GreenScreen® Chemical Assessment

Potassium Oxide (12136-45-7)

Method Version: GreenScreen® for Safer Chemicals v1.4

Assessment Details:

Assessment Type:	Certified
Assessment Prepared By:	WAP Sustainability Consulting, LLC (Eric Rosenblum Ph.D. DABT Donald K. Ward, NSF International)
Assessment Prepared For:	Tile Council of North America (TCNA)
Date Assessment Completed:	January 31st, 2021
Assessment Expiration Date:	January 31st, 2026
Assessor Type:	Licensed GreenScreen® Profiler

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GREENSCREEN BENCHMARK™ SUMMARY:

This chemical assessment report includes a GreenScreen Benchmark™ score and results for Potassium oxide (12136-45-7) only. No marketing claims can be made without licensing through Clean Production Action.

GreenScreen Benchmark Score: BM-2

Potassium oxide (12136-45-7) was assigned a Benchmark Score of 2 ("Use but Search for Safer Substitutes) as it has Very High Group II Human Toxicity (skin irritation and eye irritation). This corresponds to GreenScreen® benchmark classification 2e in CPA 2018. Data gaps (DG) exist for endocrine activity, single dose neurotoxicity and respiratory sensitization. As outlined in CPA (2018) Annex 5 (Conduct a Data Gap Analysis to assign a final Benchmark score), Potassium oxide meets the requirements for a GreenScreen® Benchmark Score of 2 despite the data gaps. In a worst-case scenario, if Sodium Oxide were assigned a High score for data gap endocrine activity it would be categorized as a Benchmark 1 Chemical.

HAZARD CLASSIFICATION SUMMARY

Table 1. GreenScreen Hazard Summary Table:

GreenScreen Hazard Summary Table for Potassium oxide – BM-2																			
Group I Human					Group II and II* Human									Ecotox		Fate		Physical	
Carcinogenicity	Genotoxicity/Mutagenicity	Reproductive Toxicity	Developmental Toxicity	Endocrine Activity	Acute Toxicity	Systemic Toxicity		Neurotoxicity		Skin Sensitization*	Respiratory Sensitization*	Skin Irritation	Eye Irritation	Acute Aquatic Toxicity	Chronic Aquatic Toxicity	Persistence	Bioaccumulation	Reactivity	Flammability
						single	repeat*	single	repeat*	*	*								
L	L	L	L	DG	*L*	M	L	DG	L	L	DG	vH	vH	L	L	vL	vL	L	L

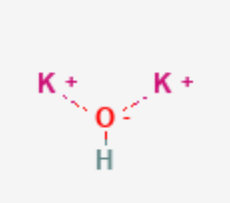
Note: Hazard levels (Very High (vH), High (H), Moderate (M), Low (L), Very Low (vL)) in italics reflect lower confidence in the hazard classification while hazard levels in BOLD font reflect higher confidence in the hazard classification. Group II Human Health endpoints differ from Group II* Human Health endpoints in that Group II Human Health endpoints have four hazard scores (i.e., vH, H, M and L) instead of three (i.e., H, M and L), and are based on single exposures instead of repeated exposures. Group II* Human Health endpoints are indicated by an * after the name of the hazard endpoint or after "repeat" for repeated exposure sub-endpoints.

SCOPE OF ASSESSMENT

Potassium oxide (12136-45-7):

Also Called: Potassium oxide (K2O); Potassium monoxide; Potassium oxide (KO); Monopotassium Monooxide (PubChem 2020)

Chemical Structure:

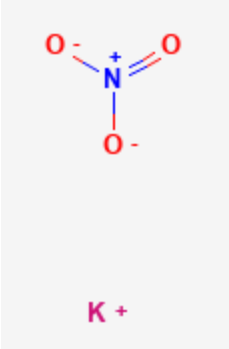


Suitable analogs or moieties of chemicals used in this assessment (CASRN(s)):

Chemical Structure(s) of suitable analog(s) and/or moieties:

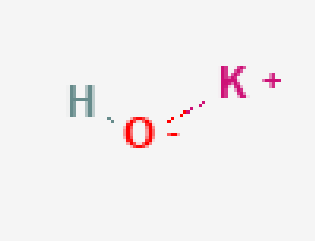
KCl

CAS# 7447-40-7



KNO3

CAS# 7757-79-1



KOH

CAS# 1310-58-3

For Inorganic Chemicals and relevant particulate organics (if not relevant, list NA)

Define Properties:

1. Particle size (e.g., silica of respirable size): N/A
2. Structure (e.g., amorphous vs. crystalline): N/A
3. Mobility (e.g., water solubility, volatility): N/A
4. Bioavailability: N/A

Identify potential applications/functional uses of the chemical:

1. Adsorbents and absorbents
2. Catalyst
3. Graveling and road bed material.
4. Intermediates
5. Process regulators
6. Processing aids, not otherwise listed
7. Other industrial function
8. Air care products
9. Building/construction materials not covered elsewhere
10. Oxygen concentrators
11. Paints and coatings

Table 2. Environmental Transformation Products Summary

Life Cycle Stage	Transformation Pathway	Environmental Transformation Product	CAS #	Feasible (Yes or No)	Relevant (Yes or No)	GreenScreen List Translator Score or GreenScreen Benchmark Score
End of life	Hydrolysis	KOH	1310-58-3	Yes	Yes	LT-P1

HAZARD CLASSIFICATION SUMMARY

GROUP I HUMAN HEALTH EFFECTS (GROUP I HUMAN)

Carcinogenicity (C): L

Potassium oxide was assigned a hazard classification level of Low for carcinogenicity based on an oral 2-year rat study using potassium chloride which found no increased incidence of tumors in exposed animals. In addition, potassium (in its ionic form) is an essential element, which is tightly regulated by the human body within its different compartments. Potassium does not exhibit any properties which would raise a concern for carcinogenic properties. The hazard conclusion is based on use of a high quality analog and therefore reported as high confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening: not included on any screening lists

Measured Data

ECHA 2020a

Valid carcinogenicity studies with animals are not available for potassium oxide. Potassium chloride (KCl) is the most common source of Potassium oxide (K2O). Therefore, the health effects of potassium chloride need to be considered in the assessment of Potassium oxide

In a long-term study performed with KCl, no carcinogenic effects were observed in male rats, Groups of 50 male F344/Slc rats were fed KCl in the diet at levels of 110, 450, 1820 mg/kg bw/day for 2 years. A comprehensive examination of the tissue revealed no evidence of treatment related carcinogenicity. Among non-tumorous lesions nephrotic lesion was predominant in all treatment groups as well as in the control group. In tumorous lesions testicular tumor (interstitial cell tumor) developed with a high incidence in all groups. However, the incidence and type of tumor in experimental and control groups were comparable to those of spontaneous tumors in the test organism (Imai 1961, UNEP 2003)

Male and female Wistar rats were fed diets containing 0 or 3 % KCL over a total period of 30 months: Examination after 13 weeks (10 rats/sex/group), after 18 months (15 rats/sex/group) and after 30 months (50 rats/sex /group). Due to the reduction of feed intake the mean test substance intake and mean body weight decreased in time. After 30 months treatment, histopathologically in the adrenals, zona glomerulosa: hypertrophy in 24/50 treated rats versus 4/50 in controls, and in the urinary bladder: cystitis (males: 3/59; females 3/50) and single epithelial hyperplasia (males 3/50; females 2/50)were the only observed changes

ECHA 2020b

Valid carcinogenicity studies with animals are not available for potassium hydroxide. An old long-term study (reliability 3) of 25-46 weeks, consisting of painting 3-6% KOH solutions on mouse skin, has been performed (Narat, 1925). The results were ca. 15% occurrence of cancer at the application site. As discussed by Ingram and Grasso (1991), such a production of skin cancer is

due to a non-genotoxic mechanism secondary to repeated application and prolonged inflammation, by indirect hyperplasia as a consequence of severe skin damage. Any kind of prolonged irritation possibly would have produced the same result. HCl solution painting produced also cancer in mice. Moreover, such an exposure causing repeated skin damage and increased cell proliferation to repair the chronic injury, is not relevant for man.

Moreover, systemic carcinogenicity is not expected to occur because potassium hydroxide is not expected to be systemically available in the body under normal handling and use conditions. Finally, no suitable studies are available to assess the risk on local carcinogenic effects.

There is therefore no evidence KOH to be carcinogenic in exposure situations that are relevant for man.

Estimated Data- None

Mutagenicity/Genotoxicity (M): L

Potassium oxide was assigned a hazard classification level of Low for mutagenicity based on no evidence for mutagenic activity in the available in vitro and the in vivo genetic toxicity tests for Dipotassium oxide and high-quality analogs. The hazard score is based on studies in potassium oxide and in high-quality analog and therefore reported as high confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening: not included on any screening lists

Measured Data

ECHA 2020a

An assessment was conducted based on an examination of the composition of the substance and the potential of its constituents to induce chromosomal aberration in mammalian cells. This assessment was supported by data from a QSAR prediction on the registered substance and its components. An assessment of the available literature information on Dipotassium oxide, predicted to be the most abundant of the species Dipotassium oxide will dissociate into in the known pH range of mammalian cells, as well as a QSAR predictions of each substance, demonstrates that Dipotassium oxide is not expected to cause chromosomal aberrations in mammalian cells

An assessment was conducted based on an examination of the composition of the substance and the potential of its constituents to induce gene mutation in mammalian cells. This assessment used data from a QSAR prediction on the registered substance and its constituents, which was found to be negative in all cases. The results of the QSAR prediction for Dipotassium oxide and its constituents show that Dipotassium oxide is not expected to induce gene mutation in mammalian cells. On the basis of these results an experimental study on the potential of Dipotassium oxide to induce gene mutation in mammalian cells is not considered necessary since it is unlikely to provide additional relevant data about the in vivo mutagenicity potential of Dipotassium oxide in accordance with ECHA's Endpoint Specific Guidance on Genotoxicity (ECHA, 2015), which states that the potential of a substance to induce gene mutation in mammalian cells does not need to be evaluated,

if it can be demonstrated that it will not provide any further useful information about the potential in vivo mutagenicity of a substance

In vitro Studies

The results of an Ames assay study with Salmonella typhimurium TA 97 and TA 102, with and without metabolic activation and up to 1 mg KOH/plate, were negative (Fujita et al., 1992).

KCl has been classified as non-genotoxic in a bacterial reverse mutation assay with S. Typhimurium TA 100, TA 1535, TA 1537 and TA 9, at 0, 100, 333, 1000, 3333 and 10000 µg/plate, with andwithout metabolic activation (Mortelmans et al., 1986). In a DNA damage and repair assay (SOS Chromotest Institut Pasteur) with E. coli PQ 37, at 1-100000 nM/ml, without metabolic activation, KCl was negative (Olivier and Marzin, 1987).

Two publications (Myhr and Caspary, 1988; Mitchell et al., 1988) report the genotoxic effect of KCl in a mammalian cell gene mutation assay with mouse lymphoma cell L5178Y, TK+/-

heterozygote, at 0-5000 µg/ml, with and without metabolic activation (OECD guideline 476). The result was positive only at high KCl concentration with metabolic activation. This has been attributed by the authors to the changed physical environment of the cells (increased osmotic pressure; K+ effects on sequestering of Mg2+ ions required for chromatin integrity), rather than to a direct genotoxic effect. Several authors confirm this type of non-specific genotoxic effect (Brusick, 1986; Seeberg, 1988).

Potassium nitrate has been classified has non-genotoxic in an Ames test with Salmonella typhimurium strains TA 1535, TA 1537, TA 98 and TA 100 and TA92, at up to 20 mg/plate, with and without metabolic activation (liver S-9 mix of Fischer rats) (Ishidate et al., 1984).

Potassium nitrate has been classified has non-genotoxic in a cytogenetic assay with Chinese hamster fibroblasts (CHL cells) at up to 1 mg/ml, without metabolic activation (Ishidate et al., 1984).

Based on QSAR examination of the constituents of the substance and the potential of its constituents to induce structural chromosomal aberration in mammalian cells, it is concluded that Dipotassium oxide is not expected to induce structural chromosomal aberration in mammalian cells.

Based on QSAR examination of the constituents of the substance and the potential of its constituents to induce gene mutation in mammalian cells, it is concluded that Dipotassium oxide is not expected to induce gene mutation in mammalian cells.

In vivo Studies

No studies were identified regarding the “in vivo” genotoxicity.

Potassium nitrate tested negative in a dormant lethal assay.

The dominant lethal test produced no consistent responses to suggest that potassium nitrate is mutagenic to rats.

Based on QSAR in vivo mutagenicity (Micronucleus) examination of the constituents of the substance and the potential of its constituents to induce structural chromosomal aberration in mammalian cells, it is concluded that Dipotassium oxide is not expected to induce structural chromosomal aberration in mammalian cells.

ECHA2020b

The in vitro genetic toxicity tests for potassium hydroxide indicated no evidence of mutagenic activity. In addition, both the in vitro and the in vivo genetic toxicity tests with the structurally related sodium hydroxide indicated no evidence of mutagenic activity. According to the REACH Regulation, further in vivo mutagenicity studies shall only be considered in case of a positive result in genotoxic (in vitro) studies (column 2, Annexes VII, VIII). Therefore, further in vivo testing for mutagenicity is not relevant for potassium hydroxide. Moreover, it is technically not feasible to perform genotoxicity tests at physiological pH with KOH.

Estimated Data- None

Reproductive Toxicity (R): L

Potassium oxide was assigned a hazard classification level of Low for reproductive toxicity based on negative reproductive results reported in guideline studies using potassium chloride and potassium nitrate. The hazard conclusion is based on quality studies using high quality analogs and therefore is reported as high confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening: not included on any screening lists

Measured Data

ECHA 2020a

OECD Guideline 421 (Reproduction / Developmental Toxicity Screening Test) A one generation study with female mice and rats exists for KCl. Doses of 2.35 – 235 mg/kg bw/day in mice and 3.1 – 310 mg/kg bw/day in rats were administered to groups of 21-24 animals by single daily oral intubation. Body weights were recorded during 17 days for mice, with a post exposure period of 2 days and during 20 days for rats, with a post exposure period of 5 days. Body weights were recorded during 17 days for mice, with a post exposure period of 2 days and during 20 days for rats, with a post exposure period of 5 days. No significant effects were observed on mice and rat's survival and reproductive organs, or on offspring survival, weight, sex ratio and congenital defects. There were no treatment-related changes in precoital time, mating index, fertility index and pregnancy index. Results for F0- Precoital time, fertility and mating data: There were no statistically significant differences compared with the controls.- Gross findings: There were no chemical- related findings in all treatment groups.- Histopathological findings: There were no chemical- related findings in all treatment groups.

OECD Guideline 422 (Combined Repeated Dose Toxicity Study with the Reproduction / Developmental Toxicity Screening Test) using KNO3 Animals on the study were divided between two subgroups (toxicity and reproductive subgroups). The exposure period for males and females in the toxicity subgroup was 28 days. The exposure period for reproductive subgroup males was at most 28 days. The exposure period for reproductive subgroup females was at most 53 days (14 days pre-mating, 14 days mating, and gestational and lactational periods up to lactation day 4). Reproductive subgroup: There were no treatment-related deaths and no signs of overt clinical toxicity. There were no effects on body weight, food consumption, or food efficiency. Mating

performance and fertility were unaffected by treatment. All animals mated within 4 days. There were no treatment-related effects on gestation length, gestation index, litter size, offspring survival indices, sex ration, offspring bodyweight, or macro pathology for offspring. NOAEL: 1,500 mg/kg/day (general toxicity) 1,500 mg/kg/day (reproduction/developmental toxicity) LOAEL: No adverse effects were seen on general toxicity endpoints. No adverse effects were seen on reproduction/developmental toxicity endpoints.

Estimated Data- None

Developmental Toxicity incl. Developmental Neurotoxicity (D): L

Potassium oxide was assigned a hazard classification level Low for Developmental Toxicity incl. Developmental Neurotoxicity based on negative results reported in guideline studies using potassium chloride and potassium nitrate. The hazard conclusion is based on quality studies using high quality analogs and therefore is reported as high confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening: not included on any screening lists

Measured Data

ECHA 2020a

Pregnant female rats received 3.1, 14.4, 66.8, 310.0 mg/kg bw /day from gd 6 to gd 15 by gavage. the controls were sham treated with the vehicle at a level equivalent to the group receiving the highest dose. The administration of 3.1 up to 310.0 mg/kg bw of KCl to pregnant Wistar rats for 10 consecutive days(gd 6 -gd15, sacrifice on gd20) had no clearly discernible effect on nidation or on maternal of fetal survival. The number of abnormalities seen in either soft or skeletal tissues of the test groups did not differ from the number occurring spontaneously in the sham-treated controls

OECD Guideline 422 (Combined Repeated Dose Toxicity Study with the Reproduction / Developmental Toxicity Screening Test) using KNO3 Animals on the study were divided between two subgroups (toxicity and reproductive subgroups). The exposure period for males and females in the toxicity subgroup was 28 days. The exposure period for reproductive subgroup males was at most 28 days. The exposure period for reproductive subgroup females was at most 53 days (14 days pre-mating, 14 days mating, and gestational and lactational periods up to lactation day 4). Reproductive subgroup: There were no treatment-related deaths and no signs of overt clinical toxicity. There were no effects on body weight, food consumption, or food efficiency. Mating performance and fertility were unaffected by treatment. All animals mated within 4 days. There were no treatment-related effects on gestation length, gestation index, litter size, offspring survival indices, sex ration, offspring bodyweight, or macropathology for offspring. NOAEL: 1,500 mg/kg/day (general toxicity) 1,500 mg/kg/day (reproduction/developmental toxicity) LOAEL: No adverse effects were seen on general toxicity endpoints. No adverse effects were seen on reproduction/developmental toxicity endpoints.

The administration of 2.35 up to 235 mg/kg bw of KCl to pregnant CD-1 mice for 10 consecutive days (gd 6 -gd15, sacrifice gd17) had no clearly discernible effect on nidation or on maternal of fetal

survival. The number of abnormalities seen in either soft or skeletal tissues of the test groups did not differ from the number occurring spontaneously in the sham-treated controls.

Estimated Data- None

Endocrine Activity (E): DG

Potassium oxide was assigned a hazard classification level of Data Gap for endocrine activity based on based on the lack of available studies that directly address endocrine activity following exposures to the compound. While some data was available regarding estrogen receptor binding, this data alone is not sufficient to fill the data requirements for the endpoint. No analog data was available to fill the data gap.

Lists

- Authoritative: not included on any authoritative lists
- Screening: not included on any screening lists

Measured Data

ECHA 2020a

Estrogen receptor (ER) binding is a molecular initiating event much like protein binding that leads to a series of adverse outcomes, which are typically considered reproductive and development hazards. It is an endpoint where several comprehensive databases exist, which has lead to the development of several approaches for using (Q)SARs to predict ER-binding and possible endocrine disruption. Popular among these are the "four phase" assessment that includes Comparative Molecular Field Analysis (CoMFA) and the Common Reactivity Pattern Approach (COREPA). Since the RE-binding is a receptor mediated event, particular organic functional groups, size and shape are critical to binding potency. Dipotassium oxide have a molecular weight of less than 500, but do not possess a cyclic structure is reported to non-binders to the

No binding to Estrogen Receptor Alpha (Log RBA <-3) for the Potassium oxide (CAS# 12136-45-7)

Estimated Data- None

GROUP II AND II* HUMAN HEALTH EFFECTS (GROUP II AND II* HUMAN)

Note: Group II and Group II* endpoints are distinguished in the v1.4 Benchmark system (the asterisk indicates repeated exposure). For Systemic Toxicity and Neurotoxicity, Group II and II* are considered sub-endpoints. See GreenScreen Guidance v1.4, Annex 2 for more details.

Acute Mammalian Toxicity (AT): L

Potassium oxide was assigned a hazard classification level of Low for acute mammalian toxicity based on exposure studies using analogs and professional judgement. The low toxicity conclusion (outside of the corrosive nature of the compound) is supported by the low acute toxicity reported for Potassium Sodium Nitrate and potassium chloride which report high oral and dermal LD50s. Potassium oxide is not expected to be systemically available and the corrosive effects reported for potassium hydroxide are expected to be due to pH changes and not due to systemic effects. The hazard score is based in part on professional judgement (discounting the corrosive effects) and therefore is reported as low confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening: not included on any screening lists

Measured Data

ECHA 2020a

Acute oral toxicity:

The acute oral LD50 in male/female rats is >2000 mg/kg bw. All animals appeared active and healthy throughout the study period. No signs of toxicity were observed. This show that Potassium oxide (the result was read across from Potassium Sodium Nitrate) is not toxic for acute Oral toxicity.

In an acute oral toxicity study (Boyd 1969, UNEP 2003) female Wistar rats received 0, 2100, 2400, 2700, 3300, 3600, 3900 mg/kg bw potassium chloride dissolved in water. The LD50 value has been determined 3020 mg/kg bw. Clinical signs of intoxication in animals that died included convulsions followed by exhaustion and respiratory failure Autopsy revealed signs of irritant gastro-enteritis and necrosis appeared in the renal tubular epithelium. Animals that survived had convulsive movements diarrhea or sonstipation loss of appetite increase thirst and urine excretion and fever, Measurements of clinical signs were within the normal range within 2 -3 days and organ weights and water levels returned to normal limits in survivors at 2 weeks after administration of potassium chloride .

Acute Dermal Toxicity:

An LD50 value of >5000 mg/kg was obtained for Potassium oxide (the result was read across from Potassium Nitrate). The dermal LD50 in male and female rats is >5000 mg/kg.

Potassium oxide is not likely to be absorbed through the skin because of the low octanol-water partition coefficient of the substance. A reliable QSAR method predicts a value for the partition co-efficient (logKow) of -5.08 for this substance.

ECHA 2020b

According to the OECD SIDS on KOH (2002), KOH has a moderate acute oral toxicity, which is essentially due to its corrosivity. The observed systemic effects could be regarded as secondary effects.

Bruce (1987) performed a study on acute oral toxicity in rats: Potassium hydroxide shows moderate acute oral toxic effects, which are essentially due to its corrosivity (local effects) (OECD SIDS on potassium hydroxide, 2002). An LD50 of 273 mg/kg bw/d (214-324) was calculated based on the conventional method after a 2 weeks observation period (Bruce, 1987). Since potassium hydroxide is a strong alkaline substance, effects may occur even after a longer observation period due to the corrosive effects of the substance, leading to organ damage. However, this effect cannot be considered as an acute effect.

Human data in the OECD SIDS further support this conclusion. The only real effects of KOH ingestion are gastrointestinal burns. The mechanism of injury is one of liquefactive necrosis. Thrombosis of local blood vessels contributes to tissue damage. Tran mural necrosis can occur with frightening rapidity and injury often extrudes through the esophagus to involve adjacent mediastinal and peritoneal structures. When alkali enters the stomach, there may be some neutralization by gastric acid, which can limit the injury to this organ. Perforation of the stomach can occur with peritonitis and caustic injury to the contiguous organs including the colon, pancreas, liver and spleen. If sufficient quantities of alkali pass through the pylorus, there may be substantial duodenal damage including perforation. Lye constitutes a greater danger than solid granules, which tend to adhere on contact to mucous membranes without travelling further. The severity of damage depends on concentration of the agent, but also on the quantity swallowed. Aspiration of the alkali into the airway can result in live-threatening injuries to the larynx, the tracheobronchial passages, and the lungs.

Estimated Data- None

Systemic Toxicity/Organ Effects incl. Immunotoxicity (ST-single) M

Potassium oxide was assigned a hazard classification level of Moderate for Systemic Toxicity/Organ Effects incl. Immunotoxicity based on professional judgement. Potassium oxide is not expected to be systemically available and the corrosive effects are expected to be due to pH changes and not due to systemic effects. A low systemic toxicity conclusion (outside of the corrosive nature of the compound) is supported by potassium chloride classification as GRAS, however, a moderate score has been assigned based on the corrosive nature of the substance which will likely result in gastrointestinal and respiratory tract irritation. The moderate score is supported by the transient effect reported following exposures to potassium chloride. It is estimated herein the potassium oxide fulfills the H335 classification. The hazard score is based on professional judgement and therefore is reported as low confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening:
  - EU - Manufacturer REACH hazard submissions H335 - May cause respiratory irritation (unverified)

Measured Data

ECHA 2020a

Acute oral toxicity:

The acute oral LD50 in male/female rats is >2000 mg/kg bw. All animals appeared active and healthy throughout the study period. No signs of toxicity were observed. This show that Potassium oxide (the result was read across from Potassium Sodium Nitrate) is not toxic for acute Oral toxicity. All animals were observed for mortality, signs of gross toxicity, and behavioral changes at least once daily for 14 days after dosing. Bodyweights were recorded prior to administration and again on days 7 and 14 (termination) after dosing. Necropsies were performed on all animals at terminal sacrifice.

In an acute oral toxicity study (Boyd 1969, UNEP 2003) female Wistar rats received 0, 2100, 2400, 2700, 3300, 3600, 3900 mg/kg bw potassium chloride dissolved in water. The LD50 value has been determined 3020 mg/kg bw. Clinical signs of intoxication in animals that died included convulsions followed by exhaustion and respiratory failure Autopsy revealed signs of irritant gastro-enterisis and necrosis appeared in the renal tubular epithelium. Animals that survived had convulsive movements diarrhea or constipation loss of appetite increase thirst and urine excretion and fever, Measurements of clinical signs were within the normal range within 2 -3 days and organ weights and water levels returned to normal limits in survivors at 2 weeks after administration of potassium chloride .

Acute Dermal Toxicity:

Potassium oxide is not likely to be absorbed through the skin because of the low octanol-water partition coefficient of the substance. A reliable QSAR method predicts a value for the partition coefficient (logKow) of -5.08 for this substance.

ECHA 2020b

According to the OECD SIDS on KOH (2002), KOH has a moderate acute oral toxicity, which is essentially due to its corrosivity. The observed systemic effects could be regarded as secondary effects.

Bruce (1987) performed a study on acute oral toxicity in rats: Potassium hydroxide shows moderate acute oral toxic effects, which are essentially due to its corrosivity (local effects) (OECD SIDS on potassium hydroxide, 2002). Since potassium hydroxide is a strong alkaline substance, effects may occur even after a longer observation period due to the corrosive effects of the substance, leading to organ damage. However, this effect can not be considered as an acute effect.

Human data in the OECD SIDS further support this conclusion. The only real effects of KOH ingestion are gastrointestinal burns. The mechanism of injury is one of liquefactive necrosis. Thrombosis of local blood vessels contributes to tissue damage. Tran mural necrosis can occur with frightening rapidity and injury often extrudes through the oesophagus to involve adjacent mediastinal and peritoneal structures. When alkali enters the stomach, there may be some neutralization by gastric acid, which can limit the injury to this organ. Perforation of the stomach can occur with peritonitis and caustic injury to the contiguous organs including the colon, pancreas, liver and spleen. If sufficient quantities of alkali pass through the pylorus, there may be substantial duodenal damage including perforation. Lye constitutes a greater danger than solid granules, which tend to adhere on contact to mucous membranes without travelling further. The severity of damage

depends on concentration of the agent, but also on the quantity swallowed. Aspiration of the alkali into the airway can result in life-threatening injuries to the larynx, the tracheobronchial passages, and the lungs.

Estimated Data- None

Systemic Toxicity/Organ Effects incl. Immunotoxicity (ST-repeat) (Group II*) L

Potassium oxide was assigned a hazard classification level of Low for repeated dose systemic toxicity/organ effects based on no significant toxicity occurring in repeat dose study using potassium chloride. While repeat oral exposures to KCl have reported adrenal gland hypertrophy, nephritis, and gastritis these occur at concentrations >100 mg/kg. In addition, the toxicological significance of these effects are unclear. The hazard score is based on data for high quality analogs and therefore is reported as high confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening: not included on any screening lists

Measured Data

ECHA 2020a

Groups of male F344/Slc rats received 110, 450, 1820 mg/kg bw/day of KCl in feed over a period of 2 years and were observed for clinical signs and mortality and were examined for gross and histopathological changes at the end of the treatment period (Imai 1961) The NOAEL was set 1820 mg/kg bw/day since nephritis was reported in all treatment groups as well as in the control group. With respect to the observed gastritis which is regarded to be a local effect a LOAEL (local) of 110 mg/kg bw/d was determined (Imai 1961, UNEP 2003).

OECD Guideline 422 (Combined Repeated Dose Toxicity Study with the Reproduction / Developmental Toxicity Screening Test) Toxicity subgroup: There were no treatment-related deaths and no signs of overt clinical toxicity. There were no effects on body weight, food consumption, or food efficiency. Functional observational battery (FOB) and motor activity tests identified no treatment-related changes in behavior, function, or motor activity. A slight increase in blood urea nitrogen was observed in male and female rats at 1,500 mg/kg/day and in female rats at 750 mg/kg/day. Although outside the range of the historical control, the absence of other indicators of renal dysfunction (e.g., creatinine) discounted the clinical significance of this endpoint. Minimal changes in electrolyte levels in male rats (e.g., 10% decrease in potassium, 4% decrease in calcium, and 22% increase in phosphorus) and female rats (3% decrease in chloride, 4% decrease in magnesium) were observed at 1,500 mg/kg/day. These changes were within the range of historical control and were not considered to be of biological significance. No treatment-related histopathological changes were reported.

Male and female Wistar rats were fed diets containing 0 or 3 % KCL over a total period of 30 months: Examination after 13 weeks (10 rats/sex/group), after 18 months (15 rats/sex/group) and after 30 months (50 rats/sex /group). Due to the reduction of feed intake the mean test substance intake and mean body weight decreased in time. After 30 months treatment, histopathologically in the adrenals, zona glomerulosa: hypertrophy in 24/50 treated rats versus 4/50 in controls, and in the

urinary bladder: cystitis (males: 3/59; females 3/50) and single epithelial hyperplasia (males 3/50; females 2/50)were the only observed changes .

14 female Wistar rats received in their drinking water 2.5% potassium chloride for a period of 105 days (controls received tp water). 10 female rats were then sacrificed for the examination of heart, kidneys and adrenals. 4 rats served as recovery group (tap water) for 1 month. Compared to concurrent control rats increase in kidney weights, decrease in heart weight and adrenal glomerular zone hyperactivity, recovery after cessation of treatment

Estimated Data- None

Neurotoxicity (N-single) DG

Potassium oxide was assigned a hazard classification level of Data Gap for single dose neurotoxicity based on insufficient available data. While nonspecific signs and symptoms of neurotoxicity (convulsive movements diarrhea or constipation loss of appetite increase thirst and urine excretion and fever) were reported in female Wistar rats following acute oral exposure to potassium chloride, it is not known whether these observations represent direct effects of the compound on the nervous system. No data was located for analogs to address this data gap.

Lists

- Authoritative: not included on any authoritative lists
- Screening: not included on any screening lists

Measured Data

ECHA 2020a

Acute oral toxicity:

The acute oral LD50 in male/female rats is >2000 mg/kg bw. All animals appeared active and healthy throughout the study period. No signs of toxicity were observed. This show that Potassium oxide (the result was read across from Potassium Sodium Nitrate) is not toxic for acute Oral toxicity.

In an acute oral toxicity study (Boyd 1969, UNEP 2003) female Wistar rats received 0, 2100, 2400, 2700, 3300, 3600, 3900 mg/kg bw potassium chloride dissolved in water. The LD50 value has been determined 3020 mg/kg bw. Clinical signs of intoxication in animals that died included convulsions followed by exhaustion and respiratory failure Autopsy revealed signs of irritant gastro-enterisis and necrosis appeared in the renal tubular epithelium. Animals that survived had convulsive movements diarrhea or constipation loss of appetite increase thirst and urine excretion and fever, Measurements of clinical signs were within the normal range within 2 -3 days and organ weights and water levels returned to normal limits in survivors at 2 weeks after administration of potassium chloride .

Acute Dermal Toxicity:

Potassium oxide is not likely to be absorbed through the skin because of the low octanol-water partition coefficient of the substance. A reliable QSAR method predicts a value for the partition co-efficient (logKow) of -5.08 for this substance.

ECHA 2020b

According to the OECD SIDS on KOH (2002), KOH has a moderate acute oral toxicity, which is essentially due to its corrosivity. The observed systemic effects could be regarded as secondary effects.

Human data in the OECD SIDS further support this conclusion. The only real effects of KOH ingestion are gastrointestinal burns.

Estimated Data- None

Neurotoxicity (N-repeated) (Group II*) L

Potassium oxide was assigned a hazard classification level of Low for repeated dose neurotoxicity based on no effects reported in a functional observational battery (FOB) and motor activity tests which identified no treatment-related changes in behavior, function, or motor activity following oral exposures at exposures of 1,500 mg/kg KNO3. The low hazard score is based on a guideline study using a high-quality analog and is therefore reported as with high confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening: not included on any screening lists

Measured Data

ECHA 2020a

OECD Guideline 422 (Combined Repeated Dose Toxicity Study with the Reproduction / Developmental Toxicity Screening Test) Toxicity subgroup: There were no treatment-related deaths and no signs of overt clinical toxicity. There were no effects on body weight, food consumption, or food efficiency. Functional observational battery (FOB) and motor activity tests identified no treatment-related changes in behavior, function, or motor activity at 1,500 mg/kg/day KNO3.

Estimated Data- None

Skin Sensitization (SnS) (Group II*) L

Potassium oxide was assigned a hazard classification level of LOW for skin sensitization based on study data for potassium hydroxide. The hazard score is based on quality studies using a high-quality analog and therefore is reported as high confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening: not included on any screening lists

Measured Data

ECHA 2020a

Skin sensitization: in vivo (non-LLNA) repeated insult test : similar to test described by Landsteiner and Jacobs (1935) the test substance was injected intracutaneously to separate skin sites of the animals (3x weekly, in total 9 treatments) Two weeks after the last injection, a challenge dose (0.1ml) was administered (test and control animals). Skin reactions were examined at 24, 48 and 72 hr following the challenge dose. Potassium Hydroxide (0.1%) does not induce cutaneous sensitization in guinea pigs When Potassium oxide is hydrated is produced Potassium hydroxide. Therefore, the health effects of Potassium hydroxide need to be considered in the assessment of Potassium oxide.

Estimated Data- None

Respiratory Sensitization (SnR) (Group II*) DG

Potassium oxide was assigned a hazard classification level of Data Gap for respiratory sensitization based on the lack of available studies that directly address respiratory sensitization following exposures to the compound. No analog data was available to fill the data gap.

Lists

- Authoritative: not included on any authoritative lists
- Screening: not included on any screening lists

Measured Data- None

Estimated Data- None

Skin Irritation/Corrosivity (IrS) vH

Potassium oxide was assigned a hazard classification level of very High for skin irritation/corrosivity based on test results reported for potassium hydroxide. Specifically, 10% KOH is classified as "corrosive" (R34). The hazard score is based on quality studies using a high-quality analog and therefore is reported as high confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening:
  - EU - Manufacturer REACH hazard submissions H314 - Causes severe skin burns and eye damage (unverified)

Measured Data

ECHA 2020a

OECD TG 431 (In vitro skin corrosion: human skin model test), study was performed prior to the adoption of the test guideline 10% KOH is classified as "corrosive" (R34), 5% KOH as noncorrosive using The Skin² model ZK1350

OECD Guideline 404 (Acute Dermal Irritation / Corrosion)-The results of the study (Nixon GA et al.. 1975) indicate that Potassium oxide (the result was read across from Potassium hydroxide) is corrosive to skin at concentrations of 10% according to OECD 404 guidelines.

Potassium Hydroxide 5% and 10% is a moderately irritating in rabbits. When Potassium oxide is hydrated is produced Potassium hydroxide. Therefore, the health effects of Potassium hydroxide need to be considered in the assessment of Potassium oxide
KOH 5% PII score = (moderate to) severe irritation
KOH 10% PII score = severe irritation

Solid Potassium hydroxide is corrosive. Depending on the concentration, solutions of Potassium hydroxide are non-irritating, irritating or corrosive and they cause direct local effects on the skin and eyes. Systemic effects are not to be expected. Solutions with concentrations higher than 2% are corrosive, while concentrations of about 0.5 to about 2.0 % are irritating.

Result: corrosive to skin at concentrations of 10%

Estimated Data- None

Eye Irritation/Corrosivity (IrE): vH

Potassium oxide was assigned a hazard classification level of very High for eye irritation/corrosivity based on test results reported for Potassium hydroxide. Specifically, potassium hydroxide is corrosive to eye at concentrations of 5%. The hazard score is based on quality studies using a high-quality analog and therefore is reported as high confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening:
  - EU - Manufacturer REACH hazard submissions H318 - Causes serious eye damage (unverified)

Measured Data

ECHA 2020a

OECD Guideline 405 (Acute Eye Irritation / Corrosion)-The acute eye irritation study(Johnson, 1975)indicates that Potassium oxide (the result was read across from Potassium hydroxide) is corrosive to eye at concentrations of 5 % according to OECD 405 guidelines. Several concentrations of Potassium hydroxide were tested by a Draize test on rabbits by instilling 0.1 ml, rinsing after 5 minutes or 24 hours of exposure and examining with the aid of fluorescein at 1, 24, 48 and 72 hours, 7 days, and eventually 14-21 days. The results were as follows: 5%/5 min.: extremely irritant and corrosive 1%/5 min.: irritant 0.5%/24 hr: marginal 0.1%/24 hr: no ocular reactions in the eye Result: corrosive Result: corrosive to eye at concentrations of 5% .

Estimated Data

ECOTOXICITY (ECOTOX)

Acute Aquatic Toxicity (AA): L

Potassium oxide was assigned a hazard classification level of Low for acute aquatic toxicity based on LC50 and EC50 values in all three trophic levels reported at >100 mg/L. The hazard score is based on well reported guideline studies using quality analogs and therefore is reported as high confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening: not included on any screening lists

Measured Data

ECHA 2020a

Summary of Aquatic Toxicity of Potassium oxide/ Dipotassium oxide on Aquatic Organisms

Aquatic Toxicity	Organism	Species	Test Duration	Result(mg/L)	Test material	Reference
Short-term toxicity to fish	Fish	Labeo rohita	96hr	LC_{50}= 917.6 mg/l	Potassium oxide	Palanisamy,R., and G. Kalaiselvi.,1992
		Pimephales promelas	96hr	LC_{50}=880 mg/l	Potassium chloride	Mount, D.R.,et al, 1997
		Fishes species (freshwater fish)	96hr	LC_{50}= 16062.827 mg/l(calculated)	Potassium oxide	EPA, 2011
Short-term toxicity to aquatic invertebrates	Invertebrates	Daphnia magna	48hr	EC_{50}= 660mg/l	Potassium chloride	Mount, D.R., Gulley, D.D., Hockett, J.R., Garrison, T.D., Evans, J.M., 1997
		Daphnid species	48hr	LC_{50}= 6675.5 mg/l(calculated)	Potassium oxide	EPA, 2011
		Ceriodaphnia dubia	48hr	EC_{50}= 630mg/l	Potassium chloride	Mount, D.R., Gulley, D.D., Hockett, J.R., Garrison, T.D., Evans, J.M., 1997
Toxicity to aquatic algae and cyano-bacteria	Algae	Nitscheria linearis	120hr	EC50=1337mg/l (growth rate)	Potassium chloride	Patrick, , R.,, Jr., J., Scheier, A.,1968

Estimated Data- None

Chronic Aquatic Toxicity (CA): L

Potassium oxide was assigned a hazard classification level of Low for chronic aquatic toxicity based on based on NOEC values in all three trophic levels reported at >10 mg/L. The hazard score is based on well reported guideline studies using quality analogs and therefore is reported as high confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening: not included on any screening lists

Measured Data

ECHA 2020a

Summary of Aquatic Toxicity of Potassium oxide/ Dipotassium oxide on Aquatic Organisms

Aquatic Toxicity	Organism	Species	Test Duration	Result(mg/L)	Test material	Reference
Long-term toxicity to fish	Fish	Fishes species (freshwater fish)	30 day	NOEC =1086.545 mg/l (calculated)	Potassium oxide	EPA, 2011
		Heteropneustes fossilis	40 day	NOEC = 2000 mg/l	Potassium oxide	Srivastava,P.N., and A.S. Narain, 1985
		Pimephales promelas	7 day	NOEC=500 mg/l	Potassium chloride	,Q.H., J.M. Lazorchak, and K.L. Winks, 1996
Long-term toxicity to aquatic invertebrates	Invertebrates	Daphnid species	30 day	NOEC = 273.134 mg/l (calculated)	Potassium oxide	EPA, 2011
		Daphnia magna	21 day	EC50 =87mg/l	Potassium chloride	Biesinger, K., Christensen, G.L..1972
Toxicity to aquatic algae and cyano-bacteria	Algae	Nitscheria linearis	120hr	EC50=1337mg/l (growth rate)	Potassium chloride	Patrick, , R.,, Jr., J., Scheier, A.,1968
		Green Algae	30 day	NOEC = 178.832 mg/l (calculated) EC50= 1368.296 mg/l (calculated)	Potassium oxide	EPA, 2011
Toxicity to aquatic plants other than algae	aquatic plants	aquatic plants other than algae	30day	NOEC = 178.832 mg/l (calculated) EC50= 1368.296 mg/l (calculated)	Potassium oxide	EPA, 2011

Estimated Data- None

ENVIRONMENTAL FATE (FATE)

Persistence (P): vL

Potassium oxide was assigned a hazard classification level of Very Low for persistence based on professional judgement. Specifically, when water is added to Potassium oxide, KOH is produced. The hazard score is based on physical/chemical properties of the substance and is therefore reported as high confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening:
  - EC - CEPA DSL Persistent

Measured Data

ECHA 2020a

The high water solubility and low vapour pressure indicate that Potassium oxide will be found predominantly in the aquatic environment. Potassium oxide is present in the environment as potassium and Oxygen ions, which implies that it will not adsorb on particulate matter or surfaces and will not accumulate in living tissues. It is obvious that both potassium and Oxygen ions have a wide natural occurrence (UNEP, 1995).

Atmospheric emissions as Potassium oxide aerosols should be rapidly neutralized by carbon dioxide, or other acids and the salts (e.g. potassium carbonate) will be washed out by rain. For this reason potential atmospheric emissions of Potassium oxide are considered of no concern. Significant emissions to the terrestrial environment are not expected during normal handling and use of Potassium oxide. Small terrestrial emissions will be neutralized by the buffer capacity of the soil. For this reason the environmental assessment can be limited to the aquatic compartment.

Because Potassium oxide does occur in the environment as Potassium and Oxygen a separate environmental assessment of both the potassium and the Oxygen ion is needed.

Oxygen is the most abundant chemical element by mass in the Earth's biosphere, air, sea and land. Oxygen is the third most abundant chemical element in the universe, after hydrogen and helium.

Potassium is essential constituent and one of the most abundant ions in all animal species. In adult humans, the total body potassium is approx. 3.5 mol (135 g). 98 % of this is located intracellular (150 mmol/l), the extracellular potassium concentration is approx. 4 mmol/l.

Both K+ and O- ions are normal constituents of the body fluids. K+ plays an essential role in the human physiology but starts to be toxic at levels exceeding 200 – 250 mg/l. Its concentration in the blood is regulated principally by renal excretion/reabsorption and controlled by an efficient feedback auto-regulation system. An excessive pH of the blood is prevented by the bicarbonate buffer system, respiration and renal compensation mechanisms.

Hydrolysis is not expected to be an important environmental fate process since this compound lacks functional groups that hydrolyze under environmental conditions.

Hydrolysis is a chemical reaction during which molecules of water (H2O) are split into hydrogen cations (H+, conventionally referred to as protons) and hydroxide anions (OH−) in the process of a chemical mechanism).

Potassium oxide is a basic oxide and reacts with water violently to produce the caustic potassium hydroxide

When water is added to Potassium oxide, KOH is produced.

- $K2O+H2O \rightarrow KOH$

Estimated Data- None

Bioaccumulation (B): vL

Potassium oxide was assigned a hazard classification level of Very Low for bioaccumulation based on professional judgement. Specifically, potassium is a naturally occurring element that is prevalent in the environment and to which organisms are exposed regularly, for which they have some capacity to regulate the concentration in the organism. Log P_{ow} is not applicable for an inorganic compound that dissociates. In addition, the hazard score is based on professional judgement and therefore reported as low confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening: not included on any screening lists

Measured Data

ECHA 2020a

Potassium oxide does not have any bioaccumulation potential.

Potassium is essential constituent and one of the most abundant ions in all animal species. In adult humans, the total body potassium is approx. 3.5 mol (135 g). 98 % of this is located intracellular (150 mmol/l), the extracellular potassium concentration is approx. 4 mmol/l.

Oxygen is the most abundant chemical element by mass in the Earth's biosphere, air, sea and land. Oxygen is the third most abundant chemical element in the universe, after hydrogen and helium

Both K+ and O- ions are normal constituents of the body fluids. K+ plays an essential role in the human physiology but starts to be toxic at levels exceeding 200 – 250 mg/l. Its concentration in the blood is regulated principally by renal excretion/reabsorption and controlled by an efficient feedback auto-regulation system. An excessive pH of the blood is prevented by the bicarbonate buffer system, respiration and renal compensation mechanisms.

Estimated Data- None

PHYSICAL HAZARDS (PHYSICAL)

Reactivity (Rx): L

Potassium oxide was assigned a hazard classification level of Low for reactivity based on the structure of the compound. Specifically, the chemical structure lacks chemical groups associated with explosive properties and there are no functional groups or other structural alerts present that would indicate that this substance would promote oxidation. The hazard score is based on the physical properties of the compound and therefore reported as high confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening: not included on any screening lists

Measured Data

ECHA 2020a

Due to the absence of chemical groups associated with Explosive properties or other structural alerts this substance is not considered to exhibit Explosiveness.

From the compositional description and the structural formula given, it can be concluded that this substance CAS 12136-45-7, (EINECS) 235-227-6 does not exhibit oxidizing properties (will not cause or contribute to the combustion of other materials by virtue of its own inherent oxidation or reduction potential). There are no functional groups or other structural alerts present that would indicate that this substance would promote oxidation and thus should not be classified as dangerous according to the criteria for evaluating oxidizing properties. Furthermore, this is confirmed by long term handling experience.

Estimated Data- None

Flammability (F): L

Potassium oxide was assigned a hazard classification level of Low for flammability based on the structure of the compound. Specifically, there are no functional groups or other structural alerts present that would support any concern that this substance should be classified as dangerous according to the criteria for evaluating flammability, which is furthermore confirmed by long term handling experience. The hazard score is based on the physical properties of the compound and therefore reported as high confidence.

Lists

- Authoritative: not included on any authoritative lists
- Screening: not included on any screening lists

Measured Data

ECHA 2020a

From the compositional description and the structural formula given, it can be concluded that this substance CAS 12136-45-7 , (EINECS) 235-227-6 is not pyrophoric (will not ignite or emit flammable gases on contact with air, damp air, or water) nor does it exhibit the property of flammability (will not easily burn when ignited or contribute to the combustion of other materials). There are no functional groups or other structural alerts present that would support any concern that this substance should be classified as dangerous according to the criteria for evaluating flammability, which is furthermore confirmed by long term handling experience.

Inchem 2006

- Not combustible

Estimated Data- None

REFERENCES

ECHA 2020a Registered Substance Database. Dipotassium oxide. Available at https://echa.europa.eu/registration-dossier/-/registered-dossier/16981/1.

ECHA 2020b Registered Substance Database. Potassium hydroxide. Available at https://echa.europa.eu/registration-dossier/-/registered-dossier/15804/1.

PubChem 2020. Potassium oxide. Available at https://pubchem.ncbi.nlm.nih.gov/compound/Potassium-oxide-_K2O#section=Related-Substances

Inchem 2006. Potassium oxide. Available at http://www.inchem.org/documents/icsc/icsc/eics0769.htm

APPENDIX A: HAZARD CLASSIFICATION ACRONYMS

(AA) Acute Aquatic Toxicity

(AT) Acute Mammalian Toxicity

(B) Bioaccumulation

(C) Carcinogenicity

(CA) Chronic Aquatic Toxicity

(D) Developmental Toxicity

(E) Endocrine Activity

(F) Flammability

(IrE) Eye Irritation/Corrosivity

(IrS) Skin Irritation/Corrosivity

(M) Mutagenicity and Genotoxicity

(N) Neurotoxicity

(P) Persistence

(R) Reproductive Toxicity

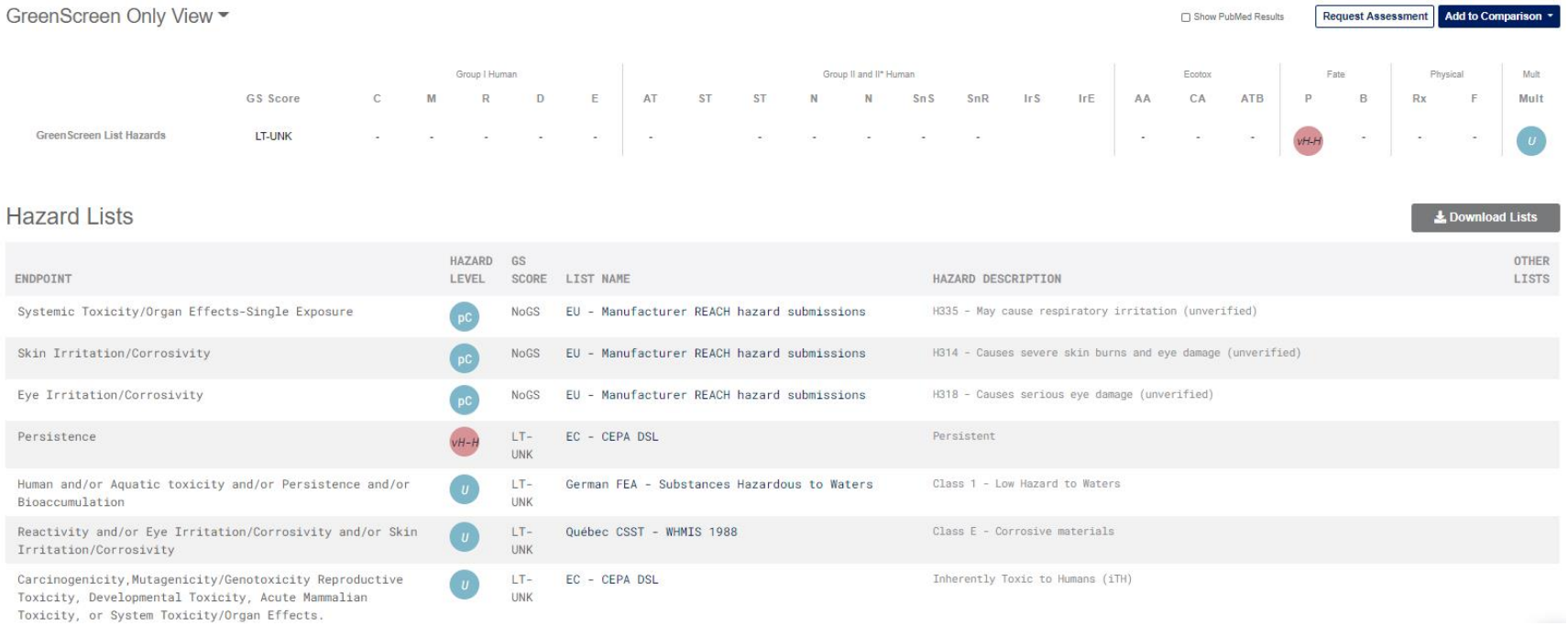
(Rx) Reactivity

(SnS) Sensitization- Skin

(SnR) Sensitization- Respiratory

(ST) Systemic/Organ Toxicity

APPENDIX B – PHAROS OUTPUT FOR POTASSIUM OXIDE (12136-45-7)



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		Group I Human					Group II and II* Human									Ecotox			Fate		Physical		Mult
	GS Score	C	M	R	D	E	AT	ST	ST	N	N	SnS	SnR	IrS	IrE	AA	CA	ATB	P	B	Rx	F	Mult
GreenScreen List Hazards	LT-UNK	-	-	-	-	-	-		-	-	-	-	-			-	-	-	vH-H	-	-	-	U

Hazard Lists

Download Lists

ENDPOINT	HAZARD LEVEL	GS SCORE	LIST NAME	HAZARD DESCRIPTION	OTHER LISTS
Systemic Toxicity/Organ Effects-Single Exposure	pC	NoGS	EU - Manufacturer REACH hazard submissions	H335 - May cause respiratory irritation (unverified)	
Skin Irritation/Corrosivity	pC	NoGS	EU - Manufacturer REACH hazard submissions	H314 - Causes severe skin burns and eye damage (unverified)	
Eye Irritation/Corrosivity	pC	NoGS	EU - Manufacturer REACH hazard submissions	H318 - Causes serious eye damage (unverified)	
Persistence	vH-H	LT-UNK	EC - CEPA DSL	Persistent	
Human and/or Aquatic toxicity and/or Persistence and/or Bioaccumulation	U	LT-UNK	German FEA - Substances Hazardous to Waters	Class 1 - Low Hazard to Waters	
Reactivity and/or Eye Irritation/Corrosivity and/or Skin Irritation/Corrosivity	U	LT-UNK	Québec CSST - WHMIS 1988	Class E - Corrosive materials	
Carcinogenicity,Mutagenicity/Genotoxicity Reproductive Toxicity, Developmental Toxicity, Acute Mammalian Toxicity, or System Toxicity/Organ Effects.	U	LT-UNK	EC - CEPA DSL	Inherently Toxic to Humans (iTH)	