Your Brain on Plastics

by Bennett Andrassy

*"Beach strewn with plastic debris" by U.S. Fish and Wildlife Service Headquarters. 2009. Wikimedia-**commons.**.*

You almost certainly have plastic in your brain. In 2018, human beings produced about 380 million tons of plastic [1]. The global plastic recycling rate hovers around 9%, while only 5% of plastic products are recycled in America [2]. That leaves over 300 million tons of annual plastic waste to be distributed across… well, everywhere. Humans have a few main strategies for disposing of plastic waste. Predictably, none of these strategies are particularly well thought-out; they address only the most superficial element of waste disposal, which is to just put the waste somewhere else. Somewhere around 80% of annual plastic waste is put in landfills. The remainder is either incinerated, or mysteriously ends up in Earth's oceans and waterways [1].

These latter two practices cause huge quantities of tiny little plastic particles to be released throughout the air and water of our planet. As every known life form on Earth lives in either air or water, these tiny plastics enter the bodies of a huge variety of animals, and ultimately cause damage to their nervous systems [3]. Our treatment of plastic pollutants is the species-level equivalent of us cramming heaps of dirty clothes under our beds, and then feeling as though we have actually cleaned our rooms. Here, we address the neurological consequences of anthropogenic (i.e. human-caused) plastic pollution, and how we might spare our own brains from plastic-induced neurotoxicity.

The Polymer Problem: We Are Consuming an Absurd Amount of Plastic

The introduction of plastics to the human species came in 1907, when inventor Leo Hendrik Baekeland created the first synthetic plastic polymer, out of phenol and formaldehyde. (Polymer = big molecule made of smaller molecules called monomers). Baekeland's creation was termed Bakelite [4], it was prized for its durability and ability to be molded into diverse shapes. Despite Bakelite's practicality, plastics were not mass-produced until the conclusion of the second world war. Annual plastic production in the late 1940s and early 1950s surpassed one million tons globally [1], as more and more commodities originally made of metal or glass, like bottles, were replaced with plastic versions. A few key societal factors enabled this transition. Firstly, progressive increases in fossil fuel mining throughout the latter half of the 20th century caused plastic production to become quite inexpensive. As over 99% of all plastic is derived from crude oil [5], increases in oil's availability without any real price changes meant that the material needed to produce plastics were made abundant and predictably priced. Another crucial factor to plastic's explosive popularization was the wide range of commercial products that could be made from it. Snack packaging. Radios. Car bumpers. Clothing. Turned to plastic. By 1989, annual global plastic production exceeded 100 million tons. Over the past three decades, that number has tripled [1].

Plastics themselves seem pretty innocuous. They're not particularly reactive, meaning they don't create major chemical reactions when contacting everyday substances, and they don't show any signs of being overtly toxic, like causing skin irritation. These traits, along with some more convoluted metrics, have led to the current designation of plastics as "inert" [6]. This term implies that plastics do not cause any significant reaction when biological systems are exposed to them. While technically correct, identifying plastics as inert is misleading.

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Superficially, it seems easy to avoid consuming plastic. While nonhuman animals like fish may lack the necessary wisdom to avoid eating whole chunks of plastic waste ("macroplastics"), people do not typically go about their days accidentally eating garbage. This leads to an interesting psychological effect among humans: we often feel as though our plastic waste is dangerous to animals other than ourselves, but not particularly dangerous to us. This line of thinking is fallacious. It would be "nice", actually, if the extent of plastic pollution's effects was just a bunch of sea turtles being strangled by those plastic rings that hold six-packs together. Unfortunately, it's becoming clear that most of the plastic that's consumed by animals, including ourselves, is microscopic [7]. That means it's invisible to the naked eye, undetectable without microscopy or laboratory testing. That also means that when it comes to plastic's potential to damage the nervous system, size matters. Perhaps counterintuitively, smaller plastic particles pose a greater threat to animal brains than larger ones. If you were to eat, say, a LEGO (don't), there's little chance that doing so would cause brain damage. It might be uncomfortable on the way out, but the LEGO probably isn't going to get lodged in your brain and cause an inflammatory response that damages DNA and kills brain cells. However, if you were to eat a LEGO's worth of microscopic plastics ("microplastics"), those aforementioned neurotoxic effects [3] would be much more likely to occur.

Microplastics are a macro - problem. They are tough to detect, yet have been detected almost everywhere that researchers have looked for them. Oceans, lakes, rivers, and rain have all been found to be contaminated [8]. The literal air of human cities has tiny plastics suspended all throughout it. These microplastics are only getting smaller, and more spread out. In 2019, the World Wide Fund for Nature estimated that the average human consumes about 5 grams of microplastics weekly [9]. Five grams is the typical weight of US nickel, or a credit card. Although there is a range in the amount of plastic we are consuming, at the high end, we are eating about a credit card's worth of microplastics every week. That is a gargantuan quantity to be consuming, for something that provides no physiological benefits and isn't food.

How Microplastics Got to Our Heads

Microplastics are not manufactured as such, they are instead formed through the degradation of larger plastic pollutants into small particles. The following is a set of typical examples of how a piece of plastic waste can be broken down and ultimately infiltrate an animal's brain: Imagine that you are a Sprite bottle. A human person has consumed the Sprite (along with, surely, a little dose of microplastics), and discarded you. You now have a slim chance of being recycled into an expensive tote bag or maybe a notebook. More likely, you will be incinerated. When you're burned, not all of you is fully combusted. Some of you melts apart into microplastics, and floats into the atmosphere. You are subsequently inhaled by terrestrial animals. Alternatively, perhaps you were littered and ended up in an aquatic ecosystem. Once you're in the water, you become degraded by weathering and sun exposure. This is when your status as a stable polymer begins to threaten animals like fish and molluscs. The nature of plastic polymers is as such that you can be broken down to near-infinitely small components, while still retaining the molecular structure of a plastic. Therefore you are not destroyed in the water, you are merely diffused, and now contaminating the very environment that dispersed you. Aquatic animals living in that environment are now going to eat, drink, or breathe you in. Yikes.

Let's assume that you, now an aquatic microplastic pollutant, have been ingested by a fish. Recent research indicates that you caused some toxicity to the gills when you were ingested, as well as the gastrointestinal tract (gut) [10]. You damaged cells composing intestinal lining and gills, causing them to detach from one another, and making the tissue loose and

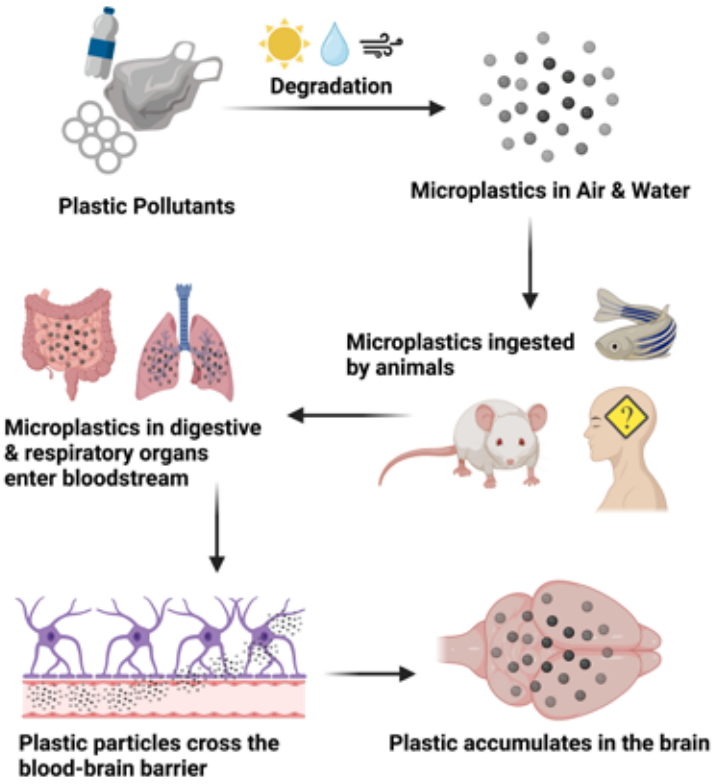

***Figure 1**: How microplastics enter the brain. Created by Bennett Andrassy in BioRender. Figure adapted from Shan et al. 2022 and Prüst et al. 2020. Wind icon created by Iyi Kon: https://www.vecteezy.com/vector-art/442866-wind-vector-icon.*

disorganized. The major cause of this toxicity is thought to be a microplastic-induced inflammatory response [3]; this general mechanism of microplastic toxicity is also reflected in the nervous system. Thus, regardless of whether you were eaten or inhaled, you toxified the tissue that uptook you, and you're now able to interact with the circulatory system. Blood vessels closely border both the gills and the gut lining, to allow for gas and nutrient exchanges with the blood, respectively. The interfaces between these organ systems have evolved to ensure that harmful microorganisms cannot pass into the bloodstream, but these "borders" between systems still contain gaps large enough for microplastics to pass through. With relative ease, you can enter the bloodstream, where you will be circulated all throughout the fish's body. This is where things get even freakier. There's an extraordinarily important region between the nervous and circulatory systems known as the blood-brain barrier, which is exactly what it sounds like. A specialized type of nervous system cell called astrocytes form the blood-brain barrier, by wrapping themselves around both blood vessels and neurons (brain cells), and acting as a buffer between them. The blood-brain barrier holds the critical responsibility of keeping toxins, viruses, and microorganisms out of the brain. A set of studies from 2022 have shown that, if you're a microplastic, the blood-brain barrier is no real obstacle [11],[12]. You can pass from the environment into an animal, throughout its circulatory system, and then straight into the brain. Each of the body's physical "checkpoints" designed to keep you out of the blood and brain has failed (Figure 1).

Passage of microplastics through the blood-brain barrier is currently regarded as the primary mechanism through which microplastics accumulate in the brain [11]. However, there are a few other avenues that lead to the same end. A recent study that used mouse models found that the negative neurological effects of microplastic exposure could be reversed by severing the vagus nerve [12], which connects the brain to the nervous system of the gut (the enteric nervous system). This finding demonstrated that microplastics can travel from the mouse gut directly to the brain, via the vagus nerve. Even more disquieting, perhaps, is the capacity for microplastics to contaminate the brains of animals' offspring, through what is called a transgenerational exposure. Transgenerational exposure to microplastics occurs when parent animals that are already harboring microplastics in their bodies reproduce, and transfer plastic contaminants to their offspring. In fish, transgenerational microplastic exposure occurs through contamination of the germ line (i.e. sperm and eggs). Then the entire fish embryo develops with microplastics already inside [13]. Mice, conversely, can be transgenerationally exposed to microplastics through contaminated maternal blood entering the placenta [14]. The severity of these exposures in newborn mice can be increased if the mouse pups feed upon microplastic-contaminated milk from their mothers. The first report of microplastics in the human placenta [15] was published last year, demonstrating that humans are also at risk of transgenerational microplastic exposures. Importantly, each avenue of microplastic exposure discussed above results in the accumulation of plastic particles in the brain.

Okay, *technically*, microplastics haven't been shown to enter the human brain. This is a

tenuous statement, however, since current knowledge in the field strongly suggests that they do. The composition of the blood-brain barrier is highly similar between rodent models and humans; in fact, physiological similarity to humans is one of the main advantages of using rodents as experimental models. It is therefore quite unlikely that microplastics are able to penetrate the blood-brain barriers of fish and rodents, without being able to do the same in humans. Also, though researchers have had trouble with exact quantifications, humans appear to have more exposure to microplastics than almost any other animal [9],[16]. Given this, human brains may actually be at greater risk of microplastic neurotoxicity than those of common experimental animals. Again, it's tough to make conclusions on this. So far, only one study [17] has demonstrated that microplastics harm cultured human neurons, by inducing inflammation. Human subjects research, or at least more studies on human cell cultures, would be needed in order to conclusively show that microplastics enter the human brain. Studies on human brains are predictably much more difficult to fund, ethically approve, and conduct than studies that use animal models. So for now, our best bet at understanding what microplastics might be doing to our nervous systems is through understanding their neurological effects on other animals.

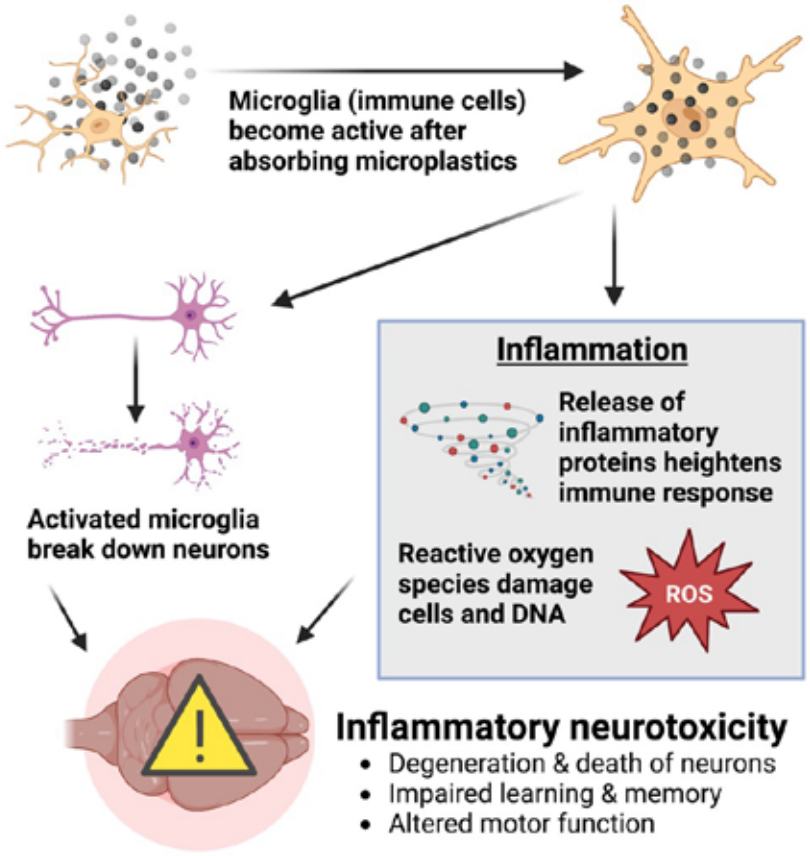

Figure 2: *How microplastics cause brain damage. Created in Biorender by Bennett Andrassy. Figure adapted from Shan et al. 2002 and Prüst et al. 2020.*

Don't Freak Out, but Microplastics definitely cause brain damage

This might be a good point to slow down and point out that this whole microplastic situation could be worse. Before unpacking the cellular and molecular mechanisms through which microplastics induce brain damage, we will attempt to contextualize and rationalize this current public health debacle. We humans have been inadvertently poisoning our brains with commercial products for well over a century. Many of these products were far more toxic than plastics, and plenty of people exposed to them led full, healthy lives. Here's an example: about half of the living US population has been exposed to toxic levels of lead throughout their lifetimes [18]. The population most affected were those born between 1950 and 1980, when household piping and a great many paints contained unsafely high lead levels. Lead is a potent neurotoxin, and exposures have been linked to drastic decreases in human IQ scores. My parents probably still have some lead stuck in their brains, but they seem pretty much fine. This is all to say: the human species has done worse things than contaminating our environment with tiny insidious bits of plastic. We have also come out on the other side of similar crises with a better knowledge of how to maintain our neurological health. As daunting as the issue of microplastic neurotoxicity may be, it's not going to cripple human civilization anytime soon; as we'll discuss at this article's conclusion, there are a few promising ways to damage-control for the neurotoxic effects of microplastic exposure. Bearing this cautious optimism in mind, let's try and understand how plastics are harming our heads.

Microplastic neurotoxicity has as much to do with immune function as nervous system func-

tion. Actually, the nervous system might not be damaged by microplastic exposures, if said exposures didn't cause an immune response in the brain. Here's how that happens: once microplastics cross the blood-brain barrier, they are recognized as an invading enemy by a type of cell called microglia. Microglia are the immune cells of the nervous system, they belong to a class of cells called macrophages (roughly meaning "big eater"). Essentially, microglia can exist in two states: an inactive anti-inflammatory state, and an active pro-inflammatory state. When inactive, microglia are a lazy cell type—their appearance is that of a globby cell body with many spindly appendages. When microglia encounter a foreign substance in the brain, such as a microplastic, they physically absorb the offender and attempt to destroy it with enzymes, in a process called phagocytosis. Microglial phagocytosis of microplastics (say that five times, fast) causes the microglia to become activated, where they expand and release signals that promote inflammation in nearby brain cells (Figure 2). This mechanism was discovered in 2022 [11], when the microglia of plastic-exposed mice were found to have microplastics inside of them. In the same study, plastic-exposed mouse brains were removed and examined by the researchers. When examining them microscopically, researchers found that mouse brains that were exposed to microplastics had holes in them, in a pathology not dissimilar to those seen in human patients with Alzheimer's and Parkinson's Disease [16]. While it's clear that holes in the brain aren't good for neurological function, there's some mystery surrounding how microglial activation causes them. The most likely culprit is a function of microglia called pruning, which occurs when activated microglia try to remove neurons that are either damaged or unused. When microglia are first activated through "eating" microplastics, they release proteins that tell surrounding cells to become inflamed (i.e. swollen and hotter). When this happens in neurons, microglia may detect their inflamed state and begin eating the neurons themselves, ultimately causing the formation of empty pockets in the brain where there should be functioning cells.

An important element of inflammation in the brain is that it's cyclical: inflammation breeds more inflammation. While the inflammatory signaling proteins released by activated microglia inflame neurons, they also help activate other microglia. Therefore, not every microglia in a plastic-contaminated brain needs to physically harbor microplastics to be in its active, proinflammatory state. In order to determine that microplastics cause neuroinflammation in fish and rodents, recent studies [11],[19],[20] have measured the levels of various proteins involved in generating inflammation. These proteins include the inflammatory signaling proteins that microglia release, called cytokines, as well as proteins that are necessary for cytokine production. Researchers have found that these inflammatory proteins were unusually abundant in mice and fish that were exposed to microplastics, when compared to unexposed animals. Measures of protein content are not the only means through which scientists can gauge neuroinflammation, however. Another common measure of inflammation is to assess the abundance of toxic compounds called reactive oxygen species (ROS). When a cell is under some kind of inflammatory stress, its energy production machinery begins to backfire. Specifically, a cellular structure called the mitochondria, which is responsible for cellular respiration (i.e. energy production), begins to produce oxygen-based compounds that have a negative electrical charge. For example, an inflamed cell might produce greater amounts of the charged compound OH- than the neutral compound O2. These charged oxygen compounds, or ROS, can then travel around the cell and "steal" electrons from other important components of the cell, like the cell membrane or the cell's DNA. At high levels, ROS are known to kill brain cells and cause damage to DNA. Much like in the case of inflammatory cytokines, animals exposed to microplastics had greater levels of ROS than those that weren't [10],[12]. Furthermore, scientists have found abnormal gene expression patterns in both fish and mice exposed to microplastics [21],[22], showing that microplastic-dependent increases in ROS functionally alter the DNA of exposed animals.

While the molecular mechanisms of how microplastics cause inflammatory neurotoxicity are certainly useful to neuroscientists, they don't bear much weight when viewed in a vacuum. This begs the question: what are the effects of microplastic neurotoxicity on the overall health and behaviors of exposed animals? Well, the good news is that microplastic exposures aren't lethal. Or, rather, they are lethal so infrequently that analyses on the survival of animals exposed to them rarely turn up statistically significant results [3]. However, a bunch of bad stuff still happens when animals get microplastics in their brains. In fish, microplastics cause alteration in movement. The normalcy and coordination of their swimming decreases in response to microplastics; researchers also correlated this finding to increased molecular markers of neuroinflammation [23]. Furthermore, the

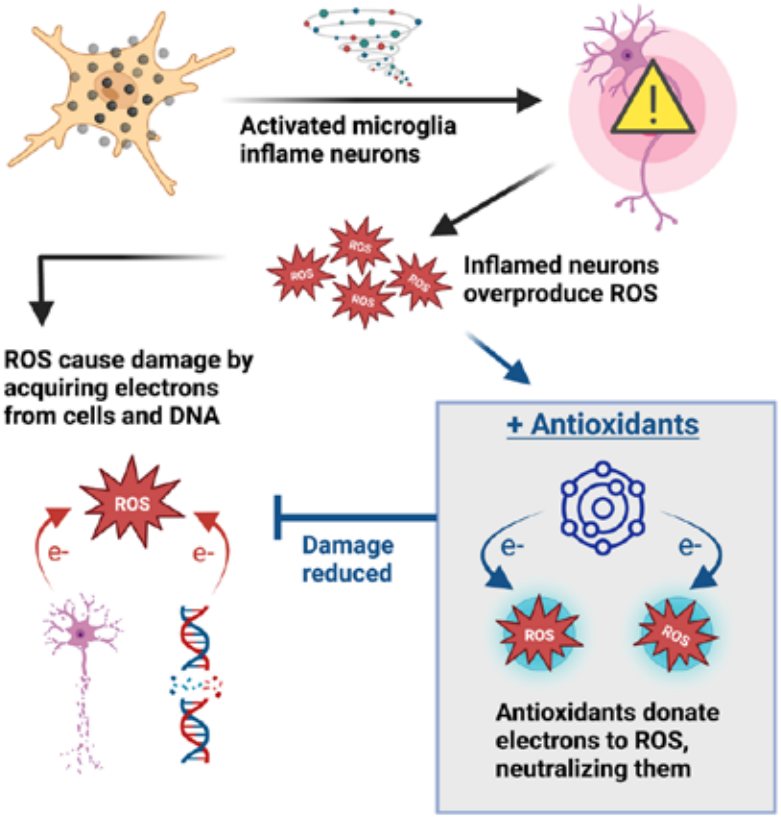

Figure 2: *How antioxidants fight inflammation. Created in Biorender by Bennett Andrassy. Antioxidant icon created by Gohzi Muhtarom : *add link**

swim bladders of developing fish exposed to microplastics often fail to inflate [13], which is a well-stereotyped sign of general toxicity. Since fish are a poor model for studies of cognition, our knowledge on the cognitive effects of microplastic exposures comes from studies that used mouse models. A study on mice that were fed microplastics found that the exposed mice had decreased performances on several tests of learning and memory, while their social behaviors remained normal [22]. This finding goes hand in hand with another, which is that microplastics seem to disproportionately damage a brain region called the hippocampus [11]. The hippocampus, in most vertebrates, is a brain region necessary for the production of new memories, and thus learning new information. The hippocampuses of microplastic-exposed mice developed holes in them, and the mice performed worse on learning and memory tests; this checks out logically, but the human implications of these findings are pretty nasty. If microplastics cause inflammation in neurons, specifically affect the hippocampus, and cause learning deficits in mice, it's quite likely that our frequent consumption of microplastics is harming our cognitive abilities. A 2022 study [24] began to explore this likelihood, by testing the effects of microplastic exposures on synthetic mini-brains grown from human stem cells. As the synthetic brains developed, the ones that were exposed to microplastics saw more changes in gene expression, as well as more dead or dying neurons. Thus, it's unlikely that our species will avoid microplastic-induced neurotoxicity. This problem will not go away without intervention.

Taking Your Vitamins and Other Coping Mechanisms

As microplastics keep getting smaller and more abundant, we are faced with the increasingly important task of maintaining our brains. As of now, nobody knows how to physically get microplastics out of brains. However, antioxidants present a viable means through which animals might combat the effects of having plastics permanently stuck in their brains (Figure 3). As microplastic neurotoxicity hinges on inflammatory responses in the brain, interventions targeting inflammation itself have been shown to be strikingly effective in reversing negative neurological effects in micro-

plastic-exposed mice [12]. In the experiment, researchers exposed mice to microplastics and tested their learning and memory, determining that it was impaired. Next, the researchers gave the same mice a dose of Vitamin E, which is a potent natural antioxidant found in many types of squash, seeds, and beans. When the plastic-exposed mice were given Vitamin E, their performances on learning and memory tests were restored to normal levels. Antioxidants like Vitamin E reduce inflammation through "donating" electrons to ROS, thus making them electrically neutral. When ROS are neutralized, they can no longer steal electrons from other components of a cell—this minimizes damage and the snowball effects of inflammation. Since it hasn't been confirmed that microplastics reach the human brain or impair its cognitive performance, antioxidants have yet to be implicated as a protective measure against microplastic neurotoxicity. However, it is known that antioxidants help humans cope with other inflammatory brain disorders, such as major depression, which is also characterized by impaired functioning of the hippocampus. While neuroscientists aren't sure that taking vitamins or supplements that contain antioxidants will help humans to minimize microplastic neurotoxicity, doing so certainly won't hurt.

While antioxidants are a promising non-clinical treatment for the brain-damaging effects of microplastic exposure, there are also some measures that we can take to minimize the amount of microplastics that animals are exposed to in the first place. Most obviously, we could recycle more of our plastic waste, which would mean that less microplastics would enter Earth's air and water annually [2]. As individuals, we can also opt to purchase less plastic products, thus reducing the amount of plastic we are exposed to through food and drink packaging. Some scientists have proposed more esoteric solutions. For instance, some laboratories are focused on developing plastic-eating bacteria [25] through genetic engineering. While these bacteria can feasibly clean up quite a bit of plastic pollution, they're currently too expensive to be used on a large scale. Also, there is an emerging trend towards creating "bioplastics", which are a subtype of plastics that are not derived from oil, and can fully biodegrade without becoming microplastics [26]. Bioplastics have not gotten particularly popular because they, uh, don't last very long compared to normal plastics. Regardless, it's heartening that people are making an effort. Overall, microplastics are being consumed by a wide variety of animals, including ourselves. Every piece of experimental data available points to an uncomfortable probability: microplastics are infiltrating our brains and damaging them. But again, don't freak out. Recent breakthrough studies on fish and mouse models have afforded us a working understanding of how microplastics damage the brain, and that's a solid start considering that microplastics weren't even thought to be neurotoxic a decade ago. That being said, now may be a good time to ditch your plastic water bottle.

"Recycling" taken by Jeff Attaway. Liscensed under CC BY 2.0.

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