Additional comments	The default allocation factor of 20% has been used to account for the fact that the available food exposure data, which suggest that exposure via this route is low, do not generally include information from developing countries, where exposure via this route may be higher, and as potential exposure via inhalation from indoor air resulting from use of dichlorvos as a domestic insecticide is unknown
	Guidance on interpreting the health-based value and deciding when to monitor can be found in section 8.5.3
Assessment date	2016
Principal references	WHO (2012). *Pesticide residues in food – 2011 evaluations* WHO (2016). *Dichlorvos in drinking-water*

* When a formal guideline value is not established, a "health-based value" may be determined in order to provide guidance to Member States when there is reason for local concern. Establishing a formal guideline value for such substances may encourage Member States to incorporate a value into their national standards when this may be unnecessary.

** For more information on acute health-based values, see section 8.7.5.

As with other organophosphorus insecticides, the inhibition of cholinesterase activity, causing neurotoxicity, is the most sensitive toxicological end-point following acute or repeated exposures to dichlorvos. Dichlorvos is unlikely to be genotoxic in vivo or to pose a carcinogenic risk to humans. Some reproductive toxicity has been observed in rats, but dichlorvos was not found to cause developmental toxicity or to be teratogenic.

Dicofol

Dicofol (CAS No. 115-32-2) is an organochlorine acaricide that has been registered for broad-spectrum contact, non-systemic control of plant-eating mites in cotton, tea and a wide variety of fruit, vegetable and ornamental crops. Products containing dicofol, which is manufactured from DDT, are being phased out in the USA and are no longer approved for use in the European Union. Dicofol is unlikely to reach water, but may do so if bound to particulate matter subject to runoff. Dicofol is only slightly soluble in water and binds strongly to soil. There are few data on the occurrence of dicofol in water. Exposure from food varies widely, depending on local circumstances and usage. Dicofol has been proposed as a persistent organic pollutant under the Stockholm Convention.

Reason for not establishing a guideline value	Unlikely to be found in drinking-water or drinking-water sources*
Health-based value**	0.01 mg/l
Acute health-based value***	6 mg/l
Occurrence	Not detected in limited groundwater monitoring
ADI	0–0.002 mg/kg bw, based on a NOAEL of 0.22 mg/kg bw per day for histopathological changes in the liver and adrenal gland in a 2-year toxicity and carcinogenicity study in rats and application of a safety factor of 100

ARfD	0.2 mg/kg bw, based on a NOAEL of 15 mg/kg bw for decreased body weight and decreased feed intake in an acute neurotoxicity study in rats and application of a safety factor of 100
Limit of detection	Solvent extraction followed by GC-ECD may be effective (limit of quantification 5 ng/l)
Treatment performance	Should be removed by adsorption onto activated carbon, and any dicofol adsorbed onto particulate matter would likely be removed during coagulation
Health-based value derivation	
• allocation to water	20% of the upper bound of the ADI
• weight	60 kg adult
• consumption	2 litres/day
Acute health-based value derivation	
• allocation to water	100% of the ARfD
• weight	60 kg adult
• consumption	2 litres/day
Additional comments	The default allocation factor of 20% has been used to account for the fact that the available food exposure data, which suggest that exposure via this route is low, do not generally include information from developing countries, where exposure via this route may be higher
	Guidance on interpreting the health-based value and deciding when to monitor can be found in section 8.5.3
Assessment date	2016
Principal references	WHO (2012). *Pesticide residues in food – 2011 evaluations* WHO (2016). *Dicofol in drinking-water*

* Although dicofol does not fulfil one of the three criteria for evaluation in the Guidelines, a background document has been prepared, and a health-based value has been established, in response to a request from Member States for guidance.

** When a formal guideline value is not established, a "health-based value" may be determined in order to provide guidance to Member States when there is reason for local concern. Establishing a formal guideline value for such substances may encourage Member States to incorporate a value into their national standards when this may be unnecessary.

*** For more information on acute health-based values, see section 8.7.5.

The primary effects of dicofol after short- or long-term exposure of experimental animals were body weight reduction associated with decreased feed intake, and increased liver weight accompanied by changes in liver enzyme activities. Dicofol caused liver tumours in male mice at doses associated with significant enzyme induction and liver hypertrophy. However, on the basis of the absence of genotoxicity in an adequate range of in vitro genotoxicity and in vivo chromosomal aberration tests, the absence of carcinogenic effects in rats and the expectation that the adenomas present in mice will exhibit a threshold, dicofol is unlikely to pose a carcinogenic risk to humans at anticipated dietary exposure levels. There is a margin of 20 000 between the upper bound of the ADI and the LOAEL for liver adenomas in the male mouse.