elements can produce reactions in the body; the elements move out of the lungs into the bloodstream and elsewhere in the body. However, there is insufficient evidence to show that these toxic effects apply when these elements are combined as an alloy with iron in welding fume.

There is some evidence that manganese combined with iron cannot readily cross from the blood into the brain — necessary for a toxic reaction — as pure manganese can. In this sense, the iron in steel welding fume, which always combines with manganese, may itself provide protection (Refs. 4, 5). Nevertheless, always limit exposure to welding fume as much as possible, because even nontoxic iron oxide creates a burden on the lungs, which is undesirable.

Studies have shown different degrees of lung damage induced by different fumes. In a study by Hicks, *et al.* (Ref. 6), using rats, inhalation of stainless steel welding fume caused greater and more prolonged lung damage than did mild steel welding fume. Still, the mild steel fume induced a significant amount of lung inflammation in these studies. Evidence of pulmonary fibrosis, a buildup of nonfunctional tissue, was also observed in the animals treated by either fume. In contrast, epidemiological studies have reported no incidence of fibrosis among humans exposed to welding fumes. However, the rats in Hicks's study received much higher doses of welding fumes than any welder would normally encounter.

Results of another study (Ref. 7) about the effects of both mild steel and stainless steel welding fumes on the lungs of rats are presented here. This work was performed to understand the pneumotoxicity and lung clearance of these fumes in a living animal.

The Experiment

Gas metal arc welding was performed using a power source capable of producing both conventional spray transfer and pulsed mode currents. The voltage used was 26 V at a current level of 220 A. Stainless (AWS designation ER308-A59) and mild steel (AWS designation ER70S-3) electrodes of 1.14 mm diameter were used. The electrode feed speed was 127 mm/s, while the welding was performed on a 12.7-mm-thick plate. The plate was rotated at a steady rate to provide a constant linear velocity of 5.3 mm/s; 96% Ar, 3% CO_2 and 1% H_2 was used with the stainless steel, while 92% Ar and 8% CO_2 was used for the carbon steel. The gas flow rate was 0.275 L/s.

Welding was performed in a metal enclosure and the fumes were collected using 0.2-μm Nuclepore filters for 2 min during welding and 2 min after welding. Approximately 200 mg of each sample was collected. The use of a pulsed current required additional welding passes since insufficient fume was formed during the 4 min of particle collection. It has been known for several years that pulsed-current welding generally produces less welding fume than constant-current welding (Ref. 8).

The welding fumes were mixed with saline solution and controlled amounts were instilled in the lungs of laboratory rats — 1 mg of fume per 100 g of body weight. This one-time dose is roughly equivalent to the amount of fume that a welder would be expected to breathe over a 30-day period, when working 8 h a day under the most serious fume conditions permitted by current OSHA (Occupational Safety and Health Administration) standards. A lower dose of one-fifth this value was also studied, but with no measurable effects. A higher dose (2 mg of fume per 100 g of body weight) was also evaluated, but the animals, so burdened with the particles in their lungs, simply could not breathe. Note that the dose rates used in this study are 30 times higher than a human would be expected to experience in a day of welding.

Silicon dioxide and iron oxide particles were used as positive and negative controls. It is well known that silica has a toxic effect on lungs and iron oxide does not.

After injected with the welding fume, the rats were put under anesthesia while their lungs were washed with a water solution for 30 s. The procedure was repeated 12 times, with samples of the washing fluid collected each time. Fluid samples were evaluated for the presence of cells, enzymes and albumin. Such tests were performed on separate groups of animals at 1-, 7-, 14- and 35-day intervals.

Table 1 — Size and Composition of the Welding Fume Samples

Sample	Count Mean Diameter (μM)	Metal Composition (% weight)
SS-Spray	1.38 ± 0.16	52.3% Fe, 2.3% Si, 18.3% Mn 22.2% Cr, 4.9% Ni
MS-Spray	1.22 ± 0.11	89.2% Fe, 2.6% Si, 8.2% Mn
MS-Pulse	1.12 ± 0.12	88.6% Fe, 3.8% Si, 7.6% Mn

Note: Diameter values are means ± SE; n = 200 particles/sample. No significant differences were observed in the size of the three welding fume samples: stainless steel spray (SS-Spray), mild steel spray (MS-Spray) and mild steel pulse (MS-Pulse).

In addition, since the welding fume has an easily measurable magnetism (ferromagnetism), the amount of magnetism from the animal's chest was measured at regular intervals to determine how rapidly the animal's body was removing the fume from its lungs.

What Happened

Table 1 provides the composition and mean particle size of the three types of fumes studied. Figure 1 shows the concentration of albumin in the animals' lungs over a period of time, while Fig. 2 shows similar results for an enzyme produced by the body.[1] All groups had significant elevations in albumin levels when compared to the saline control, but only the silica and mild steel pulsed current (MS-Pulse) groups had significant increases in enzyme activity. Animals from the stainless steel

Table 2 — Bronchoalveolar Lavage Cell Profiles

		TOTAL NUMBER (10^6)	
Treatment	Macrophage	Neutrophil	Lymphocyte
1 DAY			
Saline	5.1 ± 1.0	0.3 ± 0.1	0.1 ± 0.0
Silica	5.7 ± 0.5	13.4 ± 1.8$^{(a)}$	0.6 ± 0.2$^{(b)}$
SS-Spray	4.7 ± 0.3	6.2 ± 0.4$^{(b)}$	0.3 ± 0.1$^{(b)}$
MS-Spray	4.5 ± 0.3	8.3 ± 1.1$^{(b)}$	0.6 ± 0.3$^{(b)}$
MS-Pulse	4.6 ± 0.7	8.6 ± 0.7$^{(b)}$	0.5 ± 0.2$^{(b)}$
Iron Oxide	3.8 ± 1.0	6.6 ± 0.7$^{(b)}$	0.3 ± 0.1$^{(b)}$
7 DAYS			
Saline	4.7 ± 0.6	0.1 ± 0.0	0.0 ± 0.0
Silica	7.2 ± 0.4$^{(c)}$	10.3 ± 0.6$^{(a)}$	0.7 ± 0.1$^{(d)}$
SS-Spray	9.3 ± 0.3$^{(a)}$	4.0 ± 0.7$^{(c)}$	1.1 ± 0.3$^{(d)}$
MS-Spray	5.7 ± 0.7	1.5 ± 0.7$^{(b)}$	1.4 ± 0.7$^{(d)}$
MS-Pulse	5.6 ± 0.3	1.6 ± 0.6$^{(b)}$	0.2 ± 0.1
Iron Oxide	5.5 ± 0.8	1.6 ± 0.6$^{(b)}$	1.3 ± 0.3$^{(d)}$
14 DAYS			
Saline	4.8 ± 0.2	0.2 ± 0.0	0.2 ± 0.0
Silica	6.8 ± 1.1$^{(c)}$	20.4 ± 1.4$^{(a)}$	1.6 ± 0.2$^{(a)}$
SS-Spray	9.8 ± 0.5$^{(a)}$	7.3 ± 1.3$^{(c)}$	1.0 ± 0.1$^{(c)}$
MS-Spray	4.7 ± 0.5	0.3 ± 0.1	0.1 ± 0.0
MS-Pulse	4.2 ± 0.8	0.3 ± 0.1	0.1 ± 0.0
Iron Oxide	4.1 ± 0.3	1.0 ± 0.6	0.2 ± 0.1
35 DAYS			
Saline	3.7 ± 0.2	0.2 ± 0.1	0.1 ± 0.0
Silica	8.7 ± 0.7$^{(a)}$	41.4 ± 5.2$^{(a)}$	2.8 ± 0.5$^{(a)}$
SS-Spray	5.5 ± 1.2$^{(c)}$	1.8 ± 0.7$^{(c)}$	0.2 ± 0.1
MS-Spray	3.0 ± 0.3	0.3 ± 0.1	0.1 ± 0.1
MS-Pulse	3.4 ± 0.4	0.2 ± 0.1	0.1 ± 0.1
Iron Oxide	3.3 ± 0.5	0.2 ± 0.1	0.1 ± 0.0

Note: Values are means ± Se; n = 4.
(a) Significantly greater than all other groups ($p < 0.05$).
(b) Significantly greater than saline control group ($p < 0.05$).
(c) Significantly greater than MS-Spray, MS-Pulse, iron oxide and saline groups ($p < 0.05$).
(d) Significantly greater than MS-Pulse and saline groups ($p < 0.05$).

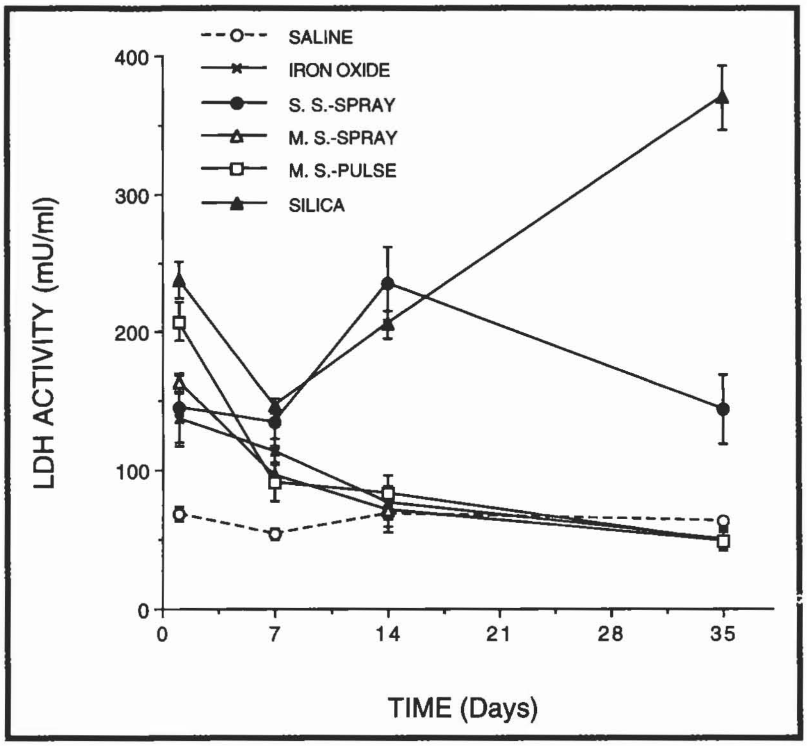

Fig. 3 — Lactate dehydrogenase (LDH) activity of the cell-free bronchoalveolar lavage fluid from the lungs of rats 1, 7, 14 and 35 days after the intratracheal instillation of different welding fumes: stainless steel spray transfer, mild steel spray transfer and mild steel pulsed current.

Table 3 — Pulmonary Clearance of Welding Fumes

Sample	Lung Burden (mg/Lung)	% Initial Dose
SS-Spray		
0 days	1.95 ± 0.46	100
1 day	1.74 ± 0.19	89.2
7 days	2.06 ± 0.26	105.6(a)
14 days	1.68 ± 0.31	86.2(a)
35 days	1.09 ± 0.07	55.9(a)
MS-Spray		
0 days	2.84 ± 0.31	100
1 day	2.60 ± 0.13	91.6
7 days	2.34 ± 0.17	82.3
14 days	1.83 ± 0.37	64.5
35 days	0.73 ± 0.07	25.6

Note: Lung burden values are means ± SE, n = 4–5. The animals received a single dose of 1.0 mg/100 g body weight of the stainless steel spray (SS-Spray) and the mild steel spray (MS-Spray) welding fumes. The initial dose (0 days) of welding fumes delivered to the lungs was measured 1 h after instillation. The percent of the initial dose in the lung of the SS-Spray group was significantly different from the values for the MS-Spray group.
(a) $p < 0.05$.

spray transfer (SS-Spray) and silica groups had significant elevations in albumin levels and enzyme activity at 7 and 14 days when compared with all the other groups. By 14 days, there were no differences in response among the mild steel spray transfer (MS-Spray), MS-Pulse, iron oxide and saline groups. By 35 days, levels of albumin and the enzyme for the SS-Spray group were still significantly elevated, but still below the silica control group. Injury induced by silica in the control animals continued to rise at 35 days and was significantly increased compared to all other groups. More activity from another enzyme (LDH) was also observed for all animals one day after the fume was instilled into their lungs; LDH activity was compared with the saline control group — Fig. 3.[1] At this point, values for the silica and MS-Pulse groups were significantly higher than the SS-Spray, MS-Spray and iron oxide groups. For the MS-Spray, MS-Pulse and iron oxide groups, the increases in activity were diminished by seven days and no different than saline control levels by fourteen days. Both the silica and SS-Spray groups had substantial elevations in LDH activity at both 14 and 35 days when compared with all the other groups. However, LDH levels were still rising for the silica group, but falling for the SS-Spray group at 35 days.

Table 2 shows the rate at which the animals' lungs were cleared of the welding fumes. For the MS-Spray group, particles were cleared quickly. On the other hand, the SS-Spray group cleared significantly slower, with 86% of fume particles still present in the lungs after 14 days. After 35 days, 56% of the SS-Spray welding fume remained in the lungs. An exponential decay model for the fume particles gave half-lifes of 47 days for the SS-Spray and 18 days for the MS-Spray groups. Microscopic analysis of the lung tissue after 35 days showed that almost all mild steel particles had been cleared from the lungs.

Toxic, or Not Toxic?

According to this experiment, mild steel welding fume, using either pulsed or conventional spray transfer current, gives essentially the same lung response as iron oxide, a nontoxic irritant. The experiment is consistent with previous epidemiological research (Ref. 9) showing that welders have no serious chronic health problems when inhaling mild steel welding fume. On the other hand, the responses of silica and stainless steel fume were virtually identical for 14 days. It was also found that stainless steel fumes take longer to clear the lungs (Table 3). After 35 days, however, the stainless steel fume response declined, approaching the level of the iron oxide and mild steel fume.

Therefore, according to this data, the conclusion can be made that stainless steel fume is a stronger lung irritant than mild steel fume, but the long-term toxic effect of the stainless steel fumes on the body is questionable.

Conclusions

According to this study, welding fumes generated from different electrodes produce different lung responses and are cleared from the lungs at different rates. It was necessary to use a very large dose to elicit any response. Doses typical of those experienced by welders on a daily basis showed no response under these tests. Using higher doses, the stainless steel spray transfer (SS-Spray) fume was more toxic and was retained in the lung longer than mild steel fumes. Unlike silica, it appears that the SS-Spray particles are eventually cleared from the lungs and thus, the potential for toxic effects in the lungs is low if fume exposure ceases or if doses are reduced.

Mild steel fumes induced a similar response as iron oxide, considered only a nuisance and nontoxic. For mild steel fume, altering the power supply (spray transfer vs. pulsed) had a negligible effect on the size and elemental composition of the fume

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particles, as well as their ability to produce injury and inflammation. However, since pulsed welding is associated with reduced fume production (Ref. 10), pulsed welding should reduce the exposure of welders and help reduce any lung irritation.

This experiment suggests that mild steel fume has no greater effect on the health of welders than does the ingestion of iron oxide. Nevertheless, take care to reduce exposure even to nuisance dust. These tests do suggest that stainless steel fumes produce a more significant response in the lung. However, further study is needed to determine whether this response has long-term significance. It is also yet to be determined whether the lung's response would be measurable at more the conventional doses experienced by welders. ◆

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