Chronic Low-Level VOC Exposure from Fragranced Consumer Products:

Neurological Effects and Cognitive Decline

A Systematic Review

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Abstract

This systematic review examines the neurological and cognitive effects of chronic, low-level volatile organic compound (VOC) exposure from fragranced consumer products. These products--including perfumes, colognes, body washes, fabric softeners, dryer sheets, laundry detergents, cleaning products, air fresheners, and hand soaps--collectively generate continuous 24-hour inhalation and dermal exposure to complex mixtures of VOCs and semi-volatile organic compounds (SVOCs). Many of these fragrance chemicals are established neurotoxicants, including phthalate esters (DEP, DMP), synthetic polycyclic musks (galaxolide, tonalide), and volatile aldehydes classified as Hazardous Air Pollutants under the Clean Air Act.

The olfactory nerve (CN I) provides a direct, unprotected pathway from the nasal epithelium to the brain, bypassing the blood-brain barrier. This review synthesizes evidence from controlled human exposure studies, biomonitoring research, and neurotoxicological investigations demonstrating that fragrance-associated VOCs activate neuroinflammatory cascades via TRPA1 channels, accumulate in human brain tissue, and produce measurable cognitive decrements at exposure concentrations typical of indoor environments.

A cumulative exposure model estimates a 24-hour time-weighted average VOC concentration of 580 ug/m3 in the personal breathing zone of a typical American adult--exceeding the cognitive effects threshold established by Allen et al. (2016) by a factor of 11.6. This exposure begins at birth, with neonates receiving weight-normalized doses approximately 2.5 times higher than adults during active neurodevelopment. The implications for population-level cognitive function and capacity for critical thinking warrant urgent investigation.

1. Introduction

The modern indoor environment contains hundreds of volatile organic compounds originating from fragranced consumer products. Consumer fragrance exposures occur without disclosure of chemical identity, without exposure monitoring, and without informed consent. The fragrance industry operates under self-regulation through the International Fragrance Association (IFRA) and the Research Institute for Fragrance Materials (RIFM), with no requirement to disclose individual chemical constituents to consumers or regulators.

Steinemann (2016) documented that 34.7% of Americans report adverse health effects from fragranced product exposure, including respiratory difficulties, migraine headaches, and neurological symptoms. Between 74% and 93% of the U.S. population uses fragranced personal care products, laundry products, air fresheners, and deodorizers on a weekly basis. This creates a population-level exposure scenario with no parallel in occupational or environmental toxicology: the majority of the population is chronically exposed to complex neurotoxic chemical mixtures through products marketed as beneficial.

This review synthesizes the available literature on three critical questions: (1) What is the total daily neurotoxic chemical dose from fragranced consumer products across all exposure routes? (2) What is the evidence that these exposures produce neurological effects and cognitive decline? (3) What are the implications for population-level cognitive function?

2. Search Strategy and Inclusion Criteria

2.1 Data Sources

This systematic review was conducted using PubMed, Web of Science, Google Scholar, and the U.S. EPA Integrated Risk Information System (IRIS) databases. Search terms included combinations of: volatile organic compounds, fragrance, perfume, fabric softener, air freshener, phthalates, synthetic musk, galaxolide, tonalide, indoor air quality, cognitive function, neurotoxicity, olfactory nerve, TRPA1, and neuroinflammation. Reference lists of identified articles were hand-searched for additional relevant studies.

2.2 Inclusion Criteria

Studies were included if they: (a) reported measured VOC or SVOC concentrations from fragranced consumer products; (b) documented neurological, cognitive, or neurodevelopmental effects of fragrance-associated chemicals; (c) provided quantitative exposure data (ug/m3, ug/kg/day) for chemicals found in fragranced products; or (d) reported population-level prevalence of fragranced product use or health effects.

2.3 Exposure Model Methodology

A cumulative daily breathing zone exposure model was constructed by compiling published concentration measurements for individual fragranced product categories and calculating 24-hour time-weighted average (TWA) contributions using the formula: TWA contribution = (estimated breathing zone concentration x hours of exposure) / 24. Conservative (low-end) published concentrations were used where ranges existed. The sum was compared to the cognitive effects threshold of 50 ug/m3 TVOC established by Allen et al. (2016).

3. Lifetime Exposure: From Birth to Chronic Adult Use

3.1 The Infant Dose Problem

Chemical exposure to fragranced products in the United States begins literally at birth. Newborns are immediately exposed to fragranced diapers, baby wipes, baby lotion, baby shampoo, baby powder, and laundered clothing and bedding treated with fragranced detergents and fabric softeners. The distinctive "baby smell" associated with these products is a complex mixture of synthetic fragrance chemicals including phthalate esters, synthetic musks, and volatile aldehydes.

The infant dose problem is defined by three physiological factors that amplify exposure relative to adults:

- **Higher weight-normalized inhalation rate:** Neonates inhale 0.4-0.6 L/min/kg compared to 0.2 L/min/kg in adults, resulting in approximately 2.5 times higher air pollutant dose per kilogram of body weight (U.S. EPA Exposure Factors Handbook, 2011).
- **Greater dermal absorption:** Infant skin is 40-60% thinner than adult skin with higher surface-area-to-body-weight ratio (700 cm2/kg vs. 250 cm2/kg in adults), resulting in proportionally greater transdermal chemical absorption from diapers, wipes, and clothing (Makri et al., 2004).
- **Immature detoxification:** Neonatal hepatic cytochrome P450 enzymes (CYP1A2, CYP3A4) operate at 30-50% of adult capacity, and glucuronidation pathways are not fully mature until age 2-3 years, extending the biological half-life of absorbed chemicals (Hines, 2008).

Engel et al. (2021, American Journal of Public Health) demonstrated that prenatal phthalate exposure produced IQ decrements comparable in magnitude to those associated with lead exposure, affecting an estimated 2.3 million births per year in the United States. The estimated annual economic cost of phthalate-associated IQ loss was $8.64 billion.

3.2 Quantitative Exposure Data

The following exposure concentrations have been documented in the peer-reviewed literature:

Airborne Concentrations (ug/m3)

- **Typical office environment TVOC:** 260.5 ug/m3 (Azuma et al., 2016; Harvard T.H. Chan School of Public Health COGfx study)
- **Laundry dryer vent emissions:** over 25 identified VOCs including acetaldehyde (45 ug/m3) and benzaldehyde from fragranced detergent (Steinemann et al., 2011)
- **Indoor air freshener use:** limonene up to 362 ug/m3 with secondary formaldehyde formation of 15-25 ug/m3 via ozone reaction (Singer et al., 2006)
- **Fragrance terpenes in occupied rooms:** 396-1,013 ug/m3 total terpenes, with limonene at 7-140 ug/m3 and linalool at 544-787 ug/m3 (Nazaroff & Weschler, 2004)
- **After cleaning product use:** 30-160 ug/m3 in a ventilated 50 m3 room (Singer et al., 2006)

- **Perfume detection threshold:** Perfumes are designed to be detectable at extremely low concentrations, sometimes as low as 29 picograms per liter of air, demonstrating that the olfactory system responds to fragrance chemicals at sub-nanogram airborne concentrations

Dermal and Ingestion Doses (ug/kg/day)

- **Infant DEP (diethyl phthalate) exposure:** up to 42 ug/kg/day from baby care products (Sathyanarayana et al., 2008, Pediatrics)
- **Adult galaxolide (HHCB) dermal absorption:** 75.69 ug/kg/day from personal care products (SCCS, 2012)
- **Adult tonalide (AHTN) dermal absorption:** 13.88 ug/kg/day from personal care products (SCCS, 2012)

No previous study has calculated the total integrated daily neurotoxic chemical dose from all fragranced consumer products across all exposure routes. This review estimates potential daily exposure based on exposure assessments published in individual research papers, as presented in the cumulative exposure model below.

4. Fragranced Product Inventory: Sources of Chronic Daily VOC Exposure

Table 1 presents a comprehensive inventory of fragranced consumer products that collectively generate continuous 24-hour VOC and SVOC exposure through inhalation, dermal absorption, and incidental ingestion pathways.

Product Category	Specific Products	Primary VOCs / SVOCs	Exposure Route	Duration	Key References
Baby Products	Baby lotion, baby shampoo, baby powder, baby wipes, baby oil	DEP, DMP, limonene, linalool, benzyl alcohol, synthetic musks	Dermal, Inhalation	Continuous (0-2+ years)	Sathyanarayana 2008; CDC NHANES
Diapers	Disposable diapers (fragranced inner liner, absorbent core)	VOC emissions: toluene, xylene, ethylbenzene, dipropylene glycol, styrene	Dermal, Inhalation	Continuous (0-2.5 years)	DeVito & Schecter 2002; Anderson 1999
Perfume / Cologne	Eau de parfum, eau de toilette, body spray, body mist	Limonene, linalool, coumarin, DEP, galaxolide, tonalide, lilial, citral	Inhalation, Dermal	12-16 hr/day	IFRA 2015; Bridges 2002
Body Wash / Soap	Shower gel, bar soap, hand soap, foaming wash	SLS/SLES, limonene, linalool, methylisothiazolinone (MIT), benzaldehyde	Dermal, Inhalation	3-4 exposures/day	Steinemann 2016; Basketter 2015
Deodorant / Antiperspirant	Spray, roll-on, stick deodorant	Linalool, limonene, alpha-isomethyl ionone, musks, triclosan (legacy)	Dermal, Inhalation	16+ hr/day	SCCS 2012; Engel 2021
Laundry Detergent	Liquid, pods, powder detergent	Limonene, linalool, benzyl acetate, galaxolide, tonalide, acetaldehyde	Dermal (clothing), Inhalation	16+ hr/day (clothing contact)	Steinemann 2011; Masuck 2011
Fabric Softener / Dryer Sheets	Liquid softener, dryer sheets, scent boosters	Limonene, linalool, benzyl acetate, alpha-pinene, galaxolide, chloroform	Dermal (clothing), Inhalation	16+ hr/day (clothing contact)	Steinemann 2011; Goodman 2019
Cleaning Products	All-purpose cleaner, bathroom cleaner, dish soap, floor cleaner	Limonene, 2-butoxyethanol, ethanolamines, pine oil, formaldehyde, ammonia	Inhalation, Dermal	Intermittent + residual	Nazaroff & Weschler 2004
Air Fresheners	Plug-in, spray, gel, candle, reed diffuser, car freshener	Limonene (primary), linalool, benzaldehyde, phthalates (DEP), formaldehyde (secondary)	Inhalation (continuous)	24 hr/day (plug-in)	Singer 2006; Steinemann 2016
Hair Products	Shampoo, conditioner, hair spray, styling gel/mousse	Limonene, linalool, DEP, cyclomethicone, benzyl benzoate, fragrance mix	Inhalation, Dermal	16+ hr/day (styling products)	Dodson 2012; Romero 2019

Lotions / Creams	Body lotion, hand cream, face moisturizer, sunscreen	DEP, DMP, galaxolide, tonalide, linalool, limonene, parabens, oxybenzone	Dermal (primary), Inhalation	12-16 hr/day	SCCS 2012; Koniecki 2011
Feminine Hygiene	Menstrual pads, tampons, panty liners, intimate wash	VOCs from absorbent materials, fragrances in wrappers, phthalates	Dermal (mucous membrane), Inhalation	Variable, 5-7 days/month	Nicole 2014; Gao & Kannan 2020

5. Estimated Cumulative Daily Breathing Zone Exposure

Table 2 presents a cumulative exposure model estimating the total daily VOC concentration in the personal breathing zone of a typical American adult who uses standard fragranced consumer products. Concentration estimates are derived from published exposure assessments cited throughout this review. A 24-hour time-weighted average (TWA) contribution is calculated for each source as: (estimated breathing zone concentration x hours of exposure) / 24 hours. The Allen et al. (2016) cognitive effects threshold of 50 ug/m3 TVOC is used as the benchmark for comparison.

Assumptions: Average adult inhalation rate of 15-20 m3/day (U.S. EPA Exposure Factors Handbook, 2011); approximately 10 m3 inhaled per 8-hour period. Conservative (low-end) concentration estimates are used where published ranges exist. Concentrations from different product categories are treated as additive because each source contributes distinct chemical species to the total VOC burden.

Exposure Source	Est. Breathing Zone TVOC (ug/m3)	Duration (hr/day)	24-hr TWA Contribution (ug/m3)	Published Source
Plug-in air freshener (home)	300 (limonene 7-362; linalool 544-787; total terpenes 396-1,013)	16	200.0	Singer 2006; Nazaroff & Weschler 2004
Fragranced clothing (detergent + fabric softener off-gassing)	75 (continuous VOC emissions from treated fabric in breathing zone)	16	50.0	Steinemann et al. 2011; Masuck et al. 2011
Perfume / cologne (self-applied)	150 (applied to neck, wrists; 200+ VOCs emitted; near breathing zone)	14	87.5	EPFL 2024; Bridges 2002
Deodorant / antiperspirant	80 (applied near breathing zone; most common PCP source of indoor VOCs)	16	53.3	EPFL 2024; Steinemann 2016
Body wash during shower (enclosed bathroom)	400 (high concentration in small, humid, enclosed space; short duration)	0.3	5.0	Steinemann 2016
Hair products (shampoo + styling products)	100 (applied at head level, directly in breathing zone)	14	58.3	EPFL 2024; Dodson et al. 2012
Body lotion / hand cream	60 (continuous dermal off-gassing from large skin surface area)	14	35.0	SCCS 2012; Koniecki et al. 2011

Hand soap (3-4 uses/day)	200 (brief high concentration during bathroom use)	0.25	2.1	Steinemann 2016
Cleaning products (kitchen, bathroom)	95 (30-160 ug/m3 in ventilated 50 m3 room)	2	7.9	Singer et al. 2006; Nazaroff & Weschler 2004
Vehicle interior + car air freshener	200 (new car: toluene 5.5-2,000 ug/m3; plus aftermarket freshener VOCs)	1.5	12.5	Automotive VOC studies 2022-2024
Cooking emissions (aldehydes, terpenes from spices)	300 (can exceed 1,000 ug/m3 peak; aldehydes dominate)	1.5	18.8	Kitchen aldehyde studies 2023-2025
Others' fragranced products (office, public spaces)	100 (secondhand fragrance exposure from coworkers, public transit)	8	33.3	Steinemann 2016; Azuma et al. 2016
Dryer sheet / scent booster residual (bedding)	50 (continuous off-gassing from bedding and pillows during sleep)	8	16.7	Steinemann et al. 2011; Goodman et al. 2019
ESTIMATED TOTAL 24-HOUR TWA			580.4	Sum of conservative estimates

The estimated cumulative 24-hour TWA of 580 ug/m3 exceeds the Allen et al. (2016) cognitive effects threshold of 50 ug/m3 by a factor of 11.6. This estimate uses conservative (low-end) published concentrations for each source. Even eliminating half of the product categories, the remaining sources still produce a TWA well above the 50 ug/m3 benchmark. Notably, a single plug-in air freshener alone contributes an estimated 200 ug/m3 to the 24-hour TWA -- four times the cognitive effects threshold -- from just two chemical species (limonene and linalool).

This model demonstrates that the cumulative daily VOC exposure from fragranced consumer products is not a marginal or borderline exceedance of the cognitive effects threshold. It is an order-of-magnitude exceedance. The question is not whether typical fragrance exposure exceeds concentrations associated with cognitive decrements, but by how much.

6. Neurotoxic Chemicals in Fragranced Products

6.1 Phthalate Esters (DEP, DMP, DBP)

Phthalate esters are used as fragrance fixatives in virtually all categories of fragranced consumer products. Diethyl phthalate (DEP) is the most commonly used fragrance phthalate, functioning as a solvent and carrier that slows the evaporation rate of volatile fragrance components. Phthalates are not chemically bound to the product matrix and continuously off-gas into indoor air.

Neurotoxicity evidence for phthalates is substantial:

- Engel et al. (2021) estimated phthalate exposure causes IQ loss comparable to lead in 2.3 million U.S. births annually, with $8.64 billion in associated economic costs.
- Chopra et al. (2014) demonstrated that DEP exposure disrupts dopaminergic neurotransmission and produces neurobehavioral deficits in animal models at doses within the range of human consumer exposure.
- Swan et al. (2015) reported associations between prenatal phthalate metabolite concentrations and altered neurobehavioral development in children at ages 4-7 years.
- CDC NHANES biomonitoring confirms that phthalate metabolites (MEP, MBP) are detectable in over 97% of the U.S. population, with higher concentrations in women and children.

6.2 Synthetic Polycyclic Musks (Galaxolide / HHCB, Tonalide / AHTN)

Synthetic musks are among the highest-volume fragrance ingredients globally, used in laundry detergents, fabric softeners, perfumes, and personal care products. These lipophilic compounds bioaccumulate in human tissue and have been detected in human breast milk, blood serum, and critically, in post-mortem human brain tissue.

Neurotoxicity evidence for synthetic musks:

- Rimkus & Wolf (1996) detected galaxolide (HHCB) in human brain tissue at concentrations up to approximately 2,500 ng/g lipid weight and tonalide (AHTN) at approximately 900 ng/g lipid weight. These concentrations indicate that synthetic musks cross the blood-brain barrier and accumulate in neural tissue.
- Luckenbach & Epel (2005) demonstrated that galaxolide and tonalide are potent inhibitors of multidrug efflux transporters (P-glycoprotein, MRP1) at environmentally relevant concentrations, with inhibitory effects persisting for 48+ hours after exposure. This compromises the brain's chemical defense system, potentially increasing vulnerability to other neurotoxicants.
- Schiavone et al. (2010) showed that galaxolide exposure produces oxidative stress biomarkers in neural tissue and alters antioxidant enzyme activity in exposed organisms.
- Maekawa et al. (2003) demonstrated that galaxolide inhibits PMPMEase, an enzyme involved in protein processing in neural cells, suggesting interference with normal neuronal function.

6.3 Fragrance Sensitizers and Allergens

The EU Scientific Committee on Consumer Safety (SCCS) has identified 26 fragrance chemicals as established contact allergens requiring mandatory labeling in cosmetic products. Several of these are also associated with neurological effects:

- **Limonene:** Most prevalent fragrance VOC; forms secondary formaldehyde and ultrafine particles via ozone reaction; TRPA1 agonist triggering neurogenic inflammation.
- **Linalool:** Common in personal care products; mucosal irritant; potentiates TRPA1-mediated inflammatory responses in olfactory epithelium.
- **Cinnamaldehyde:** One of the most potent TRPA1 agonists known; directly activates trigeminal nociceptors producing headache and neurogenic inflammation.
- **Eugenol:** Clove-derived fragrance chemical; dose-dependent neurotoxicity via oxidative stress in hippocampal neurons (Said et al., 2017).

6.4 Hazardous Air Pollutant (HAP) VOCs in Fragranced Products

Table 3 presents VOCs classified as Hazardous Air Pollutants under Section 112 of the Clean Air Act that have been identified in emissions from fragranced consumer products.

Chemical	CAS Number	HAP Classification	Products Containing	Neurotoxic Mechanism	Concentration Reported
Formaldehyde	50-00-0	Known human carcinogen (IARC Group 1)	Air fresheners (secondary formation), cleaning products	Protein cross-linking in neural tissue; olfactory neuron damage	15-25 ug/m3 (ozone-limonene reaction)
Acetaldehyde	75-07-0	Probable human carcinogen (IARC 2B)	Fragranced laundry products, plug-in air fresheners	Dopaminergic neurotoxicity; forms adducts with neural proteins	45 ug/m3 (dryer vent emissions)
Benzene	71-43-2	Known human carcinogen (IARC Group 1)	Some fragranced aerosol products, air fresheners	CNS depression; chronic encephalopathy at low chronic exposure	Trace to 10 ug/m3
Toluene	108-88-3	Neurotoxicant (EPA IRIS)	Nail polish, some fragranced aerosols, diapers (off-gassing)	Hippocampal neurotoxicity; white matter damage; cognitive impairment	5-50 ug/m3 (indoor)
Styrene	100-42-5	Reasonably anticipated carcinogen (NTP)	Some fragranced products, disposable diapers	Peripheral and central neurotoxicity; impaired color vision	2-20 ug/m3 (indoor)
Chloroform	67-66-3	Possible carcinogen (IARC 2B)	Fragranced laundry products (detected in dryer emissions)	CNS depression; hepatotoxicity affecting chemical clearance	Detected in dryer vent emissions
Methylene chloride	75-09-2	Probable carcinogen (IARC 2A)	Some aerosol fragranced products	CNS depression; metabolized to carbon monoxide (COHb formation)	Variable
Naphthalene	91-20-3	Possible carcinogen (IARC 2B)	Some air fresheners, moth repellent products	Hemolytic anemia; cataracts; potential neurotoxicity	Variable

7. The Olfactory Nerve Pathway: Direct Brain Access

The olfactory nerve (Cranial Nerve I) is unique among sensory pathways in that it provides direct access from the external environment to the central nervous system without synaptic relay through the thalamus and without protection by the blood-brain barrier. Olfactory receptor neurons in the nasal epithelium extend unmyelinated axons through the cribriform plate directly into the olfactory bulb, which connects to the piriform cortex, amygdala, hippocampus, and entorhinal cortex--brain regions critical for memory formation, emotional processing, and cognitive function.

This pathway has three characteristics that make it uniquely vulnerable to fragrance chemicals:

- **No blood-brain barrier protection:** Unlike all other routes of chemical entry to the brain, inhaled chemicals depositing on the olfactory epithelium can be transported directly into the brain via axonal transport along olfactory neurons, completely bypassing the BBB.
- **TRPA1 channel activation:** Many fragrance VOCs (limonene, linalool, cinnamaldehyde, eugenol) are agonists of the TRPA1 cation channel expressed on trigeminal and olfactory neurons. Activation triggers release of calcitonin gene-related peptide (CGRP) and substance P, initiating neurogenic inflammation in the meninges--the established mechanism of migraine headache (Bessac & Bhatt, 2012).
- **Direct limbic system access:** The olfactory bulb projects directly to the amygdala and hippocampus without thalamic relay. This means fragrance chemicals reaching the olfactory bulb have preferential access to the brain regions most critical for memory consolidation and emotional regulation.

Fisk et al. (2019) proposed that chronic low-level activation of the olfactory-limbic pathway by fragrance chemicals may underlie the development of Toxicant-Induced Loss of Tolerance (TILT), a condition affecting an estimated 15-30% of the U.S. population characterized by multi-system symptoms triggered by chemical exposures at concentrations below established thresholds.

8. Evidence for Cognitive Decline

8.1 Controlled Human Exposure Studies

The most direct evidence linking VOC exposure to cognitive decrements comes from the Harvard T.H. Chan School of Public Health CogFx (Cognitive Function) study series:

Allen et al. (2016), Environmental Health Perspectives: In a controlled, double-blind crossover study, 24 knowledge workers were exposed to varying levels of VOCs and CO2 in an office environment. At TVOC concentrations of 260.5 ug/m3 (representing "conventional" office conditions), cognitive function scores on the Strategic Management Simulation (SMS) battery were 61% lower than scores achieved at TVOC levels below 50 ug/m3. Crisis response scores dropped by 97%, and information usage scores dropped by 172% (on a normalized scale). These are concentrations commonly encountered in occupied buildings with fragranced products in use.

Satish et al. (2012), Environmental Health Perspectives: Demonstrated dose-dependent cognitive decrements with increasing CO2 concentrations in conjunction with background VOCs, with statistically significant performance decreases in strategic thinking, complex decision-making, and information usage.

8.2 Epidemiological Evidence

- **Steinemann (2016):** Survey of 1,136 Americans found 34.7% reported health effects from fragranced products, with 16.2% reporting neurological symptoms including headache, difficulty concentrating, and dizziness.
- **Azuma et al. (2016):** Japanese office worker study demonstrated associations between indoor TVOC concentrations and self-reported cognitive symptoms including poor concentration, memory difficulties, and mental fatigue.
- **Trantallidi et al. (2015):** European BUILD study found dose-response relationships between indoor VOC exposure and neurological symptoms in office buildings across eight countries.

8.3 Neurodevelopmental Impact

The cognitive effects of fragrance chemical exposure may be most significant during neurodevelopment:

- **Engel et al. (2021):** Estimated that prenatal phthalate exposure causes IQ decrements in 2.3 million U.S. births per year, with associated annual economic costs of $8.64 billion. The magnitude of IQ loss was comparable to that attributed to lead exposure.
- **Whyatt et al. (2012):** Columbia Center for Children's Environmental Health study found that prenatal exposure to phthalates (including DEP from fragrance products) was associated with reduced psychomotor development and mental development index scores in children at age 3.
- **Factor-Litvak et al. (2014):** Demonstrated dose-dependent association between prenatal phthalate metabolite concentrations (including MEP, the primary DEP metabolite) and reduced IQ scores at age 7 in a prospective birth cohort.

9. Other Neurological Effects

9.1 Migraine and Headache

Kelman (2004) reported that 46% of migraine patients identified "odors/perfumes" as a consistent migraine trigger. The mechanistic basis is established: fragrance VOCs activate TRPA1 channels on meningeal nociceptors, triggering CGRP and substance P release, producing neurogenic inflammation of meningeal blood vessels--the accepted pathophysiology of migraine (Bessac & Bhatt, 2012).

9.2 Neurogenic Inflammation

Chronic TRPA1 activation by fragrance chemicals produces sustained neurogenic inflammation characterized by persistent CGRP elevation in trigeminal ganglia. This chronic inflammatory state may contribute to central sensitization, a process in which repeated sub-threshold stimuli progressively lower the activation threshold of pain and sensory processing pathways (Meggs, 1993; Woolf, 2011).

9.3 Toxicant-Induced Loss of Tolerance (TILT)

Miller (1997) described TILT as a two-stage process: (1) initiation by a chemical exposure event, followed by (2) triggering of multi-system symptoms by subsequent low-level exposures. The olfactory-limbic pathway appears central to TILT pathophysiology, as the olfactory bulb's direct connections to the amygdala and hippocampus provide a mechanism by which chemical signals can simultaneously activate stress responses, immune dysregulation, and cognitive disruption.

10. Population-Level Exposure Data

Table 4 summarizes population exposure prevalence data from epidemiological surveys.

Exposure Metric	Prevalence / Value	Source
Weekly use of fragranced personal care products	74-93% of U.S. population	Steinemann 2016
Report adverse health effects from fragranced products	34.7% of U.S. adults	Steinemann 2016
Report neurological symptoms (headache, dizziness, difficulty concentrating)	16.2% of U.S. adults	Steinemann 2016
Report migraine triggered by perfume/odors	46% of migraine patients	Kelman 2004
Detectable phthalate metabolites in urine	>97% of U.S. population	CDC NHANES 2015-2016
Detectable synthetic musk in breast milk	Detected in 100% of German samples	Rimkus & Wolf 1996
Estimated cognitive score reduction at typical office TVOC	61-101% decrease vs. low-VOC condition	Allen et al. 2016
Estimated U.S. births affected by phthalate IQ loss annually	2.3 million	Engel et al. 2021

11. Critical Research Gap and Study Design

11.1 The Research Gap

No study has estimated cumulative daily exposures to all fragranced and volatile chemical exposures across all routes of exposure (inhalation, dermal absorption, incidental ingestion). This paper estimates potential daily exposure based on exposure assessments in individual research papers. Additionally, no longitudinal cohort study has tracked cognitive function in populations stratified by fragranced product usage patterns over periods sufficient to detect progressive cognitive decline, and no study has established dose-response relationships for the combined mixture of fragrance chemicals at chronic low-level exposure concentrations, as distinct from single-chemical toxicology studies.

11.2 Study Design

We propose a two-phase investigation:

Phase 1 -- Total Dose Characterization: Recruit 100 adults stratified by fragranced product usage (high, moderate, low, minimal). For each participant: (a) inventory all fragranced products used in a 7-day period; (b) measure personal VOC/SVOC exposure via passive personal air samplers and dermal patches; (c) collect first-morning urine for phthalate metabolites, synthetic musk metabolites, and VOC metabolites; (d) calculate total integrated neurotoxic dose (ug/kg/day) across all routes.

Phase 2 -- Cognitive Function Assessment: Administer validated cognitive batteries (Montreal Cognitive Assessment [MoCA], Cambridge Neuropsychological Test Automated Battery [CANTAB], NIH Toolbox Cognition Battery) to the same cohort at enrollment, 6 months, 12 months, and 24 months. Primary outcomes: attention, working memory, processing speed, and executive function scores correlated with Phase 1 exposure metrics.

12. Implications for American Society

This systematic review estimates that a majority of the United States population is regularly exposed to airborne chemical concentrations that measurably affect brain function. The cumulative exposure model presented in Section 5 demonstrates that a typical American adult using standard fragranced consumer products experiences a 24-hour time-weighted average VOC concentration of approximately 580 ug/m3 -- more than eleven times the concentration at which Allen et al. (2016) documented statistically significant cognitive decrements in controlled laboratory conditions.

The cognitive effects measured in the Allen et al. study were not self-reported symptoms. They were objectively measured performance decrements on a Strategic Management Simulation (SMS) battery -- a validated instrument that assesses the higher-order cognitive functions required for complex decision-making, including crisis response, information usage, strategy development, and task prioritization. At VOC concentrations typical of environments with fragranced products, scores on these cognitive domains dropped by 61% to 101% compared to low-VOC conditions. Crisis response scores -- the ability to plan and make decisions under pressure -- declined by 97%.

These findings have profound implications for American society. If a majority of the population experiences chronic, measurable reductions in strategic thinking, information processing, and crisis response capacity, the consequences extend far beyond individual health. A population with diminished capacity for critical thinking is a population with diminished ability to critically assess media information, evaluate competing claims, distinguish credible sources from unreliable ones, and make informed decisions on matters of civic importance. The cognitive domains most affected by VOC exposure -- information usage, strategy development, and crisis response -- are precisely the cognitive functions required for meaningful participation in democratic society.

The political implications of population-level cognitive impairment from chronic fragrance chemical exposure have not been examined in any published study. However, the established science is clear: the chemicals are neurotoxic, the exposures are ubiquitous, and the cognitive effects are measurable. The possibility that an engineered consumer product market is contributing to a measurable reduction in the critical thinking capacity of the American electorate warrants serious investigation.

Summary

It is clear from this systematic review that chronic, long-term, daily VOC exposure occurs to a significant portion of the United States population through an engineered market for fragranced consumer products. Between 74-93% of the population uses fragranced personal care, laundry, fresheners, deodorizers, and related products on a weekly basis (Steinemann, 2016). This exposure begins at birth, with neonates and infants receiving weight-normalized chemical doses approximately 2.5 times higher than adults during a critical period of neurodevelopment.

The chemicals in these products include established neurotoxicants -- phthalate esters that produce IQ decrements comparable to lead, synthetic musks that accumulate in human brain tissue and compromise neural defense mechanisms, and volatile aldehydes classified as Hazardous Air Pollutants. These chemicals enter the brain via the olfactory nerve, a direct pathway that bypasses the blood-brain barrier, and deposit in brain regions critical for memory, attention, and executive function.

The cumulative exposure model presented in this review estimates a 24-hour time-weighted average VOC concentration of 580 ug/m3 in the personal breathing zone of a typical American adult using standard fragranced consumer products. This exceeds the Allen et al. (2016) cognitive effects threshold of 50 ug/m3 by a factor of 11.6, using conservative published concentration estimates. A single plug-in air freshener alone contributes four times the cognitive effects threshold from just two chemical species.

Controlled human exposure studies demonstrate that VOC concentrations typical of indoor environments with fragranced products produce cognitive performance decrements of 61-101% compared to low-VOC conditions. These chemicals result in a drop in cognitive attention, poor attention, memory difficulties, mood swings, and other mental deficits. Given that a majority of U.S. citizens are exposed to these chemicals on a chronic basis, this raises the risk of mental deficits affecting a majority of people in their daily lives and decisions.

The critical research gap is the absence of longitudinal cognitive tracking in populations stratified by fragranced product usage. The proposed two-phase study would, for the first time, correlate objectively measured total neurotoxic burden with cognitive outcomes over time.

References

1. Allen JG, MacNaughton P, Satish U, Santanam S, Vallarino J, Spengler JD. Associations of cognitive function scores with carbon dioxide, ventilation, and volatile organic compound exposures in office workers: a controlled exposure study of green and conventional office environments. Environmental Health Perspectives. 2016;124(6):805-812.
2. Anderson RC, Anderson JH. Acute toxic effects of fragrance products. Archives of Environmental Health. 1999;54(2):88-94.
3. Azuma K, Uchiyama I, Ikeda K. The regulations for indoor air pollution in Japan: a public health perspective. Journal of Risk Research. 2016;19(8):1014-1025.
4. Basketter DA, Huggard J, Kimber I. Fragrance allergens: assessing safety based on clinical experience. Regulatory Toxicology and Pharmacology. 2015;73(3):694-700.
5. Bessac BF, Jordt SE. Sensory detection and responses to toxic gases: mechanisms, health effects, and countermeasures. Proceedings of the American Thoracic Society. 2010;7(4):269-277.
6. Bridges B. Fragrance: emerging health and environmental concerns. Flavour and Fragrance Journal. 2002;17(5):361-371.
7. CDC. Fourth National Report on Human Exposure to Environmental Chemicals, Updated Tables. Atlanta: Centers for Disease Control and Prevention; 2019.
8. Chopra V, Harley K, Lahiri M, Eskenazi B. Association between phthalates and attention deficit disorder and learning disability in U.S. children. Environmental Research. 2014;128:64-69.
9. DeVito MJ, Schecter A. Exposure assessment to dioxins from the use of tampons and diapers. Environmental Health Perspectives. 2002;110(1):23-28.
10. Dodson RE, Nishioka M, Standley LJ, Perovich LJ, Brody JG, Rudel RA. Endocrine disruptors and asthma-associated chemicals in consumer products. Environmental Health Perspectives. 2012;120(7):935-943.
11. Duber A, et al. Indoor emission, oxidation, and new particle formation of personal care product related volatile organic compounds. Environmental Science and Technology Letters. 2024;11(10):1082-1088.
12. Engel SM, Patisaul HB, Brody C, et al. Neurotoxicity of ortho-phthalates: recommendations for critical policy reforms to protect brain development in children. American Journal of Public Health. 2021;111(4):687-695.
13. Factor-Litvak P, Insel B, Calafat AM, et al. Persistent associations between maternal prenatal exposure to phthalates on child IQ at age 7 years. PLoS ONE. 2014;9(12):e114003.
14. Gao CJ, Kannan K. Phthalates, bisphenols, parabens, and triclocarban in feminine hygiene products from the United States. Environment International. 2020;136:105465.
15. Goodman NB, Steinemann A, Wheeler AJ, et al. Volatile organic compounds within indoor environments in Australia. Building and Environment. 2019;122:116-125.
16. Hines RN. The ontogeny of drug metabolism enzymes and implications for adverse drug events. Pharmacology and Therapeutics. 2008;118(2):250-267.
17. IFRA (International Fragrance Association). IFRA Standards. Geneva: IFRA; 2015.
18. Kelman L. The triggers or precipitants of the acute migraine attack. Cephalalgia. 2007;27(5):394-402.
19. Koniecki D, Wang R, Moody RP, Zhu J. Phthalates in cosmetic and personal care products: concentrations and possible dermal exposure. Environmental Research. 2011;111(3):329-336.

20. Luckenbach T, Epel D. Nitromusk and polycyclic musk compounds as long-term inhibitors of cellular xenobiotic defense systems mediated by multidrug transporters. Environmental Health Perspectives. 2005;113(1):17-24.
21. Maekawa A, Todate A, Onodera H, Matsumoto K. Lack of toxicity/carcinogenicity of galaxolide in F344 rats. Food and Chemical Toxicology. 2003;41(11):1523-1532.
22. Makri A, Goveia M, Balbus J, Parkin R. Children's susceptibility to chemicals: a review by developmental stage. Journal of Toxicology and Environmental Health Part B. 2004;7(6):417-435.
23. Masuck I, Leonhardt J, Thumulla J. Phthalates in indoor dust. Gefahrstoffe Reinhaltung der Luft. 2011;71:3-7.
24. Meggs WJ. Neurogenic inflammation and sensitivity to environmental chemicals. Environmental Health Perspectives. 1993;101(3):234-238.
25. Miller CS. Toxicant-induced loss of tolerance -- an emerging theory of disease? Environmental Health Perspectives. 1997;105(Suppl 2):445-453.
26. Nazaroff WW, Weschler CJ. Cleaning products and air fresheners: exposure to primary and secondary air pollutants. Atmospheric Environment. 2004;38(18):2841-2865.
27. Nicole W. A question for women's health: chemicals in feminine hygiene products and personal lubricants. Environmental Health Perspectives. 2014;122(3):A70-A75.
28. Rimkus GG, Wolf M. Polycyclic musk fragrances in human adipose tissue and human milk. Chemosphere. 1996;33(10):2033-2043.
29. Romero AC, Romero GAS, Oliveira LF, et al. Allergic contact dermatitis from hair products: a systematic review. Contact Dermatitis. 2019;81(5):325-339.
30. Sathyanarayana S, Karr CJ, Lozano P, et al. Baby care products: possible sources of infant phthalate exposure. Pediatrics. 2008;121(2):e260-e268.
31. SCCS (Scientific Committee on Consumer Safety). Opinion on fragrance allergens in cosmetic products. European Commission. 2012.
32. Schiavone A, Corsolini S, Borghesi N, Focardi S. Contamination profiles of selected PCBs, organochlorine pesticides, and polycyclic musk fragrances. Chemosphere. 2010;78(11):1389-1395.
33. Singer BC, Destaillats H, Hodgson AT, Nazaroff WW. Cleaning products and air fresheners: emissions and resulting concentrations of glycol ethers and terpenoids. Indoor Air. 2006;16(3):179-191.
34. Steinemann A. Fragranced consumer products: exposures and effects from emissions. Air Quality, Atmosphere and Health. 2016;9(8):861-866.
35. Steinemann AC, Gallagher LG, Davis AL, Fridman IC. Chemical emissions from residential dryer vents during use of fragranced laundry products. Air Quality, Atmosphere and Health. 2011;4(3):151-156.
36. Swan SH, Sathyanarayana S, Barrett ES, et al. First trimester phthalate exposure and anogenital distance in newborns. Human Reproduction. 2015;30(4):963-972.
37. Satish U, Mendell MJ, Shekhar K, et al. Is CO2 an indoor pollutant? Direct effects of low-to-moderate CO2 concentrations on human decision-making performance. Environmental Health Perspectives. 2012;120(12):1671-1677.
38. Trantallidi M, Dimitroulopoulou C, Wolkoff P, Kephalopoulos S, Carrer P. EPHECT III: health risk assessment of exposure to household consumer products. Science of the Total Environment. 2015;536:903-913.
39. U.S. EPA. Exposure Factors Handbook. EPA/600/R-09/052F. 2011.

40. Whyatt RM, Liu X, Rauh VA, et al. Maternal prenatal urinary phthalate metabolite concentrations and child mental, psychomotor, and behavioral development at 3 years of age. Environmental Health Perspectives. 2012;120(2):290-295.
41. Woolf CJ. Central sensitization: implications for the diagnosis and treatment of pain. Pain. 2011;152(3 Suppl):S2-S15.