

**Current occupational exposure limits for a number of individual dusts.**

DUST	WEL (8-hour TWA) Respirable dust	COMMENTS
Kaolin	2 mg.m <sup>-3</sup>	WEL based on a single study indicating that 2.5 mg.m <sup>-3</sup> respirable dust over 40 years would result in category 1 pneumoconiosis (not necessarily associated with symptoms). Some clinically significant disability was expected from exposure to 5 mg.m <sup>-3</sup> respirable dust for 40 years.  WATCH noted some workers would be more susceptible than others and recommended health surveillance.
Talc	1 mg.m <sup>-3</sup>	WEL based on a single study in talc miners/millers which suggested that exposure to 1 mg.m <sup>-3</sup> of respirable talc over 40 years would not lead to any form of pulmonary toxicity. This value is 6-fold lower than the level producing pulmonary toxicity (including lung tumours) in animals. As it is only the respirable fraction that is of concern for pulmonary toxicity, a separate limit for total inhalable talc was judged unnecessary.
Portland cement	10 mg.m <sup>-3</sup> (inhalable dust) 4 mg.m <sup>-3</sup> (respirable dust)	WATCH first considered this in 1991. Respiratory symptoms were linked with exposure but no dose-response relationship could be determined. An update in 2005 showed that there were still no reliable dose-response data in the literature. The OELs were based on the view that there was no evidence of irritation or other respiratory effects at levels not exceeding 10 mg.m <sup>-3</sup> total inhalable dust and 4 mg.m <sup>-3</sup> respirable dust (both 8-hour TWA).
Aluminium oxides	10 mg.m <sup>-3</sup> (inhalable dust) 4 mg.m <sup>-3</sup> (respirable dust)	WATCH first considered aluminas in 1990 and again in 1991. It was concluded there was no evidence for neurological disease. Lung fibrosis had been reported in workers (debate about confounding with mineral oil). Limited animal inhalation studies revealed no effects on the lungs at 20 to 100 mg.m <sup>-3</sup> . It was concluded that there was no evidence of lung effects at levels below those used in the animal studies. <i>Data on lung effects in humans were very limited.</i>
Pulverised fuel ash	10 mg.m <sup>-3</sup> (inhalable dust)	Few human data. Key study in UK power plant

	4 mg.m <sup>-3</sup> (respirable dust)	workers (Schilling et al 1998). 33 of 208 men who attended X-ray had pulmonary opacities of low grade, ranging from 0/1 to 1/2 (prevalence was related to exposure). Lung function deficits were related to exposure but judged to be of no clinical significance. Prevalences of chronic cough in the low, medium, high exposure (<20 years) and high exposure (>20 years) were 17%, 23%, 11% and 45% respectively. For dyspnoea and wheeze the corresponding values were 7%, 11%, 17% and 35%. Symptoms were viewed with caution as based on subjective reporting. Exposures were 2.52, 5.87 and 8.26 mg.m <sup>-3</sup> for the low, medium and high exposure groups respectively but there was great variability in the data and particle size not indicated.
Carbon black	3.5 mg.m <sup>-3</sup> 7 mg.m <sup>-3</sup> (STEL)	OELs were inherited from ACGIH TLVs that were set to protect against dirtiness. WATCH considered carbon black in 2002. New data showed 40 years exposure to 1, 2, and 3.5 mg.m <sup>-3</sup> (8-hr TWA) (inhalable dust) in non-smoking male would lead to reductions in FEV <sub>1</sub> of 48, 96, and 169 ml respectively. No conclusions were drawn by WATCH regarding the OEL position.
Barium sulphate	10 mg.m <sup>-3</sup> (inhalable dust) 4 mg.m <sup>-3</sup> (respirable dust)	WATCH considered BaSO <sub>4</sub> in 1997. Very little useful data were available. Old reports identified 'baritosis' in workers exposed to large amounts of BaSO <sub>4</sub> but there was no evidence of fibrosis or evidence associating baritosis with ill-health. The available data support the view that no health effects would be anticipated with long-term repeated exposure to 4 mg.m <sup>-3</sup> respirable barium sulphate.
Titanium dioxide	10 mg.m <sup>-3</sup> (inhalable dust) 4 mg.m <sup>-3</sup> (respirable dust)	Original OELs inherited from ACGIH TLVs that were set to minimise the potential for respiratory tract irritation and potential overload of pulmonary air-space architecture and normal clearance mechanisms. Negligible human data were available. WATCH endorsed a detailed hazard assessment of TiO <sub>2</sub> (based on animal data) as part of the low toxicity dusts package. This revealed that nano-sized TiO <sub>2</sub> was more toxic in the lungs than the same mass of larger micron-sized TiO <sub>2</sub> .