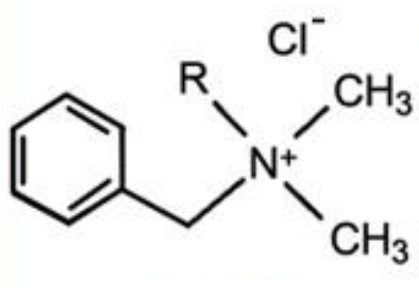
Updated EHS Summary of Alkyl Dimethyl Benzyl Ammonium Chloride (ADBAC) for the MA TURA Science Advisory Board Meeting – May 20, 2021

CAS #: 68424-85-1 Representative CAS # for ADBAC  	**NAME: Alkyl Dimethyl Benzyl Ammonium Chloride (ADBAC)** **Synonym[1]s:** n-alkyl (C14 50%; C12 40%; C16 10%) dimethyl benzyl ammonium chloride; Alkyl(C12-16)dimethylbenzylammonium chloride; Ammonium, alkyl(C12-C16)dimethylbenzyl-, chlorides; Barquat MB 80; Benzyl-C12-C16-alkyldimethyl ammonium chlorides; Bioquat 501; Bioquat 80; Black alg aetrine; BTC 835; Catigene T80; Cyncal 80; EINECS 270-325-2; Gardiquat 1250AF; Hyamine 3500; Maquat MC 1412; Protek Q; Rolquat cdm/BC; Tret-O-lite WF 828; Tret-O-lite WF 88; C12-16-Alkyldimethylbenzylammonium chloride **RTECS #[1]:** UZ2995000 **EINECS #[2]:** 270-325-2 **Molecular Weight[3]**: 377.83 **Molecular Formula[4]:** $RC_9H_{13}NCl$ R= n-alkyl (C_{12} 40%, C_{14} 50%, C_{16} 10%) **EPA PC Code[4]:** 069105 **Related CAS #'s: (See list at end of summary)**
PHYSICAL CHARACTERISTICS	
Primary Use	ADBAC is an antimicrobial used in agricultural, food handling, commercial/ institutional/ industrial, residential and public access, and medical settings[4]. There are 667 EPA-registered products that contain ADBAC as an active ingredient, 664 of which are registered as antimicrobial products and 3 that are conventional products. Production volume data for the years 2011 through 2014 indicate that approximately 198 million pounds of ADBAC are sold per year in the United States[3]. "Due to their amphiphilic nature, QACs act as detergents or surface-active agents against microorganisms. QACs target bacterial cell membranes through electrostatic interactions between the positively charged head group and negatively charged cytoplasmic membrane, adsorption, and then permeation of side chains into the intramembrane region. The lipid layer of enveloped viruses makes them sensitive to the hydrophobic activity of QACs" (Hora, 2020)[5].
Physical state, odor at room temperature & pressure	Crystalline sold in pure form Clear yellow to straw colored liquid with an amine odor[4]
Melting point; Boiling point	**MP:** 241.02˚C **BP:** 560.84˚C[4]
Solubility	Soluble in water and alcohols[3] 2.2 mg/L at 25˚C[6]

Specific Gravity	
SAFETY/PHYSICAL HAZARDS	
Vapor Pressure	3.53x 10^{-12} mm Hg[4]
Flammability	
Flashpoint	
Flammability Rating	
Auto Ignition Point	
Combustion products	
Explosivity (UEL, LEL, shock sensitive)	
Oxidizer	
Corrosivity	**Industrial Concentrates:** 50-80% of active QAC ingredient. Acute Tox, Inhalation – Category 2 Skin Corrosion/Irritation – Category 1 Serious eye damage/eye irritation – Category 1 Hazardous to the aquatic environment, acute & chronic hazard – Cat 1 **Ready-to-use Products:** .08-20% of active QAC ingredient. Skin corrosion/irritation – Category 2 Serious eye damage/eye irritation – Category 1 e.g., MD Stetson 105 Sanitizer RTU - 5% ADBAC, 5% DDAC **Institutional Concentrates:** e.g., Alpha BAC 10 - 10% ADBAC; serious eye damage Cat 1; skin corrosion Cat 1A.
pH	7.59[4]
Reactivity	
Viscosity	
Odor Threshold	
Particle size, shape, respirable fraction	
Other physical hazards associated with process: Heat, gases under pressure, noise, vibration, ergonomic hazard	
HEALTH HAZARDS	
Acute Toxicity	
Oral LD_{50}	LD_{50} Rat = 304.5 mg/kg (combined) LD_{50} Rat = 510.9 mg/kg (males) 280.8 mg/kg (females)[4] EPA Toxicity Category II[4]

Dermal LD_{50}	LD_{50} Rat = 930 mg/kg (combined) LD_{50} Rat = 1100 mg/kg (males) 704 mg/kg (females)[4] EPA Toxicity Category III[4]
Inhalation LC_{50}	0.054 < LC_{50} (Rat) < 0.51 mg/L[4] EPA Toxicity Category II[4]
Intraperitoneal LD_{50}	
Chronic or Sub-chronic Toxicity	
IARC rating	Not classified by IARC Monographs, Volumes 1-123
Carcinogenicity	The carcinogenic effects of DDAC and C12–C16 ADBAC have been investigated in multiple chronic oral toxicity studies conducted with mice and rats. All available studies indicate that neither DDAC nor ADBAC are carcinogenic via the oral exposure route, which is consistent with the conclusions of both EPA (2006a,b) and ECHA (2015a,b). Furthermore, EPA's Cancer Assessment Review Committee classified C12–C16 ADBAC and DDAC as "not likely to be carcinogenic to humans" and "Group E – evidence of non-carcinogenicity for humans," respectively (EPA, 2018). (Luz et al., 2020)
Neurotoxicity	"The goal of this study was to use targeted and untargeted mass spectrometry and transcriptomics to investigate the effect of BACs on sterol and lipid homeostasis and to predict the mechanism of toxicity of BACs on neurodevelopmental processes. After maternal dietary exposure to 120mg BAC/kg body weight/day, we quantified BAC levels in the mouse neonatal brain, demonstrating for the first time that BACs can cross the blood-placental barrier and enter the developing brain. Transcriptomic analysis of neonatal brains using RNA sequencing revealed alterations in canonical pathways related to cholesterol biosynthesis, liver X receptor-retinoid X receptor (LXR/RXR) signaling, and glutamate receptor signaling. Mass spectrometry analysis revealed decreases in total sterol levels and downregulation of triglycerides and diglycerides, which were consistent with the upregulation of genes involved in sterol biosynthesis and uptake as well as inhibition of LXR signaling. In conclusion, these findings demonstrate that BACs target sterol and lipid homeostasis and provide new insights for the possible mechanisms of action of BACs as developmental neurotoxicants."[7] "Significantly, even at non-cytotoxic concentrations, BACs can potently inhibit Dhcr7. This suggests that exposure of BACs could have severe consequences even when there is no apparent acute toxicity. It has been reported that orally administered BAC can cross the blood-brain barrier in rats (a 1.2 lg/g concentration in serum leads to 0.2 lg/g in brain) (Xue et al., 2002, 2004), which suggests that BAC could pose

	threat to the central nervous system."[8]
Developmental/Reproductive Toxicity	"Decreased reproductive performance in laboratory mice coincided with the introduction of a disinfectant containing both ADBAC and DDAC. QACs were detected in caging material over a period of several months following cessation of disinfectant use. Breeding pairs exposed for six months to a QAC disinfectant exhibited **decreases in fertility and fecundity:** increased time to first litter, longer pregnancy intervals, fewer pups per litter and fewer pregnancies. Significant morbidity in near term dams was also observed." Increased resorptions were recorded.[9] "Investigated gender-specific reproductive effects from ADBAC + DDAC. Female reproduction was assessed through ovulation, oocyte implantation, and estrus cycling. Male reproductive function was assessed by sperm concentration, motility, and viability. Numbers of corpora lutea were not different after 2 weeks, but decreased after 8 weeks of ADBAC + DDAC exposure. Dams exposed for 5 weeks to ADBAC + DDAC spent significantly less time in estrus. **ADBAC + DDAC exposed males exhibited declines in both sperm concentration and motility, but not sperm viability**. Subfertility in mice from ADBAC + DDAC exposure is, therefore, mediated through reproductive disturbances in both females and males. The study demonstrated that ADBAC + DDAC exposure is toxic to both male and female fertility. Female mice exposed to ADBAC + DDAC exhibited decreased reproductive capacity with reduced ovulation and fewer estrus cycles."[10] Neural tube defects (NTD) were seen in both rats and mice following ambient exposure to the QAC's containing disinfectant in the mouse room. NTDs were also observed in mice dosed with the disinfectant at 60 or 120 mg/kg/day in feed, or with ADBAC+DDAC chemical by gavage at 7.5, 15, or 30 mg/kg/day. Mice received ambient exposure for 2 weeks or ambient and gavage. NTDs were seen with ADBAC and DDAC dosed acutely by oral gavage, chronically in feed, and ambiently through the use of disinfectant in the mouse room. Both ambient and ambient and gavage groups exhibited significantly increased levels of NTDs compared with unexposed controls (Hrubec, 2017).[11] Note also QAC industry critique of Hrubec 2017 study[12] "The effects of the spermicide benzalkonium chloride (BKC) were studied on the conceptus of rat. Single doses (0, 25, 50, 100 or 200 mg kg-1) of aqueous solutions of BKC were administered intravaginally (1

	ml kg-1) on gestational day 1. The vulval metallic clips, used to prevent leakage of the solution, were removed 24 h post-treatment. Fetuses were obtained and examined for malformations on day 21 of gestation. Slight to copious amounts of vaginal discharge and vaginitis were noticed in rats treated with the two largest doses of BKC. **A dose-related increase in resorptions and fetal death, reduction in litter size and weight were observed in BKC-treated dams.** The conceptus loss seemed to occur both before and after implantation. BKC did not cause any discernible visceral malformations, although minor sternal defects occurred in fetuses exposed to 100 and 200 mg kg-1 of the spermicide. These results suggest that single vaginal application of BKC is embryo- and fetocidal in the rat at a dose about 143 times higher than that recommended for controlling conception in women."[13] "Some QACs have historically been used in the US as the active compound in fertility control. In an in vitro screening of organic compounds, Holzaepfel et al. (1959) identified some QA salts as having high spermicidal activity, including benzylhexadecyldimethyl ammonium chloride, n-octadecyldimethylbenzyl ammonium chloride (C18), and alkyldimethylbenzyl ammonium chloride. A US patent from the 1970s describes QACs (including BACs, DADMACs, ATMACs, and QAC mixtures) as having the capability of controlling fertility if administered at the time of mating or within an effective period after mating (Dalgard and Coval, 1975). This patent described dog and rat studies that indicated QACs administered via the diet may be embryocidal, ovicidal, and/or spermicidal. Benzalkonium chloride is used as the active spermicidal ingredient in some sponges and vaginal creams and capsules currently sold in Europe (Aubeny et al., 2000; Creatsas et al., 2001; Pharma GDD website, accessed 2020). Its spermicidal mechanism of action occurs through destruction of the sperm cell plasma membrane (Creatsas et al., 2001). Plasma membrane disruption is also the general mechanism of action by which QACs, including benzalkonium chloride, are effective as preservatives, disinfectants, and biocides (Gilbert and Moore, 2005; Wessels and Ingmer, 2013)."[14] "The prenatal developmental toxicity potential of ADBAC and DDAC was evaluated in regulatory compliant studies. Pregnant female CD® rats (25/group) and New Zealand White rabbits (16/group) were administered ADBAC (0, 10, 30 or 100 mg/kg/day and 0, 1, 3 or 9 mg/kg/day, respectively), or DDAC (0, 1, 10 or 20 mg/kg/day and 0, 1, 3

	or 10 mg/kg/day, respectively), by oral gavage on gestation days (GD) 6-15 for rats and GD 6-18 for rabbits. At scheduled termination (GD 21 for rats; GD 29 for rabbits), maternal necropsies were conducted and live fetuses were weighed and examined for external, visceral, and skeletal malformations and variations. Clinical signs of maternal toxicity were observed in rats and rabbits dosed with ADBAC, resulting in no-observed-adverse-effect levels (NOAELs) of 10 and 3 mg/kg/day, respectively. Despite the treatment-related maternal toxicity of ADBAC, the NOAEL for prenatal developmental toxicity was 100 and 9 mg/kg/day for rats and rabbits, respectively, the highest doses evaluated. **Repeated oral doses of DDAC resulted in maternal toxicity in both species at the top two doses, with 25% mortality noted in rabbits at 10 mg/kg/day. No teratogenic effects were observed at any dose for either species. However, increased incidence of dead fetuses per litter and decreased fetal body weights were observed in rabbits at the maternally lethal dose of 10 mg/kg/day.** The NOAEL for maternal toxicity of DDAC was 1 mg/kg/day for both species and the NOAEL for prenatal developmental toxicity was 20 and 3 mg/kg/day, for rats and rabbits, respectively."[15,16,17,18]
Genotoxicity/Mutagenicity	"We tested two important representatives, namely, benzalkonium chloride (BAC) and dimethyldioctadecyl-ammonium bromide (DDAB) in four genotoxicity tests. With BAC, a moderate but significant effect was found with an exposure concentration of 1.0 mg/l while DDAB caused damage at lower doses (0.3 mg/l). Our findings show that both chemicals induce moderate but significant genotoxic effects in eukaryotic cells at concentrations which are found in wastewaters and indicate that their release into the environment may cause genetic damage in exposed organisms. Furthermore, the direct contact of humans to QAC-containing detergents and pharmaceuticals that contain substantially higher concentrations than those which were required to cause effects in eukaryotic cells in the present study should be studied further in regard to potential DNA-damaging effects in man (Ferk, 2007)."[19] The genotoxicity and mutagenicity of DDAC and C12–C16 ADBAC has been investigated in a number of in vitro and in vivo test systems (Bacterial Reverse Mutation Test, Mammalian Cell Gene Mutation Test, Mammalian Chromosome Aberration Test, Unscheduled DNA Synthesis Assay, In Vivo Mammalian Bone Marrow Chromosome Aberration Test) that adhere to current OECD and OCSPP test guidelines. Collectively, available studies indicate that DDAC and C12–C16 ADBAC are non-

	mutagenic and non-clastogenic, and do not cause unscheduled DNA synthesis. See "ADBAC ISC Provided Comments on ADBAC EHS Summary" for additional information and summary table of results.
Endocrine Disruption	Females exposed to ADBAC + DDAC demonstrated significantly decreased ovulatory capacity, spent less time in estrus, and progressed through fewer estrus cycles compared to controls. It is, therefore, conceivable that ADBAC + DDAC reduced estrus length and frequency through disruption of estrogen-regulated processes. ADBAC + DDAC treated mice had significantly fewer estrus cycles over the evaluation period. This correlates directly with our 6 month breeding trial which observed significantly fewer litters produced in ADBAC + DDAC treated mice. These two findings reinforce that ADBAC + DDAC disrupt reproductive function in female mice (Melin, 2016).[20] "Based on a thorough review of all identified data pertinent to the potential endocrine activity and ED–mediated adversity of ADBAC, it is concluded that the substance is not an endocrine disruptor according to the criteria laid down in Regulation (EU) 2018/605. There is no evidence for ADBAC to cause adverse effects as a consequence of an endocrine mode of action."[21] Alkyl (C12-16) dimethylbenzyl ammonium chloride does not fulfil criterion (d) of Article 5(1) and is not considered an endocrine disruptor by the EU.[22] "Endocrine disruption potency of BKC was compared with those of other frequently used preservatives using human adrenal (H295R) and rat pituitary (GH3) cells. High concentration of BKC was detected at locations near the pharmaceutical manufacturing plants, i.e., 35.8 ug/L for dodecyl benzyl dimethyl ammonium chloride (BKC-C_{12}), and 21.6 ug/L tetradecyl benzyl dimethyl ammonium chloride (BKC-C_{14}). In Daphnia, 48 h immobilization EC_{50} and 21 d reproduction NOEC were determined at 41.1 ug/L and ug/L 10.8 mg/L, respectively. For O. *latipes*, 96 h LC_{50} was determined at 246 ug/L while the growth inhibition NOEC was 113.4 ug/L following early life stage exposure. **BKC significantly up-regulated vitellogenin gene of juvenile fish, indicating its endocrine disrupting potential in fish. Exposure to BKC increased steroid hormone level in H295R cells, and induced cytotoxicity in GH3 cells.** HQ values of BKC were determined at greater than one in the ambient water near pharmaceutical manufacturing facilities."[23]

	See Excel download of EPA Comp Tox Data with active endocrine disruption assays listed and Table of Comptox Results summary. The CompTox data include results of testing for activity associated with nuclear receptors, a subset of which are known to be involved in the regulation of lipid and cholesterol homeostasis. We include these assay results because of the research showing/suggesting the effects of QACs on lipid and cholesterol homeostasis. Presented in the summary table are the biological activity data reported for QACs with interaction with nuclear receptors that are well known to be involved in regulation of lipid and cholesterol homeostasis. These include the androgen receptor (AR), estrogen receptor α (ERα) and thyroid hormone receptor β (THRβ) are most common across the tested QACs. Also included are data from interactions with glucocorticoid receptor (GR) and liver X receptor (LXR). The biological activity data reported in CompTox provide additional evidence to suggest that multiple QACs could impact the ability of cells to synthesize/metabolize/transport lipids and cholesterol.
Thyroid	
Immunotoxicity	"This study demonstrates that the Th2-dependent adjuvanticity of QACs requires a minimum length of the alkyl chains. The adjuvant effect was not related to the suppressive effect on antibody production. Simultaneous administration of TEA, a QAC without adjuvant effect, and DDA, a QAC with adjuvant effect, increased the adjuvanticity. This may be of practical importance, since many QAC-containing products contain mixtures of several QACs."[24]
Liver	The repeated dose oral toxicity of C12–C16 ADBAC has been investigated in four subchronic 90-day studies with rats and beagles, and in four chronic studies conducted with beagles (52-week study), mice (78-week study), and rats (two 104-week studies). Reported NOAELs range from 3.7 to 188 mg/kg-day C12–C16 ADBAC. Toxicological effects most consistently reported are characterized by reduced food consumption, and reduced body weight and reduced weight gain. These effects are consistent with the MoA for an irritating/corrosive chemical (EPA, 2006b,d; ECHA, 2015b). (Luz et al., 2020) A reduction in absolute and relative liver weights were observed in males (5000 ppm) was a non-critical effect observed in a 90-day OECD 208 study. (Luz et al., 2020) In a second OECD TG 416 study, male and female Sprague Dawley rats

	(25/sex/dose) were fed an admixture containing 0, 500, 2000, or 4000 ppm ADBAC (purity 49.9%; CIT, 2008c unpublished study, as cited in ECHA, 2015b, 2019b). Parental toxicity was reported in both sexes of the P0 and P1 generations and included reduced liver weight (≥2000 ppm in P0; ≥500 ppm in P1). (Luz et al., 2020)
Other organ toxicity	"Kinetic characteristics and toxic effects of benzalkonium chloride (BZK) following injection via jugular vein (JV), femoral artery (FA) and oral administration (PO) were experimentally investigated using rats. The BZK concentrations in blood and tissues (lung, liver and kidney) were determined by high-performance liquid chromatography with solid phase extraction. Toxic doses of 15 and 250 mg/kg of BZK were used for intravascular (JV and FA) and PO administration, respectively. The fatal effects appeared soon after the dose in JV-rats, while delayed in FA- or PO-rats. The blood BZK concentrations and the elimination half-lives were similar between JV- and FA-rats, while the distribution of BZK in tissues was slightly different. In PO administration, the rats that aspirated BZK into their lungs had some symptoms, while the rats that did not aspirate BZK appeared to be normal. The BZK concentrations in blood and tissues were significantly higher in the aspirated PO-rats. The toxic degree of BZK was correlated with the BZK concentration in orally dosed rats. **Lung and kidney had higher BZK concentrations compared to blood or liver, and they could be the target organs of BZK**."[25]
Skin, Eye and Respiratory Effects	
*Irritant – **S**kin, **E**ye, or **R**espiratory*	"Our study showed BAK to be an allergen of increasing importance. From 1998 through 2000, 2001 through 2005, and 2006 through 2010, the rate of allergic patch test results to BAK increased. More than half of the reactions in each period studied were graded as macular erythema, with at least one third of all reactions deemed to be relevant. Irritancy rates were consistently low (Wentworth, 2016)."[26] "This analysis of routine clinical data and a number of previous reports add further, if weak, evidence to the notion that BAC is a contact allergen, albeit a very rare one (Uter, 2008)."[27] Two OCSPP 870.2500 studies have been conducted with C12–C16 ADBAC. In the first study, 0.5 mL of a formulation containing 82.26% ADBAC was applied to the skin of one female rabbit (Product Safety Labs, 1999a unpublished study, as cited in EPA, 2006d). Well-defined erythema and moderate edema were observed within 1 h of the exposure, and clear signs of dermal corrosion were noted by 72 h. In a second study, a formulation containing C12–C16 ADBAC (purity 49.2%)

	was applied to the skin of six rabbits for 24 h, and then the rabbits were observed for an additional 72 h (Bio-Toxicology Laboratories, Inc. 1975a unpublished study, as cited in ECHA, 2015b, 2019b). Severe edema and erythema were observed, and the test substance was classified as irritating to skin. (Luz et al., 2020)
*Corrosive – **S**, **E**, or **R***	Skin Corr. 1B; H314: causes severe skin burns and eye according to CLP (EC 1272/2008) criteria. Rabbit eye using undiluted test substance produced severe and irreversible damage in rabbit eyes (Wallace, 1975).[28]
*Permanent Damage – **S**, **E**, or **R***	
*Sensitizer– **S** & **R***	"Hypersensitivity to benzalkonium chloride was asserted by allergologic exploration in the three cases reported. Contact allergy, a frequent phenomenon, was diagnosed in two cases. The third case represents one of the few cases described in ophthalmology of immediate hypersensitivity associated to vital risk: chemosis and angioneurotic edema is reported in a woman who had been already presented an anaphylactic shock to a quaternary ammonium family anaesthetic (Chiambaretta, 1997)."[29] Unpublished studies cited by EPA and ECHA: Two OCSPP 870.2600 studies (Buehler Method) with guinea pigs have been carried out to investigate the skin-sensitizing potential of C12–C16 ADBAC (Product Safety Labs, 2005, 1999b unpublished studies, as cited in EPA 2006d & ECHA, 2015b, 2019b). Both studies concluded that C12–C16 ADBAC is not a dermal sensitizer, while a third non-guideline study concluded that C12–C16 ADBAC is not a photosensitizer (Hill Top Biolabs Inc. 1988 unpublished study, as cited in EPA, 2006d; ECHA, 2015b). (Luz et al., 2020)
Asthmagen – Initiator or Exacerbator	AOEC: Ashthmagen (Rs) – sensitizer-induced[30] Two papers were cited to support this conclusion (Bernstein et al, 1994; Purohit et al, 2000). "Exposure to QACs increased significantly the risk of reported physician-diagnosed asthma and nasal symptoms at work (adjusted OR = 7.5 and 3.2, respectively). No significant association was found with other exposures such as latex glove use, chlorinated products/bleach or glutaraldehyde".[31] "The three patients in this study, all of which were female nurses manifested asthma symptoms upon handling disinfectant solutions containing benzalkonium chloride. Work-related fall in PEFR (Peak

	expiratory flow rate) was observed in all of them. The diagnosis was confirmed by challenge tests where the patients were exposed, in a closed chamber, to suspected disinfectant contained in a tray. All of the women developed early or delayed symptoms upon exposure. Similar challenge tests to placebo or other disinfectants devoid of quaternary ammonium compound were negative (Purohit, 2000)."[32] "We describe a case of occupational asthma caused by prolonged exposure to a cleaning solution containing benzalkonium chloride in the workplace. Single-blind, placebo-controlled, open-room challenges were performed to determine the specific agent responsible for the patient's symptoms. Pulmonary function and epicutaneous challenge tests were also performed. The patient had positive responses to challenges with a liquid toilet bowl cleaner containing benzalkonium chloride but was unreactive to other agents tested. Removal from the workplace resulted in complete resolution of symptoms. The exact mechanisms responsible for occupational asthma induced by quaternary amines remain unknown; however; this case emphasizes the importance of recognizing reactive chemicals as possible causes[33]." Benzalkonium chloride is used as a preservative in some multidose nebulized bronchodilators. BACs bronchoconstrictive properties in relation to this use are well documented.[34] "Challenge exposure to the suspected cleaning agents elicited a ≥20% fall in forced expiratory volume in 1 s (FEV1) in 17 (39%) participants. The cleaning products that induced a positive specific inhalation challenge (SIC) contained quaternary ammonium compounds (n=10), glutaraldehyde (n=3), both of these agents (n=1) and ethanolamines (n=2). Positive SICs were associated with a significant decrease in the median (IQR) value of the provocative concentration of histamine causing a 20% fall in FEV1 (PC20) from 1.4 (0.2–4.2) mg/mL at baseline to 0.5 (0.4–3.0) mg/mL after the challenge and a significant increase in sputum eosinophils from 1.8 (0.8–7.2)% at baseline to 10.0 (4.1–15.9)% 7 h after the challenge exposure while these parameters did not significantly change in participants with a negative SIC. Overall, 11 of 17 participants with positive SICs showed greater than threefold decrease in post challenge histamine PC20 value, a >2% increase in sputum eosinophils, or both of these outcomes. Conclusions: **These data indicate that a substantial proportion of workers who experience asthma symptoms related to cleaning materials show a pattern of**

	bronchial reaction consistent with sensitiser-induced occupational asthma. The results also suggest that quaternary ammonium compounds are the principal cause of sensitiser-induced occupational asthma among cleaners."[35] "BAC (4.1–4.5 mg/m^3, sprayed from 0.5% solution) promoted pulmonary cell damage and inflammation as depicted by the increase in total protein, lactate dehydrogenase, polymorphonuclear leukocytes, and macrophage inflammatory protein-2 in the bronchoalveolar lavage fluid."[36] "High-level exposure, evaluated by the JTEM, to several specific disinfectants (ie, glutaraldehyde, bleach, hydrogen peroxide, alcohol, and quaternary ammonium compounds) was significantly associated with COPD incidence, with adjusted hazard ratios ranging from 1.25 (95% CI, 1.04-1.51) to 1.36 (95% CI, 1.13-1.64). Associations were not modified by smoking or asthma status (P for interaction > .15)."[37] "Weekly use of disinfectants to clean surfaces only (23% exposed) or to clean medical instruments (19% exposed) was not associated with incident asthma (adjusted hazard ratio [95%CI] for surfaces, 1.12 [0.87–1.43]; for instruments, 1.13 [0.87–1.48])."[38] "Atopic sensitization (defined as increased production of IgE to common allergens) was found to occur more frequently in farmers who used disinfectants containing quaternary ammonium compounds (QACs) (odds ratio (OR) 7.4; 95% confidence interval (95% CI) 1.3–43.1)."[39] "The onset or aggravation of asthma in this group could be related to an irritant-induced mechanism or to specific sensitization. The main sensitizers contained in cleaning products are disinfectants, quaternary ammonium compounds (such as benzalkonium chloride), amine compounds, and fragrances."[40] "The QACs gave rise to concentration-dependent decreases in the tidal volume (VT) and a concomitant increase in respiratory rate indicating pulmonary irritation. The potencies of the QAC to induce these effects were in the order: BAC > HTA = CPC > DDA. Furthermore, inhalation of BAC and CPC aerosols gave rise to pulmonary inflammation as apparent from bronchoalveolar lavage."[41]

	A study evaluated work related asthma (WRA) in France from 2001-2009 and found a significant inscrease in WRA related to exposure to QACs in high risk industries like healthcare.[42]
Skin Absorption, Kp	No in vivo studies investigating the dermal absorption of C12–C16 ADBAC were identified. An OECD TG 428 (Skin Absorption: In Vitro Method) study to investigate the absorption of 0.03% and 0.3% (weight for weight (w/w) in water) 14C-ADBAC across human skin membranes has been conducted (Charles River Laboratories, 2006, as cited in ECHA, 2015b, 2019b). Only 0.03–0.05% of the administered dose was found to fully penetrate human skin within a 24-h period, while 8.3% of the active substance was detected in the receptor fluid and skin after stratum corneum removal, which led ECHA to conclude that dermal absorption does not exceed 10% at non-corrosive doses. Similarly, based on molecular weight, lack of lipophilicity, and ionic state, EPA (2006d) concluded that dermal absorption of C12–C16 ADBAC does not exceed 10% (Luz et al., 2020). Study investigated skin permeability for single and repeated exposure of nine different compounds, including ADBAC and DDAC. "Of these nine compounds, only the quaternary ammonium chlorides ADBAC and DDAC had a clear and consistent influence on skin permeability of the marker compounds tritiated water and [^{14}C]propoxur. For these compounds, repeated exposure increased skin permeability more than single exposure. At high concentrations the difference between single and repeated exposure was quantitatively significant: repeated exposure to 300 mg/L ADBAC increased skin permeability two to threefold in comparison to single exposure. Therefore, single and repeated exposure to specific biocidal products may significantly increase skin permeability, especially when used undiluted."[43] (Buist, 2005)
LOAEL	Inhalation LOAEC < 0.08 mg/m^3 Chronic Dietary (Rat) – 88 mg/kg/day Incidental Oral (Rat) – 30 mg/kg/day[4]
NOAEL	Chronic Dietary (Rat) – 44 mg/kg/day Incidental Oral (Rat) – 10 mg/kg/day Short-Term Dermal (Guinea Pigs) – 20 mg ai/kg/day[4]
Benchmark Dose Response (BMD)	
Toxicokinetics	"In this study, we aim to identify environmental molecules that can inhibit cholesterol biosynthesis, potentially leading to the same biochemical defects as observed in cholesterol biosynthesis disorders,

	which are often characterized by congenital malformations and developmental delay. Using the Distributed Structure-Searchable Toxicity (DSSTox) Database Network developed by EPA, we first carried out in silico screening of environmental molecules that display structures similar to AY9944, a known potent inhibitor of 3b hydroxysterol-D7-reductase (DHCR7)—the last step of cholesterol biosynthesis. Molecules that display high similarity to AY9944 were subjected to test in mouse and human neuroblastoma cells for their effectiveness in inhibiting cholesterol biosynthesis by analyzing cholesterol and its precursor using gas chromatography-mass spectrometry. We found that a common disinfectant mixture, benzalkonium chlorides (BACs), exhibits high potency in inhibiting DHCR7, as suggested by greatly elevated levels of the cholesterol precursor, 7-dehydrocholesterol (7-DHC). Subsequent structure-activity studies suggested that the potency of BACs as Dhcr7 inhibitors decrease with the length of their hydrocarbon chain: C10>C12>>C14>C16. Real-time qPCR analysis revealed upregulation the genes related to cholesterol biosynthesis and downregulation of the genes related to cholesterol efflux, suggesting a feedback response to the inhibition. Furthermore, an oxidative metabolite of 7-DHC that was previously identified as a biomarker in vivo was also found in cells exposed to BACs by liquid chromatography-mass spectrometry."[44] "We performed a high-through put screen of 1,600 antiseptics, additives, and drugs, and found that, of the numerous structural classes of compounds included in the screen, the QUATS were the most mitochondrially toxic class, both in terms of inhibition of ATP synthesis and mitochondrial O_2 consumption. QUATS have the structure of lipophilic cations, which are known to be preferentially taken up by mitochondria (Murphy and Smith 2007). In summary, our findings suggest that the QUATS CPC and BAK, which are used as disinfectants in consumer products, inhibit mitochondrial complex 1 and show antiestrogenic activity in vitro at low (micromolar) concentrations that may be physiologically relevant. We hypothesize a mechanistic relationship between these outcomes, where by the antiestrogenic activity of these compounds is mediated by mitochondrial inhibition."[45] "QAC cytotoxicity to MDCK II cells *in vitro* is initiated by mitochondrial dysfunction at sub-lethal concentrations, followed by mitochondrial fragmentation and decreased cellular energy charge at slightly higher

	concentrations. In isolated mitochondria all of the QAC tested were shown to act via a common mechanism involving inhibition of NADH ubiquinone oxidoreductase (complex I) and of mitochondrial ADP-phosphorylation. QAC-induced mitochondrial dysfunction results in apoptosis (concentrations ≤ LD90), followed by a shift to necrotic cell death at concentrations above LD90."[46] "This study tested whether QAC concentrations could be detected in the blood of 43 random volunteers, and whether QAC concentrations were associated with markers of inflammation, mitochondrial function, and cholesterol synthesis in a dose dependent manner. **QAC concentrations were detected in 80% of study participants, and were associated with decreased mitochondrial function and an increase in inflammatory cytokines in a dose dependent manner.** Cholesterol synthesis pathway intermediaries were generally increased, indicating disruption in cholesterol homeostasis. This is the first study to demonstrate that chronic exposure to QACs results in measurable concentrations in human blood, and to also demonstrate significant correlations between QAC level and meaningful biomarkers related to health."[47] "Rats were treated intravenously with BZK solution (dose, 13.9 mg/kg) containing equal amounts of the three homologues. Kinetic parameters in the blood were assessed, and BZK distribution in the blood and tissues was examined both in rapid intravenous (IV) and drip intravenous (DIV) administrations. BZK concentrations were analyzed by liquid chromatography-tandem mass spectrometry (LC-MS/MS). BZK with longer alkyl chains showed lower elimination tendencies and remained in the blood for a longer duration. Concentrations of BZK were higher in the heart, lung, spleen, and kidney than those in the blood, and lower in the brain and fat. In both the IV and DIV groups, the lung, liver, spleen, and fat samples showed higher concentrations of the longer alkyl chains (BZKC12 < -C14 < -C16), and the opposite trend was observed in the kidney (BZK-C16 < -C14 < -C12). Only the heart and muscle samples displayed the homologues in ratios comparable to the original administered solutions. Differences between IV and DIV groups could be identified by comparing concentrations of BZK homologues in the heart, lung, spleen, and kidney samples. We found that the kinetics and distribution of BZK were influenced by the alkyl chain length."[48]
Metabolites	"BAC metabolism in human liver microsomes was NADPH-dependent

	and displayed apparent half-lives that increased with BAC alkyl chain length ($C_{10} < C_{12} < C_{14} < C_{16}$), suggesting enhanced metabolic stability of the more lipophilic, longer chain BACs. Metabolites of d_7-benzyl labeled BAC substrates retained all deuteriums and there was no evidence of *N*-dealkylation. Tandem mass spectrometry fragmentation of BAC metabolites confirmed that oxidation occurs on the alkyl chain region. Major metabolites of C_{10}-BAC were identified as ω-hydroxy-, (ω-1)-hydroxy-, (ω, ω-1)-diol-, (ω-1)-ketone-, and ω-carboxylic acid-C_{10}-BAC by liquid chromatography-mass spectrometry comparison with synthetic standards. CYP-mediated oxidation of C_{10}-BAC mitigates the potent inhibition of cholesterol biosynthesis exhibited by this short-chain BAC."[49]
Synergistic or Antagonistic Effects	
Environmental and Human Health Exposure and Risk Values	
RfC/RfD	US EPA has established that the chronic population adjusted dose (cPAD) for ADBAC is 0.44 mg/kg/day; cPAD is essentially equivalent to a reference dose (RfD) defined as a daily oral exposure that is likely to be without appreciable risk of deleterious effects during a lifetime (EPA, 2017).
ATSDR-MRL	
Adverse Effect Levels: DNEL, PNEC, PNEL	
Health Based Exposure Limits	
NIOSH-REL/IDLH/Ceiling Limits	Recently published Occupational Exposure Limit (OEL) = 0.1 mg/m3 (Dotson, et al., 2020).[50]
OSHA-PEL	
ACGIH TLV-TWA	
TLV-STEL	
Biomonitoring Action Limits	California Biomonitoring Scientific Guidance Panel Meeting voted on March 8, 2021 to include the class of Quaternary Ammonium Compounds to the Priority Chemicals List.
Drinking Water Standards	
Other	Since the 2006 RED, 2237 individual human health incidents have been reported for ADBAC in OPP's Incident Data System (IDS) from September 1, 2006 to March 3, 2017. The largest number of incidents (814) occurred when handling liquid concentrate products, followed by ready to use (RTU) spray products (337), RTU trigger sprayer products (244), and RTU wipes (235). A few incidents are summarized below: • A maintenance worker at a gas station used an ADBAC/DDAC disinfectant product. Another worker there was allegedly exposed to it

	and developed respiratory distress and ultimately died. She had previously had chronic obstructive pulmonary disease. • An airline employee developed respiratory distress resulting in death. Chemical exposure to an ADBAC/DDAC product and other three [sic] cleaning products was the potential cause. No other details were provided. • A two year old asthmatic child who used a breathing machine died after removing an ADBAC RTU household cleaning and disinfectant product from an unlocked cabinet and spilling it on toys. • A 34 year old diabetic resident of a nursing home had a heart attack and later died at the hospital. A partially used can of ADBAC foam product was found in her room. The director of nursing indicated that this patient had previously used this product to clean her room without incident. • A 68 year dementia patient in a nursing home ingested an ABDAC/DDAC [sic] disinfectant product that was being used to clean wheelchairs during the overnight shift.[51] "The case is described of a 44 year old pharmacist who developed severe occupational asthma threatening his continued employment, confirmed by serial measurement of peak expiratory flow at home and work. The cause was found to be the cleaning agent used in his office when it was unoccupied. Bronchial challenge testing identified the specific agent to which he was sensitized as lauryl dimethyl benzyl ammonium chloride, a constituent of the floor cleaner. Substitution of this floor cleaner by a simple detergent cleaner led to a substantial improvement in his asthma, confirmed by repeated serial peak flow measurements."[52] "Alkyl (C12-16) Dimethylbenzylammonium Chloride was assigned a **Benchmark Score of 1** based on criteria 1c. very high persistence combined with very high aquatic toxicity and very high toxicity for Group II Human endpoints (acute toxicity, skin irritation, and eye irritation). It may be noted that fewer data gaps would not impact this Benchmark score."[53]
ENVIRONMENTAL & ECO-SYSTEM HAZARDS	
PBT	
Persistence	"We have determined the occurrence of 19 QACs in residential dust collected before and during the COVID-19 pandemic. QACs were detected in >90% of the samples collected during the pandemic at concentrations ranging from 1.95 to 531 µg/g (n = 40; median of 58.9

	µg/g). The total QAC concentrations in these samples were significantly higher than in samples collected before the COVID-19 pandemic ($p < 0.05$; n = 21; median of 36.3 µg/g). Higher QAC concentrations were found in households that generally disinfected more frequently ($p < 0.05$). Disinfecting products commonly used in these homes were analyzed, and the QAC profiles in dust and in products were similar, suggesting that these products can be a significant source of QACs. Our findings indicate that indoor exposure to QACs is widespread and has increased during the pandemic."[54]
Bioaccumulation	
BAF	
BCF	The whole body BCF in fish was estimated to be 79.[2] "There is potential for acute exposure to aquatic organisms in the water column because of the high solubility of ADBAC in water… However, bioconcentration in aquatic organisms is not expected despite the high log K_{ow} of 3.91 (>3) because ADBAC is highly soluble in water and, being a positively-charged compound, is tightly sorbed to soil and sediment, which are typically negatively-charged."[3]
BMF	
Ecological Toxicity	ADBACs have a strong tendency to bind to sediment/soil (K_{ads} >5000, K_{oc} >600,000) and chronic exposure to benthic invertebrates is expected. One chronic sediment toxicity study is available for the midge: 80% ai; 28-d NOAEC - 520 mg/kg sediment; 28-d LOAEC - 1200 mg/kg sediment; 14-d LC50 = 548 mg/kg sediment. Chronic studies also are anticipated to be required for a freshwater amphipod and an estuarine/marine amphipod.[3] Additional testing anticipated to be required for aquatic plant toxicity and pollinators (honeybees). [3]
Aquatic Toxicity: LC_{50}, EC_{50}, ErC_{50}, NOAEC/NOEC	On an acute exposure basis, ADBAC is **highly toxic** to freshwater and marine/estuarine fish and freshwater invertebrates. Freshwater invertebrates are especially sensitive to ABDAC on an acute exposure basis, as the acute toxicity classification for ABDAC is very highly toxic. **Freshwater fish LC_{50}** = 280 ug ai/L; **NOAEC** = 32.2 ug ai/L **Estuarine/marine fish LC_{50}** = 310 ug ai/L **Freshwater invertebrates EC_{50}** = 5.9 ug ai/L; **NOAEC** = 4.15 ug ai/L[3,20] Exposures to benzalkonium chloride or benzethonium chloride at concentration ranges (0.05 - 5 mg/L) resulted in toxicity effects to zebrafish embryos and in germline toxicity in C. *elegans*. **These effects**

	included delayed hatching, embryonic mortality, morphological malformations, and neurotoxicity. BAC was the most toxic, with acute lethal toxicity occurring at environmentally relevant concentrations (hundreds of mg/L), which is comparable to other banned chemicals including triclocarban and triclosan.[55]
Mammalian Toxicity: LC_{50}, EC_{50}, ErC_{50}, NOAEC/NOEC	ADBAC is categorized as slightly toxic to mammals on an acute basis **(LD_{50} = 430 mg/kg-bw)** and a chronic **NOAEC** of 44 mg/kg/day was established.[4]
Wildlife Toxicity: LC_{50}, EC_{50}, ErC_{50}, NOAEC/NOEC	The results of the dietary avian studies categorized ADBAC as moderately toxic to birds on an acute basis **(LC_{50} = 136 mg/kg-bw),** with no chronic data available.[4]
Breakdown/degradation /combustion products	
Anaerobic degradation	"There is uncertainty about whether sorption or biodegradation of ADBAC would predominate during wastewater treatment. In the absence of data on the extent for ADBAC to sorb to sludge biomass during wastewater treatment, data from an Activated Sludge Sorption Isotherm (ASSI) study are needed."[3]
Aerobic degradation	"Based on a biodegradation study, ADBAC readily degrades into 60% carbon dioxide in 13 days. ADBAC is immobile in soil. The available soil mobility study shows that ADBAC has a strong tendency to bind to sediment/soil with Freundlich Kads values range from 5,123 to 32,429 depending on the soil type whereas the corresponding Koc values range from 640,389 to 6,171,657 depending on the soil type. Due to its strong adsorption to soils, ADBAC is not expected to contaminate surface and ground waters."[4]
Other observable ecological effects (e.g. BOD)	
Fate and Transport: Aquatic	Quaternary ammonium compounds have been detected worldwide in domestic wastewater, sludge, treated effluent, surface water, and sediment. It is anticipated that the majority of QAC applications leads to their eventual release into sewers and WWTPs. These compounds have been detected in aquatic environments, and at higher concentrations in locations downstream of the discharge of municipal WWTP effluents, hospitals, and industrial effluents (Hora, 2020). A solid phase extraction method was developed and used for determination of Benzalkonium chloride (BAC - CAS 8001-54-5) in the

	environment. "Concentration levels of BAC - ranging from 1.2 to 36.6 micrograms per liter - were found in [untreated wastewater and] surface-water samples collected downstream from different wastewater treatment discharges, thus indicating its input and persistence through the wastewater-treatment process." Note: for untreated wastewater, sample results for combined C_{12}BAC and C_{14}BAC varied from 12 µg/L to 42 µg/L, while stream surface water samples varied from below the detection limit (5 out of 7 locations) to 3.7 µg/L.[56] Presence in surface water and sediments upstream and downstream of WWTPs in Austria: Σ C_{12}-C_{18} BAC: Surface water ranges from 0.04 - 2.61 µg/L; sediment ranges from 7 - 5840 µg/kg dm Samples downstream of WWTP often were less than samples taken upstream - both for surface water and sediment, so presence was not heavily impacted by WWTP discharge. Study also sampled waste water from different businesses; hospitals and laundries had the highest values for C_{10}-C_{18} DDAC (max 176 µg/L) and C_{12}-C_{18} BAC (max 3929 µg/L) [57] Another study using the same dataset from Austria: Surface water concentrations are mostly below 0.1 ug/L, one instance of 0.69 ug/L (BAC-C16) and 1.9 ug/L (BAC-C-12). WWTP effluent is higher at 4.1 ug/L (BAC-C-12). "It needs to be stressed that the lowest actual ecotoxicological effective concentrations determined (41mg BAC/L) exceeded the highest concentrations measured in surface waters (1.9mg BAC/L) and effluents (1.6mg BAC/L) by only one order of magnitude.".[58] "Concentration in effluents varied over a large range. A majority of the "high concentration" effluents showed, even after ten times dilution, concentrations of ATACs and/or BACs that could pose a risk to the aquatic environment. Ecotoxicological data is lacking especially for long chained ATACs. All the selected compounds were present in all sludges. Concentrations in sludge varied much less than in effluents. Patterns of individual compound concentrations were not clearly different among countries."[59]

	QAC use in de-mossing urban surfaces and roofs. Also looked at four different rain events in July – Oct. 2011 with 1-2 cm of rain. Storm water concentration of 7-28 ug/L (greater than LC50 values for macroinvertebrates), but dissolved fraction was 0.2 ug/L. Stormwater samples were collected, "from the storm drain at the outlet of a strictly residential area (total surface area 2.1 km2)." Study took place in France.[60] "The developed method was applied to determine the QACs in 27 vegetable samples collected from Guangzhou in southern China, showing very high detection frequency with a concentration of 23–180 μg/kg (dw)."[61] "The analysis of sewage sludge samples and soil samples revealed that BAC-C12 was the most abundant QAAC with concentrations up to 38600 μg kg-1 in sewage sludge and up to 81 μg kg-1 in a Mexican soil that was irrigated with wastewater. Overall, the presented methods open perspectives for effectively studying fate and effects of QAACs in soils."[62] "The distributions of wastewater-derived quaternary ammonium compounds (QACs) were determined in surficial sediments (n = 47) collected from the urbanized lower Hudson River basin. The most abundant class of QACs were dialkyldimethylammonium compounds (DADMACs, with C8 to C18 carbon chain lengths; median ΣDADMAC) 26 μg/g), followed by benzylalkyldimethylammonium compounds (BAC, C12-C18; 1.5 μg/g), and alkyltrimethylammonium compounds (ATMAC, primarily C16 and C18; 0.52 μg/g). The concentrations of total QACs are higher than those of other conventional organic contaminants determined on the same sampless (e.g., median ΣPAH level of 2.1 μg/g). Comparatively high concentrations, correlations with sewage derived contaminants, and the relatively constant compositions of QACs observed over large areas suggest that many sediment-sorbed QACs can be relatively persistent in receiving waters. Unusually large concentration-dependent sorption is considered as a mechanism that likely affects persistence of these intrinsically biodegradable chemicals under field conditions."[63]

Fate and Transport: Terrestrial	
Fate and Transport: Atmospheric	
Transport Issues	"ADBAC is completely soluble in water, and based on its low vapor pressure and Henry's Law value, is not expected to partition from soil and water into air. ADBAC is stable to hydrolysis at pH values of 5, 7, and 9 (MRID 40835602), with half-lives ranging from 150 to 379 days, and stable to photodegradation in pH 7 buffered aqueous solutions, but degraded in water in the presence of a photosensitizer with a half-life of 7 days (MRID 40835603)".[3]
Factors affecting bioavailability	
Global Environmental Impacts	
Ozone Depletion Potential (ODP)	
Global Climate Change	
Greenhouse Gas Production	
Acid Rain Formation	
Special Reports	
EU/Other Countries	**EU Hazard Classification & labeling:** *Danger!* According to the classification provided by companies to ECHA in CLP notifications this substance is toxic if swallowed, causes severe skin burns and eye damage, is very toxic to aquatic life, is toxic in contact with skin, is very toxic to aquatic life with long lasting effects and causes serious eye damage.[2]

**Not all comments from QRG were added to this EHS Summary; please see "ADBAC ISC Provided Comments on ADBAC EHS Summary" on the Libguide for additional information and references.

Number of EPA Registered Products that contain ADBAC – 2017 FWP

PC code	CAS Number	Ingredient Name	Number of active AD registrations as of	Number of active RD registrations as of
069104	53516-76-0	Alkyl (60%C_{14}, 30%C_{16}, 5%C_{18}, 5%C_{12}) dimethyl benzyl ammonium chloride	271	2
069105	68424-85-1	Alkyl (50%C_{14}, 40%C_{12}, 10%C_{16}) dimethyl benzyl ammonium chloride	329	1
069106	8001-54-5	Alkyl (50%C_{12}, 30%C_{14}, 17%C_{16}, 3%C_{18}) dimethyl benzyl ammonium chloride	2	0
069107	139-08-2	Alkyl (100% C_{14}) dimethyl benzyl ammonium chloride	4	0
069111	8045-21-4	Alkyl (50%C_{12}, 30%C_{14}, 17%C_{16}, 3%C_{18}) dimethyl ethylbenzyl ammonium chloride	13	0
069119	73049-75-9	Dialkyl (60% C_{14}, 30% C_{16}, 5% C_{18}, 5% C_{12}) methyl benzyl ammonium chloride	37	0
069122	121-54-0	Benzenemethanaminium, N,N-dimethyl-N-(2-(2-(4-(1,1,3,3-tetramethylbutyl)phenoxy)ethoxy)ethyl)-, chloride	11	0
069125	1330-85-4	Dodecylbenzyl trimethyl ammonium chloride	1	0
069137	68424-85-1	Alkyl (60%C_{14}, 25%C_{12}, 15%C_{16}) dimethyl benzyl ammonium chloride	9	0
069140	61789-71-7	Alkyl (61% C_{12}, 23% C_{14}, 11% C_{16}, 2.5% C_{18}, 2.5% $C_{10,}$ trace C_8) dimethyl benzyl ammonium chloride	1	0
069141	68424-85-1	Alkyl (58%C_{14}, 28%C_{16}, 14%C_{12}) dimethyl benzyl ammonium chloride	23	0
069154	85409-23-0	Alkyl (68%C_{12}, 32%C_{14}) dimethyl ethylbenzyl ammonium chloride	203	2
069167	68956-79-6	Alkyl (60%C_{14}, 30%C_{16}, 5%C_{12}, 5%C_{18}) dimethyl ethylbenzyl ammonium chloride	2	0
069171	68989-01-5	Alkyl (50% C_{14}, 40% C_{12}, 10% C_{16}) dimethyl benzyl ammonium saccharinate	7	0
069175	68391-01-5	Alkyl (67%C_{12}, 25%C_{14}, 7%C_{16}, 1%C_{18}) dimethyl benzyl ammonium chloride	29	0
069184	68424-85-1	Alkyl (95%C_{14}, 3%C_{12}, 2%C_{16}) dimethyl benzyl ammonium chloride	16	0
069195	68391-01-5	Alkyl (41%C_{14}, 28%C_{12}, 19%C_{18}, 12%C_{16}) dimethyl benzyl ammonium chloride	1	0
128928	63449-41-2	Alkyl (67%C_{12}, 25%C_{14}, 7%C_{16}, 1%C_8, C_{10}, and C_{18}) dimethyl benzyl ammonium chloride	7	0
129012	61789-18-2	Alkyl (as in fatty acids of coconut oil) trimethyl ammonium chloride	1	0

[1] Toxplanet Chemical Identity page for Alkyl dimethylbenzyl ammonium chloride: https://chemical-search.toxplanet.com/#/product-search/chem-id/ei-fts-search/2813d580-b882-466f-9b5b-36d2c5a3d526

[2] European Chemicals Agency (ECHA) Substance Infocard for Quaternary ammonium compounds, benzyl-C12-16-alkyldimethyl, chlorides accessed at: https://echa.europa.eu/substance-information/-/substanceinfo/100.063.913

[3] United States Environmental Protection Agency (2017) Alkyls Dimethyl Benzyl Ammonium Chloride (ADBAC) Final Work Plan, Registration Review: Initial Docket, Case Number 03050, Docket Number EPA-HQ-OPP-2015-0737. See Appendix C for additional ecotoxicity results.

[4] United States Environmental Protection Agency (2006) Prevention, Pesticides and Toxic Substances; Reregistration Eligibility Decision for Alkyl Dimethyl Benzyl Ammonium Chloride (ADBAC) EPA's Pesticide Docket EPA-HQ-OPP-2006-0339

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