Methods and Applications

Recommended Occupational Exposure Limits for GMA Using Benchmark Dose and Bayesian Model Averaging

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ABSTRACT

Introduction: Glycidyl methacrylate (GMA) is a widely used industrial polymerization material. Current occupational exposure limits (OELs) for GMA in China show significant disparities compared to those established by international regulatory bodies, including the United States, the European Union, and Japan. A comprehensive revision of GMA exposure limits is crucial for ensuring optimal worker protection.

Methods: This investigation analyzed data from a 104-week inhalation carcinogenicity study of GMA in mice conducted in Japan. This study identified statistically significant pathological endpoints and employed benchmark dose (BMD) analysis to evaluate meaningful endpoints, focusing on those with the lowest benchmark dose lower bound values. The final recommendations were optimized using Bayesian model averaging (BMA) methodology to establish appropriate OELs.

Results: Our analysis recommends a time-weighted average allowable concentration of 0.01 ppm for GMA, which aligns with international standards established by the European Chemicals Agency (0.016 ppm), Japan Society for Occupational Health (0.012 ppm), and American Conference of Governmental Industrial Hygienists (0.01 ppm).

Conclusion: The combined application of BMD and BMA methodologies represents a scientifically robust approach for deriving points of departure in risk assessment. These evidence-based OELs are essential for effective occupational hazard management and worker health protection.

Glycidyl methacrylate (GMA) serves as a crucial industrial component in composite and epoxy polymer manufacturing and is classified as a high-volume chemical by the Organization for Economic Cooperation and Development screening information dataset (*1*). Following the publication of a 104-week inhalation carcinogenicity study in mice by the Japan Society for Occupational Health (JSOH) in 2015, JSOH (*2*) established an occupational exposure limit of 0.01 ppm for GMA (0.06 mg/m^3, with conversion factors at 25 °C and 760 torr: 1 ppm=5.81 mg/m^3; 1 mg/m^3=0.172 ppm). Subsequently, in 2019, the International Agency for Research on Cancer (IARC) (*2*) classified GMA as a "probable human carcinogen" (Class 2A) based on this study and additional toxicological evidence. This classification prompted JSOH (*3*), the European Chemicals Agency (ECHA)(*4*), and the American Conference of Governmental Industrial Hygienists (*5*) to revise their respective occupational exposure limits (OELs) for GMA to 0.016 ppm and 0.01 ppm. In contrast, China's current occupational exposure limit for GMA remains at a maximum permissible concentration of 5 mg/m^3 (*5*), with no established time-weighted average concentration (PC-TWA). This standard has remained unchanged for over three decades, creating a significant disparity between Chinese regulations and those of Europe, the United States, and Japan.

The regulatory framework for OELs in China differs fundamentally from international standards, as Chinese standards are primarily mandatory rather than recommended guidelines. Given China's specific industrial context, there is an urgent need to develop and revise the OELs for GMA to align with contemporary scientific understanding and international best practices.

The benchmark dose (BMD), introduced by Crump (*6*), represents the statistically derived lower confidence limit of the dose that produces a predetermined benchmark dose response (BMR, typically 1%–10%). This methodology was developed to address significant limitations inherent in the traditional no observed adverse effect level (NOAEL) approach. The scientific committee (SC) endorses the BMD approach as scientifically superior to the NOAEL method for determining a point of departure (PoD)(*7*). In 2017,

the European Food Safety Authority (EFSA)(8) published updated guidance recommending model averaging as the preferred methodology for calculating BMD confidence intervals. The SC's updated guidance reaffirms that the BMD approach, particularly model averaging, should be the primary method for deriving PoDs from critical dose-response data when establishing health-based guidance values and margins of exposure.

This investigation aims to establish scientifically robust OELs for GMA through the application of BMD analysis and Bayesian model averaging (BMA) techniques. Our methodology employs BMD analysis to identify potential toxic effect endpoints for GMA, selecting those with the lowest benchmark dose lower bound ($BMDL_{10}$) as critical effects. To enhance the precision and reliability of GMA risk assessment, this study further refined these critical effect outcomes using BMA.

METHODS

Dataset and Endpoint Selection

This investigation utilized data from a 104-week inhalation carcinogenicity study conducted in Japan. The experimental design comprised four groups: three treatment groups and one control group, with 50 female and 50 male rats per group, totaling 400 animals. Subjects were exposed to GMA via inhalation for 6 hours daily, 5 days weekly, throughout the 104-week period. The administered concentrations were 0 (control), 0.6, 2.5, and 10 ppm for both sexes. Detailed study information is accessible at https://anzeninfo.mhlw.go.jp/user/anzen/kag/pdf/gan/0795MAIN.pdf.

Given the absence of definitive human epidemiological evidence for GMA exposure, with existing case reports and occupational investigations lacking precise exposure quantification and being confounded by other potential sensitizing agents, these studies were deemed unsuitable for OEL determination. The selected study adhered to Good Laboratory Practice standards, featured appropriate duration, and employed the relevant exposure route (inhalation), as acknowledged in IARC's assessment. Furthermore, this study serves as the foundational evidence for GMA OELs established by JSOH, ECHA, and other regulatory bodies, validating its selection as the primary toxicological evidence for establishing GMA OELs.

For OEL assessment, chronic toxicity and carcinogenicity were identified as the primary critical effects of GMA. The study results were systematically analyzed, incorporating various uncertainty factors and categorizing endpoints into non-neoplastic and neoplastic lesions, with stratification by sex. Carcinogenicity outcomes were classified by overall and terminal rates, all of which were incorporated into the BMD analysis. Only endpoints demonstrating statistical significance ($P<0.05$) were included in the analysis.

BMD Modeling

The BMD methodology employs statistical models to estimate toxic response probabilities at specified doses, facilitating the identification of dose-response relationships and determination of lower safe doses. Using BMDS software (version 3.3.2, EPA, the United States), this study analyzed statistically significant endpoints ($P\leq0.05$) using nine models: Dichotomous Hill, Gamma, Log-Logistic, Multistage Degree, Weibull, Logistic, Log-Probit, Probit, and Quantal Linear. The BMR level was set at 0.1, with a confidence level of 0.95. The endpoint yielding the lowest $BMDL_{10}$ was selected as the BMD for GMA. Model fit assessment incorporated goodness-of-fit analysis, statistical testing, residual analysis, Akaike information criterion (AIC) value evaluation, P value examination, and nested testing.

BMA and Determining PoD

This study utilized BMDS software for modeling key effects. The software assigns prior probabilities to each model based on model selection criteria, with equal default weights assigned to all models. Using binomial sampling for dichotomous endpoints and Normal or Lognormal distributions for continuous data, the software employs Laplace approximation to correct prior density. It then performs BMD estimation through maximum a posteriori probability estimation, computes posterior probabilities across multiple models, and assigns differential weights for model averaging calculations. Bayesian model averaging enhances estimation accuracy by combining results from multiple models. The incorporation of prior information substantially reduces BMD estimation uncertainty and prevents the selection of extreme models that might occur when relying solely on AIC values. By comprehensively evaluating all candidate models, this approach minimizes model

selection bias, thereby improving result accuracy and calculation reliability (*9*).

A PoD represents the dose at which an adverse effect manifests following specific exposure, whether determined empirically or through dose-response modeling (*10*). In our analysis, we employed BMA to assign differential weights across models for averaging calculations and selected the $BMDL_{10}$ as the PoD, following EFSA and EPA recommendations for quantal data.

Application of Uncertainty Factors and Calculation of OEL

Our analysis incorporated uncertainty factors (UFs) for interspecies and intraspecies differences, along with effect severity. This study applied an interspecies factor of 2.5 and, following ECHA recommendations, an intraspecies factor of 5 for worker populations (compared to 10 for general populations) when establishing derived ineffective response levels. For GMA-induced non-neoplastic lesions, which are reversible, this study applied an uncertainty factor of 1. However, given the severity of GMA-induced neoplastic lesions, this study implemented an uncertainty factor of 10.

The final PoD serves as the basis for OEL calculation. Using equal prior probabilities for all models, this study derived GMA OEL values using the following equation:

$$OEL = PoD / UFs$$

where, OEL means occupational exposure limit (ppm); PoD means point of departure; UFs means uncertainty factors.

RESULTS

BMD Analysis Results

Analysis of non-neoplastic lesions in male mice revealed 10 endpoints with $BMDL_{10}$ values ranging from 0.100 to 8.157 ppm. The respiratory metaplasia of the nasal cavity olfactory epithelium yielded the lowest BMDL (0.103 ppm), with optimal fit achieved using the Log-Probit model. In female mice, nine non-neoplastic endpoints produced $BMDL_{10}$ values between 0.077 and 6.825 ppm, with nasopharyngeal eosinophilic change showing the lowest $BMDL_{10}$ (0.077 ppm) using the Dichotomous Hill model. Detailed BMD information is presented in Table 1.

Analysis of tumorigenic endpoints revealed 16 distinct endpoints in male mice, with $BMDL_{10}$ values ranging from 0.756 to 10.197 ppm. The terminal rate of nasal cavity hemangioma demonstrated the lowest $BMDL_{10}$, best fitted by the Dichotomous Hill model. Female mice exhibited 16 neoplastic endpoints with $BMDL_{10}$ values ranging from 0.791 to 7.434 ppm, with uterine histiocytic sarcoma showing the lowest $BMDL_{10}$, optimally fitted using the Log-Logistic model. Table 2 summarizes these findings, which identify the respiratory system as the primary target organ for GMA toxicity in female rats.

BMA Results

Figure 1 illustrates the dose-response relationships derived from Bayesian model averaging across different endpoints. Table 3 presents the posterior probabilities and BMD values post-model averaging. The model averaging approach, which incorporated all viable alternative models while excluding extreme cases, yielded more robust results than classical single-model analysis. For male rats' olfactory epithelial nasal cavity respiratory metaplasia, the Multistage, Quantal Linear, and Weibull models demonstrated superior fit and significantly influenced $BMDL_{10}$ calculations, receiving greater computational weight and yielding a model-averaged $BMDL_{10}$ of 0.118 ppm. Female rats' nasopharyngeal eosinophilia change was best characterized by the Multistage, Quantal Linear, and Log-Logistic models, producing a $BMDL_{10}$ of 0.157 ppm. For mice carcinogenicity endpoints, the Probit, Multistage, and Quantal Linear models provided optimal fit, yielding $BMDL_{10}$ values of 1.733 and 1.081 ppm for males and females, respectively. The lower PoD for non-carcinogenic effects compared to carcinogenic effects indicates that intranasal lesions represent the most sensitive endpoint for GMA inhalation exposure. Application of UFs to the model-averaged results produced OEL values of 0.0094, 0.0126, 0.0139, and 0.0086 ppm, aligning with established limits in the EU (0.016 ppm), Japan (0.012 ppm), and the US (0.01 ppm). Current evidence supports 0.01 ppm as a protective PC-TWA for occupational GMA exposure.

DISCUSSION

This study employed BMD analysis and BMA for GMA risk assessment, utilizing animal studies to identify primary sites of toxic effects. The results demonstrated that GMA's principal adverse effects

TABLE 1. Benchmark dose analysis results of non-neoplastic lesions in mice.

Endpoints	Sex	BMR	Recommended model	*P*	AIC	BMD (ppm)	$BMDL_{10}$ (ppm)
Death	Male	0.1	Log-logistic	0.243	265.728	0.964	0.441
	Female	0.1	–	<0.1	–	–	–
Nasal cavity angiectasis	Male	0.1	Quantal linear	0.968	34.937	16.821	8.157
	Female	0.1	Weibull	1.000	42.496	9.803	6.825
Nasal cavity eosinophilic change: olfactory epithelium	Male	0.1	Log-logistic	0.354	174.269	3.732	2.000
	Female	0.1	Weibull	0.485	221.871	8.783	2.561
Nasal cavity eosinophilic change: respiratory epithelium	Male	0.1	Log-probit	0.578	218.022	1.308	0.435
	Female	0.1	–	<0.1	–	–	–
Nasal cavity respiratory metaplasia: olfactory epithelium	Male	0.1	Log-probit	0.704	189.927	0.256	0.103
	Female	0.1	–	<0.1	–	–	–
Nasal cavity hyperplasia: transitional epithelium	Male	0.1	Multistage degree 3	0.977	86.385	4.505	3.048
	Female		Quantal linear	0.908	49.388	9.380	5.338
Nasal cavity regeneration: respiratory epithelium	Male	0.1	Multistage degree 1	0.730	96.060	3.1808	2.232
	Female	0.1	Gamma	1.000	121.602	1.929	1.166
Nasopharynx eosinophilic change	Male	0.1	Weibull	0.466	141.740	9.471	2.763
	Female	0.1	Dichotomous hill	0.224	227.750	0.294	0.077
Nasal cavity inflammation: respiration epithelium	Male	0.1	Multistage degree 2	0.994	67.035	6.171	4.340
	Female	0.1	–	<0.1	–	–	–
Nasal cavity respiratory metaplasia: gland	Male	0.1	Multistage degree 2	0.819	195.185	0.261	0.194
	Female	0.1	–	<0.1	–	–	–
Nasal cavity squamous cell metaplasia: respiratory epithelium	Male	0.1	–	<0.1	–	–	–
	Female	0.1	Weibull	1.000	61.295	9.388	5.310
Nasa cavity necrosis: olfactory epithelium	Male	0.1	–	<0.1	–	–	–
	Female	0.1	Weibull	0.365	52.707	9.921	6.430
Uterus nodule	Female	0.1	Multistage degree 3	0.968	222.173	8.403	3.053
Ovary enlarged	Female	0.1	Log-probit	0.334	127.124	7.082	2.002

Note: Maximum multistage degree is 3. "-": due to the goodness-of-fit $P<0.1$, the models are poorly fitted and we do not recommend any of them.

Abbreviation: BMR=benchmark response; AIC=akaike information criterion; BMD=benchmark dose; BMDL=benchmark dose lower confidence limit.

manifest at initial exposure sites, specifically the foregut following oral exposure and respiratory tract after inhalation exposure. Chronic GMA exposure in mice induced carcinogenic effects, evidenced by increased tumor incidence in multiple sites including the nasal cavity, lungs, stomach, and uterus. Animal studies (*3*) have also established GMA's reproductive toxicity, while several case reports (*11*–*12*) have documented allergic reactions in humans exposed to GMA.

The BMD approach demonstrates superior sensitivity compared to the NOAELs/LOAELs methodology, ensuring comprehensive identification of potentially sensitive endpoints for risk assessment. Our analysis yielded $BMDL_{10}$ values of 0.103 and 0.077 ppm as general toxicity PoDs, substantially lower than the NOAEL/LOAEL-derived PoD (0.6 ppm). These results produced OEL values (0.01 ppm) slightly below ECHA's 8h-TWA (0.016 ppm) and JSOH's OEL-M (0.012 ppm). The BMD approach offers distinct advantages: it transcends experimental dose limitations, shows reduced sensitivity to dose spacing, and incorporates both dose-response curve characteristics and statistical uncertainties from data quality. When statistical power is constrained by limited data points or high variability, the BMD approach provides more robust conclusions by considering the complete dose-response curve and addressing statistical limitations more effectively than NOAEL. Consequently, our derived PoD incorporates more comprehensive information and better reflects GMA's actual toxic

TABLE 2. Benchmark dose analysis results of neoplastic lesions in mice.

Site	Tumor	Overall rates			Terminal rates		
		Recommended model	$BMDL_{10}$	AIC	Recommended model	$BMDL_{10}$	AIC
Male							
Nasal cavity	Adenoma	Weibull	9.119	24.697	Weibull	4.612	13.483
	Hemangioma	Dichotomous hill	1.437	72.664	Dichotomous hill	0.756	34.532
	Hemangiosarcoma	Log-probit	4.029	63.888	Weibull	10.197	7.205
	Hemangioma, hemangiosarcoma	Multistage degree 1	2.101	4.815	Dichotomous hill	0.841	36.029
	Hemangioma, hemangiosarcoma, adenoma	Log-probit	2.023	98.715	Quantal linear	1.069	35.197
Lung	Bronchiolar-alveolar adenoma	Weibull	7.626	97.267	Weibull	3.241	45.252
Stomach	Squamous cell papilloma	Weibull	9.245	38.711	Weibull	4.612	13.483
Harderian gland	Adenoma	Log-logistic	4.457	91.079	Log-logistic	1.150	42.070
Female							
Nasal cavity	Hemangioma	Dichotomous hill	1.129	69.193	Multistage degree 1	1.649	22.193
	Hemangiosarcoma	Quantal linear	6.929	40.167	–	–	–
	Hemangioma, hemangiosarcoma	Multistage degree 3	2.797	84.368	Multistage degree 1	1.649	22.193
	Hemangiosarcoma, adenocarcinoma	Quantal linear	6.029	44.961	Weibull	4.947	8.279
	Adenocarcinoma, hemangioma, hemangiosarcoma	Multistage degree 1	2.629	86.773	Weibull	3.973	9.535
Lung	Bronchiolar-alveolar carcinoma	Weibull	7.092	55.751	Weibull	3.973	9.534
	Bronchiolar-alveolar adenoma, bronchiolar-alveolar carcinoma	Weibull	7.434	10.416	Log-logistic	1.447	47.627
Uterus	Histiocytic sarcoma	Logistic	3.448	227.296	Log-logistic	0.791	2.140
Harderian gland	Adenoma	Log-logistic	6.704	68.333	–	–	–

Note: Overall rates represent the number of tumor-bearing animals relative to total animals examined at the site. Terminal rates indicate tumor incidence at terminal kill. Maximum multistage degree is 3. "–": due to the goodness-of-fit $P<0.1$, the models are poorly fitted and we do not recommend any of them.
Abbreviation: AIC=akaike information criterion; BMDL=benchmark dose lower confidence limit.

effect profile. The alignment of final OEL values, despite different methodological approaches (ECHA and JSOH applying a 10-fold uncertainty factor for LOAEL to NOAEL extrapolation), validates 0.01 ppm as a reasonable PC-TWA for GMA under current evidence.

The BMA methodology employed here utilizes a comprehensive dose-response model with weighted mathematical components to calculate model means, generating reliable estimates and confidence intervals. This approach leverages prior information to enhance parameter estimation precision while accounting for model uncertainty. Traditional single-model statistical approaches risk introducing "model selection error (*13*)," which can be mitigated through model averaging techniques (*14*). The method's inherent capacity to address model uncertainty provides enhanced flexibility and reliability in both model selection and parameter estimation (*9*), ultimately establishing a more robust foundation for risk quantification (*7*).

This study was subject to some limitations. The selection of appropriate prior distributions for BMA in BMDS software presents challenges, particularly without comprehensive background knowledge. Furthermore, our reliance on animal test data for OEL recommendations may not fully reflect actual plant operational conditions, necessitating additional field studies for developing more practical OELs.

Our recommended PC-TWA of 0.01 ppm for GMA represents a conservative approach to worker protection. According to ECHA's (*4*) T25 methodology dose-response relationship, this concentration corresponds to approximately 40 additional cancer cases per 100,000 exposed workers. Given GMA's demonstrated high sensitization potential in animal tests and case reports, dermal exposure remains an important area for future research. While modern closed-system manufacturing processes under controlled conditions (*2*) facilitate maintaining low workplace GMA concentrations, current national OELs require revision. Specifically, establishing PC-

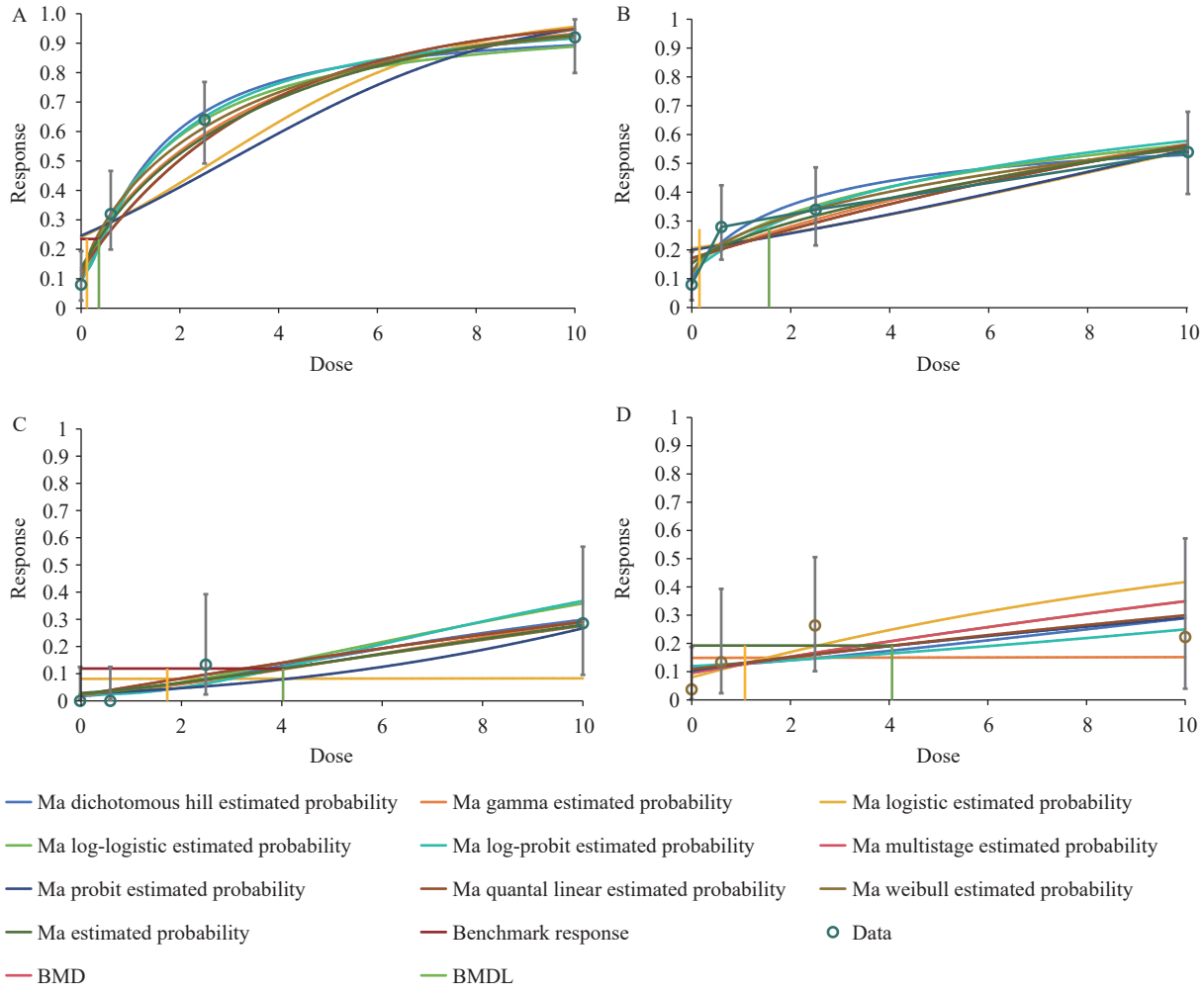

FIGURE 1. Dose-response relationships of bayesian modeling average. (A) Male mice nasal cavity respiratory metaplasia: olfactory epithelium. (B) Female mice Nasopharynx eosinophilic change. (C) Male mice nasal cavity hemangioma. (D) Female mice uterus histiocytic sarcoma.
Abbreviation: Ma=model average; BMD=benchmark dose; BMDL=benchmark dose lower confidence limit.

TABLE 3. Bayesian model averaging results for minimal effect endpoints in mice benchmark dose analysis.

Model	Male mice nasal cavity respiratory metaplasia: olfactory epithelium		Female mice nasopharynx eosinophilic change		Male mice nasal cavity hemangioma		Female mice uterus histiocytic sarcoma	
	Posterior probability	$BMDL_{10}$	Posterior probability	$BMDL_{10}$	Posterior probability	$BMDL_{10}$	Posterior probability	$BMDL_{10}$
Dichotomous hill	0.035	0.119	0.063	0.076	0.155	1.398	–	–
Gamma	0.030	0.064	0.026	0.290	0.061	2.034	0.068	1.248
Logistic	0	0.709	0.041	2.161	0.001	3.714	0.034	6.380
Log-logistic	0.043	0.096	0.092	0.091	0.076	1.575	0.065	0.431
Log-probit	0.023	0.146	0.007	0.176	0.024	1.964	–	–
Multistage	0.391	0.289	0.318	1.112	0.204	1.760	0.274	1.403
Probit	0	0.796	0.061	2.072	0.219	3.994	0.206	3.185
Quantal linear	0.391	0.289	0.318	1.112	0.204	1.760	0.274	1.403
Weibull	0.087	0.053	0.074	0.068	0.057	4.098	0.080	0.518
Model average	–	0.118	–	0.157	–	1.733	–	1.081
OELs value	–	0.0094	–	0.0126	–	0.0139	–	0.0086

Note: Maximum multistage degree is 3. "–": Model not fitted or no data.
Abbreviation: BMDL=benchmark dose lower confidence limit.

TWA and revising maximum allowable concentration are crucial steps toward enhanced worker health protection.

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