

*Short-term dietary exposure*

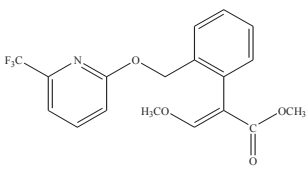
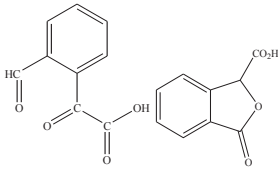
The International Estimated Short term Intake (IESTI) for bentazone was calculated for all food commodities (and their processed fractions) for which recommendations were made by the 2013 Meeting and for which consumption data were available. The results are shown in Annex 4 of the 2016 Report.

For bentazone the IESTI represented 0-1% of the ARfD (0.5 mg/kg bw) for the general population and 0-3% of the ARfD for children. On the basis of information provided to the Meeting it was concluded that the short-term exposure to residues of bentazone, when used in ways that have been considered by the JMPR, is unlikely to present a public health concern.

**3.2.2 Picoxystrobin (258)*****Background***

Picoxystrobin was evaluated as a new compound by the 2012 JMPR for toxicology and residues. The 2012 JMPR established an ADI of 0-0.09 mg/kg bw for picoxystrobin and an ARfD of 0.09 mg/kg bw.

The 2012 JMPR proposed a residue definition for enforcement of picoxystrobin and estimated a number of maximum residue levels. However, the 2012 JMPR was unable to conclude on the toxicological relevance of two metabolites IN-H8612 and 2-(2-formylphenyl)-2-oxoacetic acid tentatively identified in plant metabolism studies, for which IEDIs were above the threshold of toxicological concern of 0.15 µg/person/day for compounds with alerts for genotoxicity. As a result, it was not possible to propose a residue definition for dietary risk assessment or calculate dietary intakes, and maximum residue levels were not recommended.

Common names	Chemical name	Structure
Picoxystrobin, ZA 1963, DPX-YT669	Methyl (E)-3-methoxy-2-[2-(6-trifluoromethyl-2-pyridyloxymethyl)-phenyl]acrylate	
IN-H8612	1,3-Dihydro-3-oxoisobenzofuran-1-carboxylic acid	
	2-(2-Formylphenyl)-2-oxoacetic acid	

The 2013 JMPR received additional toxicological data (a mouse micronucleus study) for IN-H8612 which showed no evidence of genotoxicity. Conservative estimates for chronic and acute exposure to IN-H8612 were both below the relevant TTC values for Cramer class III compounds with no evidence of genotoxicity. The 2013 JMPR concluded that there was no concern for dietary exposure to IN-H8612. However, no toxicological data were submitted for 2-(2-formylphenyl)-2-oxoacetic acid, as the compound was unable to be synthesised in sufficient amounts. Although argument was provided that levels in soya beans were likely to be extremely low, the 2013 JMPR concluded that genotoxicity data or additional residues information would be required to allow further evaluation of 2-(2-formylphenyl)-2-oxoacetic acid.

***Assessment of new data***

During the current Meeting, the FAO panel received a new metabolism study for picoxystrobin in soya bean intended to address the concerns regarding 2-(2-formylphenyl)-2-oxoacetic acid, which was reported as a metabolite in mature seed in the soya bean metabolism study considered by the 2012 JMPR.

A preliminary evaluation of the new study indicates that the metabolic pathway for picoxystrobin in soya beans is broadly similar to that observed in the earlier study. Metabolites identified in the new soya bean study were mostly also identified in the plant metabolism studies provided to the 2012 JMPR (for wheat, canola, soya bean and rotational crops).

The 2-(2-formylphenyl)-2-oxoacetic acid metabolite was not identified in the new soya bean study. The Meeting noted that IN-H8612 was a significant metabolite in soya bean matrices in the new study, particularly mature seed. Further, IN-H8612 is a structural isomer of 2-(2-formylphenyl)-2-oxo-acetic acid, and in chromatography conducted for the new metabolism study, IN-H8612 was reported as eluting as two peaks.

***Conclusion***

The Meeting concluded that further information was required on the possible interconversion of IN-H8612 and 2-(2-formylphenyl)-2-oxoacetic acid, possibly through ring-chain tautomerism.

