SECTION-1: Identification of the substance / mixture and the company / undertaking

Catalogue Number	CS-O-11267
Product Name	Vigabatrin
CAS No.	60643-86-9
Category	API
Synonyms	; Gamma-Vinyl-GABA
Brand	Clearsynth Labs Ltd.
Identified uses	Laboratory Chemicals
Uses advised against	Not available
Company	Clearsynth Labs Ltd. Mumbai, India
Emergency Phone #	+91-22-245045900
REACH No.	Not available

SECTION 2: Hazards identification

Disclaimer: This is sample MSDS. Please email **sales@clearsynth.com** for more details.

2.1 Classification of the substance or mixture-Regulation (EC) No 1272/2008:

Skin irritation (Category 2)
Serious eye damage/eye irritation (Category 2)

2.2 Label Elements

Signal Word: Warning

Hazard Statement(s)

Code	Statement
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H335	Not available

Precautionary Statement(s)

Code	Statement
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.
P264	Wash hands thoroughly after handling.
P264+P265	Not available
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if presen
P319	Get medical help if you feel unwell.
P321	Specific treatment (see … on this label).
P332+P317	If skin irritation occurs: Get medical help.
P337+P317	If eye irritation persists: Get medical help.
P362+P364	Take off contaminated clothing and wash it before reuse.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.
P405	Store locked up.
P501	Dispose of contents/container in accordance with local/regional/national/international regulatio

SECTION 3: Composition / information on ingredients

3.1 Substance

Component : Vigabatrin
CAS Number : 60643-86-9
Molecular Formula : C6H11NO2
Molecular Weight : 129.2
Parent Chemical : Vigabatrin
Synonyms : ; Gamma-Vinyl-GABA
Concentration : Not available

SECTION 4: First aid measures

SECTION 4: First-aid measures

4.1 Description of first aid measures
General advice:

- Seek medical attention if symptoms occur or persist.
- Show this Safety Data Sheet to the physician in attendance.

Inhalation:
- Move person to fresh air.
- If breathing is difficult, seek medical attention.

Skin contact:
- Wash with plenty of soap and water.
- Remove contaminated clothing and wash before reuse.
- Seek medical attention if irritation develops.

Eye contact:
- Rinse cautiously with water for several minutes.
- Remove contact lenses if present and easy to do; continue rinsing.
- Seek medical attention if irritation persists.

Ingestion:
- Rinse mouth with water.
- Do not induce vomiting unless directed by medical personnel.
- Seek medical attention.

4.2 Most important symptoms and effects, both acute and delayed
- Not available.

4.3 Indication of any immediate medical attention and special treatment needed
- Treat symptomatically.
- No data available.

SECTION 5: Firefighting measures

SECTION 5: Fire-fighting measures

5.1 Extinguishing media
Suitable extinguishing media:
- Water spray, alcohol-resistant foam, dry chemical, or carbon dioxide.
Unsuitable extinguishing media:
- Not available.

5.2 Special hazards arising from the substance or mixture
- No data available.
- Hazardous combustion products: Not available.

5.3 Advice for firefighters
- Wear self-contained breathing apparatus and full protective gear.
- Use water spray to cool unopened containers.
- Avoid inhalation of combustion products.

SECTION 6: Accidental release measures

SECTION 6: Accidental release measures

6.1 Personal precautions, protective equipment and emergency procedures
- Avoid breathing dust.
- Avoid contact with skin and eyes.
- Use appropriate personal protective equipment (see Section 8).
- Ensure adequate ventilation.

6.2 Environmental precautions
- Avoid release to the environment.
- Prevent further leakage or spillage if safe to do so.

6.3 Methods and material for containment and cleaning up
- Avoid generating dust.
- Collect spilled material using suitable means (e.g., HEPA-filtered vacuum or damp method) and place in a suitable, closed container for disposal.
- Clean contaminated area with water and detergent as appropriate.

6.4 Reference to other sections
- For disposal considerations, see Section 13.
- For personal protective equipment, see Section 8.

SECTION-7: Handling and storage

SECTION 7: Handling and storage

7.1 Precautions for safe handling
- Handle in accordance with good industrial hygiene and safety practice.
- Avoid formation of dust and aerosols.
- Avoid contact with skin, eyes, and clothing.
- Do not breathe dust.
- Use with adequate ventilation.

7.2 Conditions for safe storage, including any incompatibilities
- Store in a tightly closed container.
- Store in a cool, dry, well-ventilated place.
- Protect from moisture.
- Incompatible materials: Not available.

7.3 Specific end use(s)
- API / laboratory and research use. Not for food, drug, or household use unless otherwise specified by the supplier.

SECTION 8: Exposure controls / personal protection

SECTION 8: Exposure controls/personal protection

8.1 Control parameters
Occupational exposure limits:
- Not available.
Biological limit values:
- Not available.

8.2 Exposure controls

Engineering controls:
- Use local exhaust ventilation or other engineering controls to maintain airborne levels below applicable exposure limits.
- If no exposure limits are established, use general ventilation and minimize dust generation.

Personal protective equipment (PPE):
Eye/face protection:
- Safety glasses with side shields or chemical splash goggles.
Skin protection:
- Protective gloves (material not available).
- Lab coat or protective clothing.
Respiratory protection:
- If dust is generated or ventilation is inadequate, use a suitable particulate respirator.
Hygiene measures:
- Wash hands thoroughly after handling.
- Do not eat, drink, or smoke when using this product.

SECTION 9: Physical and chemical properties

9.1 Information on basic physical and chemical properties

Test	Result
Appearance	No data available
IR spectrum	No data available
pH	No data available
Solubility	No data available

Property	Value
a) Physical State	No data available
b) Color	No data available
c) Odor	No data available
d) pH	No data available
e) Vapour Pressure	No data available
f) Viscosity	No data available
g) Initial Boiling Point and boiling range	No data available
h) Melting Point / Freezing Point	No data available
i) Auto Ignition Temperature	No data available

Property	Value
j) Flash Point	No data available
k) Explosion Limit, Lower	No data available
l) Explosion Limit, Upper	No data available
m) Decomposition Temperature	No data available
n) Loss on Drying	No data available
o) Relative Density	No data available
p) Solubility (in DMSO)	No data available
q) Oxidizing Properties	No data available

SECTION 10: Stability and reactivity

SECTION 10: Stability and reactivity

10.1 Reactivity
- No data available.

10.2 Chemical stability
- Stable under recommended storage conditions.

10.3 Possibility of hazardous reactions
- No data available.

10.4 Conditions to avoid
- Avoid dust formation.
- Avoid exposure to moisture (if applicable).
- Other conditions: Not available.

10.5 Incompatible materials
- Not available.

10.6 Hazardous decomposition products
- Not available.

SECTION 11: Toxicological information

11.1 Information on toxicological effects
- Acute toxicity: Signs and Symptoms of Overdose Vigabatrin toxicity typically develops gradually as a result of prolonged treatment. One case of acute toxicity involved a 25-year-old patient who attempted suicide by consuming 120 vigabatrin tablets (500 mg each). She had a history of refractory seizures, for which a temporal lobectomy was performed. After the surgery, she was placed on phenytoin, carbamazepine, and vigabatrin. The patient was admitted to the hospital after consuming the tablets. She was found to be very agitated and combative, requiring physical restraint. She had impaired concentration and was disoriented to time and place. Given the findings, she received a diagnosis of vigabatrin-induced delirium. No specific antidote was administered to reverse the toxicity. She was treated symptomatically with diazepam and haloperidol. Forty-eight hours later, the patient recovered but

could not recall the series of events that occurred. Her renal and hepatic parameters remained normal throughout the admission. Unconsciousness, drowsiness, or coma have been described in most cases of vigabatrin overdose. Less commonly reported symptoms include psychosis, vertigo, bradycardia, apnea, respiratory depression, agitation, headache, irritability, confusion, hypotension, abnormal behavior, increased seizure activity, speech disorder, or status epilepticus. These symptoms were resolved with supportive care. Management of Overdose During a vigabatrin overdose, supportive measures include monitoring vital signs and assessing the patient's clinical condition regularly to ensure proper management and intervention as needed. Given vigabatrin's pharmacokinetic profile, hemodialysis would significantly accelerate drug extraction and reduce vigabatrin plasma concentrations by 40% to 60%, making it a potential intervention for overdoses. LD50, oral, rat: 3000 mg/kg
- Skin corrosion/irritation: No data available.
- Serious eye damage/eye irritation: No data available.
- Respiratory or skin sensitization: No data available.
- Germ cell mutagenicity: IDENTIFICATION AND USE: Vigabatrin is a structural analog of gamma-aminobutyric acid (GABA), the primary inhibitory neurotransmitter in the CNS. Vigabatrin is commercially available as a racemic mixture of 2 enantiomers; the S enantiomer is pharmacologically active and the R enantiomer is inactive. HUMAN STUDIES: Visual field defects, including permanent vision loss, have been reported in infants, children, and adults receiving vigabatrin. Based on clinical studies in adults, bilateral concentric visual field constriction ranging in severity from mild to severe may occur in 30% or more of patients receiving the drug. Severe cases may be characterized by tunnel vision to within 10 degrees of visual fixation, which can lead to disability. In some cases, vigabatrin can also damage the central retina and decrease visual acuity. Coma, unconsciousness, and/or drowsiness were described in the majority of cases of vigabatrin overdose. Other less commonly reported symptoms included vertigo, psychosis, apnea or respiratory depression, bradycardia, agitation, irritability, confusion, headache, hypotension, abnormal behavior, increased seizure activity, status epilepticus, and speech disorder. These symptoms resolved with supportive care. ANIMAL STUDIES: Vigabatrin showed no carcinogenic potential in mouse or rat when given in the diet at doses up to 150 mg/kg/day for 18 months (mouse) or at doses up to 150 mg/kg/day for 2 years (rat). Vigabatrin (300 or 450 mg/kg) was administered by intraperitoneal injection to a mutant mouse strain on a single day during organogenesis (day 7, 8, 9, 10, 11, or 12). An increase in malformations (including cleft palate) was observed at both doses. In rats, oral administration of vigabatrin (50, 100, or 150 mg/kg) throughout organogenesis resulted in decreased fetal body weights and increased incidences of fetal anatomic variations. Oral administration of vigabatrin (50, 100, 150 mg/kg) to rats from the latter part of pregnancy through weaning produced long-term neurohistopathological (hippocampal vacuolation) and neurobehavioral (convulsions) abnormalities in the offspring. Administration of vigabatrin (oral doses of 50 to 200 mg/kg) to pregnant rabbits throughout the period of organogenesis was associated with an increased incidence of malformations (cleft palate) and embryo-fetal death; these findings were observed in two separate studies. No adverse effects on male or female fertility were observed in rats at oral doses up to 150 mg/kg/day. Oral administration of vigabatrin (5, 15, or 50 mg/kg) to young rats during the neonatal and juvenile periods of development (postnatal days 4-65) produced neurobehavioral (convulsions, neuromotor impairment, learning deficits) and neurohistopathological (brain vacuolation, decreased myelination, and retinal dysplasia) abnormalities in treated animals. The early postnatal period in rats is generally thought to correspond to late pregnancy in humans in terms of brain development. Vigabatrin was negative in in vitro (Ames, CHO/HGPRT mammalian cell forward gene mutation, chromosomal aberration in rat lymphocytes) and in in vivo (mouse bone marrow micronucleus) assays.
- Carcinogenicity: IDENTIFICATION AND USE: Vigabatrin is a structural analog of gamma-aminobutyric acid (GABA), the primary inhibitory neurotransmitter in the CNS. Vigabatrin is commercially available as a racemic mixture of 2 enantiomers; the S enantiomer is pharmacologically active and the R enantiomer is inactive. HUMAN STUDIES: Visual field defects, including permanent vision loss, have been reported in infants, children, and adults receiving vigabatrin. Based on clinical studies in adults, bilateral concentric visual field constriction ranging in

severity from mild to severe may occur in 30% or more of patients receiving the drug. Severe cases may be characterized by tunnel vision to within 10 degrees of visual fixation, which can lead to disability. In some cases, vigabatrin can also damage the central retina and decrease visual acuity. Coma, unconsciousness, and/or drowsiness were described in the majority of cases of vigabatrin overdose. Other less commonly reported symptoms included vertigo, psychosis, apnea or respiratory depression, bradycardia, agitation, irritability, confusion, headache, hypotension, abnormal behavior, increased seizure activity, status epilepticus, and speech disorder. These symptoms resolved with supportive care. ANIMAL STUDIES: Vigabatrin showed no carcinogenic potential in mouse or rat when given in the diet at doses up to 150 mg/kg/day for 18 months (mouse) or at doses up to 150 mg/kg/day for 2 years (rat). Vigabatrin (300 or 450 mg/kg) was administered by intraperitoneal injection to a mutant mouse strain on a single day during organogenesis (day 7, 8, 9, 10, 11, or 12). An increase in malformations (including cleft palate) was observed at both doses. In rats, oral administration of vigabatrin (50, 100, or 150 mg/kg) throughout organogenesis resulted in decreased fetal body weights and increased incidences of fetal anatomic variations. Oral administration of vigabatrin (50, 100, 150 mg/kg) to rats from the latter part of pregnancy through weaning produced long-term neurohistopathological (hippocampal vacuolation) and neurobehavioral (convulsions) abnormalities in the offspring. Administration of vigabatrin (oral doses of 50 to 200 mg/kg) to pregnant rabbits throughout the period of organogenesis was associated with an increased incidence of malformations (cleft palate) and embryo-fetal death; these findings were observed in two separate studies. No adverse effects on male or female fertility were observed in rats at oral doses up to 150 mg/kg/day. Oral administration of vigabatrin (5, 15, or 50 mg/kg) to young rats during the neonatal and juvenile periods of development (postnatal days 4-65) produced neurobehavioral (convulsions, neuromotor impairment, learning deficits) and neurohistopathological (brain vacuolation, decreased myelination, and retinal dysplasia) abnormalities in treated animals. The early postnatal period in rats is generally thought to correspond to late pregnancy in humans in terms of brain development. Vigabatrin was negative in in vitro (Ames, CHO/HGPRT mammalian cell forward gene mutation, chromosomal aberration in rat lymphocytes) and in in vivo (mouse bone marrow micronucleus) assays. /LABORATORY ANIMALS: Chronic Exposure or Carcinogenicity/ Vigabatrin showed no carcinogenic potential in mouse or rat when given in the diet at doses up to 150 mg/kg/day for 18 months (mouse) or at doses up to 150 mg/kg/day for 2 years (rat). These doses are less than the maximum recommended human dose (MRHD) for infantile spasms (150 mg/kg/day) and for refractory complex partial seizures (3 g/day) on a mg/sq m basis.

- Reproductive toxicity: IDENTIFICATION AND USE: Vigabatrin is a structural analog of gamma-aminobutyric acid (GABA), the primary inhibitory neurotransmitter in the CNS. Vigabatrin is commercially available as a racemic mixture of 2 enantiomers; the S enantiomer is pharmacologically active and the R enantiomer is inactive. HUMAN STUDIES: Visual field defects, including permanent vision loss, have been reported in infants, children, and adults receiving vigabatrin. Based on clinical studies in adults, bilateral concentric visual field constriction ranging in severity from mild to severe may occur in 30% or more of patients receiving the drug. Severe cases may be characterized by tunnel vision to within 10 degrees of visual fixation, which can lead to disability. In some cases, vigabatrin can also damage the central retina and decrease visual acuity. Coma, unconsciousness, and/or drowsiness were described in the majority of cases of vigabatrin overdose. Other less commonly reported symptoms included vertigo, psychosis, apnea or respiratory depression, bradycardia, agitation, irritability, confusion, headache, hypotension, abnormal behavior, increased seizure activity, status epilepticus, and speech disorder. These symptoms resolved with supportive care. ANIMAL STUDIES: Vigabatrin showed no carcinogenic potential in mouse or rat when given in the diet at doses up to 150 mg/kg/day for 18 months (mouse) or at doses up to 150 mg/kg/day for 2 years (rat). Vigabatrin (300 or 450 mg/kg) was administered by intraperitoneal injection to a mutant mouse strain on a single day during organogenesis (day 7, 8, 9, 10, 11, or 12). An increase in malformations (including cleft palate) was observed at both doses. In rats, oral administration of vigabatrin (50, 100, or 150 mg/kg) throughout organogenesis resulted in decreased fetal body weights and increased incidences of fetal anatomic variations. Oral administration of vigabatrin (50, 100, 150 mg/kg) to rats from the latter part of pregnancy through weaning produced

long-term neurohistopathological (hippocampal vacuolation) and neurobehavioral (convulsions) abnormalities in the offspring. Administration of vigabatrin (oral doses of 50 to 200 mg/kg) to pregnant rabbits throughout the period of organogenesis was associated with an increased incidence of malformations (cleft palate) and embryo-fetal death; these findings were observed in two separate studies. No adverse effects on male or female fertility were observed in rats at oral doses up to 150 mg/kg/day. Oral administration of vigabatrin (5, 15, or 50 mg/kg) to young rats during the neonatal and juvenile periods of development (postnatal days 4-65) produced neurobehavioral (convulsions, neuromotor impairment, learning deficits) and neurohistopathological (brain vacuolation, decreased myelination, and retinal dysplasia) abnormalities in treated animals. The early postnatal period in rats is generally thought to correspond to late pregnancy in humans in terms of brain development. Vigabatrin was negative in in vitro (Ames, CHO/HGPRT mammalian cell forward gene mutation, chromosomal aberration in rat lymphocytes) and in in vivo (mouse bone marrow micronucleus) assays. /LABORATORY ANIMALS: Developmental or Reproductive Toxicity/ Vigabatrin (300 or 450 mg/kg) was administered by intraperitoneal injection to a mutant mouse strain on a single day during organogenesis (day 7, 8, 9, 10, 11, or 12). An increase in malformations (including cleft palate) was observed at both doses.
- STOT-single exposure: No data available.
- STOT-repeated exposure: IDENTIFICATION AND USE: Vigabatrin is a structural analog of gamma-aminobutyric acid (GABA), the primary inhibitory neurotransmitter in the CNS. Vigabatrin is commercially available as a racemic mixture of 2 enantiomers; the S enantiomer is pharmacologically active and the R enantiomer is inactive. HUMAN STUDIES: Visual field defects, including permanent vision loss, have been reported in infants, children, and adults receiving vigabatrin. Based on clinical studies in adults, bilateral concentric visual field constriction ranging in severity from mild to severe may occur in 30% or more of patients receiving the drug. Severe cases may be characterized by tunnel vision to within 10 degrees of visual fixation, which can lead to disability. In some cases, vigabatrin can also damage the central retina and decrease visual acuity. Coma, unconsciousness, and/or drowsiness were described in the majority of cases of vigabatrin overdose. Other less commonly reported symptoms included vertigo, psychosis, apnea or respiratory depression, bradycardia, agitation, irritability, confusion, headache, hypotension, abnormal behavior, increased seizure activity, status epilepticus, and speech disorder. These symptoms resolved with supportive care. ANIMAL STUDIES: Vigabatrin showed no carcinogenic potential in mouse or rat when given in the diet at doses up to 150 mg/kg/day for 18 months (mouse) or at doses up to 150 mg/kg/day for 2 years (rat). Vigabatrin (300 or 450 mg/kg) was administered by intraperitoneal injection to a mutant mouse strain on a single day during organogenesis (day 7, 8, 9, 10, 11, or 12). An increase in malformations (including cleft palate) was observed at both doses. In rats, oral administration of vigabatrin (50, 100, or 150 mg/kg) throughout organogenesis resulted in decreased fetal body weights and increased incidences of fetal anatomic variations. Oral administration of vigabatrin (50, 100, 150 mg/kg) to rats from the latter part of pregnancy through weaning produced long-term neurohistopathological (hippocampal vacuolation) and neurobehavioral (convulsions) abnormalities in the offspring. Administration of vigabatrin (oral doses of 50 to 200 mg/kg) to pregnant rabbits throughout the period of organogenesis was associated with an increased incidence of malformations (cleft palate) and embryo-fetal death; these findings were observed in two separate studies. No adverse effects on male or female fertility were observed in rats at oral doses up to 150 mg/kg/day. Oral administration of vigabatrin (5, 15, or 50 mg/kg) to young rats during the neonatal and juvenile periods of development (postnatal days 4-65) produced neurobehavioral (convulsions, neuromotor impairment, learning deficits) and neurohistopathological (brain vacuolation, decreased myelination, and retinal dysplasia) abnormalities in treated animals. The early postnatal period in rats is generally thought to correspond to late pregnancy in humans in terms of brain development. Vigabatrin was negative in in vitro (Ames, CHO/HGPRT mammalian cell forward gene mutation, chromosomal aberration in rat lymphocytes) and in in vivo (mouse bone marrow micronucleus) assays. /LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ Long-term administration of antiepileptic drugs may be connected with the risk of impairment of bone remodeling. Contrary to the reported unfavorable effect of classic antiepileptic drugs on bone metabolism, little is known about

the effect of the next generation antiepileptics on bone remodeling. The aim of the present study was to investigate the effect of vigabatrin, as a representative of new antiepileptics, on the skeletal system of young rats, in comparison with conventional drugs--phenytoin and valproic acid. The experiments were carried out on 4-week-old male Wistar rats, divided into the control rats, and rats receiving vigabatrin (250 mg/kg p.o. daily), phenytoin (20 mg/kg p.o. daily) or valproic acid (250 mg/kg p.o. daily). The drugs were administered for 28 days. Histomorphometric parameters of the tibia and femur, mechanical properties of the femur, and bone length, diameter, mass, content of mineral substances and calcium were examined. After administration of phenytoin or valproic acid, the investigated bone parameters did not significantly differ from those observed in the control rats. Administration of vigabatrin caused profound impairment of bone accrual with impairment of bone histomorphometric parameters, along with the significant decrease in the body mass gain.
- Aspiration hazard: No data available.

Likely routes of exposure
- /SIGNS AND SYMPTOMS/ Confirmed and/or suspected vigabatrin overdoses have been reported during clinical trials and in post marketing surveillance. No vigabatrin overdoses resulted in death. When reported, the vigabatrin dose ingested ranged from 3 g to 90 g, but most were between 7.5 g and 30 g. Nearly half the cases involved multiple drug ingestions including carbamazepine, barbiturates, benzodiazepines, lamotrigine, valproic acid, acetaminophen, and/or chlorpheniramine. Coma, unconsciousness, and/or drowsiness were described in the majority of cases of vigabatrin overdose. Other less commonly reported symptoms included vertigo, psychosis, apnea or respiratory depression, bradycardia, agitation, irritability, confusion, headache, hypotension, abnormal behavior, increased seizure activity, status epilepticus, and speech disorder. These symptoms resolved with supportive care.

Symptoms related to the physical, chemical and toxicological characteristics
- IDENTIFICATION AND USE: Vigabatrin is a structural analog of gamma-aminobutyric acid (GABA), the primary inhibitory neurotransmitter in the CNS. Vigabatrin is commercially available as a racemic mixture of 2 enantiomers; the S enantiomer is pharmacologically active and the R enantiomer is inactive. HUMAN STUDIES: Visual field defects, including permanent vision loss, have been reported in infants, children, and adults receiving vigabatrin. Based on clinical studies in adults, bilateral concentric visual field constriction ranging in severity from mild to severe may occur in 30% or more of patients receiving the drug. Severe cases may be characterized by tunnel vision to within 10 degrees of visual fixation, which can lead to disability. In some cases, vigabatrin can also damage the central retina and decrease visual acuity. Coma, unconsciousness, and/or drowsiness were described in the majority of cases of vigabatrin overdose. Other less commonly reported symptoms included vertigo, psychosis, apnea or respiratory depression, bradycardia, agitation, irritability, confusion, headache, hypotension, abnormal behavior, increased seizure activity, status epilepticus, and speech disorder. These symptoms resolved with supportive care. ANIMAL STUDIES: Vigabatrin showed no carcinogenic potential in mouse or rat when given in the diet at doses up to 150 mg/kg/day for 18 months (mouse) or at doses up to 150 mg/kg/day for 2 years (rat). Vigabatrin (300 or 450 mg/kg) was administered by intraperitoneal injection to a mutant mouse strain on a single day during organogenesis (day 7, 8, 9, 10, 11, or 12). An increase in malformations (including cleft palate) was observed at both doses. In rats, oral administration of vigabatrin (50, 100, or 150 mg/kg) throughout organogenesis resulted in decreased fetal body weights and increased incidences of fetal anatomic variations. Oral administration of vigabatrin (50, 100, 150 mg/kg) to rats from the latter part of pregnancy through weaning produced long-term neurohistopathological (hippocampal vacuolation) and neurobehavioral (convulsions) abnormalities in the offspring. Administration of vigabatrin (oral doses of 50 to 200 mg/kg) to pregnant rabbits throughout the period of organogenesis was associated with an increased incidence of malformations (cleft palate) and embryo-fetal death; these findings were observed in two separate studies. No adverse effects on male or female fertility were observed in rats at oral doses up to 150 mg/kg/day. Oral administration of vigabatrin (5, 15, or 50 mg/kg) to young rats during

the neonatal and juvenile periods of development (postnatal days 4-65) produced neurobehavioral (convulsions, neuromotor impairment, learning deficits) and neurohistopathological (brain vacuolation, decreased myelination, and retinal dysplasia) abnormalities in treated animals. The early postnatal period in rats is generally thought to correspond to late pregnancy in humans in terms of brain development. Vigabatrin was negative in in vitro (Ames, CHO/HGPRT mammalian cell forward gene mutation, chromosomal aberration in rat lymphocytes) and in in vivo (mouse bone marrow micronucleus) assays.

SECTION 12: Ecological information

SECTION 12: Ecological information

12.1 Toxicity
- No data available.

12.2 Persistence and degradability
- No data available.

12.3 Bioaccumulative potential
- No data available.

12.4 Mobility in soil
- No data available.

12.5 Results of PBT and vPvB assessment
- Not available.

12.6 Endocrine disrupting properties
- Not available.

12.7 Other adverse effects
- No data available.

SECTION 13: Disposal considerations

SECTION 13: Disposal considerations

13.1 Waste treatment methods
Product:
- Dispose of contents/container in accordance with local/regional/national/international regulations.
- Do not discharge to drains.

Contaminated packaging:
- Dispose of as unused product.

Waste code:
- Not available.

SECTION 14: Transport information

SECTION 14: Transport information

14.1 UN number

- Not available.

14.2 UN proper shipping name
- Not available.

14.3 Transport hazard class(es)
- Not available.

14.4 Packing group
- Not available.

14.5 Environmental hazards
- Not available.

14.6 Special precautions for user
- Not available.

14.7 Maritime transport in bulk according to IMO instruments
- Not available.

SECTION 15: Regulatory information

SECTION 15: Regulatory information

15.1 Safety, health and environmental regulations/legislation specific for the substance or mixture
- Not available.

15.2 Chemical safety assessment
- No data available.

SECTION 16: Other information

SECTION 16: Other information

Product identifier:
- Product name: Vigabatrin
- CAS No.: 60643-86-9
- Catalog No.: CS-O-11267
- Synonyms: Gamma-Vinyl-GABA
- Supplier: Clearsynth Labs Ltd., Mumbai, India
- Emergency phone: +91-22-245045900

Revision information:
- Not available.

Disclaimer:
- The information provided is believed to be accurate based on available data; however, no warranty is expressed or implied. The user is responsible for determining suitability for a particular purpose and for complying with applicable laws and regulations.

DISCLAIMER

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