5.17 FENVALERATE (119)

TOXICOLOGY

Fenvalerate is the ISO-approved common name for (*RS*)-α-cyano-3-phenoxybenzyl (*RS*)-2-(4-chlorophenyl)-3-methylbutyrate (IUPAC), for which the CAS number is 51630-58-1. It is a broad-spectrum pyrethroid with neurotoxic effects on insect pests.

Fenvalerate is a racemic mixture of four stereoisomers ([2*S*,α*S*], [2*S*,α*R*], [*2R*,α*S*] and [2*R*,α*R*]) found in approximately equal proportions owing to the presence of two chiral centres. One of these four chiral isomers, esfenvalerate, is the [2*S*,α*S*] or A-α isomer and has been developed separately in the knowledge that it was the biologically active component of the fenvalerate racemic mixture. Fenvalerate is classified, according to its structure, as a type II pyrethroid.

Fenvalerate was reviewed by JMPR on four previous occasions. Temporary ADIs of 0–0.06, 0–0.007 and 0–0.02 mg/kg bw were established by the Meeting in 1979, 1981 and 1984, respectively, and an ADI of 0–0.02 mg/kg bw was established by the Meeting in 1986.

Fenvalerate is being reviewed by the present Meeting at the request of CCPR.

As the compound was not supported by a company that would provide toxicological studies for review, access to the toxicological studies for the current evaluation was provided by the USEPA. The evaluation of fenvalerate was based on the previous reviews by JMPR, an IPCS evaluation, the esfenvalerate review by JMPR, studies submitted to the USEPA and published studies from the open literature.

Most of the studies do not comply with GLP, as most of the data were generated before the implementation of GLP regulations. Overall, the Meeting considered that the database was adequate for the risk assessment.

Biochemical aspects

Metabolism studies have been conducted in rats, mice and dogs using ^{14}C-labelled esfenvalerate and fenvalerate. Fenvalerate was rapidly absorbed in these mammals, widely distributed to organs and tissues and rapidly metabolized. Excretion of an isomeric mixture of fenvalerate and esfenvalerate was very rapid in rats and mice, with 63–94% of the administered label being excreted within 1 day after oral dosing. Approximately equal quantities of radioactivity were eliminated in the urine and faeces. Tissue residue concentrations were generally very low, with residue levels being higher in mice than in other species. Fenvalerate and its esters concentrate in adipose tissue, adrenal gland, intestinal mucosa, skin and hair. The cyano moiety remains in the body (particularly in skin and hair) longer than other components. A placental transfer study of fenvalerate and esfenvalerate in rats indicated that there was virtually no transfer of radioactivity from maternal blood to the fetus and no evidence of accumulation in the fetus or amniotic fluid. In dogs administered fenvalerate orally, the total recovery of radioactivity in excreta was less than that in mice or rats, but the elimination half-life was similar to those found in the rodents (0.5–0.6 day).

Fenvalerate undergoes several major metabolic reactions, including cleavage of the ester linkage, hydroxylation in the acid and alcohol moieties and conversion of the cyano group to thiocyanate and carbon dioxide. The pattern of hydroxylation was different in rats and dogs, and the glycine conjugate, 3-phenoxybenzylglycine, was the major conjugate of the alcohol moiety in dogs, whereas it was a minor one in rats. The proportions of glucuronides formed at the acid moiety and its hydroxy derivatives were also greater in dogs. There were no major sex differences in the metabolism of fenvalerate.

In a 28-day feeding study in mice, the major metabolites in the liver and kidney of animals fed [^{14}C-chlorophenyl]esfenvalerate and [^{14}C-chlorophenyl]fenvalerate were chlorophenylisovaleric acid (CPIA) and the hydroxylated derivative of CPIA. These disappeared after administration of

untreated diets. In addition, "CPIA-cholesterol ester" was found in mice fed [^{14}C-chlorophenyl]fenvalerate, but not in mice fed [^{14}C-chlorophenyl]esfenvalerate.

Photolytic degradation on plants can produce a decarboxylated fenvalerate not known to occur in mammals.

Toxicological data

Clinical signs, such as choreoathetosis (coarse tremors progressing to sinuous writhing), sedation, salivation, dyspnoea and/or clonic seizures and sometimes body tremors and prostration, were observed in acute studies. These signs, which are typical of a type II pyrethroid, have been observed in various mammalian species tested with either esfenvalerate or fenvalerate and are characteristic of a strong excitatory action on the nervous system.

The oral LD_{50} in rats was greater than or equal to 451 mg/kg bw (vehicle dependent), whereas in mice, it was greater than or equal to 100 mg/kg bw (vehicle dependent). The dermal LD_{50} in rats was 5000 mg/kg bw. The inhalation LC_{50} in rats was greater than 101 mg/m^3 (3-hour exposure). No data are available on skin and eye irritation or skin sensitization. However, data are available on esfenvalerate, which is not irritating to the skin and minimally irritating to the unwashed eyes of rabbits. It was a skin sensitizer in guinea-pigs using the Magnusson & Kligman maximization test, but was not a sensitizer using the Buehler test method.

In a 90-day dietary toxicity study of fenvalerate in rats, the NOAEL was 125 ppm (equivalent to 12.5 mg/kg bw per day), based on increased relative kidney weight at 500 ppm (equivalent to 50 mg/kg bw per day). Although survival was reduced at 2000 ppm (equivalent to 200 mg/kg bw per day), the highest dose tested in this study, no degeneration of the sciatic nerve was observed.

The overall NOAEL in dogs fed fenvalerate for 3 or 6 months was 250 ppm (equivalent to 18.7 mg/kg bw per day), based on hepatic multifocal microgranulomas and histiocytic infiltration of the mesenteric lymph nodes in females at 500 ppm (equivalent to 35.5 mg/kg bw per day).

Three 18-month dietary studies of fenvalerate toxicity and carcinogenicity in mice have been reported. In the first study, the NOAEL was 300 ppm (equivalent to 15 mg/kg bw per day), based on reduced body weight gain, clinical signs of hyperactivity, mortality, clinical chemistry changes and microscopic changes in liver, kidney and mesenteric lymph nodes observed at 1000 ppm (equivalent to 50 mg/kg bw per day). In the second study, the NOAEL was 10 ppm (equivalent to 1.5 mg/kg bw per day), based on an increased incidence of microgranulomatous changes in mesenteric lymph nodes and other visceral and peripheral lymph nodes observed at 50 ppm (equivalent to 7.5 mg/kg bw per day). In the third study, the NOAEL was 30 ppm (equal to 3.48 mg/kg bw per day), based on slightly decreased erythrocyte counts, decreased serum glucose concentrations and increased histiocytic infiltration (liver and lymph nodes) and granulomatous changes in the liver and lymph nodes at 100 ppm (equal to 12.3 mg/kg bw per day). The overall NOAEL for long-term dietary exposure of mice to fenvalerate was 30 ppm (equal to 3.48 mg/kg bw per day), based on histopathology in various organs, but most consistently in lymph nodes, at 50 ppm (equivalent to 7.5 mg/kg bw per day).

There was no evidence for carcinogenicity from any of these studies in mice at fenvalerate concentrations up to 3000 ppm (equivalent to 450 mg/kg bw per day).

In a published oral gavage study of carcinogenicity of fenvalerate in mice, no evidence for carcinogenicity was observed at the highest dose of 80 mg/kg bw per day.

Four chronic toxicity and carcinogenicity studies of fenvalerate have been performed in rats, but two of the studies are inadequate for the assessment of carcinogenic potential.

Groups of Sprague-Dawley rats were administered diets containing fenvalerate at either 0 or 1000 ppm (equivalent to 50 mg/kg bw per day) for 2 years. No group difference in the incidence of any specific tumours was reported.

In a second 2-year study in rats (Wistar/SCL strain), there were observations of body weight gain depressions and giant cell infiltration of lymph nodes and adrenals and reticuloendothelial cell proliferation in the lymph nodes at 500 ppm (equivalent to 25 mg/kg bw per day).

The overall NOAEL for long-term toxicity of fenvalerate in rats was 150 ppm (equivalent to 7.5 mg/kg bw per day), on the basis of body weight gain reduction in males and giant cell infiltration of lymph nodes and adrenals and reticuloendothelial cell proliferation in the lymph nodes at 500 ppm (equivalent to 25 mg/kg bw per day). In males, Leydig cell atrophy, Leydig cell hyperplasia and Leydig cell adenomas were significantly increased in some groups, but there was no clear dose–response relationship. Similar to the Fischer 344 strain rats, Leydig cell adenomas are particularly common and variable in the Wistar/SCL strain (which is not to be confused with the Wistar strain, in which incidences of these tumours are low). Consequently, variation in the incidence of this tumour type cannot be used with any confidence in carcinogen evaluation. There was no evidence of carcinogenicity in rats at doses up to 1500 ppm (equivalent to 75 mg/kg bw per day), the highest dose tested.

The Meeting concluded that fenvalerate was not carcinogenic in mice or rats.

Fenvalerate was tested for genotoxicity in an adequate range of assays, both in vitro and in vivo. No evidence of genotoxicity emerged from the in vitro assays, but inconsistent results were obtained in the male mouse dominant lethal assays and the in vivo cytogenetic assays.

Available data allowed the Meeting to conclude that fenvalerate is unlikely to be a deoxyribonucleic acid (DNA) reactive compound, but no firm conclusion could be reached on its in vivo clastogenicity.

On the basis of the absence of carcinogenicity in mice and rats and the absence of DNA reactivity, the Meeting concluded that fenvalerate is unlikely to pose a carcinogenic risk to humans at expected dietary levels.

In a three-generation reproduction study in rats, the NOAEL for parental toxicity was 25 ppm (equivalent to 1.7 mg/kg bw per day), based on reduced mean body weights seen at 250 ppm (equivalent to 16.7 mg/kg bw per day). The NOAEL for reproductive and offspring toxicity was 250 ppm (equivalent to 16.7 mg/kg bw per day), the highest dose tested.

In a developmental toxicity study in mice, the NOAEL for maternal toxicity was 15 mg/kg bw per day, based on irregular respiration, hypersensitivity, tremors and salivation after administration of the compound (first 30–60 minutes after dosing only) seen at 50 mg/kg bw per day. The NOAEL for developmental toxicity was 50 mg/kg bw per day, the highest dose tested.

In a developmental toxicity study in rabbits, the NOAEL for maternal toxicity was 25 mg/kg bw per day, based on reduced body weight of dams seen at 50 mg/kg bw per day. The NOAEL for developmental toxicity was 50 mg/kg bw per day, the highest dose tested.

The Meeting concluded that fenvalerate is not teratogenic in mice or rabbits.

A study of the neurotoxic potential of esfenvalerate and fenvalerate in corn oil was conducted in rats following a single oral gavage dose. The NOAEL was 5 and 20 mg/kg bw for esfenvalerate and fenvalerate, respectively, based on the toxic signs typical of type II pyrethroids. Signs were observed within 2 hours of dosing at 20 and 90 mg/kg bw for esfenvalerate and fenvalerate, respectively.

No histopathological lesions were observed in rats following a single-dose administration of fenvalerate at 200 mg/kg bw. In a separate study, rats were administered fenvalerate orally at dose levels ranging from 0 to 400 mg/kg bw per day for 7 consecutive days. A significant neurological deficit was demonstrated using an inclined plane test (expressed as the angle at which the animals cannot maintain their hold on an inclining plane). In addition to functional deficits, increases in the activity of the lysosomal enzymes β-glucuronidase and β-galactosidase in the posterior tibular nerve and trigeminal ganglia were observed.

Fenvalerate did not cause delayed neuropathy in hens at 1000 mg/kg bw per day for 5 days.

The acute intraperitoneal toxicity study results indicate that fenvalerate metabolites were less toxic than the parent compound fenvalerate.

The major photodegradation product of fenvalerate, decarboxyfenvalerate, or 2-(3-phenoxyphenyl)-3-(4-chlorophenyl)-4-methylpentanenitrile, was evaluated for its acute toxicity;

results indicated that it was less toxic than the parent compound. In a 90-day toxicity study in rats with decarboxyfenvalerate, the NOAEL was 300 ppm (equivalent to 30 mg/kg bw per day), based on reduced body weight gains in males during the first 7 weeks, a decrease in white blood cell count in females, reduced mean corpuscular volume in both sexes, increases in liver and kidney weights, and hepatocellular necrosis at 1000 ppm (equivalent to 100 mg/kg bw per day). In a developmental toxicity study with decarboxyfenvalerate in rats, the NOAEL for maternal toxicity was 300 mg/kg bw per day, based on decreases in body weights and increases in relative liver weights seen at 3000 mg/kg bw per day. The developmental toxicity NOAEL was 3000 mg/kg bw per day, the highest dose tested.

Several published studies are available that evaluate the effects of fenvalerate in mice and rats on sexual maturation, stereotyped and sexual behaviour, hormonal measurements, sperm measurements and changes in various organ weights and histopathology. These studies were conducted following single or multiple oral administrations of fenvalerate. Generally, reproductive parameters and neurobehavioural parameters were affected at about 20 mg/kg bw per day via oral doses.

Observations in humans indicate that fenvalerate causes the transient facial sensations that appear to be common to pyrethroids, particularly those possessing a cyano group.

The Meeting concluded that the existing database on fenvalerate was adequate to characterize the potential hazards to fetuses, infants and children.

Toxicological evaluation

The Meeting reaffirmed the ADI of 0–0.02 mg/kg bw on the basis of a parental systemic toxicity NOAEL of 1.7 mg/kg bw per day observed in the three-generation reproduction study in rats, based on reduced mean body weights seen at 16.7 mg/kg bw per day and using a safety factor of 100. This ADI was supported by the NOAEL of 3.5 mg/kg bw per day observed in the 2-year toxicity and carcinogenicity study in mice, based on the slight decrease in erythrocyte counts, increased histiocytes and granulomatous changes in the liver and lymph nodes (mesenteric, visceral and peripheral) at 7.5 mg/kg bw per day.

The Meeting established an ARfD of 0.2 mg/kg bw on the basis of the NOAEL of 20 mg/kg bw observed in the single oral dose neurotoxicity study in rats, based on clinical signs of toxicity (muscular fibrillation, ataxia, salivation and/or hunched posture) seen at 90 mg/kg bw and using a safety factor of 100. This ARfD was supported by the developmental toxicity study in mice in which the NOAEL was 15 mg/kg bw per day, based on irregular respiration, hypersensitivity, tremors and salivation after administration of the compound (first 30–60 minutes after dosing) seen at 50 mg/kg bw per day.

A toxicological monograph was prepared.

Levels relevant to risk assessment

Species	Study	Effect	NOAEL	LOAEL
Mouse	Eighteen-month studies of toxicity and carcinogenicity[a,b]	Toxicity	30 ppm, equal to 3.5 mg/kg bw per day	50 ppm, equivalent to 7.5 mg/kg bw per day
		Carcinogenicity	3000 ppm, equivalent to 450 mg/kg bw per day[c]	—
	Developmental toxicity study[d]	Maternal toxicity	15 mg/kg bw per day	50 mg/kg bw per day
		Embryo and fetal toxicity	50 mg/kg bw per day[c]	—
Rat	Three-year studies of toxicity and carcinogenicity[a,b]	Toxicity	150 ppm, equivalent to 7.5 mg/kg bw per day	500 ppm, equivalent to 25 mg/kg bw per day

Species	Study	Effect	NOAEL	LOAEL
		Carcinogenicity	1500 ppm, equivalent to 75 mg/kg bw per day[c]	—
	Two-generation study of reproductive toxicity[a]	Reproductive toxicity	250 ppm, equivalent to 16.7 mg/kg bw per day[c]	—
		Parental toxicity	25 ppm, equivalent to 1.7 mg/kg bw per day	250 ppm, equivalent to 16.7 mg/kg bw per day
		Offspring toxicity	250 ppm, equivalent to 16.7 mg/kg bw per day[c]	—
	Neurotoxicity study (single dose)[d]	Neurotoxicity	20 mg/kg bw	90 mg/kg bw
Rabbit	Developmental toxicity study[d]	Maternal toxicity	25 mg/kg bw per day	50 mg/kg bw per day
		Embryo and fetal toxicity	50 mg/kg bw per day[c]	—
Dog	Thirteen-week and 6-month studies of toxicity[a,b]	Toxicity	250 ppm, equal to 18.7 mg/kg bw per day	500 ppm, equal to 35.5 mg/kg bw per day

[a] Dietary administration.

[b] Two or more studies combined.

[c] Highest dose tested.

[d] Gavage administration.

Estimate of acceptable daily intake for humans

0–0.02 mg/kg bw

Estimate of acute reference dose

0.2 mg/kg bw

Information that would be useful for the continued evaluation of the compound

Results from epidemiological, occupational health and other such observational studies of human exposures

Critical end-points for setting guidance values for exposure to fenvalerate

Absorption, distribution, excretion and metabolism in mammals	
Rate and extent of oral absorption	Rapid and complete
Dermal absorption	Not available
Distribution	Widely distributed
Potential for accumulation	None
Rate and extent of excretion	Rapid and complete (half life 0.5–0.6 day)
Metabolism in animals	Extensive
Toxicologically significant compounds in animals, plants and the environment	Parent compound and decarboxylated fenvalerate
Acute toxicity	
Rat, LD_{50}, oral	≥ 451 mg/kg bw (vehicle dependent)
Rat, LD_{50}, dermal	5000 mg/kg bw
Rat, LC_{50}, inhalation	> 101 mg/m^3 (3 h)
Rabbit, dermal irritation	Not available
Rabbit, ocular irritation	Not available
Dermal sensitization	Not available

Short-term studies of toxicity	
Target/critical effect	Nervous system, clinical signs
Lowest relevant oral NOAEL	12.5 mg/kg bw per day
Lowest relevant dermal NOAEL	1000 mg/kg bw per day
Lowest relevant inhalation NOAEC	7 mg/L
Long-term studies of toxicity and carcinogenicity	
Target/critical effect	Multiple target organs
Lowest relevant NOAEL	3.5 mg/kg bw day
Carcinogenicity	Not carcinogenic in mice or rats
Genotoxicity	
	Not DNA reactive, inconsistent results in in vivo cytogenetic assay
Reproductive toxicity	
Target/critical effect	None
Lowest relevant reproductive NOAEL	16.7 mg/kg bw per day (highest dose tested)
Lowest relevant parental NOAEL	1.7 mg/kg bw per day
Lowest relevant offspring NOAEL	16.7 mg/kg bw per day (highest dose tested)
Developmental toxicity	
Target/critical effect	None
Lowest relevant maternal NOAEL	15 mg/kg bw per day
Lowest relevant embryo/fetal NOAEL	50 mg/kg bw per day
Neurotoxicity	
Target/critical effect	Clinical signs typical of type II pyrethroids
Acute neurotoxicity NOAEL	20 mg/kg bw
Subchronic neurotoxicity	No data
Other toxicological studies	
Studies on metabolites	Metabolites/degradation products less toxic than parent
Immunotoxicity	No data
Medical data	
	Transient facial sensations in humans

Summary

	Value	Study	Safety factor
ADI	0–0.02 mg/kg bw	Three-generation reproduction study (rat)	100
ARfD	0.2 mg/kg bw	Single-dose study (rat)	100

RESIDUE AND ANALYTICAL ASPECTS

Fenvalerate is a broad-spectrum pyrethroid insecticide consisting of four isomers (SS, RS, SR and RR) all being present at equal amounts in the technical material (see FAO Specification from 1993). The active substance was evaluated by JMPR several times between 1979 and 1991 for residues and toxicology.

In 2002 esfenvalerate, which is the purified SS-isomer (84%), was evaluated as a new compound by the JMPR for residues as well as for toxicology.

Fenvalerate was scheduled at the Forty-third Session of the CCPR under the periodic review program to be evaluated for toxicology and residues by the 2012 JMPR. However, no data on animal or plant metabolism, the environment, analytical methods or storage stability were submitted to the 2012 JMPR, studies normally required for a periodic re-evaluation of a compound.

The Meeting noted that in 2002 esfenvalerate was evaluated for residues as a new compound. The JMPR Evaluation, presented in a comprehensive form, was primarily based on studies for fenvalerate. It included data on animal metabolism (lactating cows), plant metabolism (apple trees, cabbage, kidney beans, lettuce, soya bean, tomato and wheat), the environment (soil photolysis, aerobic and anaerobic soil metabolism, field dissipation and rotational crops) and livestock feeding

studies (dairy cattle and laying hens). Residue analytical methods and storage stability were reported for esfenvalerate only, however all isomers of fenvalerate were covered within these studies.

The Meeting recognized that basic principles for the evaluation of key studies (animal and plant metabolism, environment, analytical methods and storage stability) have not changed significantly since 2002 and concluded that a re-evaluation of the data would result in an identical recommendation for the definition of fenvalerate residues in plant and animal commodities.

The absence of key studies normally precludes the re-evaluation of an active compound under the periodic review program by JMPR. However, the Meeting noted that its evaluation for esfenvalerate in 2002 still reflects current scientific knowledge and covers fenvalerate also. In view of the closely related chemical composition of fenvalerate and esfenvalerate the Meeting decided, as an exception, to apply its 2002 evaluation of esfenvalerate for decision making to fenvalerate without re-reviewing study data previously reported.

Definition of the residue

The 2012 JMPR considered its evaluation for esfenvalerate in 2002 and confirmed that the recommended definition of the residue for plant and animal commodities is also applicable to fenvalerate.

Definition of the residue (for compliance with MRL and for estimation of dietary intake, plant and animal commodities): *sum of fenvalerate isomers*

The residue is fat-soluble.

Results of supervised residue trials on crops

The Meeting received supervised trial data for applications of fenvalerate on mango and Chinese kale conducted in Thailand. The OECD MRL calculator was used as a tool to assist in the estimation of maximum residue levels from the selected residue data set obtained from the supervised residue trials. As a first step, the Meeting reviewed all relevant factors related to each data set in arriving at a best estimate of the maximum residue level using expert judgement. Then the OECD calculator was employed. If the statistical calculation spreadsheet suggested a different value from that recommended by the Meeting, a brief explanation of the deviation was supplied.

In trials where duplicate field samples from replicated or unreplicated plots were taken at each sampling time and analysed separately, the mean sample was taken as the best estimate of the residue from the plot.

Labels (or translation of labels) were available from Thailand, describing the registered uses of fenvalerate.

Within the periodic review program for fenvalerate only uses for mango and Chinese kale from Thailand were reported to the Meeting and evaluated.

As a consequence the Meeting withdraws its previous recommendations for fenvalerate of 20 mg/kg in alfalfa fodder, 0.1 mg/kg in beans, shells, 1 mg/kg for beans, except broad beans and soya beans, 1 mg/kg for berries and other small fruit, 2 mg/kg for broccoli, 2 mg/kg for Brussels sprouts, 3 mg/kg for cabbage, head, 2 mg/kg for cauliflower, 2 mg/kg for celery, 2 mg/kg (Po) for cereal grains, 2 mg/kg for cherries, 1 mg/kg for Chinese cabbage (type Pak-choi), 2 mg/kg for citrus fruit, 0.2 mg/kg for cotton seed, 0.1 mg/kg for cotton seed oil, crude, 0.1 mg/kg for cotton seed oil, edible, 0.02 mg/kg for cucumber, 0.02 mg/kg for edible offal (mammalian), 10 mg/kg for kale, 5 mg/kg for kiwifruit, 2 mg/kg for lettuce, head, 1 mg/kg (F) for meat (from mammals other than marine mammals), 0.2 mg/kg for melons, except watermelons, 0.1 mg/kg for milks, 5 mg/kg for peach, 0.1 mg/kg for peanut, whole, 0.1 mg/kg for peas, shelled (succulent seeds), 0.5 mg/kg for peppers, sweet (including pimento or pimiento), 2 mg/kg for pome fruit, 0.05 mg/kg for root and tuber vegetables, 0.1 mg/kg for soya bean (dry), 0.5 mg/kg for squash, summer, 0.1 mg/kg for sunflower, seed, 0.1 mg/kg for sweet corn (corn-on-the-cob), 1 mg/kg for tomato, 0.2 mg/kg for tree nuts,

0.5 mg/kg for watermelon, 5 mg/kg (Po) for wheat bran, unprocessed, 0.2 mg/kg (Po) for wheat flour, 2 mg/kg (Po) for wheat wholemeal and 2 mg/kg for winter squash.

The Meeting also withdrawn its previous recommendation given in 2004 for dried chili pepper of 5 mg/kg, which was based on the previously estimated maximum residue level of 0.5 mg/kg for peppers, sweet (including pimento or pimiento) and a default processing factor of 10 for sweet pepper to dried chili pepper derived in 2004.

Mango

GAP for mango in Thailand is foliar spraying at rates of 0.0105 kg ai/hl with a PHI of 7 days. In corresponding field trials conducted in Thailand residues of fenvalerate, sum of isomers in mangos (whole fruit) were (n=4): 0.3, 0.35, 0.43, 0.48 mg/kg.

The Meeting estimated a maximum residue level, an STMR and an HR of 1.5 mg/kg, 0.39 mg/kg and 0.48 mg/kg for fenvalerate in mango, respectively.

Chinese kale

The GAP for Chinese kale in Thailand is foliar applications at rates of 0.021 kg ai/hL with a PHI of 7 days. In corresponding field trials conducted in Thailand residues of fenvalerate, sum of isomers in Chinese kale were (n=6): 0.36, 0.36, 0.7, 0.92, 1.0, 1.8 mg/kg.

The Meeting noted that Chinese kale belongs to the Chinese broccoli commodity (VB 0401) within the Codex Classification of foods and animal feeds.

The Meeting estimated a maximum residue level, an STMR and an HR of 3 mg/kg, 0.81 mg/kg and 1.8 mg/kg for fenvalerate in Chinese broccoli, respectively.

Residues in animal commodities

As mango and Chinese kale are not potential animal feed items, no evaluation of residues in livestock was undertaken.

DIETARY RISK ASSESSMENT

Long-term intake

The evaluation of fenvalerate resulted in recommendations for MRLs and STMR values for mango and broccoli. Where data on consumption were available for the listed food commodities, dietary intakes were calculated for the 13 GEMS/Food Consumption Cluster Diets. The results are shown in Annex 3.

The IEDIs in the thirteen Cluster Diets, based on the estimated STMRs were 0–1% of the maximum ADI (0.02 mg/kg bw). The Meeting concluded that the long-term intake of residues of fenvalerate from uses that have been considered by the JMPR is unlikely to present a public health concern.

Short-term intake

The IESTI for fenvalerate calculated on the basis of the recommendations made by the JMPR represented 0–40% of the ARfD (0.2 mg/kg bw) for children and 0–20% for the general population.

The Meeting concluded that the short-term intake of residues of fenvalerate resulting from uses that have been considered by the JMPR is unlikely to present a public health concern.