American Industrial Hygiene Association Journal
Publication details, including instructions for authors and subscription information:
http://www.tandfonline.com/loi/aiha20

Estimation of Regional Pulmonary Deposition and Exposure for Fumes from SMAW and GMAW Mild and Stainless Steel Consumables

Paul Hewett [a]

[a] National Institute for Occupational Safety and Health, 1095 Willowdale Road, Morgantown, WV 26505-2845

Published online: 04 Jun 2010.

To cite this article: Paul Hewett (1995) Estimation of Regional Pulmonary Deposition and Exposure for Fumes from SMAW and GMAW Mild and Stainless Steel Consumables, American Industrial Hygiene Association Journal, 56:2, 136-142

To link to this article: http://dx.doi.org/10.1080/15428119591017169

ESTIMATION OF REGIONAL PULMONARY DEPOSITION AND EXPOSURE FOR FUMES FROM SMAW AND GMAW MILD AND STAINLESS STEEL CONSUMABLES

Paul Hewett

National Institute for Occupational Safety and Health, 1095 Willowdale Road, Morgantown, WV 26505-2845

The particle size distributions and bulk fume densities for mild steel and stainless steel welding fumes generated using two welding processes (shielded metal arc welding [SMAW] and gas metal arc welding [GMAW]) were used in mathematical models to estimate regional pulmonary deposition (the fraction of each fume expected to deposit in each region of the pulmonary system) and regional pulmonary exposure (the fraction of each fume expected to penetrate to each pulmonary region and would be collected by a particle size-selective sampling device). Total lung deposition for GMAW fumes was estimated at 60% greater than that of SMAW fumes. Considering both the potential for deposition and the fume specific surface areas, it is likely that for equal exposure concentrations GMAW fumes deliver nearly three times the particle surface area to the lungs as SMAW fumes. This leads to the hypothesis that exposure to GMAW fumes constitutes a greater pulmonary hazard than equal exposure to SMAW fumes. The implications of this hypothesis regarding the design of future health studies of welders is discussed.

In a companion article[1] (see p. 128) the methods and results were presented from a study[2] of the particle size distributions and physical characteristics (bulk fume density and specific surface area) of various common welding fumes. In this article the particle size distributions and bulk fume densities are used to estimate, using mathematical models, both regional pulmonary deposition (the fraction of each fume that deposits in each region of the pulmonary system) and regional pulmonary exposure (the fraction of each fume that penetrates to each pulmonary region and would be measured by an ideal particle size-selective sampling device). The limitations of the deposition estimates are discussed since (1) the pulmonary deposition model did not account for enhanced deposition due to hygroscopic particle growth and deposition due to interception and electrostatic attraction, and (2) there may be inaccuracies in the estimation of diffusion diameters. Assuming that the estimates of regional deposition are reasonably accurate, it is possible that there will be a different exposure-response relationship for each type of fume. This hypothesis should be of interest to anyone intending to study pulmonary disease in welders.

BACKGROUND

The welding methods and consumables studied in the companion article[1] were shielded metal arc welding (SMAW) using AWS (American Welding Society) E7018 mild steel (MS) and AWS E308–16 stainless steel (SS) welding rods, and gas metal arc welding (GMAW) using AWS E70S-3 mild steel and AWS ER308 stainless steel welding wires. The primary sampling device was a micro orifice uniform deposit (cascade) impactor (MOUDI).[3] The geometric mean (GM) for fumes from both wires was 0.25 μm aerodynamic diameter. The mass distributions for the SMAW welding wire fumes also were basically submicrometer, but included larger particles resulting in larger GMs: 0.59 μm aerodynamic diameter for the SMAW-MS welding rod and 0.46 μm aerodynamic diameter for the SMAW-SS welding rod.

Bulk density and specific surface area were determined from a bulk sample of each fume using micropycnometry and gas adsorption methods, respectively. The bulk fume densities for mild steel and stainless steel fume were similar for each welding method, but differed considerably between welding methods. The bulk density for both SMAW-MS and SMAW-SS fumes was 3.4 g/cm^3. In contrast, the bulk density for the GMAW-MS and GMAW-SS fumes averaged 5.8 g/cm^3. The specific surface area results followed a similar trend. The averages were 18.6 m^2/g for the SMAW fumes and 33.4 m^2/g for the GMAW fumes.

METHODS

The fraction of each fume that deposits in the various regions of the pulmonary system was estimated by numerically integrating

Disclaimers: Mention of company or product names does not constitute an endorsement by the National Institute for Occupational Safety and Health.

the product of the regional deposition function, the mass fraction distribution function, and an inhalability function:

$$DE_i = \int I(x)DE_i(x)f(x)dx \quad (1)$$

where DE_i represents the fraction of an aerosol that deposits in the i^{th} region of the pulmonary system; x is the particle aerodynamic diameter; I(x) is the inhalability function; $DE_i(x)$ is the deposition function for the i^{th} region; and f(x) is the mass distribution function. The regional deposition functions—DE (total pulmonary deposition); DE_l (laryngeal or extrathoracic deposition); DE_{ba} (thoracic deposition); DE_b (tracheobronchial deposition); and DE_a (alveolar deposition)—come from an empirical pulmonary deposition model described by Stahlhofen et al.[4] (This model, hereafter referred to as the German deposition model, is a revision of an earlier model.[5,6]) The inhalability function, I(x), refers to the probability that a given size particle enters the mouth during oral breathing. The best estimate of this function is the empirically derived American Conference of Governmental Industrial Hygienist (ACGIH)[7] inhalable (formerly inspirable) particulate mass (IPM) collection efficiency function.

The German deposition model[4] uses relatively simple formulae to calculate regional deposition curves for any combination of breathing mode (oral versus nasal breathing), tidal volume, breathing rate, and functional residual capacity (within the range of experimental values). The parameters entered into the model were those of the ACGIH[7] reference worker: oral breathing mode, tidal volume (1450 cm^3/breath), breathing frequency (15 breaths/min), and functional residual capacity (2200 cm^3). Figure 1 shows the pulmonary deposition curves calculated by this model.

The mass distribution function, at any x, can be calculated:

$$f_n(x) = \frac{1}{\log_{10} GSD\sqrt{2\pi}} \exp\left(\frac{-(\ln x - \ln GM)^2}{2(\ln GSD)^2}\right) \quad (2)$$

where GM and GSD are the geometric mean and geometric standard deviation of the mass distribution. The distribution function parameters for the four fumes were estimated in Reference 1 using particle size distribution data collected using a cascade impactor.

The author modified the German deposition model equations so that pulmonary deposition curves could be calculated for different particle densities, but with all diameters expressed in aerodynamic diameters. This was advantageous considering that mass distributions derived from cascade impactors are expressed in aerodynamic terms. However, as the diameter of a particle decreases, the probability of deposition is increasingly related to the particle's diffusion diameter, which is closely related to the actual size and shape of the particle.[8] The aerodynamic and diffusion diameters are different for densities other than one. For those equations in the model that estimated regional deposition due to diffusion, the aerodynamic diameter was converted to an approximate diffusion diameter by the following equation:

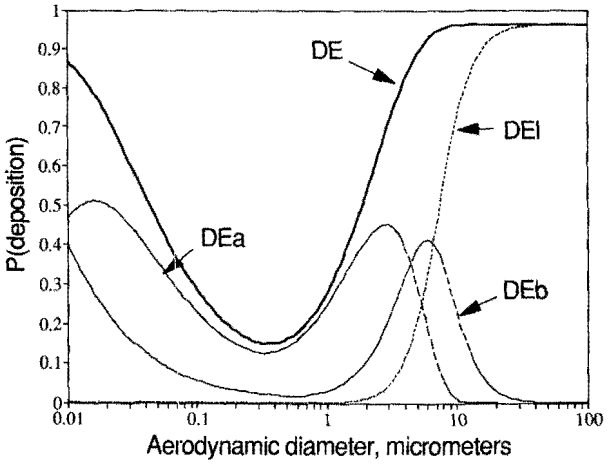

FIGURE 1. Regional pulmonary deposition curves calculated using the German deposition model.[4] Model parameters were: oral breathing, tidal volume 1450 cm^3, breathing rate 15 min^{-1}, and functional residual capacity 2200 cm^3. DE = total deposition, DE_l = laryngeal (extrathoracic) deposition, DE_b = tracheobronchial deposition, and DE_a = alveolar deposition.

$$d = \left(\frac{\rho C(d_a)}{\rho_0 C(d)}\right)^{1/2} d_a \quad (3)$$

where d is the approximate diffusion diameter; ρ is the bulk fume density; ρ_o is unit density; and C is the slip correction function evaluated for both the aerodynamic diameter, d_a, and diffusion diameter, d. This calculation required the assumption that (1) the bulk fume density and individual particle densities were similar, and (2) the particles were reasonably spherical and solid. The diameter calculated by Equation 3 will be wrong to the extent that either of these assumption do not apply. Figure 2 shows the effect of different densities on the alveolar deposition curves. The significance of this density effect is that predictions of alveolar deposition are different for identical submicrometer mass distributions (when expressed in aerodynamic diameters) when the densities are different. For example, a 0.1-μm aerodynamic diameter particle having a density of 1 g/cm^3 has a 20% probability of depositing in the alveoli; in comparison, a particle having an identical aerodynamic diameter, but a density of 6 g/cm^3, has a 50% probability of depositing in the alveoli.

The fraction of each fume that is collected by an ideal sampling device was estimated by numerically integrating the product of the appropriate particle size-selective collection efficiency function (Figure 3) and the mass fraction distribution function:

$$i^{th} \text{ fraction} = \int E_i(x)f(x)dx \quad (4)$$

where the i^{th} fraction refers to the inhalable particulate mass (IPM) fraction, thoracic particulate mass (TPM) fraction, or respirable particulate mass (RPM) fraction; x is the aerodynamic diameter); $E_i(x)$ refers to the appropriate sampling device col-

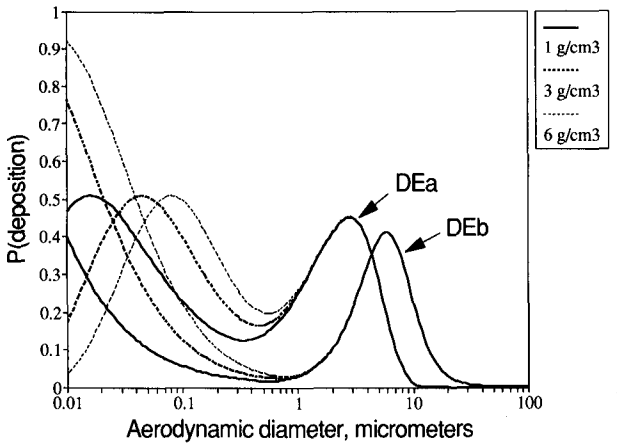

FIGURE 2. Density-specific tracheobronchial and alveolar deposition curves where the particles are spherical, and the diameter is expressed in aerodynamic terms. Curves were calculated using the regional deposition functions of the German deposition model.[4]

lection efficiency function; and f(x) is the previously mentioned mass fraction distribution function. The collection efficiency function has been defined by ACGIH for collecting inhalable (formerly inspirable), thoracic, and respirable particulate mass. (ACGIH recently recommended revising the particle size-selective collection efficiency functions.[9] Considering that each welding fume consisted mostly of submicrometer particles, Equation 4 will yield approximately the same result regardless of which collection efficiency functions are used.)

RESULTS AND DISCUSSION

Using Equations 1–3, the author estimated that between 23% and 41% of the welding fumes studied deposit in the pulmonary system, and the majority of this deposits in the alveoli (Table I). There was a sharp distinction between the deposition estimates for welding rods and wires. Predicted total lung deposition for the SMAW-MS and SMAW-SS fumes was 26% and 23%, respectively. Predicted deposition for the GMAW-MS and GMAW-SS fumes was 41% and 40%, respectively. Therefore, it is likely that for equal exposures, roughly 60% more GMAW fume deposits in the lung compared with SMAW fume. In contrast, there was no distinction between the predicted regional pulmonary exposures (Table I). Virtually 100% of the fumes of all four consumables was predicted to be collected by sampling devices meeting any of the ACGIH particle size sampling criteria.

SMAW-MS is the process most represented in past pulmonary function studies of welders. GMAW is a newer technology that is increasing in popularity. The specific surface areas for GMAW fumes were nearly twice those for SMAW fumes.[1] Considering both the differences in specific surface area and predicted deposition leads to the hypothesis that for equal exposure concentrations GMAW fumes deliver nearly three times the particle surface area to the pulmonary system compared with SMAW fumes. Since it is logical that any health effect, be it pulmonary function impairment or lung cancer, is related to the quantity of metal either released in dissolved form into the lung tissues or the area of contact between particle and tissue, two hypotheses result: (1) if the response is proportional to mass deposited, the differences in response for welders exposed to SMAW fume compared with welder exposure to GMAW fume may, for equal exposures, vary by 60%; (2) if the biological response is proportional to surface area, the differences in response may, for equal exposures, vary by a factor of three.

There have been some direct and indirect measurements of fume deposition in welders. Stahlhofen and Möller[10] used magnetopneumography to measure the lung burden of magnetic material in control subjects, SMAW welders, and GMAW welders. The GMAW welders had between 60 and 400 mg of magnetic material in their lungs, compared with roughly 10 to 30 mg for the SMAW welders and 0.1 to 1 mg for the controls. A direct comparison of these lung burdens is limited by the lack of information on exposure levels, duration of exposure, and the fraction of each fume that was magnetic. These results do, however, lend support to the hypothesis that welders exposed to GMAW fumes are, for equal exposures, at greater risk than welders exposed to SMAW fumes.

Rudell et al.[11] measured total deposition in six subjects exposed to fumes from a mild steel welding rod similar to the SMAW-MS welding rod used here. The subjects inhaled through the nose and exhaled through the mouth. The inhaled and exhaled aerosol was monitored with low pressure cascade impactors. Deposition ranged between 55% and 73%, with an average of 64%, which is considerably greater than the 26% deposition predicted here for a similar fume. Akselsson et al.[12] used methodology and equipment similar to that used by Rudell et al., and found that for nasal breathing the deposition for eight elements

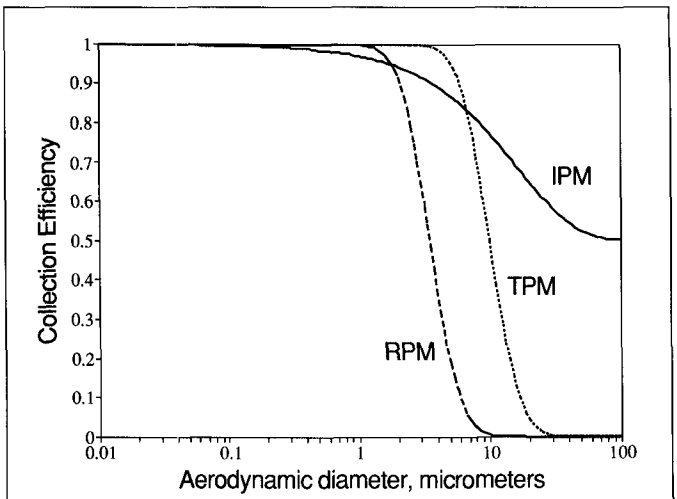

FIGURE 3. ACGIH[7]*-recommended collection efficiency curves for particulate size-selective sampling devices: inhalable particulate mass (IPM), thoracic particulate mass (TPM), and respirable particulate mass (RPM)*

TABLE I. Estimated Fractions of Fumes from Different Consumables that Deposit in or Penetrate to Various Regions of the Pulmonary System during Oral Breathing

	Regional Deposition			Regional Exposure		
Consumable	Total Deposition	Thoracic Deposition	Alveolar Deposition	Inhalable Particulate Mass	Thoracic Particulate Mass	Respirable Particulate Mass
SMAW-MS (AWS E7018)	0.26	0.26	0.22	0.98	1.00	0.99
SMAW-SS (AWS E308-16)	0.23	0.23	0.19	0.99	1.00	1.00
GMAW-MS (AWS E70S-3)	0.41	0.41	0.31	0.99	1.00	1.00
GMAW-SS (AWS ER308)	0.40	0.40	0.31	0.99	1.00	1.00

in a welding fume (type not given) averaged 30% (author's calculation from summary data in Akselsson's Table 5). Ahlberg et al.[13] also used similar equipment and found that deposition during nasal breathing was 20% or less for particles in the range of 0.25 to 0.5 μm aerodynamic diameter.

For comparison to the Rudell et al., Akselsson et al., and Ahlberg et al. measurements, the German deposition model was used to estimate total deposition for nasal breathing. The resulting estimate was 33%, similar to the averages measured by Akselsson et al. and Ahlberg et al., but well below the average observed by Rudell et al. The Rudell et al. deposition measurements (and the Akselsson et al. and Ahlberg et al. measurements) cannot be uncritically accepted. The breathing patterns of the subjects in the Rudell et al. study were not given, and breath holding or substantial pauses between inhalation and exhalation increases deposition for all particle sizes. In addition, the health status of the subjects was not discussed, and there can be considerable variation in deposition for any group, particularly when the group consists of smokers or those who have chronic obstructive pulmonary disease from prolonged exposure to welding fumes or other contaminants. However, if the deposition measurements of Rudell et al. are approximately correct, the accuracy of the deposition estimates reported here becomes questionable.

Even without the results of Rudell et al. the estimates of regional deposition in Table I should be considered rough approximations for several reasons. First, the German deposition model, as revised for this study, calculates regional deposition due to particle diffusion based on the particle diffusion diameters calculated using Equation 3. These diameters may not be good approximations, depending on how well the Equation 3 assumptions were met. Second, the German deposition model does not take into account the effects of electrostatic attraction, interception, and hygroscopic particle growth when estimating regional deposition. These effects are next examined to determine if any can explain the difference between measured and predicted deposition.

Electrostatic Deposition

Current deposition models do not take into account electrostatic deposition of charged particles. The German deposition model was based on the deposition of electrically neutral particles.[14] Johnston et al.[15] measured the electrostatic charge for several aerosols, including welding fumes. Expecting highly charged welding fume particles, they found that both SMAW and GMAW fume particles had less than two charges per particle. They attributed the low level of charging in the welding fumes to "charge-aging," which they define as the process whereby free ions in the air neutralize charged particles. They theorized that the presence of free positive and negative ions in the plasma surrounding the electric arc were responsible for this rapid neutralization. In summary, electrostatic deposition is unlikely to be a major factor in the deposition of welding fumes.

Interception

The author has found no human deposition data for chains and branched structures. Hammad and Attieh[16] found that total pulmonary deposition (nasal breathing mode) in the rat for fibers between 1 and 10 μm in length and a diameter of 0.4 μm was between 25 and 50%. No articles were found that discuss the interception of small fibers in the smallest airways. However, considering the results of Hammad and Attieh, it is possible that interception of chains and branched structures of fume particles may contribute substantially to the total deposition of welding fumes.

Hygroscopic Particle Growth

The flux coating of many mild steel and stainless steel electrodes contains calcium and sodium fluorides. Thus, it is conceivable that a fraction of the fumes is water soluble and capable of hygroscopic growth, resulting in enhanced total deposition. Rudell et al.[11] measured changes in the welding fume particle size distributions as a function of temperature and humidity. Fumes from different consumables were mixed with heated and humidified air. The particle size distributions were measured before and after mixing. For fumes from the low-hydrogen (fluoride flux) welding rod, which is similar to the SMAW-MS welding rod used here, they found that the mass median aerodynamic diameter (geometric mean) increased approximately 30%, from 0.54 μm aerodynamic diameter to 0.70 μm aerodynamic diameter when the temperature and relative humidity increased from 22°C and 45% to 37.1°C and 99%. Smaller increases were observed for the other fumes studied, none of which contained fluorides.

TABLE II. Ratio of Inhalable, Thoracic, and Respirable Particulate Exposure to Total, Thoracic, and Alveolar Deposition, Respectively, for Fumes from Various Welding Consumables

Consumable	*Inhalable Particulate Mass/Total Deposition*	*Thoracic Particulate Mass/Thoracic Deposition*	*Respirable Particulate Mass/Alveolar Deposition*
SMAW-MS (AWS E7018)	3.77	3.84	4.50
SMAW-SS (AWS E308-16)	4.30	4.35	5.26
GMAW-MS (AWS E70S-3)	2.41	2.44	3.23
GMAW-SS (AWS ER308)	2.48	2.50	3.23

To assess the effect of hygroscopic growth on deposition, the German deposition model was used to estimate oral and nasal deposition for the SMAW-MS welding fume where the geometric mean is increased by 30%, from 0.59 μm aerodynamic diameter to 0.77 μm aerodynamic diameter. Total deposition for oral breathing was predicted to be 28%, which is only slightly greater than that predicted previously. Total deposition for nasal breathing was 38%, which is an increase of 5% over the earlier estimate of total nasal deposition for the SMAW-MS welding fume. This prediction is consistent with the measured deposition fractions reported by Akselsson et al.[12] (discussed previously).

Several investigators have reported findings that make the hygroscopic fume hypothesis a plausible explanation for the high deposition values reported by Rudell et al. Ahlberg et al.[17] studied the effect of humidity on the aerodynamic and mobility diameter of welding fumes. They concluded that hygroscopic welding fume agglomerates may collect moisture in the humidified environment of the lungs and collapse to spheres. For large agglomerates this will have the interesting effect of increasing deposition due to both sedimentation and diffusion.* They concluded that lung deposition due to sedimentation (based on measurements of aerodynamic diameter) should be lower than predicted for a nonhygroscropic welding fume and higher than predicted for a hygroscopic welding fume. Kütz and Schmidt-Ott[18] showed that agglomerates tend to collapse to spheres if they are capable of being wetted by a vapor. Thus, if a portion of the welding fume is water soluble then the welding fume agglomerates—cluster, chains, and filamentous structures—may tend to absorb moisture and, due to surface tension forces, collapse to more dense spherical shapes.

The implications of these articles and the Rudell et al. article are significant. Any welding fumes that contain water-soluble components may deposit at much greater rates than those predicted. Furthermore, the use of particle sizing techniques that measure the inhaled particle size distribution in terms of aerodynamic diameter, diffusion diameter, or projected area (determined using electron microscopy), may have limited usefulness for predicting pulmonary deposition if welding fume agglomerates, particularly those containing water-soluble components, undergo such radical changes once inside the pulmonary system.

This leaves unresolved the difference between the large deposition values measured by Rudell et al. and those predicted here for a similar welding fume. However, the results of Stahlhofen and Möller,[10] Akselsson et al.,[12] and Ahlberg et al.[13] (discussed earlier) lend support to the hypothesis that SMAW fumes deposit at a lower rate, perhaps similar to that predicted here.

IMPLICATIONS FOR THE STUDY OF HEALTH EFFECTS IN WELDERS

Of particular interest are the implications these hypotheses have regarding welder epidemiology. There is the possibility that the differences in the particle size distributions affect the probability of observing a significant exposure-response relationship in pulmonary function studies of welders if, as in previous studies, the exposures are measured simply as total fume. In an earlier paper[19] the author introduced the distribution constant ($K_{(PSD)}$) and showed how it relates dose and exposure for any particular particle size distribution. The distribution constant is simply the ratio of exposure (mass collected per unit volume sampled) to dose** (mass deposited per unit volume inhaled):

$$K_{(PSD)} = \frac{\int E_i(x)f(x)dx}{\int I(x)DE_i(x)f(x)dx} \quad (5)$$

where x is the particle aerodynamic diameter; f(x) is the mass distribution function; $E_i(x)$ and $DE_i(x)$ represent the exposure sampling device collection efficiency function and the regional pulmonary deposition function, respectively, for the i^{th} region of

* The collapsed agglomerate will have both an increased aerodynamic diameter due to the extra absorbed moisture and spherical shape, and a decreased "mobility diameter" due to the more compact conformation.

** Dose also can be defined as mass deposited or mass deposited per unit time, but this requires knowledge of minute volumes and lung deposition curves for individual workers. It can be argued that even this definition should be improved to describe particulate mass that persists long enough to cause damage, either in the alveoli or at translocation sites. This requires knowledge of individual clearance rates, particle dissolution rates, and other measures of bioavailability. If no such information is available on an individual basis (and much of the above is, from a practical viewpoint, unknowable), and average values must be assumed for all workers, then these refined estimates of dose will contain no additional power to discriminate between workers. The definition of dose used here—mass deposited per unit volume inhaled for the ACGIH[7] reference worker—varies with particle size distribution and mass concentration, both of which can be measured on an individual basis.

the pulmonary system; and I(x) is the inhalability function (ACGIH[7] inhalable particulate mass collection efficiency function). The distribution constants relating total lung deposition fraction (DE) to inhalable particulate mass (IPM) fraction, thoracic deposition fraction (DE_{ba}) to thoracic particulate mass (TPM) fraction, and alveolar deposition fraction (DE_a) to respirable particulate mass (RPM) fraction are presented in Table II. These were calculated using the estimates of regional deposition and exposure from Table I. The exposure measurement overestimates the dose when the distribution constant is greater than one. The distribution constant is different for each combination of mass distribution, sampling instrument collection efficiency curve, and regional pulmonary deposition curve. Assuming that the latter two are relatively fixed, the distribution constant becomes a function of the particle size distribution.

The distribution constants were similar for the two SMAW fumes and the two GMAW fumes, but differed when compared by welding method (SMAW versus GMAW). Since the distribution constant is the slope of the curve relating exposure and deposition, these relationships can be displayed graphically. In Figure 4 the effect of particle size distribution on the IPM/DE distribution constant can be seen by considering that for identical exposure concentrations, the mass deposited (per m^3 inhaled) is substantially different when comparing SMAW to GMAW fumes. Therefore, a difference should also exist between the SMAW and GMAW exposure-response relationships. Hypothetical dose- and exposure-response curves are graphed in Figure 5. These curves were calculated using the following equation (Equation 6 of Reference 19):

$$\text{Response} = \frac{K_{(DR)}}{K_{(PSD)}} \cdot \text{Exposure} \qquad (6)$$

and the distribution constants in Table II for IPM versus total

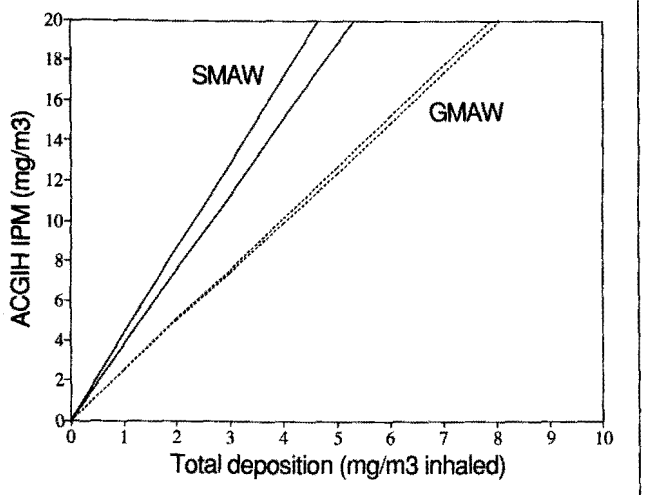

FIGURE 4. Inspirable particulate mass exposure (IPM) graphed against total lung dose for the two SMAW and GMAW fumes. Dose defined as mass deposited per unit volume inhaled.

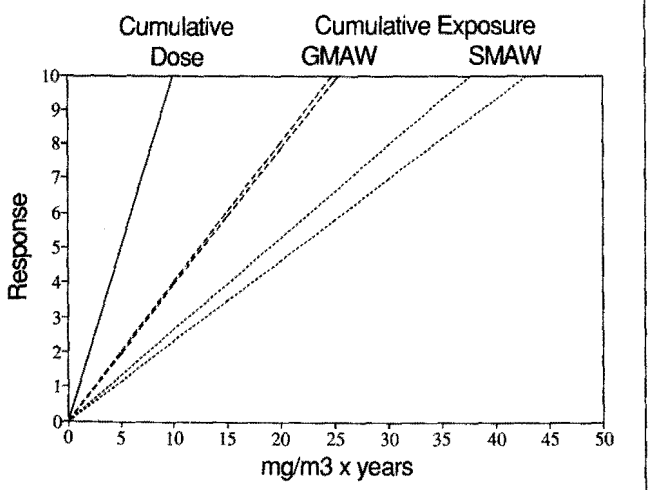

FIGURE 5. Hypothetical exposure-response curves illustrating the potential difference between SMAW and GMAW processes if response is proportional to mass deposited within the pulmonary system

deposition (DE). The author arbitrarily assumed a dose-response effect ($K_{(DR)}$) of one response unit per $mg/m^3 \cdot$ year. If the (cumulative) exposure-response curves in Figure 5 represented a real study that combined welders engaged in SMAW and GMAW using either mild steel or stainless steel consumables, then the cumulative exposure data would fall along the two separate sets of dotted lines. It can easily be seen that the cumulative exposure data would have considerable variability simply due to process differences. It is possible to conceive of a situation where the GMAW group has lower cumulative exposures than the SMAW group, but larger responses, resulting in a negative slope for the exposure-response curve. In this hypothetical example the author assumed that the response was proportional to the quantity deposited. If the response is proportional to the total (deposited) particle surface area, the difference in responses for equal exposures will be greater.

Given the results of this analysis, it is advisable, in any study of pulmonary disease in welders, to collect aerodynamic and diffusion particle size distribution data for each welding process and consumable combination. These data, coupled with total fume measurements and information on exposure duration, work rate, and breathing mode, can then be used to generate estimates of actual deposition. An additional benefit of this approach is that factors that affect deposition curves, but are usually ignored (such as work rate and nasal breathing), can now be taken into account. These estimates remain exposure estimates, as it is not possible to estimate true individual deposited dose without knowledge of individual deposition characteristics, breathing rates, and tidal volumes. However, the use of more accurate estimates of actual deposited particulate should, in principle, result in exposure-response curves with slopes closer to the true, underlying dose-response curves. In addition to the above, the bulk fume specific surface area, particle surface chemistry,[20] and lung clearance rates[21] also vary with fume type and may be

useful when distinguishing between the potential health effects of chemically similar welding fumes.

ACKNOWLEDGMENTS

This article summarizes a portion of the work done for a doctoral degree in industrial hygiene at the School of Public Health, University of Pittsburgh. The author wishes to thank NIOSH for supporting him in the pursuit of this degree, Dr. Nurtan A. Esmen for serving as his advisor, and Dr. Sidney Soderholm for his many helpful suggestions regarding the draft manuscript.

REFERENCES

1. **Hewett, P.:** The particle size distribution, density, and specific surface area of welding fumes from SMAW and GMAW mild and stainless steel consumables. *Am. Ind. Hyg. Assn. J. 56*:128-135 (1995).
2. **Hewett, P.:** "Particle Size Analysis of Welding Fumes and Prediction of Pulmonary Deposition." Ph.D. diss., School of Public Health, University of Pittsburgh, Pittsburgh, PA. Available from *Dissertation Abstracts International*, vol. 52–10B, University Microfilms, Ann Arbor, MI [Pub. #9209560], 1991.
3. **Marple, V.A., K. Rubow, and S.M. Bahm:** A microorifice uniform deposit impactor (MOUDI): description, calibration, and use. *Aerosol Sci. Technol. 14*:434–446 (1991).
4. **Stahlhofen, W., G. Rudolf, and A.C. James:** Intercomparison of experimental regional aerosol deposition data. *J. Aerosol Med. 2*:285–308 (1989).
5. **Heyder, J., J. Gebhart, G. Rudolf, C.F. Schiller, and W. Stahlhofen:** Deposition of particles in the human respiratory tract in the size range 0.005–15 μm. *J. Aerosol Sci. 17*:811–825 (1986) (erratum *18*:353 (1987)).
6. **Rudolf, G., J. Gebhart, J. Heyder, C.F. Schiller, and W. Stahlhofen:** An empirical formula describing aerosol deposition in man for any particle size. *J. Aerosol Sci. 17*:350–355 (1986).
7. **American Conference of Governmental Industrial Hygienists (ACGIH):** *Particle Size-Selective Sampling in the Workplace. Report of the ACGIH Technical Committee on Air Sampling Procedures.* Cincinnati, OH: ACGIH, 1985.
8. **Hinds, W.C.:** *Aerosol Technology.* New York: John Wiley & Sons, 1982.
9. **American Conference of Governmental Industrial Hygienists (ACGIH):** Notice of intended change—appendix d—particle size-selective sampling criteria for airborne particulate matter. *Appl. Occup. Environ. Hyg. 6*:795–796 (1991).
10. **Stahlhofen, W. and W. Möller:** Description of a biomagnetic method for detection of the behavior of magnetic aerosols in the human lung. [Abstract of a presentation at the 1988 European Aerosol Conference] *J. Aerosol Sci. 19*:1087–1091 (1988).
11. **Rudell, B., K.R. Akselsson, and M.H. Berlin:** Growth of welding aerosol particles in high relative humidity and particle deposition in the human respiratory airways. [Abstract of a presentation at the 1988 European Aerosol Conference] *J. Aerosol Sci. 19*:1153–1156 (1988).
12. **Akselsson, K.R., G.G. Desaedeleer, T.B. Johansson, and J.W. Winchester:** Particle size distribution and human respiratory deposition of trace metals in indoor work environments. *Ann. Occup. Hyg. 19*:225–238 (1976).
13. **Ahlberg, M.S., H.J. Annegarn, B.O. Dalhbäck, A.C.D. Leslie, and J.W. Winchester:** Particle size dependent aerosol deposition in the human respiratory tract. *Trace Substances Environ. Health 7*:463–471 (1978).
14. **Stahlhofen, W., J. Gebhart, and J. Heyder:** Experimental determination of the regional deposition of aerosol particles in the human respiratory tract. *Am. Ind. Hyg. Assoc. J. 41*:385–398a (1980).
15. **Johnston, A.M., J.A. Vincent, and A.D. Jones:** Measurements of electric charge for workplace aerosols. *Ann. Occup. Hyg. 29*:271–284 (1985).
16. **Hammad, Y.Y. and B. Attieh:** Pulmonary deposition of MMMF. *J. Aerosol Sci. 20*:1329–1332 (1989).
17. **Ahlberg, M.S., H-C. Hansson, and I. Fängmark:** The growth of welding fume as function of relative humidity. In *Health Hazards and Biological Effects of Welding Fumes and Gases.* R.M. Stern, et al., ed. New York: Elsevier Science Publishers, Biomedical Division, 1986.
18. **Kütz, S. and A. Schmidt-Ott:** Characterization of agglomerates by condensation-induced restructuring. *J. Aerosol Sci. 23(suppl. 1)*:S357–S360 (1992).
19. **Hewett, P.:** Limitations in the use of particle size selective sampling criteria in occupational epidemiology. *Appl. Occup Environ. Hyg. 6*:290–300 (1991).
20. **Grekula, A., E. Ristolainen, V.P. Tanninen, H-K. Hyvarinen, and P-L. Kalliomaki:** Surface and bulk chemical analysis on metal aerosols generated by manual metal arc welding of stainless steel. *J. Aerosol Sci. 17*: 1–9 (1986).
21. **Kalliomaki, P-L., M-L. Junttila, K.K. Kalliomaki, E-L. Lakomaa, and R. Kivela:** Comparison of the behavior of stainless steel and mild steel manual metal arc welding fumes in rat lung. *Scand. J. Work, Environ., Health 9*:176–180 (1983).