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OFFICE OF  
PREVENTION, PESTICIDES AND  
TOXIC SUBSTANCES

Date: December 22, 1997

## MEMORANDUM

SUBJECT: *FENOXYCARB* - Report of the Hazard Identification Assessment Review Committee.

FROM: Jess Rowland *Jess Rowland 12/22/97*  
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Science Analysis Branch, Health Effects Division (7509C)

THROUGH: K. Clark Swentzel, Chairman,  
Hazard Identification Assessment Review Committee  
Toxicology Branch II, Health Effects Division (7509C) *[Signature]*

TO: Stephen Dapson  
Branch Senior Scientist  
Toxicology Branch 2  
Health Effects Division (7509C)


PC Code: 125301

On December 12, 1997, the Health Effects Division's Hazard Identification Review committee evaluated the toxicology data base, selected doses and endpoints for acute dietary, chronic dietary (RfD) as well as occupational and residential exposure risk assessments, assessed the carcinogenic potential and addressed the sensitivity of infants and children from exposure to Fenoxycarb as required by the Food Quality Protection Act (FQPA) of 1996. The Committee's conclusions are presented in this report.

### Committee Members in Attendance

Members in attendance were Karl Baetcke, William Burnam, George Ghali, Karen Hamernik, Susan Makris, Melba Morrow, Kathy Raffaele, Jess Rowland and Clark Swentzel. Member in absentia: Nancy McCarroll. Data was presented by Stephen Dapson of Toxicology Branch 2.

Data Presentation:

  
Stephen Dapson, Ph.D.

Report Preparation:

  
Jess Rowland, M.S

## I. INTRODUCTION

On December 2, 1997, the Health Effects Division's Hazard Identification Review committee evaluated the toxicology data base, selected doses and endpoints for acute dietary, chronic dietary (RfD) as well as occupational and residential exposure risk assessments, assessed the carcinogenic potential and addressed the sensitivity of infants and children from exposure to Fenoxycarb as required by the Food Quality Protection Act (FQPA) of 1996.

## II. HAZARD IDENTIFICATION

### A. Acute Dietary (one-day)

Females 13+

Study Selected: Developmental Toxicity - Rabbit §83-3

MRID No. 00153125

Executive Summary: In a developmental toxicity study, Swiss hare rabbits (20/dose) received oral administration of Fenoxycarb (98%) at dose levels of 0, 30, 100 or 300 mg/kg/day during gestation days 7 through 19. In a second study, groups of 35 Swiss hare rabbits received Fenoxycarb at 0 or 200 mg/kg/day during gestation days 7 through 19. Maternal toxicity at 200 and 300 mg/kg/day manifested as decreased body weight gain during the dosing period; weight gain showed a rebound at 300 mg/kg/day during the rest of the gestation. For maternal toxicity, the NOEL was 100 mg/kg/day and the LOEL was 200 mg/kg/day based on decreases in body weight gain. Developmental toxicity at 300 mg/kg/day was manifested as slightly increased incidence of spinal bifida of the sacral region (3 fetuses in 3/20 litters vs none in the control groups of 53 litters) and possibly increased incidence of hypoplastic tail (4 fetuses in 3/20 litters vs. 1 per control group, 2/53 litters). Although the report stated that the incidence was stated to be outside historical control range, no historical control data were provided. For developmental toxicity, the NOEL was 200 mg/kg/day and the LOEL was 300 mg/kg/day based on increased incidence of fetal skeletal malformations.

Dose and Endpoint for Risk Assessment: Developmental NOEL=200 mg/kg/day based on increased incidence of spinal bifida and hypoplastic tail at 300 mg/kg/day.

Comments about Study and Endpoint: The fetal skeletal malformations are presumed to occur after a single exposure (dose) and thus were considered to be appropriate for this risk assessment.

**This risk assessment is required.**

General Population (Including Infants and Children): A dose and endpoint was not selected for this population since there were no effects that could be attributed to a single dose (exposure) were observed in oral toxicology studies including the developmental toxicity studies in rats or rabbits.

Acute Dietary Risk Assessment: The Committee determined that the **10 x** factor to account for enhanced sensitivity of infants and children (as required by FQPA) **should be retained**. For acute dietary risk assessment, a **Margin of Exposure (MOE) of 1000 is adequate** for the protection of the general U.S population including infants and children from acute exposure to Fenoxycarb. A MOE of 1000 is required based on the following weight-of-the-evidence considerations:

- (i) Data Gap: there are no acute or subchronic neurotoxicity studies and in the subchronic oral toxicity study measurement of cholinesterase inhibition was not well characterized.
- (ii) There is evidence for spinal neurotubule defects following *in utero* exposure as shown in the developmental rabbit study
- (iii) Data Gap: the lack of an adequate two generation reproduction toxicity study in rats. The available study is un-acceptable because a NOEL for parental systemic toxicity was not established which precluded an evaluation of sensitivity in pups when compared to adults.
- (iv) There is concern for neurotoxicity ; decreases in brain weight were observed in the F<sub>1a</sub>, F<sub>2a</sub> and F<sub>2b</sub> pups in the un-acceptable two-generation reproduction study . Decreases in brain weights were also observed in the chronic toxicity study in dogs.

#### **B. Chronic Dietary [Reference Dose (RfD)]**

The Committee determined that a Reference Dose can not be established for Fenoxycarb and concluded that the Agency should not support the registration of a first food use for this chemical. The Committee's conclusions are based on the following factors:

- (i) Data gap; lack of an adequate two generation reproduction toxicity study in rats (critical study) which precluded an evaluation of sensitivity in pups when compared to adults.
- (ii) Data gap; no subchronic neurotoxicity study. In the subchronic oral toxicity study, cholinesterase inhibition was not well characterized.
- (iii) There is evidence for spinal neurotubule defects following *in utero* exposure as shown in the developmental rabbit study.

- (iv) There is concern for neurotoxicity ; decreases in brain weight were observed in the F<sub>1a</sub>, F<sub>2a</sub> and F<sub>2b</sub> pups in the un-acceptable two-generation reproduction study . Decreases in brain weights were also observed in the chronic toxicity study in dogs.
- (v) The observance of neurotoxic effects in the developmental and the two-generation reproduction studies dictates the need for a developmental neurotoxicity study.

## **C. Occupational/Residential Exposure**

### **1. Dermal Absorption**

Dermal Absorption Factor: Due to the lack dermal absorption study a dermal absorption factor of 100% will be used for converting oral doses to dermal doses.

### **2. Short-Term Dermal - (1-7 days)**

Study Selected: Developmental Toxicity - Rabbit §83-3

MRID No. 00153125

Executive Summary: See Acute Dietary

Dose and Endpoint for Risk Assessment: Developmental NOEL=200 mg/kg/day based on increased incidence of spinal bifida and hypoplastic tail at 300 mg/kg/day (LOEL).

Comments about Study and Endpoint: In a 21-day dermal study in rats (MRID No. 00146601), repeated dermal application at 0, 20, 200 or 2000 mg/kg/day resulted in hepatotoxicity characterized by elevated liver weights and hypertrophy at 2000 mg/kg/day; the NOEL was 200 mg/kg/day. The Committee did not use the dermal study because: 1) of the concern for the developmental effects seen after 11 days of dosing which encompasses the exposure period of concern (1-7 days); 2) developmental effects are not evaluated in the dermal study; and 3) in the 21-day dermal study, hepatotoxicity was seen after three weeks of treatment which is longer than the exposure period of concern (i.e., 1-7 days). Since an oral dose was selected a dermal absorption rate of 100% (default value) should be used in risk assessments.

**This risk assessment is required.**

### 3. Intermediate-Term Dermal (7 Days to Several Months)

Study Selected: Developmental Toxicity - Rabbit §83-3

MRID No. 00153125

Executive Summary: See Acute Dietary

Dose and Endpoint for Risk Assessment: Developmental NOEL=200 mg/kg/day based on increased incidence of spinal bifida and hypoplastic tail at 300 mg/kg/day (LOEL).

Comments about Study and Endpoint: In a 21-day dermal study in rats (MRID No. 00146601), repeated dermal application at 0, 20, 200 or 2000 mg/kg/day resulted in hepatotoxicity characterized by elevated liver weights and hypertrophy at 2000 mg/kg/day; the NOEL was 200 mg/kg/day. The Committee did not use the dermal study because: 1) of the concern for the developmental effects seen after 11 days of dosing (encompasses the exposure period of concern, 1-7 days); 2) developmental effects are not evaluated in the dermal study; and 3) hepatotoxicity was seen after three weeks of treatment which is longer than the exposure period of concern (i.e., 1-7 days). Since an oral dose was selected a dermal absorption rate of 100% (default value) should be used in risk assessments.

**This risk assessment is required.**

### 4. Long-Term Dermal (Several Months to Life-Time)

Based on the use pattern, chronic dermal exposure is not anticipated and thus this risk assessment is not required.

This risk assessment is **NOT** required.

### 5. Inhalation Exposure (Any-Time period)

Study Selected: 21-Day Inhalation - Rat      Guideline: §82-3

MRID No 40355801

Executive Summary: In an inhalation study, groups of SPF-bred Wistar rats (5/sex/concentration) were exposed via inhalation (nose only) to Fenoxycarb (96.6%) at concentrations of 0, 0.01, 0.10, or 1.13 mg/L, 6 hours/day, 5 days/week for 21 days. Effects observed at 1.13 mg/L were limited to minimal decreased body weight gain in males and increased absolute liver weights in females. These were considered to be of questionable significance by the Committee and a threshold NOEL was determined to be 1.13 mg/L.

Dose and Endpoint for Risk Assessment: NOEL =1.13 mg/L (HDT).

Comments about Study and Endpoint: Due to the lack of subchronic or chronic inhalation studies, the NOEL from this study will be used for Short-, Intermediate-and Chronic inhalation risk assessments.

**This risk assessment is required.**

**D Margin of Exposure for Occupational/Residential Exposures:**

For **Short-and Intermediate Term dermal** as well as for **Inhalation (any time period)** risk assessments, **a MOE of 1000 is required** for occupational and/or residential exposures to Fenoxycarb. **Long-Term dermal** risk assessment is not required. A MOE of 1000 is required because

- (i) Data gap: lack of an adequate two generation reproduction toxicity study in rats (critical study) which precluded an evaluation of sensitivity in pups when compared to adults.
- (ii) Data gap: there are no acute or subchronic neurotoxicity studies and cholinesterase inhibition was not well characterized in the subchronic oral toxicity study.
- (iii) There is evidence for spinal neurotubule defects following *in utero* exposure as shown in the developmental rabbit study.
- (iv) There is concern for neurotoxicity ; decreases in brain weight were observed in the F<sub>1a</sub>, F<sub>2a</sub> and F<sub>2b</sub> pups in the un-acceptable two-generation reproduction study . Decreases in brain weights were also observed in the chronic toxicity study in dogs.
- (v) The observance of neurotoxic effects in the developmental and the two-generation reproduction studies dictates the need for a developmental neurotoxicity study.

#### IV. CARCINOGENICITY CLASSIFICATION

##### 1. Combined Chronic Toxicity/Carcinogenicity Study in Rats

Guideline §83-5

MRID No. 40376901

Executive Summary: In a combined chronic/oncogenicity study, Crl:CD (SD) BR Sprague-Dawley rats (50/sex/dose) were fed diets containing Fenoxycarb (94.9%), at dose levels of 0, 200, 600 or 1800 ppm (0, 8.1, 24.7, 74.4 mg/kg/day for males, and 0, 10.9, 33.1, or 100.4 mg/kg/day for females, respectively) for 24 months. A total of 10 rats/sex/group were terminated at 12 months and all remaining animals were sacrificed at 24 months of the study. For chronic toxicity, the NOEL was 200 ppm (8.1 mg/kg/day in males and 10.9 mg/kg/day in females and the LOEL was 600 ppm (24.7 mg/kg/day in males and 33.1 mg/kg/day in females) based on hepatotoxicity characterized by increases in liver enzymes (SGOT, SGPT, AP) and non-neoplastic lesions (centrilobular hypertrophy, focal necrosis, focal fibrosis, focal cystic degeneration, basophilic foci and pigmented macrophages). **There was no evidence of carcinogenicity.** The dose levels used in this study were judged to be adequate to assess the carcinogenic potential of Fenoxycarb based on the hepatotoxicity observed in both sexes at the mid and high dose group.

##### 2. Carcinogenicity Study in Mice

Guideline § 83-2

MRID No.: 40376902

In a carcinogenicity study CD-1 mice (50/sex/dose) were fed diets containing Fenoxycarb at dose levels of 0, 30, 110 or 420 ppm to males (equivalent to 0, 6, 21.7, or 81.8 mg/kg/day, respectively and at 0, 20, 80 or 320 ppm (0, 4.8, 18.2, or 71.6 mg/kg/day, respectively) for 80 weeks. For systemic toxicity, the NOEL was  $\geq$  420 ppm (81.8 mg/kg/day) in males and 320 ppm (71.6 mg/kg/day) in females; a LOEL was not established. **There was evidence of carcinogenicity.** Fenoxycarb was associated with a statistically significant increase in Harderian gland tumors as well as a statistically significant increase in lung bronchiolar/alveolar tumors in male mice at 420 ppm. Dose levels were judged to be inadequate to assess the carcinogenic potential of Fenoxycarb due to the absence of toxicity at the highest doses tested.

MRID No. 43912903

Executive Summary: In a carcinogenicity study groups of Tif:MAGf mice (60/sex/dose) were fed diets containing Fenoxycarb at dose levels of 0, 10, 50, 500 or 2000 ppm (equivalent to 0, .1.10, 5.75, 55.4 or 222 mg/kg/day for females and 0, 1.04, 5.33, 51.5 or 201 mg/kg/day for males, respectively) for 18 months. For systemic toxicity, the NOEL was 50 ppm (5.75 mg/kg/day in males and 5.33 mg/kg/day in females) and the LOEL was 500 ppm (55.4 mg/kg/day in males and 51.5 mg/kg/day in females) based on decreased body weight gains and evidence of hepatic and pulmonary toxicity.



**There was evidence of carcinogenicity.** Fenoxycarb was associated with statistically significant increases in the following tumor types: 1) hepatocellular carcinomas and/or carcinomas and hepatomas combined in males at 2000 ppm; 2) hepatocellular carcinomas in males at 500 ppm; and 3) alveolar/bronchiolar adenoma, carcinoma and combined adenoma/carcinomas in males at 500 and 2000 ppm and in females at 2000 ppm. The dose levels used were judged to be adequate to assess the carcinogenicity of Fenoxycarb.

### 3. Classification of Carcinogenic Potential

On May 10, 1995, the HED Cancer Peer Review Committee (CPRC) classified Fenoxycarb as a **Group B2 Carcinogen (Probable Human Carcinogen)** based on statistically significant increases of tumors of the lung (adenomas, carcinomas and combined adenomas/carcinomas) and adenomas of the Harderian gland in male CD-1 mice even at a dose that was not adequate to assess the carcinogenicity. Urethan, a possible metabolite of Fenoxycarb, is also associated with tumors at these same sites, and other, in multiple species and strains. For the purpose of risk characterization, the CPRC recommended, a low dose extrapolation for the quantification of human risk, based on the combined incidence of adenoma/carcinoma in the lungs of male mice.

The Hazard Identification Assessment Committee, classified Fenoxycarb as a **"likely"** human carcinogen according to EPA *Proposed Guidelines for Carcinogen Risk Assessment* (April 10, 1996). This classification was based on the lung and Harderian gland tumors seen in the first mouse study and on the lung and liver tumors seen in the second mice study. The Committee recommended that the human slope factor ( $Q_1^*$ ) be based on the combined incidence of adenomas/carcinomas in the lungs and should be used for quantification of human risk.

## V. FQPA CONSIDERATIONS

### 1. Neurotoxicity Data

No acute or subchronic neurotoxicity study in rats was available.

### 2. Determination of Susceptibility

In the prenatal developmental toxicity studies in rats and rabbits, there was no indication of increased susceptibility of the young animals to pre- and/or postnatal exposure to Fenoxycarb. However, in the two-generation reproduction study in rats, days 7 and/or 21 pup body weight decreases were observed at all dose levels tested, even at doses that were not toxic to the parental animals, suggesting increased susceptibility of the offspring following Fenoxycarb exposure. It was noted that parental toxicity, specifically liver histopathology, was not adequately examined at the low- and mid-dose levels; therefore, an adequate assessment of susceptibility could not be completed.

(i) Developmental Toxicity

In a prenatal developmental toxicity study, Albino rats received oral administration of Fenoxycarb (98%) in vehicle solution (comprised of 4% carboxymethyl cellulose, 0.9% sodium chloride, 0.5% benzylalcohol, and 0.4% Tween 80 in distilled water at 10 ml/kg) at doses of 0, 50, 150, or 500 mg/kg/day during gestation days 7 through 16. The day of mating was designated gestation day 1 and cesarean sections were performed on gestation day 21 until 15 pregnant females per group were attained; the remaining females (16-18/group) were allowed to deliver their litters and were killed at lactation day 21. No maternal toxicity was observed; for maternal toxicity, the NOEL was  $\geq 500$  mg/kg/day; a LOEL was not established. Although a significant increase in early resorptions was observed at 500 mg/kg/day, this was considered not to be biologically significant by the RfD Committee of 7/7/94. Therefore, for developmental toxicity, the NOEL was  $\geq 500$  mg/kg/day; a LOEL was not established (MRID No. 00131346).

*In utero* exposure to Fenoxycarb did not affect the postnatal growth or survival of pups in those litters that were delivered. This study was considered to be adequate at the 1994 RfD Peer Review, even though no maternal or developmental toxicity was observed at only half the limit dose and only 15 litters per group were assessed. (unspecified author, 1983) [The HIARC did not judge this study to be adequate, but did not recommend that the study be repeated, since it is unlikely that any additional critical information would be gained from a second study with dose groups up to 1000 mg/kg/day (the limit dose). The rabbit is the more sensitive species for the identification of developmental effects, and rats are assessed in the 2-generation reproduction study] (MRID No 00131346).

In a prenatal developmental toxicity study, Swiss hare rabbits (20/group) received oral administration of Fenoxycarb (98%) at doses of 0, 30, 100, 200, or 300 mg/kg/day in a vehicle solution (comprised of 4% carboxymethylcellulose, 0.9% sodium chloride, 0.5% benzylalcohol, and 0.4% Tween 80 in distilled water at 5 ml/kg) during gestation days 7 through-19. (The 200 mg/kg/day dose group was examined in a second controlled study.) Cesarean section was performed on gestation day 29. For maternal toxicity, the NOEL was 100 mg/kg/day and the LOEL was 200 mg/kg/day, based on reduced body weight gain during treatment at 200 and 300 mg/kg/day, with a rebound effect at 300 mg/kg/day. For developmental toxicity, the NOEL was 200 mg/kg/day and the LOEL was 300 mg/kg/day, based on a slightly increased incidence of fetal malformations: spina bifida (3 fetuses in 3 litters) and "possibly" hypoplastic tails (4 fetuses in 3 litters) (MRID No. 00153125)

In a two-generation reproduction study in rats Fenoxycarb (96.6%) was administered to Sprague-Dawley rats (25/sex/group) at dietary concentrations of 200, 600, or 1800 ppm (approximately 16, 47, or 140 mg/kg/day). According to the DER, the parental systemic NOEL could not be determined since livers were not evaluated histopathologically at all doses; the RfD Committee of 7/7/94 determined that the parental systemic NOEL was 200 ppm, and the parental systemic LOEL was 600 ppm, based on statistically and biologically significant reductions in P generation body weight gain. The HIARC reconsidered this finding, and determined that the conclusion of the DER (p. 10) are more accurate than those of the RfD Committee, and reversed that decision. Also, although there was significantly decreased day 7 and/or day 21 pup weight at all dose levels tested, the RfD Committee (7/7/94) determined that the NOEL for offspring toxicity was 600 ppm, and the LOEL for offspring toxicity was 800 ppm. The HIARC concluded that no offspring NOEL was established by this study. The DER presents information from the registrant which provides a derived NOEL using analysis of variance and regression; the derived-NOELs for the F1 and F2 generations were  $39 \pm 28.87$  and  $83 \pm 13.66$  ppm, respectively, based upon pup body weight decrements. **Due to the lack of adequate information to establish the parental NOEL, and in the presence of offspring toxicity at all dose levels, this study was considered unacceptable by the HIARC (MRID No. 40376903).**

### 3. Recommendation for a Developmental Neurotoxicity Study

Based on the following weight-of-the-evidence consideration, the Committee determined that a developmental neurotoxicity study is *required*. Since the two-generation reproduction study in rats was unacceptable, the Committee suggested that, at the discretion of the Registrant, the developmental neurotoxicity study in rats could be conducted in conjunction with the two-generation reproduction study.

#### (i) Evidence that support requiring a developmental neurotoxicity study:

- SAR: Fenoxycarb is a carbamate.
- Some evidence of neurotoxicity was evident in the data package but was not fully examined:
- Behavioral alterations that were indicative of neurotoxicity were noted in the developmental toxicity study in rats (nervous behavior at 150 and 500 mg/kg/day).
- Alterations to brain weight were observed in the 1-year dog study (absolute decrease in high-dose males, with a significant body weight decrement)

- Indication of brain weight deficits in F1 pups were observed in the two-generation reproduction study in rats; however, these did not occur in a dose-related manner and were not consistent across generations.
- Evidence of cholinesterase inhibition was observed in the 13-week rat study (females only; ChEI NOEL/LOEL = 80/250 mg/kg/day); it did not appear to be measured in any other study; however, from this information one could assume that Fenoxycarb is not a very potent cholinesterase inhibitor
- There was evidence of neural tube defects in the prenatal developmental toxicity study in rabbits (spina bifida and hypoplastic tails)

(ii). Evidence that do not support asking for a developmental neurotoxicity study:

- There is no evidence of neuropathology in the central or peripheral nervous system tissues (nonperfused) in the chronic or subchronic studies.
- There was no evidence of abnormalities in the development of the fetal nervous system in the prenatal developmental toxicity study in rats, although maternally toxic doses were not achieved (up to 500 mg/kg/day)
- In the two-generation reproduction study in rats, functional testing on postnatal day 21 (grip strength, papillary reflex of both eyes, visual placing response, and auditory response) did not identify any abnormalities in functional development; however, neuropathology was not evaluated in these pups, nor were they examined for behavioral, learning, or motor deficits at any time point other than postnatal day 21.

(iv). Unknown Factors

- No acute or subchronic neurotoxicity study in rats was available for review, to more adequately assess the neuropathology of Fenoxycarb in that species, following perfusion of tissues.

## VI. DATA GAPS

Acute Neurotoxicity Study §81-8

Two-Generation Reproduction Study §83-4

## VII. SUMMARY OF TOXICOLOGY ENDPOINT SELECTION

The doses and toxicological endpoints selected and Margins of Exposures for various exposure scenarios are summarized below.

EXPOSURE SCENARIO	DOSE (mg/kg/day)	ENDPOINT	STUDY	MOE
Acute Dietary	Developmental NOEL =200.0	Developmental toxicity	Developmental Toxicity - Rabbit	1000
Chronic Dietary	Not Applicable	Inadequate data base. Cannot establish a Reference Dose		
Short-Term (Dermal)	Developmental NOEL =200.0	Increased incidence of spinal bifida and hypoplastic tail	Developmental Toxicity	1000
Intermediate-Term (Dermal)	Developmental NOEL=200.0	Increased incidence of spinal bifida and hypoplastic tail	Developmental Toxicity	1000
Long-Term (Dermal)	Not Applicable	Based on use pattern, long-term dermal exposure is not anticipated; risk assessment not required		
Inhalation (Any Time Period)	NOEL= 1.13 mg/L	Systemic toxicity	21-Day Inhalation Toxicity	1000

## VIII. ACUTE TOXICITY:

Guideline No.	Study Type	MRID NO.	Results	Toxicity Category
81-1	Acute Oral	00247925	LD <sub>50</sub> = > 1000 mg/kg	IV
81-2	Acute Dermal	00247925	LD <sub>50</sub> > 2000 mg/kg	IV
81-3	Acute Inhalation	42343802	LC <sub>50</sub> = > 4.434 mg/L	III
81-4	Primary Eye Irritation	00247925	Mild redness	III
81-5	Primary Skin Irritation	NA		
81-6	Dermal Sensitization	00247925	Non-sensitizer	N/A

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<b>Chemical:</b>	<b>Fenoxycarb</b>
<b>PC Code:</b>	<b>125301</b>
<b>HED File Code</b>	<b>21100 HIARC</b>
<b>Memo Date:</b>	<b>02/25/1998</b>
<b>File ID:</b>	<b>TX012508</b>
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