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Endocrine disruptors also function as nervous disruptors and can be renamed endocrine and nervous disruptors (ENDs)

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ABSTRACT

Endocrine disruption (ED) and endocrine disruptors (EDs) emerged as scientific concepts in 1995, after numerous chemical pollutants were found to be responsible for reproductive dysfunction. The World Health Organization established in the United Nations Environment Programme a list of materials, plasticizers, pesticides, and various pollutants synthesized from petrochemistry that impact not only reproduction, but also hormonal functions, directly or indirectly. Cells communicate via either chemical or electrical signals transmitted within the endocrine or nervous systems. To investigate whether hormone disruptors may also interfere directly or indirectly with the development or functioning of the nervous system through either a neuroendocrine or a more general mechanism, we examined the scientific literature to ascertain the effects of EDs on the nervous system, specifically in the categories of neurotoxicity, cognition, and behaviour. To date, we demonstrated that all of the 177 EDs identified internationally by WHO are known to have an impact on the nervous system. Furthermore, the precise mechanisms underlying this neurodisruption have also been established. It was previously believed that EDs primarily function via the thyroid. However, this study presents substantial evidence that approximately 80 % of EDs operate via other mechanisms. It thus outlines a novel concept: EDs are also neurodisruptors (NDs) and can be collectively termed endocrine and nervous disruptors (ENDs). Most of ENDs are derived from petroleum residues, and their various mechanisms of action are similar to those of "spam" in electronic communications technologies. Therefore, ENDs can be considered as an instance of spam in a biological context.

1. Introduction

Endocrine disruption (ED) or endocrine disruptors (EDs) emerged as scientific concepts in 1995 [Colborn [1]; Lindström et al. [2]; Ginsburg [3]] after numerous chemical pollutants were found to be responsible for reproductive dysfunction. This was first posited three decades prior [Carson [4]]. EDs were reviewed more recently in a book [Seralini [5]], which advanced the understanding of the molecular bioaccumulation of identified xenobiotics, as well as their combined and long-term effects on the whole physiology of one or several generations. They have been identified in organisms at all levels of the ecosystem and are also ubiquitously found in the food chain.

The World Health Organization established in the United Nations Environment Programme [WHO [6]], a list of 176 compounds comprising materials, pesticides, and various pollutants (Table 1, columns 1–3) impacting not only reproduction, but also hormonal functions—directly or indirectly—primarily in mammals, including humans. This has enabled numerous countries to establish regulatory policies for the production and use of these chemicals or to manage contamination in food, air and water. Numerous political debates have been raised around regulatory thresholds, based on the effects of ENDs demonstrated on a population or a subpopulation of animals or humans, and published at epidemiological and molecular levels. The herbicide Roundup has been added as the 177th compound due to its widespread usage as a pesticide, combined with the relatively recent demonstration of its ED effects [Richard et al. [7]].

Epidemiology is not technically adapted to solve the questions on combined and long-term effects of molecules or mixtures on mammalian or human health [Mesnage et al. [8]]; this becomes further complicated when epigenetic and transgenerational impacts are studied [Skinner and Anway [9]]. For instance, pesticide accumulation is rarely measured in organs after death in order to ascertain whether they can be used as markers to correlate their levels with pathologies. Instead, the understanding of endocrine disruption may be aided by advances in the

Abbreviations: ED, Endocrine disruptors; ENDs, Endocrine and nervous disruptors; ND, Nervous disruptors; WHO, World Health Organization.

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combined knowledge of biochemical, cellular, organic and environmental effects in experimental animal models, farm animals, wildlife observations in contaminated areas, and occupational medicine in factories producing the chemicals in question.

The endocrine system is not limited to the control of sexual reproduction and development. Endocrine disruptors may affect the thyroid as well as the glucocorticoid axis, adrenal and pancreatic systems, adipose tissue and immune or neuroendocrine targets [Laessig et al. [10]; Masuo and Ishido [11]; Weiss [12]; Leon-Olea et al. [13]]. They even possess cognitive effects [Schantz and Widholm [14]], particularly via various neuromediator interferences.

Cells communicate via either chemical or electrical signals that are transmitted within the endocrine or nervous systems. Generally, in endocrinology, hormones may have a biphasic action dependent on the receptors' availability and concentration, resulting in time-, dose- and sex-dependent effects that vary according to the targeted tissue and the specific organism. It is thus reductionist to say, for instance, that a hormone such as estradiol "stimulates ovulation", since it can inhibit this function when used at pharmacological doses as a pill or during embryonic or foetal life. Endocrine disruptors may thus possess the potential of exerting similar biphasic ambivalent effects.

Thus, to determine whether hormonal disruptors may also interfere directly or indirectly with neural development [Grandjean and Landrigan [15]] or functioning even in adults, either through a neuroendocrine or a more general mechanism, this study examined the scientific literature to ascertain the effects of ENDs on the nervous system —including neurotoxicity, cognition, and behaviour— of the major internationally identified (according to WHO) endocrine disruptors.

2. Materials and methods

Each compound was numbered (Nb, Table 1) out of 176 known endocrine disruptors [WHO [6]], plus Roundup. Its name was associated with the keyword "nervous" or "neurotoxicity" or "cognitive" or "behavio(u)r" on the PubMed data bank, and eventually on Google Scholar. When the references were too numerous, "or" was excluded in order to directly associate the keywords. If more than 20 references were found to be published, "review" was added to the keywords and cited. Finally, a maximum of five references were indicated, focusing on the most recent research in humans or mammals, without excluding other models. The mechanisms were documented (Table 1, column 4) as direct effects on the neurons or the nervous system, or as indirect effects, including thyroid regulation.

3. Results and discussion

All of the 176 internationally identified EDs (as per WHO guidelines) were studied in the international bibliography published between 1931 and 2021, in order to assess their effects on the nervous system (Table 1). It was observed that every single one of the EDs induced neurodisruption or clear neuromodulation, while even stimulation occurred in certain rare cases. Thus, 100 % of the EDs studied were known neuromodulators, either directly or indirectly. Previously, it was generally believed that endocrine disruptors with a neuronal impact functioned via the thyroid gland, which is classically known to control nervous system development. However, this study presents substantive evidence that at least 79.1 % of the EDs are, in fact, also NDs through various other mechanisms (Table 1). 37 compounds out of the total were implicated to possess a thyroid-dependent mechanism. Therefore, the emergence of this overarching concept compelled us to propose the introduction and use of a collective abbreviation "ENDs" for endocrine and nervous disruptors.

Therapeutic hormones or pharmaceutical products (see column 3 in Table 1) that may exist as pollutants in rivers [Arya et al. [16]; Saussereau et al. [17]; Goulle et al. [18]] often comprise ENDs—such as certain natural phytoestrogens that are more biodegradable than stable petrochemistry-based xenobiotics—often resulting in bioaccumulation within organisms. Mixtures of chemicals such as Agent Orange or dioxins have also been cited as ENDs. In addition, formulants of pesticides—such as POEA (polyoxyethylene tallow amine)—have been discovered to be EDs more recently [Gasnier et al. [19]; Defarge et al. [20]] and may also possess nervous effects [Malhotra et al. [21]; Sato et al. [22]]. In some models, glyphosate alone [Martinez et al. [23]; Coullery et al. [24]], known as the declared active ingredient of a major pesticide formulation used across the world, has been shown to be less toxic or disruptive to the nervous system than its equivalent formulants present in Roundup [Mesnage et al. [8]; Aitbali et al. [25]; Gallegos et al. [26]]. This also proves to be true for non-glyphosate-based herbicides containing non-declared polycyclic aromatic hydrocarbons and heavy metals that are individually identified as EDs [Seralini and Jungers [27]].

The impacts of ENDs on physiological functions and pathologies appear to be less specific than expected, especially since they interact with several intracellular and intercellular communications simultaneously. A majority of them are derived from petroleum residues, and their mechanisms of action can be likened to those of "spam" in electronic communications technologies. Thus, ENDs can be considered as a widespread instance of spam in a biological context.

Table 1
Endocrine disruptors (according to WHO, 2013) also function as nervous system disruptors, and may thus be collectively termed as Endocrine and Nervous Disruptors (ENDs).

Nb	Endocrine disruptor	Class or use	Mechanisms of nervous disruption
1	Acetochlor	Herbicide	**Roman** [28]: antithyroid agents **Helbing et al.** [29]: thyroid hormone receptor gene expression in the brain **Zafeiridou et al.** [30]: compound action potential of the sciatic nerve
2	Alachlor	Herbicide	**Goldner et al.** [31]: hypothyroidism **Seok et al.** [32]: central nervous system symptoms **Lo et al.** [33]: severe neurological and cardiovascular outcomes after acute poisoning **Doïcheva** [34]: higher irritability, lack of coordination and orientation
3	Amitrole	Herbicide	**Sirohi et al.** [35]: specific binding to lactoperoxidase **Chilumuri et al.** [36]: inhibit neuroprotection against amyloid peptides **Pan et al.** [37]: reduction of thyroid-stimulating hormone receptors **Roman** [28]: hypothyroxinemia **Brucker-Davis** [38]: thyroid disruption in utero or direct neurotoxicity
4	Anthracene	PAH	**Palanikumar et al.** [39]: neurotoxicity by inhibition of acetylcholinesterase **Vieira et al.** [40]: increase catalase activity and superoxide dismutase, glutathione reductase and peroxidase.

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Table 1 (*continued*)

Nb	Endocrine disruptor	Class or use	Mechanisms of nervous disruption
5	Aroclor 1254	PCB mixture	**Mucio-Ramírez et al.** [41]: decrease somatodendritic vasopressin release **Wei et al.** [42]: oxidative stress in the brain **Coburn et al.** [43]: Inhibition of vasopressin release from magnocellular neuroendocrine cells **Majumdar et al.** [44]: induces oxidative stress in the brain **Tilson et al.** [45]: increases translocation of protein kinase C and decreases Ca2+-buffering in the brain
6	Arsenic (As)	Heavy metal, pesticide	**Liu et al.** [46]: cognitive impairment **Webb et al.** [47]: chronic neurological disease **Preciados et al.** [48]: influence nuclear respiratory factor 1 by genomic and epigenomic networks, this contributes in the development of complex chronic human brain disorders **Karri et al.** [49]: pro-oxidant elements dominate antioxidants factors and leads to cognitive dysfunction **Tyler at al.** [50]: alters epigenetics and hippocampal function, glutamatergic, cholinergic and monoaminergic signaling; altered adult neurogenesis; and increases Alzheimer's-associated pathologies
7	Atrazine	Herbicide	**Ma et al.** [51]: reduces dopamine levels in the substantia nigra and corpus striatum in the midbrain **Zhang et al.** [52]: severe dopamine neuron degeneration **Lin et al.** [53]: increases in striatal dopamine; decreases in perirhinal cortex serotonin **Ottinger et al.** [54]: impact on arginine vasotocin, catecholamines and gonadotropin releasing hormone.
8	Benzo(a)anthracene	PAH	**Dayal et al.** [55]: most notable symptoms are neurologic
9	Benzo(a)pyrene	PAH	**Slotkin et al.** [56]: impairs neurodifferentiation and neurite formation, development of dopamine and acetylcholine phenotypes **Sarma et al.** [57]: induces neuronal cell damage involving oxidative stress through mitochondria-mediated apoptosis pathway **Yang et al.** [58]: cause disruption of glutamate (Glu) neurotransmitter transmission by decreasing the level of Glu, reducing the expression of Glu receptors **Chepelev et al.** [59]: binds to the aryl hydrocarbon receptor, modulate the transcription of glutamate receptor subunits, decrease long-term potentiation, learning and memory **Niu et al**. [60]: reduces neurobehavioral function and monoamine, amino acid and choline neurotransmitter levels
10	BB-153	PBB	**Jacobson et al.** [61]: associations of PBBs and PCBs with thyroid disease and thyroid hormone levels. **Tilson et al.** [62]: chronic exposure produces behavioral or neurological toxicity
11	Benzyl butyl phthalate	Phthalate	**Morgenstern et al.** [63]: impairs thyroid function in preschool children **Min et al**. [64]: attenuation of the effects of cAMP Response Element-Binding Protein downstream, oxidative damage and impaired behavioral performance **Betz et al.** [65]: changes in amygdala protein s related on synaptic plasticity **Kasuya M** [66]: inhibited the outgrowth of nerve fibers and glial cells from cerebellar explants
12	BDE-209	PBDE	**Chen et al.** [67]: activation of NMDA receptors inhibits the expression of phosphodiesterases, favoring apoptosis induction and induces neurotoxicity. **Li et al.** [68]: the mRNA expressions of synaptobrevin 2, syntaxin 1A, SNAP-25, and synaptophysin are significantly decreases in the hippocampi of rat exposed to it **Sun et al.** [69]: during rat pregnancy increases hippocampal autophagy, decreases neuron viability **Vuong et al.** [70]: large prospective human cohorts demonstrates that prenatal and postnatal exposure adversely impacts externalizing behavior (e.g., hyperactivity and conduct problems)
13	BDE-47	PBDE	**Zhuang et al.** [71]: induces neurotoxicity and cognitive impairment by upregulation of nuclear TAR DNA-binding protein 43 in the hippocampus provoking neuronal apoptosis. **Chen et al.** [67]: inhibits axonal growth via ryanodine receptor-dependent mechanisms **Zhai et al.** [72]: prenatal exposure inhibits neurodevelopmental function and behavior with an increase of luteinizing hormone levels.
14	BDE-99	PBDE	**Dach et al.** [73]: reduces expression of myelin associated genes like HMBP due to olligodendrocyte reduction. **Ding et al.** [74]: prenatal exposure is associated with lower developmental quotients in young children. **Roze et al.** [75]: transplacental transfer is associated with worse fine manipulative abilities, and worse attention of children at school age.
15	Benzene	Aromatic solvent	**Bahadar et al**. [76]: exposure can lead to aberration of vital systems in the body like nervous, cardiovascular, and respiratory. **Manto M.** [77]: in humans, cerebellum is a main target of environmental toxins such as toluene/benzene derivatives. **Ritchie et al.** [78]: a number of published studies report acute or persisting neurotoxic effects of hydrocarbon fuels.
16	Benzylidene camphor	UV filter	**Ruszkiewicz et al.** [79]: potential neurotoxicity **Faass et al**. [80]: reduces proceptive and receptive behaviors and specific gene expression in ventromedial hypothalamic nucleus and medial preoptic area.
17	Bisphenol A	Plastics monomer	**Ejaredar et al.** [81]: exposure in childhood is associated with higher levels of anxiety, depression, hyperactivity, inattention, and conduct problems. **Zhou et al.** [82]: affects neuron numbers in different regions of the hypocampus altering learning and memory ability of adolescent mice. **Inadera H.** [83]: in a review, it has detrimental effects on neurological development. **Masuo et al.** [11]: locus ceruleus is enlarged in treated male rats and neurodegenerative disorders.
18	Bisphenol A diglycid ether	Plastics monomer	**Hutler et al.** [84]: the main abnormalities in amphibians larvae related to neurotoxicity.
19	Bisphenol F	Plastics monomer	**Ohtani et al.** [85]: exposure alters offspring behavior, resulting in increases in anxiety and depressive state in mice. **Rosenfeld** [86]: induces neurobehavioral disruptions. **Castro et al.** [87]: affects genes in the juvenile female rats prefrontal cortex.
20	Bisphenol S	Plastics monomer	**Wu et al.** [88]: triggers oxidative stress in the nervous system in vivo and in vitro in humans. **Castro et al.** [87]: affects genes expression and dopamin serotonin system in the prefrontal cortex of juvenile female rats.

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Table 1 (*continued*)

Nb	Endocrine disruptor	Class or use	Mechanisms of nervous disruption
21	Bromacil	Herbicide	**Lombardi et al** [89]: exposure through residential proximity to agricultural applications during pregnancy may increase the risk of childhood central nervous system tumors.
22	Butylate	Herbicide	**Lulla et al.** [90]: for Ziram: selectively toxic to dopaminergic neurons in vivo, and this toxicity is synuclein-dependent.
23	Butylated hydroxyanisole	Antioxidant for long preservation of food products	**Miyazaki et al.** [91]: quinone reductase inducer which significantly and dose dependently blocked methamphetamine-induced elevation of quinoprotein, and ameliorated methamphetamine-induced cell death. **Katsuki et al.** [92]: abolishes neurotoxic action of arachidonic acid.
24	Cadmium (Cd)	Heavy metal	**Raciti et al.** [93]: affects epigenetics with negative consequences on the development of the nervous system. **Jacobo-Estrada et al.** [94]: induces toxicity in fetus on the central nervous system. **Zhang et al.** [95]: induces autophagy in neurons promoting neurodegenerative disorders. **Sanders et al** [96]: exposure may be associated with poorer cognition. **Bo et al**. [97]: neurotoxic on the long-term.
25	Carbamazepine	Pharmaceutical anti-epileptic	**Al-Rubai et al.** [98]: reduces neurosphere size and cell migration at high doses. Reduction in glial fibrillary protein and tubulin III. **Hansen et al.** [99]: induces encephalopathy with hyperammonemia, intrinsic effects on cerebral receptors. **Gualtieri et al.** [100]: affects tests of memory, psychomotor speed, cognitive flexibility, and attention.
26	Carbaryl	Insecticide	**Lee et al.** [101]: inhibits classically acetylcholinesterase in the nervous system; induces cognitive impairments by disturbed neurodevelopment. **Freeborn et al.** [102]: affects electroencephalogram by decreasing theta area and delta frequency, increases beta frequency. **Wang et al**. [103]: acetylcholinesterase is inhibited by high dose and damages the sciatic nerve.
27	CB-15	PCB	**Cocco et al** [104]: chronic exposure to PCBs affects the development and function of the nervous system. **Lovato et al** [105]: a mixture of PCB can induce functional deficits and altered behavioral threat in zebrafish.
28	CB-77	Coplanar PCB	**Ozcan et al.** [106]: dioxin-like and non-dioxin-like PCB congeners are equally potent in causing cognitive decrements seen in children exposed prenatally to PCBs. **Howard et al.** [107]: binds the aryl hydrocarbon receptors with high affinity.
29	CB-118	PCB	**Brucker-Davis et al.** [108]: negative impact on neurocognitive development, negatively correlated on motor and expressive language in children. **Doi et al.** [109]: four-month-olds children with a low-level of prenatal exposure exhibits a preference for the upright biological motion, impairs the development functioning and brain development.
30	CB-126	Planar PCB	**Cauli et al.** [110]: impairs motor coordination at 2 months in males but not in female rats, reduces locomotor activity in females.
31	CB-132	PCB	**Uwinana et al.** [111]: does not appear to affect dopaminergic cells in cultures or levels of dopamine. To be further studied.
32	CB-138	PCB	**Boix et al.** [112]: exposition activates metabotropic glutamate receptors and that increases dopamine in females and reduces it in males. The opposite changes are observed for glutamate, in rat nucleus accumbens. **Campagna et al.** [113]: Ca2+ homeostasis and androgen receptor signaling pathways are primarily disrupted in cerebellum proteome, contributing toward a premature ageing and neurotoxicity. **Naert et al.** [114]: birds bioaccumulate in brain and the central nervous system.
33	CB-153	PCB	**Enayah et al.** [115]: neurotoxic, and affects dopamine turnover in vitro. **Cauli et al.** [110]: many motor alterations and induces hyperactivity at adulthood in rats. **Gascon et al.** [116]: deleterious effects on neuropsychological development which are mainly attributable to prenatal exposure.
34	CB-169	Planar PCB	**Morse et al.** [117]: local hypothyroidism occurs in the brains of fetal and neonatal rats exposed by increase in type II thyroxine 5'-deiodinase in the brain.
35	CB-180	PCB	**Boix et al**. [112]: affects motor activity in rats; increased glutamate release in nucleus accumbens following activation of metabotropic glutamate receptors would be involved in reduced dopamine release. **Naert et al.** [114]: birds bioaccumulate in brain and the central nervous system.
36	Chlordane	Organo-chlorine insecticide	**Kilburn** [118]: it is suggested that it causes protracted neurotoxicity in patients. **Kilburn and Thornton.** [119]: exposure is associated with protracted impairment of neurophysiological and psychological functions. The central nervous system is the most important target. **Grutsch et al**. [120]: the characteristic signs of acute toxicity are hypothermia, hyperexcitability, tremors and convulsions. In human, signs of acute toxicity are tremors and convulsions.
37	Chlordibromo-methane	Trihalo-methane	**Villanueva et al.** [121]: Minor associations observed between exposure during gestation and child neuropsychological development. **Balster et al.** [122]: effect on operant behavior in mice.
38	Chlorinated Paraffins	Flame retardants, lubricants, plasticizers	**Liu et al.** [123]: exposure could alter gene expression in the hypothalamic-pituitary-thyroid axis. **Mariussen et al.** [124]: neurobehavioral effects, indicating adverse effects on the central nervous system: alteration of neurotransmitter functions, Ca2+ homeostasis processes, induction of protein kinase C and phospholipase A2 mobilization, and oxidative stress. **Eriksson et al.** [125]: significant decrease of presynaptically sodium-dependent choline uptake in mice.
39	Chlorpyrifos	Insecticide	**Burke et al.** [126]: acute exposure of humans irreversibly inhibit acetylcholinesterase, and chronic exposure induces neurological deficits that range from cognitive impairments to tremors in childhood **Yamada et al.** [127]: inhibit neural induction via mitochondrial fusion protein mitofusin 1-mediated mitochondrial dysfunction in human stem cells. **Sogorb et al.** [128]: seems able to induce neurodevelopmental alterations in animals **Lee et al.** [101]: affects protein levels in the mice developing brain and induces persistent adult behavior and cognitive impairments; neurotoxic effects.

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Table 1 (*continued*)

Nb	Endocrine disruptor	Class or use	Mechanisms of nervous disruption
40	Citalopram	Antidepressant	**Yeo et al.** [129]: disability is improved. **Gaanderse et al.** [130]: induces dyskinesia of the tongue. **Sprowles et al.** [131]: selective serotonin reuptake inhibitor, alters spatial learning and memory, anxiety, depression in rats.
41	Clofentezine	Pesticide Acaracide	**Hurley** [132]: induces thyroid follicular cell tumors in rodents; disrupts thyroid-pituitary homeostasis.
42	Coumaphos	Pharmaceutical	**Abdelsalam** [133]: inhibition of brain of acetylcholinesterase, inhibition of brain neurotoxic esterase, plus delayed neurotoxicity. **Abou-Donia et al.** [134]: degeneration of axons and myelin in the spinal cord.
43	Coumestrol	Phytoestrogen	**Jantaratnotai et al.** [135]: suppression of interferon regulatory factor-1 and phosphorylated STAT1 expression in lipopolysaccharide-activated microglia.
44 45 46	D4 Cyclic siloxane D5 Cyclic siloxane D6 Cyclic siloxane	Material	**Andreou et al.** [136]: unusual constriction of the isolated sciatic nerve, death of nerve fibers. **Fuzzard et al.** [137]: due to silicone implants, myalgias, chronic fatigue, cognitive impairment.
47	Daidzein	Isoflavones Phytoestrogen	**Yu et al.** [138]: perinatal exposure enhances estrogen receptor alpha expression in several brain regions such as stria terminalis, arcuate hypothalamic nucleus, and central amygdaloid nucleus. **Zeng et al.** [139]: significant effects on locomotor activity, mood and social behavior after long-term consumption. **Jin et al.** [140]: the neurotoxic effect of daidzein could be due to the inhibition of the GABA(A) receptor resulting in further enhancement of excitation by glutamate and leading to cellular damage in primary rat neuronal cultures. **Román GC** [28]: inhibits thyroperoxidase that catalyzes iodination and thyroid hormone biosynthesis
48	Dibromochloropropane	Pesticides	**Teitelbaum** [141]: the product is central nervous system depressant.
49	Desethylatrazine	Herbicide Metabolite	**Liu et al.** [46]: neuroendocrine disruptor that impacts the expression of neurotoxicity-related genes such as Ache, Gap43, Gfap, Syn2a, Shha, Mbp, Elavl3, Nestin and Ngn1 in early developmental stages of zebrafish. **Gunderson et al.** [142]: Strong positive correlation between CYP19 and SF-1 transcript abundance in exposed tadpoles during brain development. **Hossain et al.** [143]: decreases striatal dopamine levels and in synaptosomes in rat.
50	2,4-D	Herbicide agent orange	**Yi et al.** [144]: Increases various neurologic diseases; systemic atrophies affecting the nervous system, including spinal muscular atrophy, Alzheimer disease, and peripheral polyneuropathies. **Bortolozzi et al** [145]: changes in various neurotransmitter systems, such as serotonin (5-HT) and dopamine (DA), were proposed to mediate some of the behavioral effects in rats **Evangelista de Duffard et al.** [146]: Increases sensitivity in dopamine D2-like brain receptor from 2,4-dichlorophenoxyacetic acid (2,4-D)-exposed and amphetamine challenged rats.
51	2,4-Dichlorophenol	Chlorophenol	**Krieg** [147]: At low concentrations, it may act at acetylcholine and γ-aminobutyric acid synapses in the central nervous system to modify neurobehavioral test performance.
52	3-Diltiazem	Pharmaceutical	**Stevens et al.** [148]: Calcium channel blocker.
53	2,4′-DDD (o,p'-DDD)	Organo-chlorine Insecticide and pharmaceutical mitotane	**Heilmann et al.** [149]: Some central nervous disorders were observed. **Lanser et al.** [150]: Neuropsychologic and neurologic side effects. **Du Rostu et al.** [151]: neurological symptoms and neurotoxicity both central and peripheral.
54	2,4′-DDT (o,p'-DDT)	Organo-chlorine Insecticide	**Kajta et al.** [152]: depressive-like symptoms after prenatal exposure possibly by DNA hypomethylation and estrogen receptor signaling pathway. **Zhang et al.** [153]: effects on glucocorticoid-receptors on the nervous system. **Eskenazi et al.** [154]: Prenatal exposure to DDT, and to a lesser extent DDE, was associated with neurodevelopmental delays during early childhood. **Halldin** [155]: altered the development of the neural system and resulted in demasculinization of male quail. **Fry** [156]: impaired differentiation of the nervous system through mechanisms of hormonal mimicking of estrogens.
55	4,4′-DDD (p,p'-DDD)	Organo-chlorine Insecticide	**Al-Saleh et al.** [157]: low parent's evaluation of developmental status of infants was significantly associated with DDD in breast milk.
56	4,4′-DDE (p,p'-DDE)	Organo-chlorine Insecticide	**Wnuk et al.** [158]: stimulation of retinoid X receptor α and retinoid X receptor β-mediated intracellular signaling plays an important role in the propagation of DDE-induced apoptosis during early stages of neural development. **Cartier et al.** [159]: p,p'-DDE exposure, both pre- and postnatally, during early childhood is associated with visual processing impairment later in life. **Eskenazi et al.** [154]: Prenatal exposure to DDT, and to a lesser extent DDE, was associated with neurodevelopmental delays during early childhood.
57	4,4′-DDT (p,p'-DDT)	Organo-chlorine Insecticide	**Kajta et al.** [152]: depressive-like symptoms after prenatal exposure possibly by DNA hypomethylation and estrogen receptor signaling pathway. **Zhang et al.** [153]: effects on glucocorticoid-receptors on the nervous system. **Eskenazi et al.** [154]: prenatal exposure to DDT, and to a lesser extent DDE, was associated with neurodevelopmental delays during early childhood **Parent et al.** [160]: DDT is involved in neuroendocrine disruption of the reproductive axis.
58	Di-(2-ethylhexyl) adipate	Plasticizer	**Lee et al.** [161]: inappropriate expression of granulin and/or p130 genes in the brains of male and female neonatal rats by perinatal exposure may exert permanent effects on the hypothalamus, thereby decreasing sexual behavior after maturation.
59	Dehydroepi-androsterone	Natural hormone	**Arbo et al.** [162]: neuroactive steroid that modulate neuronal and astroglial function and have neuroprotective effects. **Woda et al.** [163]: synthetized in nervous cells, neuroactive local factor in the central nervous system and the periphery. **Yu et al.** [164]: induced depression-like behavior in polycystic ovary syndrome mice, possibly through down-regulation of brain monoamines and/or their metabolites. **Li et al.** [165]: provides robust ischemic neuroprotection but also exerts neurotoxicity when administered during ischemia and early reperfusion.

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Table 1 (*continued*)

Nb	Endocrine disruptor	Class or use	Mechanisms of nervous disruption
60	Dexamethasone	Synthetic steroid	**Coplan et al.** [166]: glucocorticoid-induced neurotoxicity. **Yu et al.** [164]: protracted disruption of mental functions. **Lopes et al.** [167]: affects the phosphorylation state of glutamate AMPA receptors in the human limbic system. **Feng et al.** [168]: decreased both body and brain weight gain. Tapering and repeated doses increased caspase-3 activity. Impaired learning and memory capability at juvenile age. **Uno et al.** [169]: induced degeneration and depletion of the hippocampal pyramidal and dentate granular neurons in the brains of primate fetuses.
61	Dibutyl phthalate	Phthalate	**Wójtowicz et al.** [158]: Aryl hydrocarbon receptor is involved in dibutyl phthalate induced apoptosis and neurotoxicity, while the estrogen receptors and peroxysome proliferator-activated receptor gamma signaling pathways are impaired by the phthalate. **Farzanehfar et al.** [170]: could reduce total distance movement, impair memory function an induce anxiety in mice. Significant nuclei size reduction and condensation in dentate gyrus cells **Yan et al.** [171]: link between oxidative stress and anxiety-like behavior produced by dibutyl phthalate at high doses.
62	Dibutyltin	Plastics stabilizer	**Chantong et al.** [172]: potentiation of oxidative stress and pro-inflammatory cytokine expression in microglia cells **Tsuji et al.** [173]: Dibutyltin is neurotoxic and poly-L-lactides toxicity increases with the increase in tin concentration. **Jenkins et al.** [174]: developmental neurotoxicant; the incidence of apoptotic cell death, was increased in the neocortex and hippocampus. **Kobayashi et al.** [175]: synaptic parameters modulations; tributyltin metabolites inhibit various parameters of cholinergic activity with a potency ranking of tributyltin> dibutyltin> monobutyltin.
63	Dicofol	Organo-chlorine Insecticide	**Evangelista de Duffard et al.** [146]: has effects on motor, sensory, or cognitive functions. **Lessenger et al.** [176]: case report, neurological injury, cognitive and emotional difficulties persisted over an 18-mo period.
64	Dieldrin	Organochlorine Insecticide	**Cowie et al.** [177]: disrupts proteins related to oxidative respiration and mitochondrial stress in the central nervous system. **Schmidt et al.** [178]: induced neurotoxicity by impaired mitochondrial bioenergetics and endoplasmic reticulum stress in rat dopaminergic cells. **Babot et al.** [179]: Long term exposure reduces gamma-aminobutyric acid type A and N -methyl—aspartate receptor function in primary culture of mouse cerebellar granule cells. **Evangelista de Duffard et al.** [146]: motor sensory or cognitive function effects.
65	Diethyl hexyl phthalate	Phthalate	**Luu et al.** [180]: regulate microRNAs in a sex-specific manner which may interfere with proper hippocampal development in males and preserve hippocampal development in females. **Preciados et al.** [48]: induced brain health deficits by NRF1 regulated gene networks. **Park et al.** [181]:sex-dependent effect on anxiety proneness in childhood. **Quinnies et al.** [182]: transgenerational modifications in the expression of several pituitary hormones involved in the hypothalamic-pituitary-adrenal axis and in stress hormones.
66	Mono-2-ethyl-hexyl phthalate	DEHP Hydrolysis product	**Huang et al.** [183]: prenatal exposure was associated with decreased cognitive development in the young children. **Téllez-Rojo et al.** [184]: prenatal exposure creates sex specific neurodevelopmental effects.
67	Mono-n-butyl phthalate	DBP Hydrolysis product	**Doherty et al.** [185]: prenatal associations between urinary phtalates in aged mothers and brain performances in young children. **Mao et al.** [186]: induce spatial cognitive deficits through altering the expression of apoptosis-related protein. **Won et al.** [187]: increased exposure exhibited supralinear associations with social, thought and attention problems in children.
68	Diethylstilbestrol	Synthetic estrogen	**Tomihara et al.** [188]: developmental deficits may stem from both in utero toxicity and aberrant maternal care. **Frye et al.** [189]: effects on the aryl hydrocarbon receptor, the peroxisome proliferator-activated receptor and the retinoid X receptor, signal transduction pathways, and on calcium influx and/or neurotransmitter receptors. **Sato et al.** [190]: marked influence on synaptogenesis and neuronal vulnerability through mechanisms other than through estrogen receptors.
69	Diisononyl phthalate	Plasticizer	**Ma et al.** [191]: cause cognitive deficits and anxiety. **Peng L** [192]: oral exposure of mice induced brain damage, and oxidative stress, inflammation, and apoptosis. **Boberg et al.** [193]: behavioral effects, spatial learning effects in perinatally exposed rats.
70	Diphenhydramine	Antihistamine	**Kim et al.** [194]: Inhibitory effects on proton currents in microglial cells. **Mansfeild et al.** [195]:reduced attention and increased self-reported drowsiness. **Wilken et al.** [196]: caused significant decrements in vigilance and cognitive functioning.
71	Dimethyl-benz(a) anthracene	PAH	**Vaswani et al.** [197]: alterations of opioid neuropeptides such as beta endorphin, meth-enkephalin and dynorphin levels
72	Endosulfan (alpha/beta)	Organo-chlorine Insecticide	**Jang et al.** [198]:induced acute neurotoxicity via induction of oxidative stress and pro-inflammatory responses. **Caudle WM** [199]: can alter the normal development and potential function of neurotransmission in the frontal cortex **Silva et al.** [200]: neurotoxicity and developmental effects in the zebrafish. **Silva et al.** [201]:effects on brain biogenic amine levels Developmental reproductive toxicity or endocrine disruption occurs only at doses causing neurotoxicity.
73	Endrin	Organo-chlorine Insecticide	**Bagchi et al.** [202]: induced lipid peroxidation and DNA damage in brain and regional distribution of catalase activity in rat brain. **Gray et al.** [203]: alteration of central nervous system function in rats and hamsters even though endrin produces gross morphological defects only in hamsters.
74	Estradiol	Natural hormone	**Li et al.** [204]: anxiety disorders, augmentation of vulnerability factors associated with anxiety disorder development; and facilitation of the maintenance of anxious symptoms post-development. **Preciados et al.** [48]: influences NRF1 regulated gene networks in the development of complex human brain diseases. **Perez-Alvarez et al.** [205]: neuroprotective role after ischemic injury. **Rossetti et al.** [206]: neurosteroid bind specific receptors to promote essential brain functions.

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Table 1 (*continued*)

Nb	Endocrine disruptor	Class or use	Mechanisms of nervous disruption
75	Estrone	Natural hormone	**Mahmoud et al.** [207]: may influence adult hippocampal neurogenesis, with a focus on cognitive function and mood regulation. **Grimm et al.** [208]: may act upon neuronal bioenergetics in a delicate balance with an age-related effect that might be involved in mitochondrial dysfunction underlying neurodegenerative disorders.
76	Ethinylestradiol	Synthetic hormone	**Porseryd et al**. [209]: alteration in expression of genes involved in synaptogenesis and synaptic function. In female brains, produced significant effects on pathways connected to the circadian rhythm, cytoskeleton and motor proteins and synaptic proteins. In male brains effects on pathways related to cholesterol biosynthesis and synaptic proteins. **Preciados et al.** [48]: influences NRF1 signaling pathways, and epigenomic multiple networks. **Zaccaroni et al.** [210]: very low doses during development can affect key behavioral traits that are modulated by anxiety.
77	Ethylene thiourea	Herbicide	**Wang et al.** [211]: induced abnormal innervation patterns in the anorectum of fetal rats **Debbarh et al.** [212]: neurotoxic in utero, increases sensitivity to genetic and environmental risk factors for cell death and apoptosis.
78	Ethylparaben	Antifungal Preservative	**Merola et al.** [213]: provoked behavioral changes including trembling of head, pectoral fins and spinal cord of zebrafish.
79	Fadrozole	Pharmaceutical	**Lynch et al.** [214]: displayed significant fear generalization in rats. **Alward et al.** [215]: this aromatase inhibitor reduced the motivation to sing as well as song acoustic stereotypy. **Xing et al.** [216]:dopamine neuron degeneration and aromatase activity inhibition could be respectively achieved in vivo with treatments with the product in female goldfish. **Langlois et al**. [217]: induced female- and male-biased sexual development on Silurana tropicalis brain mRNA levels, and reduced brain aromatase activity in frogs.
80	Fenbuconazole	Fungicide	**Hurley et al.** [132]: disrupts thyroid hormone excretion.
81	Fenitrothion	Organophosphate Insecticide	**Geraldi et al.** [218]: affected the acquisition and, mainly, the retention of instrumental conditioning in rats. **Groszek et al.** [219]: High concentration of the pesticides was found in adipose tissue and also in the brain. Respiratory failure was the syndrome; and inhibition of acetylcholinesterase activity persisted even for 30 days from poisoning. **Ram et al.** [220]: Neurobehavioral changes in freshwater fish exposed
82	Fenoxycarb	Insecticide	**Lenkic et al.** [221]: allatostatin may be one of the effectors in the brain by which the pesticides inhibits juvenile hormone biosynthesis.in cockroach.
83	Finasteride	Pharmaceutical	**Fertig et al.** [222]: permanent sexual dysfunction and mood changes (fatigue, anxiety, depression and suicidal ideation) during treatment with this 5-alpha-reductase inhibitor. **Traish et al.** [223]: Also non-sexual adverse effects such as diabetes, psychosis, depression, and cognitive function. **Ganzer et al.** [224]: sexual libido, ejaculatory disorders, disorders of the penis and testes, cognitive symptoms, and psychological symptoms
84	Fipronil	Insecticide	**Godinho et al.** [225]: toxic interactions with the central nervous system of mammals and lead to memory impairment by modulating the GABAergic system. **Park et al.** [181]: Progressive loss of nigrostriatal dopaminergic neurons induced by inflammatory responses to the pesticide. **Magalhães et al**. [226]: acts on maternal aggressive behavior through GABA(A) receptors. **Simon-Delso et al.** [227]: disrupting neural transmission in the central nervous system of invertebrates, inhibits neuronal receptors. **Marrs et al.** [228]: 4-Aminobutyric acid (GABA) and glycine are inhibitory neurotransmitters and their antagonist, fipronil, is excitatory.
85	Fluoxetine	Pharmaceutical	**Golub et al.** [229]: provoked greater dendritic spine synapse density in prefrontal cortex of monkeys. **Hong et al.** [230]: induced predominant sympatho-excitation and depressed parasympathetic activity leading to mild hypertension, tachycardia, and impairment of baroreflex function. **Sprowles et al.** [131]: Differential effects of perinatal exposure to antidepressants on learning and memory, acoustic startle, anxiety, and open-field activity in rats.
86	Flutamide	Pharmaceutical	**Yamada et al.** [127]: the effects of postnatal treatment on brain masculinization were observed by analysis of male sexual behavior. **Svensson** [231]: induced anxiolytic-like behavior in castrated rats **Zhang et al.** [232]: Effects of neonatal treatment on hippocampal neurogenesis and synaptogenesis correlate with depression-like behaviors in preadolescent male rats. **Ahmadiani et al**. [233]: Anticonvulsant effects on seizures involvement of benzodiazepine receptors.
87	Fonofos	Organo-phosphate Insecticide	**GK Sidhu,** [234]: known to inhibit acetylcholinesterase activity, not only in insect, but in aquatic and terrestrial organisms leading to nervous abnormalities among others.
88	Formaldehyde	Solvent	**Liu et al. (2018)** [235]: Acute formaldehyde exposure induced early Alzheimer-like changes in mouse brain. Provoked the permeability of the blood-brain barrier, activation of astrocyte and microglia, oxidative stress and inflammation. **Li et al.** [236]: effects on anxiety, depression-like behavior and cognition ability which may be associated with alterations in hippocampal glucocorticoid receptors and brain tyrosine hydroxylase levels. **Zendehdel et al.** [237]: Its neurotoxic effect depend on acetylcholinesterase activity; provoked cholinergic signal reduction in cases of cognitive dysfunction. **Tulpule et al.** [238]: contribute to the impaired cognitive performance and neurodegeneration in diseases. **Songur et al.** [239]: neurotoxic characteristics; neurological diseases.
89	Furan	Solvent	**Johnston et al** [240]: exhibits a peculiar mode of attack on the central nervous system
90	Galaxolide	Synthetic musk	**Ayuk-Takem et al.** [241]: neurotoxicity may be associated with the inhibition of cellular; polyisoprenylated methylated protein methyl esterase activity; significant risk to individuals predisposed to developing degenerative disorders.

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Table 1 (*continued*)

Nb	Endocrine disruptor	Class or use	Mechanisms of nervous disruption
91	Genistein	Isoflavone Phyto-estrogen	**Patisaul HB** [242]: involve action at nuclear estrogen receptors, effects on vasopressin, innervation of the lateral septum and other brain regions. **Luo et al.** [243]: pretreatment significantly increased cell viability and protein kinase C activity, decreased the levels of intracellular calcium, and blocked caspase-3 activity in induced cells. **Román GC** [28]: Transient in utero hypothyroxinemia is related to maternal flavonoid ingestion during pregnancy and may provoke autism. **Lee et al.** [244]: mimic the actions and functions of estrogens on brain, two putative pathways; an estrogen receptor-mediated pathway and via the inhibition of tyrosine kinase. **Lephart et al**. [245]: The specific influence of dietary soy phytoestrogens is identified on consumptive, learning and memory, and anxiety-related behaviors.
92	Hexabromocyclodo-decane	Flame retardant	**Pham-Lake et al.** [246]: Impairment in the mesohippocampal dopamine circuit following exposure. **Wang et al.** [247]: main metabolic pathways perturbed, nervous system damage, and developmental disorders. **Maurice et al.** [248]: Short-term effects of a perinatal exposure in rats provoked impairments of early locomotor activity and sensory development. **Al Mousa et al.** [249]: inhibiting reticulum Ca(2+)ATPase in human neuroblastoma cells and induced cells death possibly causing neurological disorders. **Lilienthal et al.** [250]: Effects on dopamine-dependent behavior and brainstem auditory evoked potentials in rats.
93	Hexachlorobenzene	Chlorinated aromatic	**Fu et al.** [251]: exhibits through its metabolite a neurotoxic effect by inducing oxidative stress-mediated inflammatory responses. **Kyriklaki et al.** [252]: High exposure during pregnancy reduction in working memory score and reduced cognitive development at preschool age. **Reed et al.** [253]: exposure involved systemic impairment, as well as on nervous system. **Li et al.** [254]: can induce enhanced lipid peroxidation on rats, and the oxidative stress plays an important role in the mechanism of neurotoxicity. **Goldey et al.** [255]: behavioral teratogen, and suggests that human fetuses and suckling infants may be at risk because of the neurotoxic effects of the chemical.
94	Heptachlor	Organo-chlorine Insecticide	**Nyffeler et al.** [256]: neural crest cell migration was inhibited by this toxicant disturbing a key neurodevelopmental process. **Christen et al.** [257]: Strong and dose- dependent inhibition of neurite outgrowth was induced developmental neurotoxicity. **Hong et al.** [258]: induced nigral dopaminergic neuronal loss and Parkinsonism-like movement deficits in mice. **Moser et al.** [259]: perinatal exposure produced neurochemical and persistent neurobehavioral changes, including alterations in spatial learning and memory.
95	Heptachlor epoxide	Organo-chlorinei Insecticide Metabolite	**Kirby et al.** [260]: toxic effects of heptachlor epoxide may be responsible for loss of maximal dopamine uptake **Yamaguchi et al.** [261]: effects on calcium mediated transmitter release from brain synaptosomes of rats.
96	Hexachlorobutadiene	Solvent	**Badaeva et al.** [262,263]: neurotoxic effects in the postnatal period of ontogeny in the rats. **Murzakaev** [264]: small doses affected central nervous activity.
97	Heptachloro-dibenzodioxin	Dioxin	**Chen et al.** [265]: synaptic plasticity and neuro-immune system may be two principal affected areas. **Kimura et al.** [266]: over-activation of aryl hydrocarbon receptor following perinatal dioxin exposure, perturbs neuronal migration and morphological development in mammalian cortex, supporting previous observations of impaired dendritic structure, cortical dysgenesis, and behavioral abnormalities
98	HPTE	Methoxychlor Metabolite	Not specifically studied (see Methoxyclor):
99	Iodine (I)	Halogen; Essential element	**Román** [28]: Iodine deficiency as a cause of autism.
100	Kepone	Organo-chlorine Insecticide	**Evangelista de Duffard et al.** [146]: effects on motor, sensory, or cognitive function; developmental neurotoxicant. **Mactutus et al.** [267]: Neonatal exposure impairs early learning and retention of active avoidance in the rat. **Mactutus et al.** [268]: neurotoxic profile of tremor. **Mactutus et al.** [269]: effect on the development of behavioral and/or neural function.
101	Lead	Heavy metal	**Andrade et al.** [270]: can induce dyshomeostasis, potentially triggering neurodegenerative disorders, such as Alzheimer's disease and Parkinson's disease Additionally, changes in heme synthesis have been associated with neurodegeneration. **Chen et al.** [271]: exposure in the early stages of neurodevelopment results in long-lasting alterations that ultimately cognitive function and behavior. The prime targets of lead toxicity are the multipotent neural stem cells **Assi et al.** [272]: wide spectrum of toxic effects, a real threat to the public health, including on the central nervous system **Karri et al.** [49]: lead to imbalance between the pro-oxidant elements and the antioxidants, and induced cognitive dysfunction. **Caito et al.** [273]: The central nervous system is particularly vulnerable. The brain accumulates metals.
102	Levonorgestrel	Synthetic Estrogen	**Aleknaviciute et al.** [274]: induces a centrally-mediated sensitization of both autonomic and hypothalamic-pituitary-adrenal (HPA) axis. **Simone et al.** [275]: in combination with ethinyl estradiol reduced brain-derived neurotrophic factor mRNA in the hippocampus resulting in a decline in learning and memory. **Porcu et al.** [276]: Long-term administration decreased allopregnanolone levels and altered GABA(A) receptor subunit expression and anxiety-like behavior.
103	Lindane	Organo-chlorine Insecticide	**Costa** [277]: block the chloride channels of the GABA-A receptor. **Mariussen et al.** [124]: has neurotoxic potentials after both acute and chronic exposure. **Evangelista de Duffard et al.** [146]: has effects on motor, sensory, or cognitive function modifying behavior.

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Table 1 (*continued*)

Nb	Endocrine disruptor	Class or use	Mechanisms of nervous disruption
104	Linuron	Herbicide	**Quintaneiro et al.** [278]: adverse effects on neurotransmission and energy production, and interference with hypothalamic-pituitary-thyroid and -adrenal-axis. **Lichtensteiger et al.** [279]: in antiandrogenic mixtures impacted genes encoding for components of excitatory glutamatergic synapses and genes controlling migration and pathfinding of glutamatergic and GABAergic neurons, as well as genes linked with increased risk of autism spectrum disorders. **Schinn et al.** [280]: in mixture inhibited swimming activity of juvenile rainbow trout.
105	Malathion	Organophosphate Insecticide	**Richendrfer & Creton** [281]: cause abnormalities in behavior and brain size during development, zebrafish larvae had significantly smaller forebrain and hindbrain regions. **Salama et al**. [282]: affect proliferation, differentiation and viability of cultured neurospheres, **Hashjin et al.** [283]: induced chronic toxicity and anxiety-like behavior in the male adult mouse. **Rastogi et al.** [284]: In mixture provoked neurologic self-reported symptoms, headache, watering in eyes, and burning sensation in eye/face, cholinergic symptoms, such as insomnia, headache, muscle cramps, weakness, and anorexia, in children. High frequency of neurologic symptoms may be due to parasympathetic hyperactivity. **Valvassori et al.** [285]: affects the central nervous system by inhibiting acetylcholinesterase, leading to an increase of acetylcholine in the synaptic cleft, and subsequent activation of cholinergic muscarinic and nicotinic receptors, and impairs aversive-memory retention but not non-associative memory, without affecting anxiety-related behaviors.
106	Mancozeb	herbicide	**de Joode et al.** [286]: poorer verbal learning outcomes in children, may affect their neurodevelopment. **Brody et al.** [287]: behavioral dysfunction, notably serotonin-mediated egg-laying behavior in Caenorhabditis elegans. **Li et al.** [288]: potentiation on KCNQ2 potassium channels might be the possible mechanism of this product toxicity in the nervous system. **Domico et al.** [289]: acute exposure to high doses produces equipotent toxic effects in both dopamine and GABA neurons. **Kimura et al.** [290]: nerve conduction velocities and postural sway seem to be sensitive indicators of the effects on the central and peripheral nervous system.
107	Manganese	Heavy metal	**Lucchini et al.** [291]: essential metal that plays a fundamental role for brain development and functioning. Environmental exposure may lead to accumulation in the basal ganglia and development of Parkinson-like disorders. **Peres et al.** [292]: Various neurotransmitter systems may be impaired, especially dopaminergic, but also cholinergic and GABAergic. **Tarale et al.** [293]: epigenetic mechanism in product-induced neurotoxicity, development of Parkinson's disease. **Zhang et al.** [293]: overexposure amplified the role of autophagy in the mechanisms of common neurodegenerative disorders.
108	Methylsulfonyl-DDE	DDE Metabolite	**Wnuk et al.** [158]: apoptotic action during early stages of neural development with crucial involvement of retinoid X receptors. **Torres-Sanchez et al.** [294]: prenatal exposure impaired early child neurodevelopment.
109	Methoxychlor	Organo-chlorine Insecticide	**Zhang et al.** [153]: showed remarkable GR antagonistic properties, disruption of glucocorticoid-responsive genes. **Martini et al.** [295]: perinatal exposure has an organizational effect on hippocampus-dependent memory and emotional behaviors. **Schuh et al.** [296]: inhibited brain mitochondrial respiration and increases hydrogen peroxide production and CREB phosphorylation.
110	Methyl bromide	Fumigant, Pesticide	**De Souza et al.** [297]: acute and chronic progressive neurologic injury: seizures, myoclonus, ataxia or cerebral oedema, defective neurotransmitter function and abnormal oxidative phosphorylation. **Kim & Kang** [298]: chronic toxic encephalopathy. **Yang et al.** [299]: Sub chronically and chronically, principal target PAHsite appears to be the central nervous system. **Anger et al.** [300]: produce slight neurotoxic effects in fumigation, reduced performance on all cognitive tests.
111	Methyl farnesoate	Juvenile Hormone	**Moshitzky et al.** [301]: neural inhibition from the brain (drosophila) act before farnesoic acid, a precursor of the product. **Prestwich et al.** [302]: is secreted by the mandibular organs of crustaceans, role partially known.
112	Methyl triclosan	Triclosan metabolite Product	**DeLeo et al.** [303]: Effect on thyroid hormone action and stress in frog and mammalian culture systems.
113	Methylbenzylidene camphor	UV filter	**Ruszkiewicz et al.** [79]: potential neurotoxicity **Broniowska et al.** [304]: affected the viability of nerve cells, most likely by enhancing the process of apoptosis. **Li et al.** [305]: reduction of neuronal and muscular development in zebrafish embryos **Faass et al.** [80]: effect on female sexual behavior and gene expression in sexually dimorphic brain regions after pre- and postnatal exposure in rats. **Maerkel et al.** [306]:Sex- and region-specific alterations of progesterone receptor mRNA levels and estrogen sensitivity in rat brain
114	Methylcholanthrene	PAH	**Singh et al.** [307]: induces neurotoxicity in developing neurons derived from human stem cells by activation of aryl hydrocarbon receptor.
115	Mirex	Organo-chlorine Insecticide	**Puertas et al.** [308]: showed a decrease in working memory in children. The deficit found in intellectual function during early childhood suggests that prenatal exposure may have a significant impact on school performance. **Shankland** [309]: enhanced the release of neurotransmitters. Direct evidence is available on cholinergic and glutaminergic junctions, but other kinds of junctions may be affected.
116	Monosodium glutamate	Food Additive	**Foran et al.** [310]: Auditory hindbrain atrophy and anomalous calcium binding protein expression after neonatal exposure. **Sadek et al.** [311]: induced neurotoxicity by cholinergic dysfunction, Bcl-2/Bax balance, and antioxidant enzymes gene transcripts in rats **Sasaki-Hamada et al.** [312]: Changes in hippocampal synaptic functions and protein expression in obese mice. **Lau et al.** [313]: glutamate excitotoxicity has also been linked to chronic neurodegenerative disorders such as amyotrophic lateral sclerosis; evidence for the product excitotoxicity in acute neurologic diseases

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Nb	Endocrine disruptor	Class or use	Mechanisms of nervous disruption
117	n-Butylbenzene	Chemical Synthesis Intermediate	**Chalansonnet et al.** [314]: a decrease in the concentrations of free malondialdehyde in brain structures was observed after acute administration of this product.
118	Nicotine	Alkald	**England et al**. [315]: exposure during pregnancy and adolescence may contribute to cognitive and behavioral deficits in later life. Exposure during adolescence is associated with deficits in working memory, attention, and auditory processing, as well as increased impulsivity and anxiety. **Ferrea et al.** [316]: Neuroprotective and neurotoxic effects, modifications of cholinergic transmission. **Ciani et al.** [317]: neurotoxicity on rat habenulo-interpeduncular cholinergic neurons.
119	Nonachlor	Organochlorine Insecticide	**Lee et al.** [318]: chronic exposure to low doses linked to the risk of developing cognitive impairment in elderly. **Kim et al.** [319]: key role in the development of hypertension-related cognitive impairment.
120	Nonylphenol	Formulant	**Litwa et al.** [320]: RXRα, PXR and CAR xenobiotic receptors mediate the apoptotic and neurotoxic actions of the product in mouse hippocampal cells. **Tabassum et al.** [321]: potential risk of cognitive, neurochemical and histopathological perturbations; induced toxicity in frontal cortex and hippocampus of rat brain. **Jie et al.** [322]: inhibited neuronal development and differentiation as indicated by the reduction of the neurotrophic factor GAP-43. **Couderc et al.** [323]: perinatal exposure induced behavioral and neuro-developmental impairments.
121	Norfluoxetine	Pharmaceutical	**Pinna et al.** [324]: selective brain steroidogenic stimulant, reduced post-traumatic stress disorder -like behavior in mice **Pinna et al.** [325]: facilitated GABA(A) receptor neurotransmission and effectively ameliorate emotional and anxiety disorders and depression. **Matsumoto et al.** [326]: non-serotonergic mechanism of action in mood and anxiety disorders. **Pinna et al**. [327]: stereo specifically and selectively increase brain neurosteroid content.
122	Octachlorodibenzo- p-dioxin	Dioxin	**Tawara et al** [328]: fetal growth may be influenced by maternal total exposure to dioxins
123	Octachlorostyrene	Chlorinated Aromatic	**Chu et al.** [329]: 90-day toxicity in the rat: effects on thyroid. **Chu et al.** [330]: long-term toxicity in the rat: effects on thyroid.
124	Octyl-methoxycinnamate	UV filter	**Ruszkiewicz et al.** [79]: neurotoxic effect of active ingredients in sunscreen products. **Axelstad et al**. [331]: effects on auditory and neurological development of rat offspring.
125	Octylphenol	Formulant	**Bianco et al.** [332]: greater accumulation in the cerebral cortex, more accumulation in the cerebellum compared to the mesencephalus and thalamus, with consequences to neural behaviour. **Ghisari et al.** [333]: negative impact on fetal brain development, resulting in cognitive dysfunctions. **Shikimi et al.** [334]: promote Purkinje dendritic growth during neonatal life, may be mediated by estrogen receptor in the Purkinje cell.
126	Oxychlordane	Chloridane Metabolite	**Kim et al.** [335]: role of background exposure in the development of dementia should be explored **Kim et al**. [336]: greater cognitive decline with aging among elders with high serum concentrations **Jain** [337]: total serum thyroxine levels had an inverse association with the product.
127	Parathion	Organo-phosphate Insecticide	**Slotkin et al.** [56]: produced a net increase in norepinephrine emerged over the course of development in brain region. **Liu et al.** [338]: effects on endocannabinoid and endocannabinoid-like lipid metabolites in rat striatum. **Beard et al.** [339]: positively associated with depression in. male private pesticide applicators in the agricultural health study.
128	Pendimethalin	Herbicide	**Lerro et al.** [340]: long-term exposure may alter thyroid function among male pesticide applicators. **Campillo et al.** [341]: Biomarkers indicative of neurotoxicity and physiological stress in caged clams exposed to a contaminated water containing the product. **Pan et al.** [342]: thyroglobulin decreased in rats thyroid cells after exposure.
129	Pentachlorobenzene	Chlorinated Aromatic	**Den Besten et al.** [343]: severe effects on rats thyroid.
130	Pentachloro-nitrobenzene	Herbicide	**Hurley PM** [132]: disrupted thyroid-pituitary homeostasis
131	Pentachlorophenol	Herbicide, fungicide	**Cheng et al.** [344]: affected the timing and coordination of development in the central nervous system. **Krieg** [147]: may act at acetylcholine and γ-aminobutyric acid synapses in the central nervous system. **Roze et al.** [75]: worse coordination, less sensory integrity, worse attention, and worse visuomotor integration. **Jorens etal**. [345]: increased risk for nasal carcinoma
132	Perchlorate	Oxidizer	**Steinmaus et al.** [346]: affected thyroid hormone production during pregnancy and fetal neurodevelopment. **Brent GA** [347]: exposure in pregnancy impacted cognitive outcomes in children **Gilbert et al.** [348]: developmental exposure altered synaptic transmission in hippocampus of the adult rat.
133	Permethrin	Insecticide	**Hossain et al.** [349]: may directly activate microglial cells and may contribute to neurodegeneration. **Nasuti et al.** [350]: decreased levels of dopamine in the striatum, loss of dopaminergic neurons in the substantia nigra pars compacta and cognitive impairments. Motor coordination defects appeared at adult age after early life exposure. **Zakirova et al**. [351]: persistent neuroinflammation, neurobehavioral and neuropathological cognitive impairment in mouse. **Yang et al.** [352]: significant effects on the central nervous system.
134	Perfluorodecane sulfonic acid	Perfluoroalkyl substance	**Ren et al. (2016)** [353]: Binding interactions with thyroid hormone transport proteins and potential toxicological implications.
135	Perfluorohexane sulfonic acid	Perfluoroalkyl substance	**Oulhote et al.** [354]: High serum concentrations at ages 5- and 7-years, but not prenatally, were associated with parent-reported behavioral problems at age 7. **Ren et al.** [353]: Binding interactions with thyroid hormone transport proteins and potential neurotoxicological implications.

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Table 1 (*continued*)

Nb	Endocrine disruptor	Class or use	Mechanisms of nervous disruption
136	Perfluorononanoic acid	Perfluoroalkyl substance	**Jantzen et al.** [355]: males exposed showed a reduction in total distance traveled and time of immobility, and an increase in thigmotaxis behavior, aggressive attacks, and preference for the bright. Acute, embryonic exposure resulted in significant biochemical and behavioral changes in young adult zebrafish. **Lien et al.** [356]: prenatal exposure was found to associate with neurobehavioral symptoms related to attention deficit hyperactivity disorder among Asian seven-year-old children. **Oulhote et al.** [354]: sex-dimorphic associations between concentrations and strengths and difficulties.
137	Perfluorooctanoic acid	Perfluoroalkyl substance	**Jantzen et al.** [355]: embryonic exposure resulted in significant biochemical and behavioral changes in young adult zebrafish. **Oulhote et al.** [354]: significant associations were found in regard to hyperactivity, peer relationship, and conduct problems, as well as internalizing and externalizing problems and autism.
138	Perfluorooctane sulfonate	Perfluoroalkyl substance	**Ge et al.** [357]: could significantly reduce the cell viability and mediate cell apoptosis in HAPI microglia cells of rat. **Jantzen et al.** [355]: embryonic exposure resulted in significant biochemical and behavioral changes in young adult zebrafish. **Oulhote et al.** [354]: significant associations were found in regard to hyperactivity, peer relationship, and conduct problems, as well as internalizing and externalizing problems and autism.
139	Perfluorooctanesulfonyl fluoride	Perfluoroalkyl substance	**Ren et al**. [353]: Binding interactions with thyroid hormone transport proteins and potential neurotoxicological implications.
140	Phorate	Organo-phosphate Insecticide	**Starks et al.** [358]: associated with better verbal learning and memory **Vandana et al.** [359]: obvious effect on cholinesterase enzyme profile of olfactory bulb of mice after systemic administration of low doses for long terms.
141	Picloram	Herbicide	**Reddy et al.** [360]: decreased neuronal branching and degenerating neurons, probably through a mitochondrial pathway.
142	Polyvinylchloride	Polymer; PVC	**Meshchakova et al.** [361]: provoked functional disorders risk connected with cardiovascular and nervous diseases. **Podoll et al.** [362]: acute intoxication resulted in vertigo, nausea and headache up to a narcotic effect. In patients with chronic occupational exposure, neurological disturbances included sensory-motor polyneuropathy, trigeminal sensory neuropathy, slight pyramidal signs and cerebellar and extrapyramidal motor disorders. Psychiatric disturbances present as neurasthenic or depressive syndromes. Sleep disorders and disorders of sexual functions are frequently encountered.
143	8-Prenylnaringenin	Prenylflavonoid	**Urmann et al**. [363]: neurodifferentiating potential of the product. **Bagatin et al.** [364]: panicolytic effects in rats with generalized anxiety and panic disorders. **Oberbauer et al.** [365]: promote neuronal differentiation and neurite outgrowth and are neuroprotective.
144	Procloraz	Fungicide	**Vinggaard et al.** [366]: agonizes the aryl hydrocarbon receptor and inhibits aromatase activity. **Ghisari et al.** [333]: inhibitory effect on rat pituitary cell growth increasing the risk or a negative impact on fetal brain development, resulting in cognitive dysfunctions.
145	Procymidone	Fungicide	**Xiang et al**. [367]: potential to disrupt thyroid homeostasis, agonistic effects.
146	Prodiamine	Herbicide	**Radio et al** [368]: selectively increased neurite outgrowth.
147	Propylthiouracil	Thyroid inhibitor	**Gilbert et al.** [369]: an impaired capacity for hippocampal neurogenesis may contribute to impairments in synaptic plasticity and cognitive deficits **Koromilas et al.** [370]: inhibition of hypothalamic, pontine and cerebellar NaK-ATPase; a major marker of neuronal excitability and metabolic energy production as well as a regulator of important systems of neurotransmission. **Koromilas et al**. [371]: impairs neurochemical mechanisms that could be involved in the way clinical hypothyroidism could affect optimal neurodevelopment and, ultimately, cognitive function. **Vanek et al**. [372]: induced central nervous system vasculitis presenting as confusion.
148	Pyrene	Polycyclic Aromatic Hydrocarbon	**Yang et al**. [58]: Benzo[a]Pyrene (BaP) exposure caused the disruption of glutamate (Glu) neurotransmitter transmission by decreasing the level of Glu, reducing the expression of Glu receptors, enhancing the level of SNAP-25, and neurotoxicity. **Chepelev et al**. [59]: BaP correlates with impaired learning and memory in adults, and poor neurodevelopment in children. Neurotoxic endpoints and DNA damages are more sensitive than cancer endpoints. **Chen et al.** [373]: behavioral impairments resulting from postnatal BaP exposure are potentially long-lasting in rats. **Wormley et al.** [374]: neurobehavioral deficits; gestational exposure to BaP and dioxin reduced specific indices of learning and memory, including hippocampal-based synaptic plasticity mechanisms. **Takeda et al**. [375]: the fetal exposure of mice to diesel exhaust affected the emotional behaviors associated with the serotonergic and dopaminergic systems in the brain
149	Pyrimethanil	Fungicide	**Hurley PM** [132]: disrupt thyroid-pituitary homeostasis only
150	Pyriproxyfen	Juvenile hormone analog	**Truong et al.** [376]: induced craniofacial defects in zebrafish, and adverse behavioral effects. **Fourrier et al.** [377]: changes in social integration, acceptance by nestmates and social behaviors performance in bees.
151	Resorcinol	Disinfectant, Chemical intermediate	**Motonaga et al.** [378]: inhibit thyroid peroxidase to cause developmental toxicity and neurotoxicity. **Román** [28]: transient maternal hypothyroxinemia resulting from dietary and/or environmental exposure to this antithyroid agent.
152	Roundup	Main herbicide worldwide	**Defarge et al.** [20]: its formulants decrease aromatase activity below toxic levels. **Gress et al.** [379]: the product altered locomotor activity in rats. **Modesto et al** [380]: it inhibits acetylcholinesterase in fish brain.
153	Sertraline	Psychotropic	**Lee et al.** [381]: the product is used for trauma-focused psychotherapies **Frölich et al.** [382]: selective serotonin reuptake inhibitor, which has demonstrated efficacy on neuropsychiatric behavioral symptoms in general.

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Table 1 (*continued*)

Nb	Endocrine disruptor	Class or use	Mechanisms of nervous disruption
154	Short chain chlorinated paraffins	Flame retardant; plasticizer	**Liu et al.** [123]: exposure could alter gene expression in the hypothalamic-pituitary-thyroid axis and thyroid hormone levels. **Wyatt et al.** [383]: potent peroxisome proliferators; high dose shows a depressed plasma thyroxine level, with increase in thyroid stimulating hormone
155	2,4,5-T (in Agent Orange)	Herbicide	**Gunderson and Daroff** [384]: epilepsy and later on all effects of brain injury and post-traumatic stress disorders. **St Omer et al.** [385]: together with 2.4D, increased significantly the concentration of norepinephrine in whole developing brain and increased dopamine. **Yi et al.** [144]: increased the prevalence of endocrine disorders, especially in the thyroid and pituitary gland; and increased various neurologic diseases.
156	Tamoxifen	Pharmaceutical	**Denk et al.** [386]: granular neurons of the olfactory bulb and dentate gyrus, vascular cells and ependymal cells throughout the brain, and peripheral sensory neurons are modified by this treatment. **Boele et al.** [387]: Cognitive domains that rely on verbal abilities (verbal memory and fluency) seem to be at risk for deterioration after treatment.
157	Tetrabromo-bisphenol A	Flame retardant	**Park et al.** [388]: induced the loss of both zebrafish neuromasts and hair cells in the rat cochlea in a dose-dependent manner. **Chen et al.** [389]: induced apoptotic cell death, delayed cranial motor neuron development, inhibited primary motor neuron development and loosed muscle fiber during the early development in zebra fish. **Jarema et al.** [390]: may have developmental or pharmacological effects on the vertebrate nervous system. **Wojtowicz et al.** [391]: decreased the expression of PPAR-γ protein in neocortical neurons; and the mechanism of action also induced apoptotic and neurotoxic effects. v
158	testosterone	Natural hormone	**Holmes et al.** [392]: testosterone increased the expression of COX2 and apoptosis in dopamine neurons, increased incidence of Parkinson's disease in men compared with women. **Cunningham et al.** [393]: induces dopaminergic neurotoxicity via caspase-3-dependent activation of protein kinase C-delta.
159	Tetrachloro-dibenzofuran	Chlorinated dioxin	**Xu et al.** [394]: this dioxin-like compound suppresses acetylcholinesterase activity via transcriptional downregulations in vitro.
160	Tetrachloro-dibenzo-p-dioxin	Chlorinated dioxin	**Pelclova et al.** [395]: neurological and neurophysiological findings in workers with chronic intoxication 50 years after exposure. **Xu et al.** [394]: this dioxin suppresses acetylcholinesterase activity via transcriptional downregulations in vitro. **Sánchez-Martín et al.** [396]: aryl hydrocarbon receptor-dependent induction of apoptosis by the product in cerebellar granule cells from mouse.
161	PCB methyl sulfones	PCB metabolite	**Kato et al.** [397]: reduction of thyroid hormone levels by different mechanisms.
162	Tetraiodothyronine	Natural hormone *	**Chen et al.** [398]: inhibition of UDP-glucuronosyltransferases. **Dos Reis-Lunardelli et al.** [399]: can alter animal behavior and learning and memory in rats. **Zamoner et al.** [400]: reorganizes the cytoskeleton of glial cells through Gfap phosphorylation and Rhoa-dependent mechanisms.
163	Thiazopyr	Herbicide	**Hurley PM** [132]: disrupts thyroid-pituitary homeostasis only.
164	Toxaphene	Organo-chlorine Insecticide	**Calciu et al.** [401]: congeners products showed a strong inhibitory effect on the otic system development. **Waritz et al.** [402]: increases the occurrence of two thyroid tumors and increased excretion of thyroid hormones. **Kodavanti et al.** [403]: inhibits calmodulin activated adenylate cyclase in rat brain. **Brunström.** [404]: affected the growth of the chicks and had neurotoxic effects.
165	2,4,6-Tribromophenol	BFR, Natural product	**Leonetti et al.** [405]: accumulate in the placenta and potentially alter thyroid hormone function in a sex-specific manner. **Lee et al.** [406]: thyroid gland activity decreased, disrupted homeostasis and interfered with thyroid hormone system. **Lyubimov et al.** [407]: developmental neurotoxicity and immunotoxicity in rats.
166	Trenbolone	Anabolic steroid	**Quinn et al.** [408]: disrupted development of either the central nervous system or the hypothalamic-pituitary-gonadal axis.
167	Tributyltin	Fungicide	**Ishihara et al.** [409]: induces oxidative neuronal injury. **Frye et al.** [189]: effects through the aryl hydrocarbon receptor, the peroxisome proliferator-activated receptor and the retinoid X receptor, signal transduction pathways, calcium influx and/or neurotransmitter receptor. **Kotake** [410]: neurotoxic, induces behavioral abnormalities and toxic to the developing central nervous system through AMPA receptor subunit.
168	Trichloroethylene	Chlorinated solvent	**Yeung** [411]: neurotoxicity inducing anxiety in man. **Da Broi et al.** [412]: produces pleasant inebriating effects with rapid dissipation, followed by central nervous system depression, coma. **Kang et al.** [413]: provokes chronic central nervous system disorders and peripheral neuropathy. **Chiu WA et al.** [414]: carcinogenic to humans by all routes of exposure and toxic to the central nervous system. **Bale et al.** [415]: interacts directly with several different classes of neuronal receptors by generally inhibiting excitatory ions receptors/channels and potentiating the function of inhibitory receptors/channels.
169	Trichlorophenol	Fungicide	**Xu et al** [416]: urinary levels increased risk of attention deficit hyperactivity disorder among school-aged children.
170	Triclocarban	Antibacterial agent	**Dong et al.** [417]: altered expression of proteins involved in nervous system development. **Barros et al.** [418]: modified chronically female amphipod Gammarus behavior. **Wu et al.** [419]: inhibited iodide uptake, but had differential effects on the expression of thyroid hormone synthesis-related genes and the activity of thyroid peroxidase.

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Table 1 (*continued*)

Nb	Endocrine disruptor	Class or use	Mechanisms of nervous disruption
171	Triclosan	Antibacterial agent	**Ruszkiewicz et al.** [79]: few neurotoxic effects. **Szychowski et al.** [420]: activates aryl hydrocarbon receptor-dependent apoptosis and affects Cyp1a1 and Cyp1b1 expression in mouse neocortical neurons. **Wu et al.** [419]: inhibited iodide uptake, but had differential effects on the expression of thyroid hormone synthesis-related genes and the activity of thyroid peroxidase.
172	Tri-iodothyronine	Natural thyroid hormone	**Noda** [421]: impairs glial function as well as neuronal function and thus disturb the brain, which may cause mental disorders. **Zhang et al.** [422]: affects feeding behavior. **Ortiga-Carvalho et al.** [423]: regulates by negative feedback the hypothalamus pituitary-thyroid axis.
173	Triphenyl phosphate	Flame retardant	**Sun et al.** [424]: Developmental neurotoxicity in early life stages of Japanese medaka. **Kim et al.** [425]: up-regulated the expression of the genes related to the metabolism, transport, and elimination of thyroid hormones. **Jarema et al**. [390]: produced behavioral effects; may have developmental or pharmacological effects on the vertebrate nervous system. **Tanaka et al.** [426]: induced shrinkage and increased packing densities, axonal and terminal degenerations in the developing visual system of the ferret.
174	Triphenyltin	Fungicide	**Kotake** [410]: potent inhibitor of mitochondrial ATP synthase, neurotoxic and induces behavioral abnormality. **Karpiak et al.** [427]: disrupts components of glutamate homeostasis in rat astrocyte cultures. **Lin et al.** [428]: triphenyltin acetate may cause cellular dysfunction of brain without structural damage and provoked demyelinated neuropathy. **Attahiru et al**. [429]: produced significant central nervous system and respiratory depressions and produced brain congestion. **Lehotzky et al**. [430]: crosses the blood-brain barrier, and induces diminished plasticity of the central nervous system.
175	Venlafaxine	Psychotropic	**Belovicova et al.** [431]: stressed rat dams had lowered hippocampal neurogenesis, while the product treatment reversed this lowering. **Singh et al.** [432]: induced ROS-mediated apoptotic neurodegeneration in fetal neocortex, and neurobehavioral sequelae in rat offspring. **Pinzani et al.** [433]: it is an antidepressant that selectively inhibits serotonin reuptake and is a norepinephrine inhibitor.
176	Vinclozolin	Fungicide	**Gillette et al.** [434]: Debilitating effects were seen at all levels of the phenotype, including physiology, behavior, brain metabolism, gene expression, and genome-wide transcriptome modifications in specific brain nuclei, and transgenerational effects. **León-Olea et al.** [13]: can lead to severe and widespread neuroendocrine disruptions in discrete brain regions, including the hippocampus, amygdala, and hypothalamus, resulting in behavioral changes in a wide range of species. **Skinner et al.** [435]: Transgenerational epigenetic programming of the brain transcriptome and anxiety behavior are modified by the product. **André et al**. [436]: learning deficits are expressed following perinatal exposure.
177	Zearalenone	Mycotoxin	**Makowska et al.** [437]: affects calcitonin gene related peptide-like immunoreactive neurons in the pig enteric nervous system. **Obremski et al.** [438]: induced changes in the lymphoid tissue and mucosal nerve fibers in the porcine ileum. **Venkataramana et al.** [439]: showed a marked suppressive effect on the neuronal gene expression; oxidative stress is the main upstream signal leading to increased neurotoxicity due to the product.

Each chemical compound or pollutant has been numbered (Nb) out of 177 known endocrine disruptors; its name was associated with the key word "nervous" or "neurotoxicity" or "cognitive" or "behavio(u)r" on PubMed data bank, or eventually on Google Scholar. When the number of references per compound were too numerous, "or" was excluded in order to directly associate the keywords. If more than 20 references were found to be published, "review" was added to the keywords and cited as a reference. Finally, a maximum of five references were indicated, focusing on the most recent research in humans or mammals, without excluding other models. The mechanisms of nervous disruption could be direct, on the neurons or the nervous system, or indirect, through endocrine disruption interfering with neurodevelopment or nervous system functioning, including thyroid regulation. PAH, polycyclic aromatic hydrocarbon; PCB, polychlorobiphenyl; PBB, polybromobiphenyl; PBDE, polybrominated diphenyl ether; PFAS, perfluoroalkyl substances.

Declaration of Competing Interest

The authors Seralini & Jungers declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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