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

Catalogue Number	CS-ED-00324
Product Name	Perfluorohexane Sulfonic Acid
CAS No.	355-46-4
Category	Fine Chemicals
Synonyms	Perfluorohexanesulfonic acid; Perfluorohexane-1-sulphonic acid;
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)
Acute toxicity (Category 4)

2.2 Label Elements

Signal Word: Warning

Hazard Statement(s)

Code	Statement
H302	Harmful if swallowed.
H312	Harmful in contact with skin.

H314	Not available
H332	Harmful if inhaled.
H373	Not available
H315	Causes skin irritation.
H319	Causes serious eye irritation.
H335	Not available

Precautionary Statement(s)

Code	Statement
P260	Not available
P261	Avoid breathing dust/fume/gas/mist/vapours/spray.
P264	Wash hands thoroughly after handling.
P270	Not available
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves/protective clothing/eye protection/face protection.
P301+P317	Not available
P301+P330+P331	Not available
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P302+P361+P354	Not available
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P305+P354+P338	Not available
P316	Not available
P317	Not available
P321	Specific treatment (see … on this label).
P330	Not available
P362+P364	Take off contaminated clothing and wash it before reuse.
P363	Not available
P405	Store locked up.
P501	Dispose of contents/container in accordance with local/regional/national/international regulatio
P319	Get medical help if you feel unwell.

P264+P265	Not available
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if presen
P332+P317	If skin irritation occurs: Get medical help.
P337+P317	If eye irritation persists: Get medical help.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

SECTION 3: Composition / information on ingredients

3.1 Substance

Component : Perfluorohexane Sulfonic Acid
CAS Number : 355-46-4
Molecular Formula : C■HF■■O■S
Molecular Weight : 400.11
Parent Chemical : .
Synonyms : Perfluorohexanesulfonic acid; Perfluorohexane-1-sulphonic acid;
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 SDS to the physician.
Inhalation: Move person to fresh air. Keep at rest. If breathing is difficult, seek medical attention.
Skin contact: Wash with plenty of soap and water. Remove contaminated clothing and wash before reuse. Get 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. 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: Use extinguishing measures appropriate to local circumstances and the surrounding environment (e.g., water spray, dry chemical, foam, carbon dioxide).
Unsuitable extinguishing media: Not available.

5.2 Special hazards arising from the substance or mixture

Hazardous combustion products: Not available. Thermal decomposition may produce irritating and/or toxic fumes.

5.3 Advice for firefighters
Wear self-contained breathing apparatus (SCBA) and full protective gear. Cool containers with water spray if exposed to fire. 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 contact with skin and eyes. Avoid breathing dust/mist/vapors. Use appropriate personal protective equipment (see Section 8). Ensure adequate ventilation.

6.2 Environmental precautions
Avoid release to the environment. Prevent entry into drains, surface water, and soil. Notify authorities if significant contamination occurs.

6.3 Methods and material for containment and cleaning up
Contain spill. Collect using inert absorbent material and place in a suitable, labeled container for disposal. Clean contaminated area with water and detergent as appropriate. Dispose of waste in accordance with local regulations.

6.4 Reference to other sections
See Sections 8 and 13.

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 contact with skin, eyes, and clothing. Avoid breathing dust/mist/vapors. Use with adequate ventilation. Wash hands thoroughly after handling.

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

7.3 Specific end use(s)
Fine chemical. No data available.

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: Provide adequate ventilation. Use local exhaust where dust/mist/vapor may be generated.
Personal protective equipment (PPE):

- Eye/face protection: Safety glasses with side shields or chemical splash goggles.
- Skin protection: Protective gloves (material selection dependent on use conditions). Protective clothing as needed.
- Respiratory protection: If ventilation is inadequate or exposure is possible, use appropriate respiratory protection.
- Hygiene measures: Wash hands after handling. Remove contaminated clothing and wash before reuse.

Environmental exposure controls: Avoid release to the environment.

SECTION 9: Physical and chemical properties

9.1 Information on basic physical and chemical properties

Test	Result
Appearance	Low melting solid
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
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

Property	Value
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 heat, moisture, and other conditions that may cause decomposition. No data available.

10.5 Incompatible materials
Not available.

10.6 Hazardous decomposition products
Not available. Thermal decomposition may produce irritating and/or toxic fumes.

SECTION 11: Toxicological information

11.1 Information on toxicological effects
- Acute toxicity: IDENTIFICATION AND USE: Perfluorohexane sulfonate (PFHxS) is a six-carbon compound in the perfluoroalkyl family of chemicals. Perfluorohexane sulfonate was once used in firefighting foam and carpet treatment solutions. It was once widely used as a stain and water repellent but was phased out due to U.S. Environmental Protection Agency regulations. HUMAN EXPOSURE AND TOXICITY: An epidemiological study evaluated the associations between serum levels of PFHxS and diagnosis of attention deficit/hyperactivity disorder (ADHD). In another study, the associations of prenatal serum concentrations (PFHxS) with fetal and postnatal growth in girls was examined. Serum samples were obtained from mothers during pregnancy. PFHxS (median, 1.6 ng/mL) was detected in 100% of samples. On average, girls born to mothers with prenatal concentrations of PFHxS in the upper tertile weighed 108 g less at birth than girls born to mothers with concentrations in the lower tertile in adjusted models. In another study, the association between levels of PFHxS and semen volume, sperm concentration, total sperm count, motility and morphology were assessed. The proportion of morphologically normal cells was 35% lower for the third tertile of PFHxS exposure as compared with the first. In a surveillance study, the serum concentrations of perfluoroalkyl acids (PFAAs) in firefighters was evaluated, and serum concentration of PFHxS was statistically higher in firefighters. Another study reported evidence of a significant association between PFHxS and total cholesterol (TC), low-density lipoprotein cholesterol (LDL), total cholesterol/high density lipoprotein cholesterol ratio (TC/HDL) and non-HDL cholesterol as well as an elevated odds of high cholesterol. In another study, which evaluated the relationship between serum PFCs and thyroid function, increase in natural log-PFHxS was associated with an increase of total T4 by 0.26 ug/mL and total T3 by 4.074 ng/dL in women and a decrease of natural log-free T4 by 0.016 (ng/dL) in men. ANIMAL STUDIES: A subchronic study investigated the mechanism underlying the effect of PFAS surfactants on lipoprotein metabolism. Mice were fed a Western-type diet with PFHxS,

(6 mg/kg/day) for 4-6 weeks. PFHxS markedly reduced TG, non-HDL-C, and HDL-C. The decrease in very low-density lipoprotein (VLDL) was caused by enhanced lipoprotein lipase-mediated VLDL-TG clearance and by decreased production of VLDL-TG and VLDL-apolipoprotein B. Reduced HDL production, related to decreased apolipoprotein AI synthesis, resulted in decreased HDL. PFHxS increased liver weight and hepatic TG content. Hepatic gene expression profiling data indicated that these effects were the combined result of peroxisome proliferator-activated receptor alpha and pregnane X receptor activation. Another study showed that neonatal exposure to PFHxS can alter neuroprotein levels, e.g. CaMKII, GAP-43, synaptophysin and tau, which are essential for normal brain development in mice. This was measured for both males and females, in hippocampus and cerebral cortex. The results suggest that PFHxS may act as a developmental neurotoxicant and the effects are similar to that of PFOS and PFOA, but also to other substances such as PCBs, PBDEs and bisphenol A. In another study, in ovo effects of PFHxS exposure (maximum dose = 38,000 ng/g egg) on embryonic death, developmental endpoints, tissue accumulation, mRNA expression in liver and cerebral cortex, and plasma TH levels were evaluated. Pipping success was reduced to 63% at the highest dose of PFHxS; additional effects included decreased tarsus length and embryo mass. Plasma TH levels were reduced in a concentration-dependent manner following PFHxS exposure. A subsequent study evaluated the relationship between PFHxS exposure and TH-dependent neurodevelopmental pathways. PFHxS significantly altered the expression of 11 transcripts at the low dose (890 ng/g) and 101 transcripts at the high dose (38,000 ng/g). Functional enrichment analysis showed that PFHxS affected the genes involved in tissue development and morphology, cellular assembly and organization, and cell-to-cell signaling. In another study, the effects of PFHxS on the neuronal cell death and the underlying mechanisms were examined using PC12 cells as a model of dopaminergic neuron. The treatment with PFHxS reduced cell viability in a dose-dependent manner. PFHxS increased cell apoptosis, increased the activations of ERK1/2, JNK and p38 MAPK with different temporal activations. PFHxS exposure also increased ROS formation. For more Human Toxicity Excerpts (Complete) data for Perfluorohexanesulfonic acid (17 total), please visit the HSDB record page.
- Skin corrosion/irritation: No data available.
- Serious eye damage/eye irritation: No data available.
- Respiratory or skin sensitization: No data available.
- Germ cell mutagenicity: No data available.
- Carcinogenicity: No data available.
- Reproductive toxicity: IDENTIFICATION AND USE: Perfluorohexane sulfonate (PFHxS) is a six-carbon compound in the perfluoroalkyl family of chemicals. Perfluorohexane sulfonate was once used in firefighting foam and carpet treatment solutions. It was once widely used as a stain and water repellent but was phased out due to U.S. Environmental Protection Agency regulations. HUMAN EXPOSURE AND TOXICITY: An epidemiological study evaluated the associations between serum levels of PFHxS and diagnosis of attention deficit/hyperactivity disorder (ADHD). In another study, the associations of prenatal serum concentrations (PFHxS) with fetal and postnatal growth in girls was examined. Serum samples were obtained from mothers during pregnancy. PFHxS (median, 1.6 ng/mL) was detected in 100% of samples. On average, girls born to mothers with prenatal concentrations of PFHxS in the upper tertile weighed 108 g less at birth than girls born to mothers with concentrations in the lower tertile in adjusted models. In another study, the association between levels of PFHxS and semen volume, sperm concentration, total sperm count, motility and morphology were assessed. The proportion of morphologically normal cells was 35% lower for the third tertile of PFHxS exposure as compared with the first. In a surveillance study, the serum concentrations of perfluoroalkyl acids (PFAAs) in firefighters was evaluated, and serum concentration of PFHxS was statistically higher in firefighters. Another study reported evidence of a significant association between PFHxS and total cholesterol (TC), low-density lipoprotein cholesterol (LDL), total cholesterol/high density lipoprotein cholesterol ratio (TC/HDL) and non-HDL cholesterol as well as an elevated odds of high cholesterol. In another study, which evaluated the relationship between serum PFCs and thyroid function, increase in natural log-PFHxS was associated with an increase of total T4 by 0.26 ug/mL and total T3 by 4.074 ng/dL in women and a decrease of

natural log-free T4 by 0.016 (ng/dL) in men. ANIMAL STUDIES: A subchronic study investigated the mechanism underlying the effect of PFAS surfactants on lipoprotein metabolism. Mice were fed a Western-type diet with PFHxS, (6 mg/kg/day) for 4-6 weeks. PFHxS markedly reduced TG, non-HDL-C, and HDL-C. The decrease in very low-density lipoprotein (VLDL) was caused by enhanced lipoprotein lipase-mediated VLDL-TG clearance and by decreased production of VLDL-TG and VLDL-apolipoprotein B. Reduced HDL production, related to decreased apolipoprotein AI synthesis, resulted in decreased HDL. PFHxS increased liver weight and hepatic TG content. Hepatic gene expression profiling data indicated that these effects were the combined result of peroxisome proliferator-activated receptor alpha and pregnane X receptor activation. Another study showed that neonatal exposure to PFHxS can alter neuroprotein levels, e.g. CaMKII, GAP-43, synaptophysin and tau, which are essential for normal brain development in mice. This was measured for both males and females, in hippocampus and cerebral cortex. The results suggest that PFHxS may act as a developmental neurotoxicant and the effects are similar to that of PFOS and PFOA, but also to other substances such as PCBs, PBDEs and bisphenol A. In another study, in ovo effects of PFHxS exposure (maximum dose = 38,000 ng/g egg) on embryonic death, developmental endpoints, tissue accumulation, mRNA expression in liver and cerebral cortex, and plasma TH levels were evaluated. Pipping success was reduced to 63% at the highest dose of PFHxS; additional effects included decreased tarsus length and embryo mass. Plasma TH levels were reduced in a concentration-dependent manner following PFHxS exposure. A subsequent study evaluated the relationship between PFHxS exposure and TH-dependent neurodevelopmental pathways. PFHxS significantly altered the expression of 11 transcripts at the low dose (890 ng/g) and 101 transcripts at the high dose (38,000 ng/g). Functional enrichment analysis showed that PFHxS affected the genes involved in tissue development and morphology, cellular assembly and organization, and cell-to-cell signaling. In another study, the effects of PFHxS on the neuronal cell death and the underlying mechanisms were examined using PC12 cells as a model of dopaminergic neuron. The treatment with PFHxS reduced cell viability in a dose-dependent manner. PFHxS increased cell apoptosis, increased the activations of ERK1/2, JNK and p38 MAPK with different temporal activations. PFHxS exposure also increased ROS formation. /EPIDEMIOLOGY STUDIES/ Associations between perfluoroalkyl acids (PFASs) and human thyroid hormone levels remain unclear, especially during early pregnancy when small changes in maternal thyroid hormones can affect fetal brain development. The objective of this study was to examine associations between maternal serum PFAS levels and maternal thyroid hormone levels in the early 2nd trimester of pregnancy. Participants were euthyroid pregnant women (n=152) enrolled in the Chemicals, Health and Pregnancy (CHirP) study based in Vancouver, Canada. Associations between maternal serum PFASs, including perfluorohexanesulfonate (PFHxS), perfluorononanoate (PFNA), perfluorooctanoate (PFOA) and perfluorooctanesulfonate (PFOS) and repeated measures of maternal thyroid hormones, including free thyroxine (fT4), total thyroxine (TT4) and thyroid stimulating home (TSH) were examined using mixed effects linear models. Associations were considered in all women, then separately in women with high (greater than or equal to 9 IU/mL) vs normal (<9 IU/mL) levels of thyroid peroxidase antibody (TPOAb), a marker of autoimmune hypothyroidism (Hashimoto's disease). Median PFAS concentrations (ng/mL) in maternal sera were 1.0 (PFHxS), 0.6 (PFNA), 1.7 (PFOA) and 4.8 (PFOS). PFASs were not associated with fT4, TT4 or TSH among women with normal TPOAb. However, among the 9% of women with high TPOAb (n=14), interquartile range (IQR) increases of PFASs were associated with a 46-69% increase in maternal TSH (95% CIs ranging from 8% to 123%) (PFNA, PFOA and PFOS only), and with a 3% to 7% decrease in maternal fT4 (95% CIs ranging from -18% to 5%) (all 4 PFASs). PFNA was also associated with higher maternal TSH in the whole sample. PFASs were positively associated with TSH, and weakly negatively associated with fT4 in the subset of pregnant women with high TPOAb, which occurs in 6-10% of pregnancies. PFASs may exacerbate the already high TSH and low fT4 levels in these women during early pregnancy, which is a critical time of thyroid hormone-mediated fetal brain development. The clinical significance of these findings is not clear. We propose a "multiple hit hypothesis" to explain these findings; this hypothesis deserves evaluation in larger, more representative study samples. /perfluoroalkyl acids/

- STOT-single exposure: No data available.

- STOT-repeated exposure: IDENTIFICATION AND USE: Perfluorohexane sulfonate (PFHxS) is a six-carbon compound in the perfluoroalkyl family of chemicals. Perfluorohexane sulfonate was once used in firefighting foam and carpet treatment solutions. It was once widely used as a stain and water repellent but was phased out due to U.S. Environmental Protection Agency regulations. HUMAN EXPOSURE AND TOXICITY: An epidemiological study evaluated the associations between serum levels of PFHxS and diagnosis of attention deficit/hyperactivity disorder (ADHD). In another study, the associations of prenatal serum concentrations (PFHxS) with fetal and postnatal growth in girls was examined. Serum samples were obtained from mothers during pregnancy. PFHxS (median, 1.6 ng/mL) was detected in 100% of samples. On average, girls born to mothers with prenatal concentrations of PFHxS in the upper tertile weighed 108 g less at birth than girls born to mothers with concentrations in the lower tertile in adjusted models. In another study, the association between levels of PFHxS and semen volume, sperm concentration, total sperm count, motility and morphology were assessed. The proportion of morphologically normal cells was 35% lower for the third tertile of PFHxS exposure as compared with the first. In a surveillance study, the serum concentrations of perfluoroalkyl acids (PFAAs) in firefighters was evaluated, and serum concentration of PFHxS was statistically higher in firefighters. Another study reported evidence of a significant association between PFHxS and total cholesterol (TC), low-density lipoprotein cholesterol (LDL), total cholesterol/high density lipoprotein cholesterol ratio (TC/HDL) and non-HDL cholesterol as well as an elevated odds of high cholesterol. In another study, which evaluated the relationship between serum PFCs and thyroid function, increase in natural log-PFHxS was associated with an increase of total T4 by 0.26 ug/mL and total T3 by 4.074 ng/dL in women and a decrease of natural log-free T4 by 0.016 (ng/dL) in men. ANIMAL STUDIES: A subchronic study investigated the mechanism underlying the effect of PFAS surfactants on lipoprotein metabolism. Mice were fed a Western-type diet with PFHxS, (6 mg/kg/day) for 4-6 weeks. PFHxS markedly reduced TG, non-HDL-C, and HDL-C. The decrease in very low-density lipoprotein (VLDL) was caused by enhanced lipoprotein lipase-mediated VLDL-TG clearance and by decreased production of VLDL-TG and VLDL-apolipoprotein B. Reduced HDL production, related to decreased apolipoprotein AI synthesis, resulted in decreased HDL. PFHxS increased liver weight and hepatic TG content. Hepatic gene expression profiling data indicated that these effects were the combined result of peroxisome proliferator-activated receptor alpha and pregnane X receptor activation. Another study showed that neonatal exposure to PFHxS can alter neuroprotein levels, e.g. CaMKII, GAP-43, synaptophysin and tau, which are essential for normal brain development in mice. This was measured for both males and females, in hippocampus and cerebral cortex. The results suggest that PFHxS may act as a developmental neurotoxicant and the effects are similar to that of PFOS and PFOA, but also to other substances such as PCBs, PBDEs and bisphenol A. In another study, in ovo effects of PFHxS exposure (maximum dose = 38,000 ng/g egg) on embryonic death, developmental endpoints, tissue accumulation, mRNA expression in liver and cerebral cortex, and plasma TH levels were evaluated. 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PFHxS increased cell apoptosis, increased the activations of ERK1/2, JNK and p38 MAPK with different temporal activations. PFHxS exposure also increased ROS formation. /LABORATORY ANIMALS: Subchronic or Prechronic Exposure/ Perfluorobutane sulfonate (PFBS), perfluorohexane sulfonate (PFHxS), and perfluorooctane sulfonate (PFOS) are stable perfluoroalkyl sulfonate (PFAS) surfactants, and PFHxS and PFOS are frequently detected in human biomonitoring studies. Some epidemiological studies have shown modest positive correlations of serum PFOS with non-high-density lipoprotein (HDL)-cholesterol (C). This study investigated the mechanism underlying the

effect of PFAS surfactants on lipoprotein metabolism. CETP mice were fed a Western-type diet with PFBS, PFHxS, or PFOS (30, 6, and 3 mg/kg/day, respectively) for 4-6 weeks. Whereas PFBS modestly reduced only plasma triglycerides (TG), PFHxS and PFOS markedly reduced TG, non-HDL-C, and HDL-C. The decrease in very low-density lipoprotein (VLDL) was caused by enhanced lipoprotein lipase-mediated VLDL-TG clearance and by decreased production of VLDL-TG and VLDL-apolipoprotein B. Reduced HDL production, related to decreased apolipoprotein AI synthesis, resulted in decreased HDL. PFHxS and PFOS increased liver weight and hepatic TG content. Hepatic gene expression profiling data indicated that these effects were the combined result of peroxisome proliferator-activated receptor alpha and pregnane X receptor activation. In conclusion, the potency of PFAS to affect lipoprotein metabolism increased with increasing alkyl chain length. PFHxS and PFOS reduce plasma TG and total cholesterol mainly by impairing lipoprotein production, implying that the reported positive correlations of serum PFOS and non-HDL-C are associative rather than causal.
- Aspiration hazard: No data available.

Likely routes of exposure
- No data available.

Symptoms related to the physical, chemical and toxicological characteristics
- IDENTIFICATION AND USE: Perfluorohexane sulfonate (PFHxS) is a six-carbon compound in the perfluoroalkyl family of chemicals. Perfluorohexane sulfonate was once used in firefighting foam and carpet treatment solutions. It was once widely used as a stain and water repellent but was phased out due to U.S. Environmental Protection Agency regulations. HUMAN EXPOSURE AND TOXICITY: An epidemiological study evaluated the associations between serum levels of PFHxS and diagnosis of attention deficit/hyperactivity disorder (ADHD). In another study, the associations of prenatal serum concentrations (PFHxS) with fetal and postnatal growth in girls was examined. Serum samples were obtained from mothers during pregnancy. PFHxS (median, 1.6 ng/mL) was detected in 100% of samples. On average, girls born to mothers with prenatal concentrations of PFHxS in the upper tertile weighed 108 g less at birth than girls born to mothers with concentrations in the lower tertile in adjusted models. In another study, the association between levels of PFHxS and semen volume, sperm concentration, total sperm count, motility and morphology were assessed. The proportion of morphologically normal cells was 35% lower for the third tertile of PFHxS exposure as compared with the first. In a surveillance study, the serum concentrations of perfluoroalkyl acids (PFAAs) in firefighters was evaluated, and serum concentration of PFHxS was statistically higher in firefighters. Another study reported evidence of a significant association between PFHxS and total cholesterol (TC), low-density lipoprotein cholesterol (LDL), total cholesterol/high density lipoprotein cholesterol ratio (TC/HDL) and non-HDL cholesterol as well as an elevated odds of high cholesterol. In another study, which evaluated the relationship between serum PFCs and thyroid function, increase in natural log-PFHxS was associated with an increase of total T4 by 0.26 ug/mL and total T3 by 4.074 ng/dL in women and a decrease of natural log-free T4 by 0.016 (ng/dL) in men. ANIMAL STUDIES: A subchronic study investigated the mechanism underlying the effect of PFAS surfactants on lipoprotein metabolism. Mice were fed a Western-type diet with PFHxS, (6 mg/kg/day) for 4-6 weeks. PFHxS markedly reduced TG, non-HDL-C, and HDL-C. The decrease in very low-density lipoprotein (VLDL) was caused by enhanced lipoprotein lipase-mediated VLDL-TG clearance and by decreased production of VLDL-TG and VLDL-apolipoprotein B. Reduced HDL production, related to decreased apolipoprotein AI synthesis, resulted in decreased HDL. PFHxS increased liver weight and hepatic TG content. Hepatic gene expression profiling data indicated that these effects were the combined result of peroxisome proliferator-activated receptor alpha and pregnane X receptor activation. Another study showed that neonatal exposure to PFHxS can alter neuroprotein levels, e.g. CaMKII, GAP-43, synaptophysin and tau, which are essential for normal brain development in mice. This was measured for both males and females, in hippocampus and cerebral cortex. The results suggest that PFHxS may act as a developmental neurotoxicant and the effects are similar to that of PFOS and PFOA, but also to other substances such as PCBs, PBDEs and bisphenol A. In another study, in ovo effects of PFHxS exposure (maximum

dose = 38,000 ng/g egg) on embryonic death, developmental endpoints, tissue accumulation, mRNA expression in liver and cerebral cortex, and plasma TH levels were evaluated. Pipping success was reduced to 63% at the highest dose of PFHxS; additional effects included decreased tarsus length and embryo mass. Plasma TH levels were reduced in a concentration-dependent manner following PFHxS exposure. A subsequent study evaluated the relationship between PFHxS exposure and TH-dependent neurodevelopmental pathways. PFHxS significantly altered the expression of 11 transcripts at the low dose (890 ng/g) and 101 transcripts at the high dose (38,000 ng/g). Functional enrichment analysis showed that PFHxS affected the genes involved in tissue development and morphology, cellular assembly and organization, and cell-to-cell signaling. In another study, the effects of PFHxS on the neuronal cell death and the underlying mechanisms were examined using PC12 cells as a model of dopaminergic neuron. The treatment with PFHxS reduced cell viability in a dose-dependent manner. PFHxS increased cell apoptosis, increased the activations of ERK1/2, JNK and p38 MAPK with different temporal activations. PFHxS exposure also increased ROS formation.

SECTION 12: Ecological information

SECTION 12: Ecological information

12.1 Toxicity
Not available.

12.2 Persistence and degradability
Not available.

12.3 Bioaccumulative potential
Not available.

12.4 Mobility in soil
Not available.

12.5 Results of PBT and vPvB assessment
Not available.

12.6 Endocrine disrupting properties
Not available.

12.7 Other adverse effects
Not 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. Empty containers may retain residues.
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 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
Not available.

SECTION 16: Other information

SECTION 16: Other information

Product name: Perfluorohexane Sulfonic Acid
CAS No.: 355-46-4
Catalog No.: CS-ED-00324
Synonyms: Perfluorohexanesulfonic acid; Perfluorohexane-1-sulphonic acid
Supplier: Clearsynth Labs Ltd., Mumbai, India
Emergency phone: +91-22-245045900

Revision date: Not available.
Disclaimer: The information provided is believed to be accurate based on available data, but no warranty is expressed or implied. Users are responsible for determining suitability for their particular application and for compliance with applicable laws and regulations.

DISCLAIMER

This MSDS is system-generated. Please verify and confirm all data, statements, and values with the Support Team before use or distribution.