ENVIRONMENTAL TOXINS AND NEURODEGENERATIVE DISEASE

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What follows are tables showing the principal sources and neurodegenerative effects of some of the main toxins, together with their references. There are many more studies than I have shown here.

I have tried to show human studies where possible, covering the development, progression or mortality of Alzheimer's disease (AD), Parkinson's disease (PD), amyotrophic lateral sclerosis (ALS, better known as motor neurone disease) and multiple sclerosis (MS) and their association with air pollution (indoor and outdoor), persistent organic pollutants (POPs), pesticides, foods and their contaminants, toxic metals, ionising and non-ionising radiation.

The handout also covers how to avoid toxins where possible, how to protect yourself from them where possible, testing for toxins and how to remove them.

OUTDOOR AIR POLLUTION

External air pollution is a complex mixture of multiple pollutants, which may be in the form of gases or particles, the concentration of which will depend upon emission sources, weather (temperature, wind speed and direction, precipitation) and land patterns.

Principal sources of outdoor air pollution

Natural	
Radon	Radioactive gas from radium/uranium decay. Problem in Cornwall.
Vehicle emissions:	
Ozone	Formed through reaction of solar radiation and other pollutants
Nitrogen oxides	Principally nitrogen dioxide (NO2)
Carbon monoxide	Produced by incomplete combustion of carbonaceous fuels (diesel)
Particulate matter (PM)	Solid or liquid particles suspended in air, particularly from diesel emissions. Usually PM 10; PM 2.5; PM 0.1
VOCs	Hydrocarbons, formaldehyde and methanol
Industrial emissions	Smelters, foundries, chemical manufacturers, power stations
Vehicle emissions +	
Sulphur dioxide	Mainly emitted by coal combustion
Carbon dioxide	Produced by combustion of carbonaceous fuels
Toxic metals	Especially mercury and lead
Landfill sites	
Carbon dioxide	Formed by breakdown of organic matter
Hydrogen sulphide	Formed by breakdown of organic matter
Methane	Formed by breakdown of organic matter
Ammonia	
Volatile organic compounds (VOCs)	Tri- and tetra-chloroethylene, polycyclic aromatic hydrocarbons (PAHs), chlorinated hydrocarbons
Incinerators	
As industrial emissions, plus: hydrogen chloride polychlorinated biphenyls (PCBs), dioxins, furans, PAHs	Formed from burning of plastics. Forms acid on contact with H2O.

The following page lists some of the studies showing that outdoor air pollution is associated with or can cause neurodegenerative disease. You should be aware that there are also studies showing that these toxins are not associated with any adverse effect on health.

Toxin	Alzheimer's disease (AD)	Parkinson's disease (PD)	ALS	MS
Radon	Radon metabolites found in brains of AD patients[1]. Radon background radiation correlated with AD death rate[2].	Radon metabolites found in brains of PD patients[10].		
Diesel exhaust (DE)	Occupational exposure associated with higher AD risk[3]. Inhalation causes plaque formation and impaired motor function in mouse brain[4], with activation of Aβ 42[5].	In vitro study found raised intracellular dopamine levels, suggesting dopamine accumulation[11], with damaged dopaminergic neurons[12].	Truck drivers, with high exhaust exposure, at higher risk for ALS[21].	
Particulate matter (PM)	Young Mexico City residents high brain PM with inflammation, Aβ accumulation, cognitive dysfunction and other AD markers[6]. PM10 and PM 2.5 particularly associated with AD risk[7],[8].	Young Mexico City residents high brain PM, hyper-phosphorylated α-synuclein and early PD diagnosis[13]. PM 2.5 and 10 associated with increased PD risk[14],[15].		PM10 associated with increased MS risk[23] and can trigger relapses[24].
Nitrogen dioxide (NO2)		Exposure associated with increased PD risk[16] and increased PD aggravation[17].		NO2 exposure associated with increased risk[25].
Nitric oxide (NO)				Increased MS risk[26].
Sulphur dioxide (SO2)		Exposure associated with increased PD aggravation[18].		
Carbon monoxide (CO)		CO exposure associated with increased PD aggravation[19].		Associated with increased MS risk[27].
Ozone (O3)	O3 ROS produce changes in Aβ peptide folding, associated with increase in Aβ 42[9].			O3 associated with MS relapse[28].
Hydro-carbons (HC)		Meta-analysis found increased PD risk with HC exposure[20].	Exposure associated with >50% higher ALS risk[22].	

Key: ALS = Amyotrophic laterals sclerosis; MS = Multiple sclerosis; Aβ = Amyloid β; ROS = Reactive oxygen species

INDOOR AIR POLLUTION

Principal sources of indoor air pollution

Fossil fuel combustion	Carbon monoxide; sulphur dioxide; nitrogen oxides; radon, hydrogen chloride (HCl), particulates (PM); ozone; volatile organic compounds
Off-gassing of new building materials, carpets, furniture and furnishings	Formaldehyde, pesticides, brominated flame retardants and other volatile organic compounds (VOCs)
Home office equipment and supplies	VOCs: formaldehyde, benzene
Dry cleaning	Tetrachloroethylene
Mothballs, air freshener	Dichlorobenzene, naphthalene
Cleaning agents, perfume	Benzene, acetaldehydes
Tobacco smoke	Particulate matter, toxic metals, toxic gases, VOCs, nitrosamines
Dust	Toxic metals, pesticides, asbestos, fibre glass

The following pages list some of the studies showing that toxic metals are associated with or can cause neurodegenerative disease. You should be aware that there are also studies showing that these metals are not associated with any adverse effect on health.

Toxin	Alzheimer's disease (AD)	Parkinson's disease (PD)	Amyotrophic lateral sclerosis	Multiple sclerosis
Smoking	2015 meta-analysis: current smokers had a dose-dependent increased risk for dementia and AD[29].	Two meta-analyses: inverse association with PD risk[33],[34].	2011 pooled analysis: smoking confers >40% increased risk[39].	Meta-analysis: smoking confers >30% increased risk[41].
Form-aldehyde (FA)	Elevated FA is associated with impaired memory, amyloid plaques, tau protein and neuronal loss[30].	Increased FA formation in rat brain associated with decreased tyrosine hydroxylase[35].	Occupational exposure associated with increased ALS prevalence[40].	
Acet-aldehyde	Acetaldehyde associated with AD incidence[31].	Acetaldehyde associated with PD incidence[36].		
Toluene	Patients had history of exposure[32].	Patients had higher exposure[37].		
Trichloro-ethylene (TCE)		Occupational exposure is a risk factor for PD[38].		

PERSISTENT ORGANIC POLLUTANTS (POPs)

POPs are resistant to environmental degradation, taking up to 100 years to biodegrade. They bioaccumulate in the environment and in fatty tissue. Most POPs are semi-volatile i.e. may travel thousands of miles before depositing on the earth or water. They comprise some pesticides, solvents, industrial chemicals, metals and pharmaceuticals. Some POPs are also volatile organic compounds (VOCs).

The UN Binding Convention on Persistent Organic Pollutants in 2001 banned 11 POPs: www.irptc.unep.ch/pops/default.html. Many other remain in use.

Principal sources of some of the main POPs

Polychlorinated biphenyls (PCBs)	Used in insulation; found in much old electrical equipment, office products and pesticides. Found in meat, fish, dairy, cord blood and breast milk.
Phthalates	Used in any plastics: cling film and other food packaging and manufacture of cosmetics, pharmaceuticals, toiletries and pesticides. Found in air, food, water.
Bisphenol A	Used in plastics and epoxy resins: plastic water bottles, sports equipment, CDs and DVDs, lining water pipes, coating on the inside of cans and thermal paper.
Perfluoroalkyl substances	Found in drinking water, non-stick cookware, water-repellent clothing, stain resistant fabrics, cosmetics, firefighting foams, and products that resist grease, water and oil.
Polybrominated diphenyl ethers (PBDEs)	Flame retardants found in furnishings.

Following are some of the studies showing that POPs are associated with or can cause neurodegenerative disease. You should be aware that there are also studies showing that these toxins are not associated with any adverse effect on health.

POP	Alzheimer's disease	Parkinson's disease	ALS	MS
Perfluoroalkyl substances (PFAS)	Associated with AD mortality[42].	PFAS in drinking water associated with PD mortality in females only[44].		
Polychlorinated biphenyl (PCBs)		Higher PCBs in PD brains, particularly in females[45].	Increased ALS risk[47].	
Polybrominated diphenyl ether (PBDEs)			A PBDE associated with increased ALS risk[48].	
Bisphenol A (BPA)	Aβ42 and tau increased[43].	PD patients had lower BPA conjugation[46].		Higher rat MS risk[49]
Painters' solvents				Doubled risk[50]

PESTICIDES

Pesticide exposure can occur through ingestion of contaminated foods or drinking water, inhalation (including from showering or bathing) or skin contact. Indoor exposure (domestic spraying, soft furnishings, floors) is often underestimated, as is exposure in aircraft. Virtually all public buildings and spaces now have some form of pest control.

Regulation

2009 EU pesticide legislation banned certain pesticides from use in Europe; this is the strictest legislation in the world. Similarly, the EU Drinking Water Directive has a general limit for all pesticides of 0.1mcg/l, with the sum of all pesticides being below 0.5mcg/l; however, few pesticides are included in the routine monitoring of drinking water in the UK.

Principal classes of pesticides

Organochlorine insecticides	The oldest, and generally the most toxic, pesticides. Most of them are POPs and have been banned.
Organophosphate insecticides	Slightly less toxic than organochlorines. Includes malathion, dursban, diazinon, trichlorofon, parathion, chlorpyrifos, malathion and mevinphos.
Carbamate insecticides	Similar to organophosphates. Includes aldicarb, carbaryl, methomyl, propoxur, thiophanate methyl and carbofuran (N-methyl carbamate).
Pyrethroid insecticides	The newest class, based on pyrethrin from chrysanthemums. Includes allethrin, cismethrin, fenvalerate and remethrin.
Herbicides	Some (e.g. paraquat) banned in the EU. Most common: Glyphosate, developed by Monsanto as RoundUp, designed for use with GM crops.
Fungicides	Widely used in agriculture/supermarkets. Mancozeb, maneb, tributyltin.

The following pages list some of the studies showing that pesticides are associated with or can cause neurodegenerative disease. You should be aware that there are also studies showing that these toxins are not associated with any adverse effect on health.

Pesticide	Alzheimer's disease	Parkinson's disease	ALS
Various	Meta-analysis showed exposure associated with AD risk[51].	Occupational exposure associated with increased risk of PD[58],[59]. Meta-analysis found a positive association between PD and any insecticide or herbicide use[60].	Meta-analyses: exposure associated with ALS[66],[67].
Organo-chlorines (OCs)	Serum and brain DDT, DDE and others higher in AD[52],[53]. OCs increased AβPP levels and impaired degradation of Aβ peptides[54],[55].	Paraquat exposure associated with PD incidence[61]. Paraquat caused a 30% loss of dopaminergic neurons, reduced striatal tyrosine hydroxylase immunoreactivity and induced α-synuclein accumulation in the substantia nigra[62].	Occupational or residential exposure to cis-chlordane associated with ALS risk[68].
Organo-phosphates (OPs)	OPs affect metabolism of Aβ[56], with tau hyperphos-phorylation[57].	Frequent use of any household pesticide increased the odds of PD by 47%; frequent use of products containing OPs increased the risk by 71%[63].	
Pyrethroids		Long term intake of pyrethroids: decreased dopamine uptake and increased apoptosis[64].	
Carbamates		Frequent domestic use of carbamates increased the risk by 455%[65].	

Key: Aβ = Amyloid β; AβPP = Aβ precursor protein;

FOODS, FOOD CONTAMINANTS AND ADDITIVES

Principal toxic foods, food contaminants and additives

Microorganisms	Infected animals; poor food handling and storage
Drug use in animals	Antibiotics and growth hormone; sheep dip. Chemicals remain in meat.
Toxic metals	Mercury, arsenic: fish; contaminated soil or feed or aerosol deposit
Pesticides, fertiliser	Non-organic fruits and vegetables (contain PCBs, dioxins, furans, chlorinated hydrocarbons). Also stored in animal fat.
Heterocyclic aromatic amines	From browned or burned meat
Nitrosamines	Processed foods
Acrylamide	Starchy foods cooked at high temperatures
Bisphenol-A (BPA)	Metal can liners
PCBs	Very high levels in fish
Food additives	For colour, taste and as a preservative
Trans fats	Processed foods, heated polyunsaturated oils
Sugar and artificial sweeteners	Processed foods and beverages

The following pages list some of the studies showing that certain foods and food contaminants are associated with or can cause neurodegenerative disease. You should be aware that there are also studies showing that these toxins are not associated with any adverse effect on health.

Toxin	**Alzheimer's disease**	**ALS**
Trans fats (TF)	A prospective study showed increased AD risk with higher TF intake[69]. TFs increase amyloidogenic processing of AβPP, with increased incidence and aggregation of Aβ peptides[70].	Higher intake of TF associated with increased ALS risk[75].
Nitros-amines	Induces AD-type neuro-degeneration in rats via oxidative stress and DNA damage[71],[72].	
Artificial sweeteners	Higher cumulative intake of artificially sweetened soft drinks associated with increased risk of AD[73].	
Sugars	Higher intake of sugary drinks, including fruit juice, associated with lower brain volume and poorer memory, both markers of preclinical AD[74].	

Key: ALS = Amyotrophic laterals sclerosis; Aβ = Amyloid β; AβPP = Aβ precursor protein

TOXIC METALS

Principal sources of toxic metals

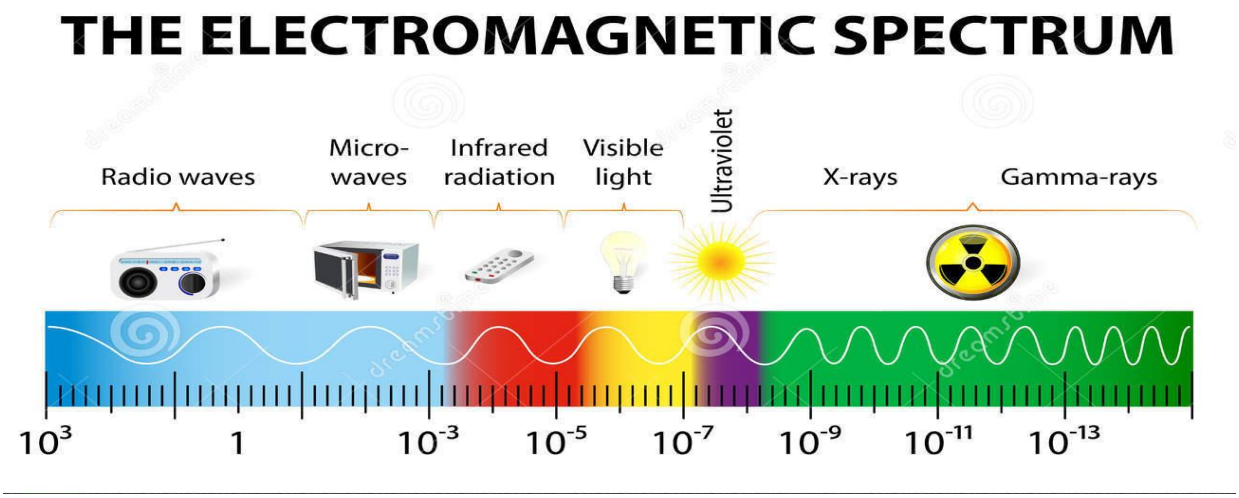
Non-organic foods and food cans/linings	Aluminium, arsenic, cadmium, chromium, copper, iron, lead, mercury, nickel, palladium, silver, titanium
Infant formula/food	Aluminium, arsenic, cadmium, lead, manganese, mercury
Cookware	Aluminium
Drinking water	Aluminium, arsenic, cadmium, chromium, copper, iron, lead, manganese, mercury, palladium, uranium
Pharmaceuticals	Aluminium, copper, mercury, palladium, titanium
Cosmetics/toothpaste	Aluminium, arsenic, cadmium, chromium, lead, mercury, nickel, silver, titanium
Toiletries	Aluminium, arsenic, nickel
Vaccines	Aluminium as adjuvant, mercury (as thimerosal)
Dental materials	Cadmium, chromium, copper, mercury, nickel, palladium
External/internal air	Aluminium, arsenic, cadmium, iron, lead, mercury, nickel, silver, titanium, uranium

The following pages list some of the studies showing that toxic metals are associated with or can cause neurodegenerative disease. You should be aware that there are also studies showing that these metals are not associated with any adverse effect on health.

Metal	Alzheimer's disease	Parkinson's disease	ALS	MS
Aluminium (Al)	Higher Al in UK drinking water correlated with increased AD incidence[76]. Meta-analysis 2018: Al elevated in AD patients[77]. Autopsy study: Al deposits corresponded with senile plaque density, Aβ42 pleated sheets, fibrillar amyloid deposits, neurofibrillary tangles[78],[79].	Al found in substantia nigra, induces aggregated α-synuclein in dopaminergic neurons[108],[109].	Patients had higher levels of CSF Al[126]. In animals: motor neurone degeneration and loss, denervation in spinal cord[127].	Increased urinary Al excretion[143]; decreased CNS Mg and Zn, esp in white matter[144].
Arsenic (As)	Autopsy study: As levels in brain ventricular fluid in AD patients significantly higher[80]. Serum As correlated with cognitive function[81].	Higher CSF As levels in patients[110]. As induces elevated α-synuclein[111].	Decreased neuro-filament content in ALS folate deficiency[128].	Blood As levels higher in MS patients; correlated with oxidised lipids[145].
Cadmium (Cd)	Meta-analysis: raised Cd levels in AD patients[82]. High blood Cd levels associated with increased risk of AD mortality[83]. Cd increases Aβ peptide and phosphorylation of tau protein[84] and can induce tau filament formation[85].	Cd induces neurotoxicity in α-synuclein and apoptosis via oxidative stress[112].	Serum and CSF Cd levels higher in ALS patients[129],[130].	MS patients had higher blood Cd[146],[147].
Cobalt (Co)	Co is cytotoxic, can induce oxidative stress and increase secretion of Aβ 1-40 and 1-42 in neuroblastoma cells[86].		Higher levels of Co in ALS CSF[131].	

Copper (Cu)	2 x meta-analyses: higher levels of serum Cu in AD patients[87],[88]. Cu-induced ROS lead to AD brain inflammation[89],[90].	Occupational Cu exposure associated with PD incidence[113].	Higher Cu in CSF of patients[132].	Increased MS with higher soil levels[148].
Iron (Fe)	In AD postmortem, cortex levels of Fe higher[91]. Fe accumulates in plaques, microglia and on myelinated fibres[92]. High CFS ferritin found with accelerated break-down of $A\beta_{42}$ and plaque formation[93].	Fe stored in the substantia nigra[114], neuron damage and lower dopamine production[115].	Serum ferritin elevated[133], transferrin reduced, suggesting disrupted Fe homeostasis[134].	
Lead (Pb)	Bone Pb levels predict poor cognitive function and smaller brain volume[94]. In animals, exposure upregulated APP and Aβ[95],[96] down-regulated DNA methylation[97], increased DNA oxidative damage and induced histone modification[98],[99]. .	Higher atmospheric Pb associated with higher PD mortality in elderly[116]. Higher bone Pb associated with PD incidence[117].	2014 meta-analysis: doubled risk of ALS with occupational exposure[135]. Higher blood and CSF Pb with higher PD rates[136].	
Manganese (Mn)	2018 meta-analysis showed elevated serum Hg in AD patients[100]; blood and CSF Hg correlated with Aβ in AD patients[101].	>20 years Mn exposure associated with PD incidence[118]. Induction of protein fibrils in α-synuclein[119].	Mn CSF levels higher in ALS patients[137].	
Mercury (Hg)	Autopsy studies found increased Hg in brain tissues of AD patients[102]. Subjects with dental amalgams had higher risk of AD[103].	Association of blood/ urinary Hg and PD[120]. Faster induction of fibrils in α-synuclein[121].	ALS patients had higher toenail Hg and CSF[138],[139]. Higher Hg in neurons in patients[140].	MS patients had higher serum and hair Hg[149],[150].
Selenium (Se)	Increased Se found in the brain of AD patients[104], as well as higher serum Se[105]. Higher CSF Se was associated with later development of AD[106].	Higher serum + CSF Se in patients[122],[123]Increased Se in brain of patients[124].	Higher CSF Se in ALS patients[141].	
Zinc (Zn)	In mouse hippocampus Zn concentrations were increased in Aβ plaques[107].	Faster growth of α-synuclein fibrils[125].	Higher levels of Zn in ALS CSF[142].	

Key: ALS = Amyotrophic laterals sclerosis; MS = Multiple sclerosis; Aβ = Amyloid β; AβPP = Aβ precursor protein; ROS = Reactive oxygen species; CSF = Cerebral spinal fluid



The electromagnetic (EM) spectrum is divided into ionising and non-ionising radiation, depending upon the extent of the energy carried. The threshold between ionising and non-ionising radiation is located roughly at the upper end of the UV-band, i.e. above 300GHz.

- Ionising radiation comprises high energy particles (photons) which have sufficient energy to break chemical bonds and form ion pairs from the matter through which they pass. This results in removing a negatively charged electron from an atom or molecule, leaving the atom or molecule positively charged.
- Non-ionising radiation energy is too weak to break chemical bonds.

Ionising radiation: sources

- Cosmic gamma rays from sun and space:
- Natural terrestrial radiation: radium and radon
- Medical and dental diagnostics and scientific research (X-rays, CT scans etc)
- Medical therapeutics: Radiation (X-ray) therapy for certain cancers
- Radioactive minerals used in building materials, phosphate fertilisers and fuels.
- Screening of luggage and passengers at port and airports
- Components of TV sets, video display terminals, smoke detectors, luminous watches etc
- Irradiation of food to extend shelf life or destroy disease-causing organisms
- Nuclear power production and nuclear waste
- Occupational exposure among workers in medical and industrial radiography, nuclear reactor construction and operation, uranium mining and enrichment

Non-ionising radiation: sources

- <u>Microwave (MW) and radiofrequency radiation (RFR):</u> Radio and TV transmitters, satellite telecommunications systems, microwave ovens, mobile phones, mobile phone masts, radar
- <u>Extremely low frequency (ELF) magnetic fields:</u> Solar activity and thunderstorms, electric power production, transmission, distribution and use (i.e. power lines, electrical wiring, transformers, motors, household appliances, video display terminals and medical devices including magnetic resonance imaging (MRI) scanners.

Electromagnetic fields (EMFs) are made up of both electrical and magnetic fields. At higher frequencies the electrical and magnetic fields are inseparable but at extremely low frequencies

(ELFs), electrical and magnetic frequencies are independent and measured separately. Both fields are produced whenever current is flowing through a wire. But when a device is merely plugged in but not switched on, it is still generating an electric field. Most obstacles such as walls and fences can shield us from electrical fields but nothing can shield us from magnetic fields, which can penetrate anything except lead.

The following pages list some of the studies showing that radiation is associated with or can cause neurodegenerative disease. You should be aware that there are also studies showing that radiation is not associated with any adverse effect on health.

Ionising radiation: neurodegenerative effects

Radiation source	**Alzheimer's disease**	**Parkinson's disease**	**MS**
Cosmic radiation	Induces cognitive impairment and increased Aβ plaque accumulation in mice[151]		
Ultra-violet radiation (UVB)		Relationship with PD incidence, modified by age[155].	Positive correlation with MS incidence[157],[158].
Radon	Associated with AD mortality[152].		
X-rays	Ionising radiation reduces ADAM10, an inhibitor of Aβ protein production[153] and increases tau phosphorylation in neurons[154].		Higher X-ray history associated with MS incidence[159].
Radiation therapy		Upregulates α-synuclein[156].	

Non-ionising radiation: neurodegenerative effects

Radiation source	**Alzheimer's disease**	**Parkinson's disease**	**ALS**
LF-EMF	Increases Aβ peptide secretion[160].		
ELF-EMF	Occupational exposure associated with increased AD mortality among males[161]. Higher dementia risk with occupational exposure[162].	50% higher PD mortality risk[165].	Occupational exposure associated with increased ALS incidence[166] and mortality[167].
900 MHz	Rats showed impaired object exploration and alteration in brain mono-amine content[163].		
Pulsed EMFs (simulating a mobile phone)	Cognitive and memory impairment in rats, with increased Aβ production[164].		

Combinations of toxins

After years of testing just one chemical at a time, scientists are now looking at two at a time. This is a small step forward….

But 2 chemicals can have an effect that is either additive or antagonistic.

Animal studies have shown:

- Synergistic effect of ultrafine PM and a pesticide to activate NADPH oxidase in microglia, leading to oxidative damage to dopaminergic neurons[168].
- PM2.5 and formaldehyde had no effect in inducing AD-like brain changes but the combination induced pathogenic changes[169].
- Single pesticide (paraquat and maneb) exposure had no effect on α-synuclein but the combination significantly increased pathology, including mitochondrial degeneration[170].
- Toxic metal increases the effect of herbicides on α-synuclein[171].
- Neurodevelopmental problems with exposure in infancy to combined mercury and gamma-radiation, whereas single exposure caused no problems[172].
- Lead and chronic stress altered adrenal function and brain structures, which was not seen in lead exposure alone[173].

A human study found that proximity to WiFi enhanced mercury release from amalgam fillings[174].

Some common mechanisms

- Generation of ROS/RNS: oxidative stress and low endogenous antioxidants
- Brain inflammation
- Heating effect (for ionising radiation)
- Endoplasmic reticulum stress from elevated intracellular calcium
- Brain insulin resistance (more on this coming up!)

In addition, the toxins may act indirectly by binding to and interfering with cell membranes, so preventing the normal functioning of the cell and its signalling, or binding to DNA as adducts, where they inhibit normal functioning.

There may also be gene-toxin interactions, making some more susceptible to particular toxins than others.

PROTECTING YOURSELF FROM TOXIN DAMAGE

- Avoidance is the key, so find out where the key toxins may impact your life and take steps to avoid them.

- Around the home, it is worth doing the following:
    - Install a water filter, preferably 'whole house' but certainly for drinking water.
    - Especially with children and dogs, remove carpets and lay wood which has had time to 'off-gas' to remove treatment chemical fumes.
    - Eat only organic food and a nutrient-dense diet with minimal processed foods. Forget '5-a-day'; we all need at least '7-a-day'.
    - Avoid trans fats, sugars and artificial sweeteners
    - Do not use a microwave oven (it denatures proteins)
    - Never cook in metal or plastic; use glass where possible: Corning Ware and Visions are good companies.
    - Toiletries, cosmetics and cleaning materials: use brands with the minimum of chemicals (Ecover and Green People are widely available). Do not use perfume or any toiletries that are fragranced.
    - When redecorating, use paints from companies such as Lakeland Paints, to minimise the VOCs.
    - Open windows whenever possible to disperse VOCs.
    - Try to put up with pests instead of using commercial pesticides in home or garden.
    - Remove outdoor shoes before coming indoors, to avoid bringing in dirt from the road or garden.
    - Use wired broadband instead of WiFi; protect bedrooms with anti-radiation paint.
    - Find a mercury-free dentist and don't accept any metal in the mouth.
    - Avoid vaccinations unless absolutely necessary.

- But no-one can protect themselves from all toxins, all the time. Your biochemical status is very important, as this determines whether the toxins will affect you or will pass out of the body without causing too much damage.
    - Ensure that you are adequately methylating DNA and histones (most people are not, and need vitamins B6, B12 and folate); diet may not provide adequate levels
    - Ensure good antioxidant status – but note that you need pro-oxidants as well, so it is the balance which is crucial. Zinc therapy can reduce cognitive decline[175].
    - Ensure excellent detoxification status.
    - Optimise the microbiome as this resets brain gene expression[176]; treat intestinal permeability.
    - Ensure all organs of elimination are open.
    - Fasting to remove lipophilic toxins from adipose tissue and stimulate autophagy to clear amyloid protein in the brain.
    - Ensure sufficient good, sound sleep – the brain uses this time to remove toxins and debris through activation of the glymphatic system. Anaesthesia does an equally good job![177]
    - The glymphatic system uses the cells' mitochondria to remove cellular waste from the brain in CSF (cf: lymph). So ensure mitochondrial function is optimized as well.[178]
    - Fasting helps remove all lipophilic toxins from adipose tissue. Fasting for at least 3 days allows autophagy to take place – this has also been shown to remove Aβ fragments from the brain.

TESTING FOR TOXINS

A detailed exposure history will give the best clue (diet, occupation, residence etc).

For metals (*Biolab or Genova*):

Blood test: only recent exposure
Urine test: only recent exposure
Hair analysis: previous 2-3 months' exposure
Urine challenge test with DMSA chelating agent: flushes metals out of tissues. No prescription necessary for DMSA but use with extreme caution.

Urine tests from Great Plains (*through Biolab*)

Non-metal chemical profile: 172 toxins
Glyphosate
Mycotox profile: 7 mycotoxins from 4 mould species

Fat biopsy for lipophilic toxins

Blood brain barrier permeability: https://www.cyrexlabs.com/

TOXIN REMOVAL FROM THE BRAIN

Note that removal of toxins may not make the patient any better, but generally patients can never recover until the toxins are removed.

It's hardly worth getting a toxin out of the body unless the avoidance measures are in place.

Ensure that all the usual detoxification procedures are under way, plus:

- Get into ketosis (fasting or ketogenic diet), as the brain mitochondria prefer ketones as a fuel. Mild ketosis has also been found to induce production of BDNF.
- Adequate methylation is vital for the brain. If unsure, test homocysteine and/or S-adenosyl methionine.
- Ensure adequate DHEA levels. DHEA enhances neuroplasticity and protects the brain from inflammation and free radical damage. If unsure, test salivary adrenal hormones.

Useful foods/supplements, plus:

- Green tea extract (EGCG): protects against neurodegenerative disease[179] and can chelate iron from neuroblastoma cells as effectively as desferrioxamine[180].
- Magnesium aids brain fibrinolytic degradation[181].
- Curcumin protects against pesticide-induced oxidative damage in the hippocampus[182].
- Resveratrol protects against formaldehyde-induced hyperphosphorylation of tau protein[183].
- Parkinson's: fish intake [also smoking and alcohol consumption] protective against multiple toxins[184]; ω3 intake is protective against pesticides[185].

Liposomal detoxification agents

- Liposomes are able to cross the BBB: liposomal anti-cancer agents have been used effectively to treat cancer of glioma cells.
- Have a look at the website of www.quicksilverscientific.com which make a liposomal EDTA for removing metals from the brain.
- Ensure there are sufficient toxin binders in the diet and organs of elimination are functioning well.

USEFUL WEBSITES AND OTHER RESOURCES

- Society for Neuroscience www.sfn.org
- www.healthy-house.co.uk for all kinds of useful products
- www.emfields.com for radiation information and testing
- www.freshwaterfilter.com for whole house or drinking water filters.

- If you want to buy just one book, buy 'Living Dangerously' by Pat Thomas, available on Amazon.

- Biolab and Genova have a lot of useful information about toxins on their websites.

Dale Bredesen papers referred to in presentation:

Bredesen 2015[186]
Bredesen 2014[187]
Mehlen 2010[188]
Lourenco 2009[189]

Anti-microbial and anti-metal amyloid papers referred to in presentation:

Van Rensburg 1997[190]
Telling 2017[191]
Everett 2014[192]
Kumar 2016[193]
Soscia 2010[194]
Luna 2013[195]
Shah 2006[196]
Giuffrida 2009[197]
Grant 2012[198]
Wang 2014[199]

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