identity theft based on the VA sensitive personal information that had been compromised;

[5] Whether private entities are required under Federal law to offer credit protection services to individuals if the same or similar data of the private entities had been similarly compromised; and

[6] The recommendations, if any, concerning the offer of, or benefits to be derived from, credit protection services in this case that are in the risk analysis report.

(Authority: 38 U.S.C. 501, 5724, 5727)

§75.117 Notification.

(a) With respect to individuals found under this subpart by the Secretary to be subject to a reasonable risk for the potential misuse of any sensitive personal information, the Secretary will promptly provide written notification by first-class mail to the individual (or the next of kin if the individual is deceased) at the last known address of the individual. The notification may be sent in one or more mailings as information is available and will include the following:

(1) A brief description of what happened, including the date[s] of the data breach and of its discovery if known;

(2) To the extent possible, a description of the types of personal information that were involved in the data breach (e.g., full name, Social Security number, date of birth, home address, account number, disability code);

(3) A brief description of what the agency is doing to investigate the breach, to mitigate losses, and to protect against any further breach of the data;

(4) Contact procedures for those wishing to ask questions or learn additional information, which will include a toll-free telephone number, an e-mail address, Web site, and/or postal address;

(5) Steps individuals should take to protect themselves from the risk of identity theft, including steps to obtain fraud alerts (alerts of any key changes to such reports and on demand personal access to credit reports and scores), if appropriate, and instruction for obtaining other credit protection services offered under this subpart; and

(6) A statement whether the information was encrypted or protected by other means, when determined such information would be beneficial and would not compromise the security of the system.

In those instances where there is insufficient, or out-of-date contact information that precludes direct written notification to an individual subject to a data breach, a substitute form of notice may be provided, such as a conspicuous posting on the home page of VA’s Web site and notification in major print and broadcast media, including major media in geographic areas where the affected individuals likely reside. Such a notice in media will include a toll-free phone number where an individual can learn whether or not his or her personal information is possibly included in the data breach.

(c) In those cases deemed by the Secretary to require urgency because of possible imminent misuse of sensitive personal information, the Secretary, in addition to notification under paragraph (a) of this section, may provide information to individuals by telephone or other means, as appropriate.

(d) Notwithstanding other provisions in this section, notifications may be delayed upon lawful requests, from other Federal agencies, for the delay of notifications in order to protect data or computer resources from further compromise or to prevent interference with the conduct of an investigation or efforts to recover the data. A lawful request is one made in writing by the entity or VA component responsible for the investigation or data recovery efforts that may be adversely affected by providing notification. Any lawful request for delay in notification must state an estimated date after which the requesting entity believes that notification will not adversely affect the conduct of the investigation or efforts to recover the data. However, any delay should not exacerbate risk or harm to any affected individual(s). Decisions to delay notification should be made by the Secretary.

(Authority: 38 U.S.C. 501, 5724, 5727)

§75.118 Other credit protection services.

(a) With respect to individuals found under this subpart by the Secretary to be subject to a reasonable risk for the potential misuse of any sensitive personal information under this subpart, the Secretary may offer one or more of the following as warranted based on considerations specified in paragraph (b) of this section:

(1) One year of credit monitoring services consisting of automatic daily monitoring of at least 3 relevant credit bureau reports;

(2) Data breach analysis;

(3) Fraud resolution services, including writing dispute letters, initiating fraud alerts and credit freezes, to assist affected individuals to bring matters to resolution; and/or

(4) One year of identity theft insurance with $20,000.00 coverage at $0 deductible.

(b) Consistent with the requirements of the Fair Credit Reporting Act (15 U.S.C. 1681 et seq.) as interpreted and applied by the Federal Trade Commission, the notice to the individual offering other credit protection services will explain how the individual may obtain the services, including the information required to be submitted by the individual to obtain the services, and the time period within which the individual must act to take advantage of the credit protection services offered.

(c) In determining whether any or all of the credit protection services specified in paragraph (a) of this section will be offered to individuals subject to a data breach, the Secretary will consider the following:

(1) The data elements involved;

(2) The number of individuals affected or potentially affected;

(3) The likelihood the sensitive personal information will be or has been made accessible to and usable by unauthorized persons;

(4) The risk of potential harm to the affected individuals; and

(5) The ability to mitigate the risk of harm.

(c) The Secretary will take action to obtain data mining and data breach analyses services, as appropriate, to obtain information relevant for making determinations under this subpart.

(Authority: 38 U.S.C. 501, 5724, 5727)

§75.119 Finality of Secretary determination.

A determination made by the Secretary under this subpart will be a final agency decision.

[FR Doc. 07–3085 Filed 6–20–07; 9:50 am]

BILLING CODE 8320–01–P

ENVIRONMENTAL PROTECTION AGENCY

40 CFR Part 180


Thiamethoxam; Pesticide Tolerance

AGENCY: Environmental Protection Agency (EPA).

ACTION: Final rule.

SUMMARY: This regulation establishes tolerances for combined residues of thiamethoxam and its metabolite (CGA–322704) in or on artichoke, globe; caneberry subgroup 13-A, hop, dried cones; grape; grape, raisin; brassica,
head and stem, subgroup 5-A; brassica, leafy greens, subgroup 5-B; vegetable, leafy, except brassica group 4.

Additionally, tolerance levels for barley, grain; barley, hay and barley, straw will be amended. Interregional Research Project Number 4 (IR-4) and Syngenta Crop Protection Inc. requested these tolerances under the Federal Food, Drug, and Cosmetic Act (FFDCA).

DATES: This regulation is effective June 22, 2007. Objections and requests for hearings must be received on or before August 21, 2007, and must be filed in accordance with the instructions provided in 40 CFR part 178 (see also Unit I.C. of the SUPPLEMENTARY INFORMATION).

ADDRESSES: EPA has established a docket for this action under docket identification (ID) number EPA–HQ–OPP–2006–0523. To access the electronic docket, go to http://www.regulations.gov, select “Advanced Search,” then “Docket Search.” Insert the docket ID number where indicated and select the “Submit” button. Follow the instructions on the regulations.gov web site to view the docket index or access available documents. All documents in the docket are listed in the docket index available in regulations.gov. Although listed in the index, some information is not publicly available, e.g., Confidential Business Information (CBI) or other information whose disclosure is restricted by statute. Certain other material, such as copyrighted material, is not placed on the Internet and will be publicly available only in hard copy form. Publicly available docket materials are available in the electronic docket at http://www.regulations.gov. or, if only available in hard copy, at the OPP Regulatory Public Docket in Rm. S-4400, One Potomac Yard (South Bldg.), 2777 S. Crystal Dr., Arlington, VA. The Docket Facility is open from 8:30 a.m. to 4 p.m., Monday through Friday, normal hours of operation (8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays). The Docket Facility telephone number is (703) 305–5805.

FOR FURTHER INFORMATION CONTACT: Barbara Madden, Registration Division (7505P), Office of Pesticide Programs, Environmental Protection Agency, 1200 Pennsylvania Ave., NW., Washington, DC 20460–0001; telephone number: (703) 305–6463; e-mail address: madden.barbara@epa.gov.

SUPPLEMENTARY INFORMATION:

I. General Information

A. Does this Action Apply to Me?

You may be potentially affected by this action if you are an agricultural producer, food manufacturer, or pesticide manufacturer. Potentially affected entities may include, but are not limited to those engaged in the following activities:

- Crop production (NAICS code 111), e.g., agricultural workers; greenhouse, nursery, and floriculture workers; farmers.
- Animal production (NAICS code 112), e.g., cattle ranchers and farmers, dairy cattle farmers, livestock farmers.
- Food manufacturing (NAICS code 311), e.g., agricultural workers; farmers; greenhouse, nursery, and floriculture workers; ranchers; pesticide applicators.
- Pesticide manufacturing (NAICS code 32532), e.g., agricultural workers; commercial applicators; farmers; greenhouse, nursery, and floriculture workers; residential users.

This listing is not intended to be exhaustive, but rather to provide a guide for readers regarding entities likely to be affected by this action. Other types of entities not listed in this unit could also be affected. The North American Industry Classification System (NAICS) codes have been provided to assist you and others in determining whether this action might apply to certain entities. If you have any questions regarding the applicability of this action to a particular entity, consult the person listed under FOR FURTHER INFORMATION CONTACT.

B. How Can I Access Electronic Copies of this Document?


C. Can I File an Objection or Hearing Request?

Under section 408(g) of the FFDCA, any person may file an objection to any aspect of this regulation and may also request a hearing on those objections. You must file your objection or request a hearing on this regulation in accordance with the instructions provided in 40 CFR part 178. To ensure proper receipt by EPA, you must identify docket ID number EPA–HQ–OPP–2006–0523 in the subject line on the first page of your submission. All requests must be in writing, and must be mailed or delivered to the Hearing Clerk as required by 40 CFR part 178 on or before August 21, 2007.

In addition to filing an objection or hearing request with the Hearing Clerk as described in 40 CFR part 178, please submit a copy of the filing that does not contain any CBI for inclusion in the public docket that is described in ADDRESSES. Information not marked confidential pursuant to 40 CFR part 2 may be disclosed publicly by EPA without prior notice. Submit this copy, identified by docket ID number EPA–HQ–OPP–2006–0523, by one of the following methods:

- Delivery: OPP Regulatory Public Docket (7502P), Environmental Protection Agency, Rm. S-4400, One Potomac Yard (South Bldg.), 2777 S. Crystal Dr., Arlington, VA. Deliveries are only accepted during the Docket’s normal hours of operation (8:30 a.m. to 4 p.m., Monday through Friday, excluding legal holidays). Special arrangements should be made for deliveries of boxed information. The Docket Facility telephone number is (703) 305–5805.

II. Petition for Tolerance

In the Federal Register of July 12, 2006 (71 FR 39316) (FRL–8074–3), EPA issued a notice pursuant to section 408(d)(3) of FFDCA, 21 U.S.C. 346a(d)(3), announcing the filing of pesticide petitions (PP 6E7060, 0F6142, and 9F5051) Interregional Research Project Number 4 (IR-4), 681 U.S. Highway #1 South, North Brunswick, NJ 08902–3390 and Syngenta Crop Protection Inc., P.O. Box 18300, Greensboro, NC 27419–8300. These petitions requested that 40 CFR 180.565 be amended by establishing a tolerance for combined residues of the insecticide, thiamethoxam [3-[(2-chloro-5-thiazolyl)methyl]tetrahydro-5-methyl-N-nitro-4H-1,3,5-oxadiazin-4-imine and its metabolite [N-(2-chloro-thiazol-5-ylmethyl)-N-methyl-N-nitro- guanidine] in or on the following commodities: PP 6E7060: Caneberry subgroup 13-A at 0.30 parts per million (ppm); hops at 0.1 ppm; grape at 0.04 ppm and amend the existing tolerance levels for barley, grain at 0.3 ppm; barley, hay at 0.4 ppm; and barley, straw at 0.4 ppm. PP 0F6142: Grapes at 0.15 ppm; grape, juice at 0.20 ppm; and raisins at 0.30 ppm.
section 18 emergency exemptions that
determined tolerance levels for
pesticide chemical residue, including
result from aggregate exposure to the
pesticide chemical residue in or on a food) only if EPA
established tolerances in
support of this action. EPA has
reviewed the available scientific data
and other relevant information in
support of this action. EPA has
sufficient data to assess the hazards of
and to make a determination on
aggregate exposure for the petitioned-for
tolerances for combined residues of the
insecticide, thiamethoxam [3-[(2-chloro-5-
thiazolyl)methyl]tetrahydro-5-methyl-
N-nitro-4H-1,3,5-oxadiazin-4-imine and
its metabolite [N-(2-chloro-thiazol-5-
yl)methyl]-N-methyl -N'-nitro-
guanidine] on artichoke, globe at 0.45
ppm; barley, grain at 0.30 ppm; barley,
hay at 0.40 ppm; barley, straw at 0.40
ppm; Brassica, head and stem, subgroup
5-A at 4.5; brassica, leafy greens,
subgroup 5-B at 3.0 ppm; caneberry
subgroup 13-A at 0.35 ppm; grape at
0.20 ppm; grape, raisin at 0.30 ppm;
hop, dried cones at 0.10 ppm; and
vegetable, leafy except Brassica, group 4
at 4.0 ppm. EPA’s assessment of
exposures and risks associated with
establishing the tolerance follows.

A. Toxicological Profile
EPA has evaluated the available
pesticide chemical residue in or on a food) only if EPA
determines that the tolerance is “safe.”
pesticide chemical residue, including
all anticipated dietary exposures and all
other exposures for which there is
reliable information.” This includes
occupational exposure. Section

408(b)(2)(C) of the FFDCA requires EPA
to give special consideration to
exposure of infants and children to the
pesticide chemical residue in
establishing a tolerance and to “ensure
that there is a reasonable certainty that
no harm will result to infants and
children from aggregate exposure to the
pesticide chemical residue. . . .” These
provisions were added to the FFDCA by
the Food Quality Protection Act (FQPA) of
1996.
Consistent with section 408(b)(2)(D)
of FFDCA, and the factors specified in
section 408(b)(2)(D) of FFDCA, EPA has
reviewed the available scientific data
and other relevant information in
support of this action. EPA has
sufficient data to assess the hazards of
and to make a determination on
aggregate exposure for the petitioned-for
tolerances for combined residues of the
insecticide, thiamethoxam [3-[(2-chloro-5-
thiazolyl)methyl]tetrahydro-5-methyl-
N-nitro-4H-1,3,5-oxadiazin-4-imine and
its metabolite [N-(2-chloro-thiazol-5-
yl)methyl]-N-methyl -N'-nitro-
guanidine] on artichoke, globe at 0.45
ppm; barley, grain at 0.30 ppm; barley,
hay at 0.40 ppm; barley, straw at 0.40
ppm; Brassica, head and stem, subgroup
5-A at 4.5; brassica, leafy greens,
subgroup 5-B at 3.0 ppm; caneberry
subgroup 13-A at 0.35 ppm; grape at
0.20 ppm; grape, raisin at 0.30 ppm;
hop, dried cones at 0.10 ppm; and
vegetable, leafy except Brassica, group 4
at 4.0 ppm. EPA’s assessment of
exposures and risks associated with
establishing the tolerance follows.

A. Toxicological Profile
EPA has evaluated the available
toxicology data and considered its validity,
completeness, and reliability as well as
the relationship of the results of the studies
to human risk. EPA has also
considered available information
concerning the variability of the
sensitivities of major identifiable
subgroups of consumers, including
infants and children. Specific
information on the studies received and
the nature of the adverse effects caused
by thiamethoxam as well as the
observed-adverse-effect-level (NOAEL)
and the lowest-observed-adverse-effect-
level (LOAEL) from the toxicity studies
can be found at http://
www.regulations.gov. The referenced
document is available in the docket
established by this action, which is
described under ADDRESSES, and is
identified as EPA–HQ–OPP–2006–
0523–0003 in that docket.

The database for thiamethoxam
indicates 4 primary targets for this
pesticide: The liver, testes, kidney, and
hematopoietic system. The testicular
effects are considered to be
toxicologically significant effects and
most of the endpoints for risk
assessment are based on these effects. In
the liver, enzyme induction and
hepatocellular hypertrophy in and of
themselves are not necessarily
considered adverse effects. However,
other effects were associated with these
observations that are considered to be
toxicologically significant. These
include necrosis of single hepatocytes,
foci of cellular alteration, apoptosis,
Kupffer cell infiltration, pigmentation
and hyperplasia, and benign and
malignant liver tumors. The majority of
the kidney effects may be attributed to
accumulation of ε2u-globulin, a protein
that is unique to males rats (it is noted
that 1 high-dose female in the
reproduction study also had similar
effects). If the effects in male rats are
related to accumulation of ε2u-globulin,
then these particular kidney effects are
not relevant to humans. The
hematological effects are observed in
three species. These include increased
spleen weights, increases in the
incidence and severity of hemosiderosis
and/or extramedullary hematopoiesis
and a slight reduction in erythrocytes,
hemoglobin and hematocrit. In the dog,
leukopenia and slight microcytic
anemia have been observed. These
effects are not considered to be as
significant as the testicular, liver, and
kidney effects. They often appear at very
high dose levels and the changes are not
dramatic.

The final rule published in the
Federal Register of January 5, 2006
http://www.epa.gov/fedrgstr/EPA-PEST/
2005/january/Day-05/p089.htm
reported that the EPA had classified
thiamethoxam as “likely carcinogen for
humans” based on increased incidence
of hepatocellular adenomas and
carcinomas in male and female mice.
Quantification of risk based on most
potent unit was based on male mouse
liver adenoma and/or carcinoma
combined tumor rate. The upper bound
estimate of unit risk, Q1* was calculated
as 3.77 × 10−3 in human equivalents.
EPA re-evaluated this determination
based on new data submitted by the
registrant indicating the mode of action
for the mouse liver tumors. EPA agreed
with the registrant that a plausible mode
of action has been established for the
development of liver tumors in a mouse
bioassay with thiamethoxam. EPA
concluded that the liver tumors in the
mouse arise through a non-genotoxic
mode of action characterized by a series
of key events that include: Perturbation
of cholesterol biosynthesis,
hepatotoxicity, cell death (both as single
cell necrosis and apoptosis) and a
sustained increase in cell replication rates. Neither the key events nor an increase in liver tumors are seen in rats fed on diets containing up to 3,000 ppm thiamethoxam. The key metabolites, CGA330050 and CGA265307, responsible for the key events in the mouse are not formed in sufficient quantities in the rat and explain the lack of a carcinogenic response in this species.

A sufficient amount of active metabolite must be produced along with persistent exposure to the active metabolite to lead to the hepatotoxic/regenerative proliferative/neoplastic response in the mouse. Limited human in vitro metabolism studies suggest that humans are more similar to the rat compared to the mouse in producing the active metabolite. The rat does not develop tumors following treatment with thiamethoxam. Thus, the mouse appears to be uniquely sensitive to this mode of action. Because of the threshold nature of the mode of action and the unique sensitivity of the mouse, it is concluded that humans are unlikely to be at risk for developing tumors following exposures to thiamethoxam.

After considering EPA’s Final Guidelines for Carcinogen Risk Assessment, the Agency classified thiamethoxam as “Not Likely to be Carcinogenic to Humans” based on convincing evidence that a non-genotoxic mode of action for liver tumors was established in the mouse and that the carcinogenic effects are a result of a mode of action dependent on sufficient amounts of a hepatotoxic metabolite produced persistently. Although humans are qualitatively capable of producing the active metabolite, thiamethoxam is unlikely to pose a cancer risk to humans unless sufficient amounts of metabolites are persistently formed to drive a carcinogenic response. Lastly, the non-cancer (chronic) assessment is sufficient protective of the key events (perturbation of liver metabolism, hepatotoxicity/regenerative proliferation) in the animal mode of action and, thus, cancer is not an issue. Thus, quantification of carcinogenic potential is not required.

In assessing the human health risks associated with the existing and proposed uses of thiamethoxam, EPA has included exposure to thiamethoxam as well as its metabolite CGA–322704 when evaluating exposure from the dietary (food only) pathway. This approach was developed when the Agency received the first food-use request for registration of thiamethoxam and determined that the CGA–322704 metabolite/degrade, as well as the parent compound, are residues of concern in food; no exposure to CGA–322704 in drinking water was considered likely following application of thiamethoxam. At the time, toxicological information regarding CGA–322704 was not available, and it was assumed that thiamethoxam and this metabolite are toxicologically equivalent for estimation of dietary risk. Subsequently, the Agency received a petition requesting registration of the insecticide clothianidin. Upon review of that petition, the Agency discovered that CGA–322704 and clothianidin are identical. With the registration of clothianidin uses, the Agency now has a complete toxicological database for both thiamethoxam and CGA–322704 (referred to in the remainder of this rule as clothianidin). While some of the toxic effects observed following dosing with the two active ingredients are similar, the available information indicate that thiamethoxam and clothianidin have different toxicological effects in mammals and should be assessed separately. A separate risk assessment of clothianidin has been completed in conjunction with the registration of clothianidin. The most recent assessment, which provides details regarding the toxicology of clothianidin is discussed in the final rule published in the Federal Register of December 13, 2006 (http://www.epa.gov/EPA-PEST/2006/December-Day-13/p20898.htm).

### B. Toxicological Endpoints

For hazards that have a threshold below which there is no appreciable risk, the toxicological level of concern (LOC) is derived from the highest dose at which no adverse effects are observed (the NOAEL) in the toxicology study identified as appropriate for use in risk assessment. However, if a NOAEL cannot be determined, the lowest dose at which adverse effects of concern are identified (the LOAEL) is sometimes used for risk assessment. Uncertainty/safety factors (UF) are used in conjunction with the LOC to take into account uncertainties inherent in the extrapolation from laboratory animal data to humans and in the variations in sensitivity among members of the human population as well as other unknowns. Safety is assessed for acute and chronic risks by comparing aggregate exposure to the pesticide to the acute population adjusted dose (aPAD) and chronic population adjusted dose (cPAD). The aPAD and cPAD are calculated by dividing the LOC by all applicable uncertainty/safety factors. Short-, intermediate, and long-term risks are evaluated by comparing aggregate exposure to the LOC to ensure that the margin of exposure (MOE) called for by the product of all applicable uncertainty/safety factors is not exceeded.

For non-threshold risks, the Agency assumes that any amount of exposure will lead to some degree of risk and estimates risk in terms of the probability of occurrence of additional adverse cases. Generally, cancer risks are considered non-threshold. For more information on the general principles EPA uses in risk characterization and a complete description of the risk assessment process, see http://www.epa.gov/fedrgstr/EPA-PEST/1997/ November/Day-26/p30948.htm.

A summary of the toxicological endpoints for thiamethoxam used for human risk assessment is shown in the Table of this unit.

### SUMMARY OF TOXICOLOGICAL DOSE AND ENDPOINTS FOR THIAMETHOXAM FOR USE IN HUMAN RISK ASSESSMENT

<table>
<thead>
<tr>
<th>Exposure/Scenario</th>
<th>Dose Used in Risk Assessment, Interspecies and Intraspecies and any Traditional FQPA, SF</th>
<th>Special FQPA SF and Level of Concern for Risk Assessment</th>
<th>Study and Toxicological Effects</th>
</tr>
</thead>
</table>
| Acute Dietary (All Populations including females 13-50 years of age, infants and children) | NOAEL = 34.5 mg/kg/day
SF = 100X
Acute RfD = 0.35 mg/kg/day | Special FQPA SF = 1X
aPAD = acute RfD Special FQPA
SF = 0.35 mg/kg/day | Rat Developmental Neurotoxicity study
LOAEL = 298.7 mg/kg/day based on delayed sexual maturation in male pups, and reduced brain morphometric measurements. |
SUMMARY OF TOXICOLOGICAL DOSE AND ENDPOINTS FOR THIAMETHOXAM FOR USE IN HUMAN RISK ASSESSMENT—Continued

<table>
<thead>
<tr>
<th>Exposure/Scenario</th>
<th>Dose Used in Risk Assessment, Interspecies and Intraspecies and any Traditional FQPA, SF</th>
<th>Special FQPA SF and Level of Concern for Risk Assessment</th>
<th>Study and Toxicological Effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chronic Dietary (All populations)</td>
<td>NOAEL= 1.2 mg/kg/day SF = 100X Chronic RfD = 0.012 mg/kg/day</td>
<td>Special FQPA SF = 1X cPAD = chronic RfD/Special FQPA SF = 0.012 mg/kg/day</td>
<td>2—Generation Reproduction study LOAEL = 1.8 mg/kg/day based on increased incidence and severity of tubular atrophy in testes of F₁ generation males. 2—Generation Reproduction study LOAEL = 3 in males based on sperm abnormalities in F₁ males. No LOAEL was determined for females.</td>
</tr>
<tr>
<td>Incidental Oral (All durations)</td>
<td>NOAEL = 8.23 mg/kg/day</td>
<td>LOC for MOE = 100 (Residential)</td>
<td>90-day Dog study LOAEL= 32 (males) 33.9 (females) mg/kg/day based on slightly prolonged prothrombin times and decreased plasma albumin and A/G ratio (both sexes); decreased calcium levels and ovary weights and delayed matura- tion in the ovaries (females); decreased cholesterol and phospholipid levels, testis weights, spermatogenesis, and spermatic giant cells in testes (males).</td>
</tr>
<tr>
<td>Dermal (All durations) (Adults) (Residential)</td>
<td>Oral study NOAE= 1.2 mg/kg/day (dermal absorption rate = 5%)</td>
<td>LOC for MOE = 100 (Residential)</td>
<td>2—Generation Reproduction study LOAEL = 1.8 mg/kg/day based on increased incidence and severity of tubular atrophy in testes of F₁ generation males. 2—Generation Reproduction study (46402904) LOAEL = 3 in males based on sperm abnormalities in F₁ males. No LOAEL was determined for females.</td>
</tr>
<tr>
<td>Dermal (All durations) (Infants/children 1-6 years old) (Residential)</td>
<td>Dermal study NOAE = 60 mg/kg/day (dermal absorption rate = 5%)</td>
<td>LOC for MOE = 100 (Residential)</td>
<td>Rat 28-Day Dermal Toxicity study LOAEL = 250 (females) mg/kg/day based on increased plasma glucose, triglyceride levels, and alkaline phosphatase activity and inflammatory cell infiltration in the liver and necrosis of single hepatocytes in females.</td>
</tr>
<tr>
<td>Inhalation (All durations) (Residential)</td>
<td>Oral study NOAE = 1.2 mg/kg/day (inhalation absorption rate = 100%)</td>
<td>LOC for MOE = 100 (Residential)</td>
<td>2—Generation Reproduction study LOAEL = 1.8 mg/kg/day based on increased incidence and severity of tubular atrophy in testes of F₁ generation males. 2—Generation Reproduction study (46402904) LOAEL = 3 in males based on sperm abnormalities in F₁ males. No LOAEL was determined for females.</td>
</tr>
</tbody>
</table>

C. Exposure Assessment

1. Dietary exposure from food and feed uses. In evaluating dietary exposure to thiamethoxam, EPA considered exposure under the petitioned-for tolerances as well as all existing thiamethoxam tolerances in (40 CFR 180.565). EPA assessed dietary exposures from thiamethoxam in food as follows:

   For both acute and chronic exposure assessments EPA combined residues of clothianidin coming from thiamethoxam with residues of thiamethoxam per se. As discussed above, thiamethoxam’s major metabolite is CGA–322704, which is also the registered active ingredient clothianidin. There is available information indicating that thiamethoxam and clothianidin have different toxicological effects in mammals and should be assessed separately, however, these exposure assessments for this action incorporated the total residue of thiamethoxam and clothianidin to estimate dietary exposure. This aggregation of thiamethoxam and clothianidin began with the initial assessment of thiamethoxam, prior to the requested registration of clothianidin as an active ingredient, and is being maintained in this action for historical purposes. In future assessments, as time and resources allow, the EPA will provide a rationale for the separate analysis of risks coming from thiamethoxam and clothianidin, and will conduct separate evaluations of exposure and risk for each chemical. The combining of these residues, as was done in these assessments, results in highly...
conservative estimates of dietary exposure and risk.

1. *Acute exposure.* Quantitative acute dietary exposure and risk assessments are performed for a food-use pesticide, if a toxicological study has indicated the possibility of an effect of concern occurring as a result of a 1-day or single exposure. In estimating acute dietary exposure, EPA used food consumption information from the USDA 1994–1996 and 1998 Nationwide Continuing Surveys of Food Intake by Individuals (CSFII). As to residue levels in food, EPA assumed maximum residues of thiamethoxam and clothianidin observed in the thiamethoxam field trials. It was also assumed that 100% of crops with registered or requested uses of thiamethoxam are treated.

   ii. *Chronic exposure.* In conducting the chronic dietary exposure assessment EPA used the food consumption data from the USDA 1994–1996 and 1998 Nationwide CSFII. As to residue levels in food, EPA assumed maximum residues of thiamethoxam and clothianidin observed in the thiamethoxam field trials. It was also assumed that 100% of crops with registered or requested uses of thiamethoxam are treated.

   A complete listing of the inputs used in these assessments can be found in the document titled Thiamethoxam Acute and Chronic Aggregate Dietary and Drinking Water Exposure and Risk Assessments for FIFRA Section 3 Registration available in the docket established by this action EPA–HQ–OPP–2006–0523.

   iii. *Cancer.* A quantitative cancer exposure assessment is not necessary because EPA concluded that thiamethoxam is “Not Likely to be Carcinogenic to Humans” based on convincing evidence that a non-genotoxic mode of action for liver tumors was established in the mouse and that the carcinogenic effects are a result of a mode of action dependent on sufficient amounts of a hepatotoxic metabolite produced persistently. Therefore, the Agency concluded that thiamethoxam is not expected to pose a carcinogenic risk and an exposure assessment pertaining to cancer risk is not necessary.

2. *Dietary exposure from drinking water.* Thiamethoxam is expected to be persistent and mobile in terrestrial and aquatic environments. These fate properties suggest that thiamethoxam has a potential to move into surface water and shallow ground water. The Agency lacks sufficient monitoring data to complete a comprehensive dietary exposure analysis and risk assessment for thiamethoxam in drinking water. Because the Agency does not have comprehensive monitoring data, drinking water concentration estimates are made by reliance on simulation or modeling taking into account data on the environmental fate characteristics of thiamethoxam. Further information regarding EPA drinking water models used in pesticide exposure assessment can be found at http://www.epa.gov/oppefed1/models/water/index.htm.

   Based on the Pesticide Root Zone Model/Exposure Analysis Modeling System (PRZM/EXAMS) and Screening Concentrations in Groundwater (SCI-GROW) models, the estimated environmental concentrations (EECs) of thiamethoxam for acute exposures are estimated to be 12.26 parts per billion (ppb) for surface water and 7.94 ppb for ground water. The EECs for chronic exposures are estimated to be 1.29 ppb for surface water and 7.94 ppb for ground water.

   The registrant has conducted small-scale postapplication ground water studies in several locations in the U.S. to investigate the mobility of thiamethoxam in a vulnerable hydrogeological setting. A review of those data shows that generally residues of thiamethoxam as well as CGA–322704 are below the limit of quantitation (0.05 ppb). When quantifiable residues are found, they are sporadic and at low levels. The maximum observed residue levels from any monitoring well were 1.0 ppb for thiamethoxam and 0.73 ppb for CGA–322704. These values are well below the modeled estimates summarized above, indicating that the modeled estimates are, in fact, protective of what actual exposures are likely to be.

   Modeled estimates of drinking water concentrations were directly entered into the dietary exposure model. For both the acute and chronic assessments the acute EEC of 12.26 ppb (0.0123 ppm) was used as a worst-case estimate of exposure via drinking water.

   3. From non-dietary exposure. The term “residential exposure” is used in this document to refer to non-occupational, non-dietary exposure (e.g., for lawn and garden pest control, indoor pest control, termiteicides, and flea and tick control on pets).

   Thiamethoxam is registered for use on turfgrass on golf courses, residential lawns, commercial grounds, parks, playgrounds, athletic fields, landscapes, interiortscapes and sod farms. Thiamethoxam is applied by commercial applicators only. Therefore, exposures from homeowner applications were not assessed. However, entering areas previously treated with thiamethoxam could lead to exposures for adults and children. As a result, risk assessments have been completed for postapplication scenarios. Short-term exposures (1 to 30 days of continuous exposure) may occur as a result of activities on treated turf. There are no use patterns for thiamethoxam that indicate intermediate-term (1 to 6 months of continuous exposure) or chronic non-dietary exposures are likely to occur.

   Dermal exposures were assessed for adults and children. Oral non-dietary ingestion exposures (i.e. soil ingestion, and hand-/object-to-mouth) were assessed for children as well. Since all postapplication scenarios occur outdoors the potential for inhalation exposure is negligible and therefore does not require an inhalation exposure assessment. For purposes of this assessment exposure from residential lawns is used to represent the worst case scenario for both dermal and oral postapplication exposure.

   Postwithdrawal when a homeowner exposed resulting from contact with treated turf was assessed using the HED Standard Operating Procedures for Residential Exposure and a chemical-specific turf transfer residue (TTR) study.

4. *Cumulative effects from substances with a common mechanism of toxicity.* Section 408(b)(2)(D)(v) of the FFDCA requires that, when considering whether to establish, modify, or revoke a tolerance, the Agency consider “available information” concerning the cumulative effects of a particular pesticide’s residues and “other substances that have a common mechanism of toxicity.”

   Thiamethoxam is a member of the neonicotinoid class of pesticides and produces, as a metabolite, another neonicotinoid, clothianidin. Structural similarities or common effects do not constitute a common mechanism of toxicity. Evidence is needed to establish that the chemicals operate by the same, or essentially the same sequence of major biochemical events (EPA, 2002). Although clothianidin and thiamethoxam bind selectively to insect nicotinic acetylcholine receptors (nACHr), the specific binding site(s)/ receptor(s) for clothianidin, thiamethoxam, and the other neonicotinoids are unknown at this time. Additionally, the commonality of the binding activity itself is uncertain, as preliminary evidence suggests that clothianidin operates by direct competitive inhibition, while thiamethoxam is a non-competitive inhibitor. Furthermore, even if future research shows that neonicotinoids share a common binding activity to a
specific site on insect nicotinic acetylcholine receptors, there is not necessarily a relationship between this pesticidal action and a mechanism of toxicity in mammals. Structural variations between the insect and mammalian nAChRs produce quantitative differences in the binding affinity of the neonicotinoids towards these receptors, which, in turn, confers the notably greater selective toxicity of this class towards insects, including aphids and leafhoppers, compared to mammals. While the insecticidal action of the neonicotinoids is neurotoxic, the most sensitive regulatory endpoint for thiamethoxam is based on unrelated effects in mammals, including effects on the liver, kidney, testes, and hematopoietic system. Additionally, the most sensitive toxicological effect in mammals differs across the neonicotinoids (e.g., testicular tubular atrophy with thiamethoxam; mineralized particles in thyroid colloid with imidacloprid). Thus, there is currently no evidence to indicate that neonicotinoids share common mechanisms of toxicity, and EPA is not following a cumulative risk approach based on a common mechanism of toxicity for the neonicotinoids. For information regarding EPA’s efforts to determine which chemicals have a common mechanism of toxicity and to evaluate the cumulative effects of such chemicals, see the policy statements concerning common mechanism determinations and procedures for cumulative effects from substances found to have a common mechanism released by EPA’s Office of Pesticide Programs on EPA’s website at http://www.epa.gov/pesticides/cumulative/.

D. Safety Factor for Infants and Children

1. In general. Section 408 of FFDCA provides that EPA shall apply an additional (10X) tenfold margin of safety for infants and children in the case of threshold effects to account for prenatal and postnatal toxicity and the completeness of the data base on toxicity and exposure unless EPA determines based on reliable data that a different margin of safety will be safe for infants and children. This additional margin of safety is commonly referred to as the FQPA safety factor. In applying this provision, EPA either retains the default value of 10X when reliable data do not support the choice of a different factor, or, if reliable data are available, EPA uses a different additional FQPA safety factor value based on the use of traditional uncertainty/safety factors and/or special FQPA safety factors, as appropriate.

2. Prenatal and postnatal sensitivity.

In the developmental studies, there is no evidence of increased quantitative or qualitative susceptibility of rat or rabbit fetuses to in utero exposure to thiamethoxam. The developmental NOAELs are either higher than or equal to the maternal NOAELs. The toxicological effects in fetuses do not appear to be any more severe than those in the dams or does. In the rat developmental neurotoxicity study, there was no quantitative evidence of increased susceptibility. There is evidence of increased quantitative susceptibility for male pups in both 2-generation reproductive studies. In one study, there are no toxicological effects in the dams whereas for the pups, reduced bodyweights are observed at the highest dose level, starting on day 14 of lactation. This contributes to an overall decrease in bodyweight gain during the entire lactation period. The reproductive effects in males appear in the F1 generation in the form of increased incidence and severity of testicular tubular atrophy (see developmental/reproductive section). These data are considered to be evidence of increased quantitative susceptibility for male pups (increased incidence of testicular tubular atrophy at 1.8 mg/kg/day) when compared to the parents (hyaline changes in renal tubules at 61 mg/kg/day; NOAEL is 1.8 mg/kg/day).

In the more recent 2-generation reproduction study, the most sensitive effect was sperm abnormalities at 3 mg/kg/day (the NOAEL is 1.2 mg/kg/day) in the F1 males. This study also indicates increased susceptibility for the offspring for this effect.

Although there is evidence of increased quantitative susceptibility for male pups in both reproductive studies, NOAELs and LOAELs were established in these studies and the Agency selected the NOAEL for testicular effects in F1 pups as the basis for risk assessment. The Agency has confidence that the NOAEL selected for risk assessment is protective of the most sensitive effect (testicular effects) for the most sensitive subgroup (pups) observed in the toxicological database. Due to the finding of quantitative sensitivity in the reproduction studies, the EPA conducted a degree of concern analysis to assess the residual uncertainties for prenatal and/or postnatal susceptibility. The Agency concluded that there is low concern for an increased susceptibility in the young given:

i. There is no increased sensitivity (qualitative or quantitative) in the rat developmental, rabbit developmental and rat developmental neurotoxicity studies; and

ii. There was a clear NOAEL identified for the effects in pups in the rat reproduction studies where sensitivity was seen; and

iii. The Agency selected this NOAEL as the basis for risk assessment.

3. Conclusion.

The final rule published in the Federal Register of January 5, 2006 (http://www.epa.gov/fedreg/EPA-PEST/2005/January/Day-05/p009.htm) reported that the EPA had determined that the 10X special safety factor to protect infants and children should be retained for thiamethoxam based on the following factors: Effects on endocrine organs observed across species; the significant decrease in alanine amino transferase levels in the companion animal studies and in the dog studies; the mode of action of this chemical in insects (interferes with the nicotinic acetylcholine receptors of the insect’s nervous system); the transient clinical signs of neurotoxicity in several studies across species; and the suggestive evidence of increased quantitative susceptibility in the rat reproduction study.

Since that determination the EPA has reviewed and reviewed a Developmental Neurotoxicity (DNT) study in rats and an additional Reproduction study in rats. Taking the results of these studies into account, EPA has determined that reliable data show that it would be safe for infants and children to reduce the FQPA safety factor to 1X. That decision is based on the following findings:

i. The toxicity database for thiamethoxam is complete.

ii. For the reasons discussed above, there is low concern for an increased susceptibility in the young.

iii. Although there is evidence of neurotoxicity after acute exposure to thiamethoxam at doses of 500 mg/kg/day including dropped palpebral closure, decrease in rectal temperature and locomotor activity and increase in forelimb grip strength, no evidence of neuropathology was observed. These effects occurred at doses at least 14-fold and 416-fold higher than the doses used for the acute, and chronic risk assessments, respectively; thus, there is low concern for these effects since it is expected that the doses used for regulatory purposes would be protective of the effects noted at much higher doses.

iv. There are no residual uncertainties identified in the exposure databases. The dietary food exposure assessments were performed based on assumption that the maximum use rates of thiamethoxam and clothianidin observed in the thiamethoxam field


trials were remaining on crops. Although there is available information indicating that thiamethoxam and clothianidin have different toxicological effects in mammals and should be assessed separately, the residues of each have been combined in these assessments to ensure that the estimated exposures of thiamethoxam do not underestimate actual potential thiamethoxam exposures. An assumption of 100% crop treated was made for all foods evaluated in the assessments. For both the acute and chronic assessments the acute EEC of 12.26 ppb (0.0123 ppm) was used as a worst-case estimate of exposure via drinking water. Compared to the results from small-scale prospective ground water studies where the maximum observed residue levels from any monitoring well were 1.0 ppb for thiamethoxam and 0.73 ppb for CGA-322704, the modeled estimates are protective of what actual exposures are likely to be. Similarly conservative Residential SOPs as well as a chemical-specific turf transfer residue (TRR) study were used to assess post-application exposure to children as well as incidental oral exposure of toddlers. These assessments will not underestimate the exposure and risks posed by thiamethoxam.

E. Aggregate Risks and Determination of Safety

Safety is assessed for acute and chronic risks by comparing aggregate exposure to the pesticide to the acute population adjusted dose (aPAD) and chronic population adjusted dose (cPAD). The aPAD and cPAD are calculated by dividing the LOC by all applicable uncertainty/safety factors. For linear cancer risks, EPA calculates the probability of additional cancer cases given aggregate exposure. Short-, intermediate, and long-term risks are evaluated by comparing aggregate exposure to the LOC to ensure that the margin of exposure (MOE) called for by the product of all applicable uncertainty/safety factors is not exceeded.

1. Acute risk. Using the exposure assumptions discussed in this unit for acute exposure, the acute dietary exposure from food and water to thiamethoxam will occupy 3% of the aPAD for children 1-2 years old, the population group with greatest exposure. Based on the use patterns proposed, chronic residential exposure to residues of thiamethoxam is not expected.

2. Chronic risk. Using the exposure assumptions described in this unit for chronic exposure, EPA has concluded that thiamethoxam from food and water will utilize 42% of the cPAD for children 1-2 years old, the

population group with greatest exposure. Based on the use patterns proposed, chronic residential exposure to residues of thiamethoxam is not expected.

3. Short-term risk. Short-term aggregate exposure takes into account residential exposure plus chronic exposure to food and water (considered to be a background exposure level). Thiamethoxam is currently registered for use that could result in short-term residential exposure and the Agency has determined that it is appropriate to aggregate chronic food and water and short-term exposures for thiamethoxam. The level of concern for the margin of exposure (MOE) is 100 for all residential uses (i.e., MOEs less than 100 indicate potential risks of concern). Using the exposure assumptions described in this unit for short-term exposures, EPA has concluded that food, water, and residential exposures aggregated result in aggregate MOEs of 730 through 2,800 for all exposure scenarios (dermal exposures, and oral non-diary ingestion) for infants, children and adults.

4. Intermediate-term risk. Intermediate-term aggregate exposure takes into account residential exposure plus chronic exposure to food and water (considered to be a background exposure level). There are no use patterns for thiamethoxam that indicate intermediate-term (1 to 6 months of continuous exposure) exposures are likely to occur.

5. Aggregate cancer risk for U.S. population. The Agency has classified thiamethoxam as not likely to be a human carcinogen based on convincing evidence that a non-genotoxic mode of action for liver tumors was established in the mouse and that the carcinogenic effects are a result of a mode of action dependent on sufficient amounts of a hepatotoxic metabolite produced persistently. Thiamethoxam is not expected to pose a cancer risk.

6. Determination of safety. Based on these risk assessments, EPA concludes that there is a reasonable certainty that no harm will result to the general population, or to infants and children from aggregate exposure to thiamethoxam residues.

IV. Other Considerations

A. Analytical Enforcement Methodology

Adequate enforcement methodology (high-performance liquid chromatography/ultraviolet (HPLC/UV) or mass spectrometry (MS) is available to enforce the tolerance expression. The method may be requested from: Chief, Analytical Chemistry Branch, Environmental Science Center, 701 Maps Rd., Ft. Meade, MD 20755–5350; telephone number: (410) 305–2905; e-mail address: residuemeethods@epa.gov.

B. International Residue Limits

There are no CODEX or Mexican maximum residue limits (MRLs) for thiamethoxam. A number of Canadian MRLs exist for this chemical and are in accord with U.S. tolerances. The new/revised tolerances established by this rule have been derived using the NAFTA Tolerance Harmonization Spreadsheet.

C. Response to Comments

Several comments were received from a private citizen objecting to establishment of these tolerances. The Agency has received similar comments from this commenter on numerous previous occasions. Refer to Federal Register 70 FR 37686 (June 30, 2005), 70 FR 1354 (January 7, 2005), 69 FR 63096–63098 (October 29, 2004) for the Agency’s response to these objections. In addition, the commenter noted several adverse effects seen in animal toxicology studies with thiamethoxam and claims because of these effects no tolerance should be approved. EPA has found, however, that there is a reasonable certainty of no harm to humans after considering these toxicological studies and the exposure levels of humans to thiamethoxam.

V. Conclusion

Based upon review of the data supporting and use of the NAFTA Tolerance Harmonization Spreadsheet the EPA has determined that tolerance levels for the following crops should be changed as follows: Artichoke, globe from 0.40 ppm to 0.45 ppm; caneberry subgroup 13-A from 0.30 ppm to 0.35 ppm; grape from 0.15 ppm to 0.20 ppm; brassica, head and stem, subgroup 5-A from 1.0 ppm to 4.5 ppm; brassica, leafy greens, subgroup 5-B from 2.0 ppm to 3.0 ppm; and vegetable, leafy, except brassica from 2.0 ppm to 4.0 ppm. EPA has also determined that a separate tolerance for grape juice is not needed since any residues in grape juice are not expected to exceed the grape tolerance of 0.20 ppm. Therefore, tolerances are established for the combined residues of thiamethoxam [3-[(2-chloro-5-thiazolyl)methyl]tetrahydro-5-methyl-N'-ylylmethyl-N-thiazolyl]-1,3,5-oxadizin-4-imine and its metabolite [N-(2-chloro-thiazol-5-ylmethyl)-N'-methyl-N-nitro- guanidine] on artichoke, globe at 0.45 ppm; barley, grain at 0.30 ppm; barley, hay at 0.40 ppm; barley, straw at 0.40 ppm; brassica, head and stem, subgroup 5-A at 4.5; brassica, leafy greens, subgroup
5-B; caneberry subgroup 13-A at 0.35 ppm; grape at 0.20 ppm; grape, raisin at 0.30 ppm; hop, dried cones at 0.10 ppm; and vegetable, leafy except brassica, group 4 at 4.0 ppm.

VI. Statutory and Executive Order
Reviews

This final rule establishes a tolerance under section 408(d) of FFDCA in response to a petition submitted to the Agency. The Office of Management and Budget (OMB) has exempted these types of actions from review under Executive Order 12866, entitled Regulatory Planning and Review (58 FR 51735, October 4, 1993). Because this rule has been exempted from review under Executive Order 12866, this rule is not subject to Executive Order 13211, Actions Concerning Regulations That Significantly Affect Energy Supply, Distribution, or Use (66 FR 28355, May 22, 2001) or Executive Order 13045, entitled Protection of Children from Environmental Health Risks and Safety Risks (62 FR 19885, April 23, 1997).

This final rule does not contain any information collections subject to OMB approval under the Paperwork Reduction Act (PRA), 44 U.S.C. 3501 et seq., nor does it require any special considerations under Executive Order 12898, entitled Federal Actions to Address Environmental Justice in Minority Populations and Low-Income Populations (59 FR 7629, February 16, 1994). Since tolerances and exemptions that are established on the basis of a petition under section 408(d) of FFDCA, such as the tolerance in this final rule, do not require the issuance of a proposed rule, the requirements of the Regulatory Flexibility Act (RFA) (5 U.S.C. 601 et seq.) do not apply.

This final rule directly regulates growers, food processors, food handlers and food retailers, not States or tribes, nor does this action alter the relationships or distribution of power and responsibilities established by Congress in the preemption provisions of section 408(n)(4) of FFDCA. As such, the Agency has determined that this action will not have a substantial direct effect on States or tribal governments, on the relationship between the national government and the States or tribal governments, or on the distribution of power and responsibilities among the various levels of government or between the Federal Government and Indian tribes. Thus, the Agency has determined that Executive Order 13132, entitled Federalism (64 FR 43255, August 10, 1999) and Executive Order 13175, entitled Consultation and Coordination with Indian Tribal Governments (65 FR 67249, November 6, 2000) do not apply to this rule. In addition, This rule does not impose any enforceable duty or contain any unfunded mandate as described under Title II of the Unfunded Mandates Reform Act of 1995 (UMRA) (Public Law 104–4).

This action does not involve any technical standards that would require Agency consideration of voluntary consensus standards pursuant to section 12(d) of the National Technology Transfer and Advancement Act of 1995 (NTTAA), Public Law 104–113, section 12(d) (15 U.S.C. 272 note).

VII. Congressional Review Act

The Congressional Review Act, 5 U.S.C. 801 et seq., generally provides that before a rule may take effect, the agency promulgating the rule must submit a rule report to each House of the Congress and to the Comptroller General of the United States. EPA will submit a report containing this rule and other required information to the U.S. Senate, the U.S. House of Representatives, and the Comptroller General of the United States prior to publication of this final rule in the Federal Register. This final rule is not a “major rule” as defined by 5 U.S.C. 804(2).

List of Subjects in 40 CFR Part 180

Environmental protection, Agricultural commodities, Pesticides and pests, Reporting and recordkeeping requirements.


Lois Rossi,
Director, Registration Division, Office of Pesticide Programs.

Therefore, 40 CFR chapter I is amended as follows:

PART 180—AMENDED

1. The authority citation for part 180 continues to read as follows:


2. Section 180.565 is amended as follows:

(a) In paragraph (a) by alphabetically adding commodities to the table;

(i) In paragraph (a) by revising the entries for Barley, grain; Barley, hay and Barley, straw in the table;

(ii) In paragraph (b) by removing the entries for Artichoke, globe; Bean, dry, seed; Bean, succulent; and Hops in the table.

The amendment read as follows:

§180.565 Thiamethoxam; tolerances for residues.

(a) * * *

Commodity Parts per million

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[FR Doc. E7–11794 Filed 6–21–07: 8:45 am]
BILING CODE 6560–50–S

DEPARTMENT OF TRANSPORTATION

National Highway Traffic Safety Administration

49 CFR Part 571

[Docket No. NHTSA–2007–27662]

Federal Motor Vehicle Safety Standards; Electronic Stability Control Systems; Correction

AGENCY: National Highway Traffic Safety Administration (NHTSA), Department of Transportation (DOT).

ACTION: Correcting amendments.

SUMMARY: In April 2007, the agency published a final rule establishing a new Federal motor vehicle safety standard on electronic stability control (ESC) systems for light vehicles. As part of that rulemaking, the final rule notice stated that NHTSA had decided to defer the standard’s requirements related to the ESC telltale and control requirements until the end of the phase-in period (i.e., until September 1, 2011). Accordingly, most of the paragraphs containing ESC telltale and control requirements were prefaced with the phrase “as of September 1, 2011.” However, that phrase was inadvertently omitted from two of the paragraphs setting forth ESC telltale and control requirements. These amendments correct this administrative error by adding the phrase “as of September 1, 2011” to those paragraphs.

DATES: This rule is effective June 22, 2007.

FOR FURTHER INFORMATION CONTACT: Mr. Patrick Boyd, Office of Crash Avoidance Systems; Correction

National Highway Traffic Safety Administration

49 CFR Part 571

[Docket No. NHTSA–2007–27662]