

2,4 D and Forest Lake for the Homeowners

2,4 D is a very commonly used herbicide in agriculture. It has a very long history of use, since the 1940's. It has been scrutinized very intensively because it was a component of Agent Orange. Ultimately it was determined that a dioxin contaminant of Agent Orange was associated with cancers and other toxicities in our service members. However, no significant toxicity from 2,4 D was identified in the agent orange studies. It has essentially no acute toxicity in humans unless ingested in large oral doses. Likewise, there is no animal toxicity unless very large doses are tested. It is listed as toxic by the EPA only because when sprayed directly into the eyes it causes significant inflammatory reaction. When 2,4 D is applied to a body of water it is broken down by the microbes in the water and does not persist, nor does it persist in sediments. Humans cannot absorb 2,4 D from their skin. We can however absorb 2,4 D via the oral route. Humans excrete 2,4 D quickly in the urine if ingested, mostly unchanged, with a half-life of about 10 hours. It does not accumulate in the human body, and it does not accumulate in the biomass of a lake. In lakes treated for milfoil with 2,4 D no trace of 2,4 D could be detected in shallow wells in a 73-day study. I have been asked to discuss 2,4 D by the Forest Lake Preservation Foundation. While the above summary is accurate, and probably reassuring, detecting toxicity of chemicals released into the environment is extremely challenging. Testing methods continue to evolve, and environmental and human toxicity issues continue to be contentious. Because of the difficulties interpreting studies I have tried to put these issues in perspective, as there is rarely any absolute certainty in this field.

The first paper will discuss how we test for toxicity, looking at the multiple methods in use and explaining the weaknesses and strengths of each form of test. The second paper will look at who is protecting us from environmental toxins and how they have performed historically (both government and industry). The third paper will look at what we can do personally to protect ourselves, our children, and the lake, followed by more comments on 2,4 D. The 4th paper is courtesy of Nikki and Colin Fitzgerald. As many of you know they are family from Forest Lake. Both have tremendous expertise on a class of chemicals in our environment, the PFOA's, also known as the "forever chemicals". I very much appreciate Nikki taking time out to answer questions particularly considering that she was expecting a baby within 2 weeks of my contacting her, and working full time.

These papers will not be referenced. I am sure that there will be questions, comments, and clarifications needed. Please email me with any questions as I am happy to discuss and if I don't know an answer I will try to find it. My goal is that when you are finished with these papers, you will have more tools to evaluate and judge for yourselves how we can protect ourselves and Forest Lake as we move into the future.

I also apologize in advance for how long this paper is. I couldn't in good conscience give you my opinion without also explaining how our testing and regulation don't always give us certainty. If you don't wish to read all 3 papers, just read my intro here and my conclusions. The DNR fact sheet is included at the end of chapter 3 and is certainly a quick read as well. Mark

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Are there harmful chemicals released into the environment? Definitely. Today we are exposed to countless new chemicals our ancestors were not exposed to. Naturally, we would like to avoid the harmful ones, and ignore the harmless chemicals. Most of us rely on others to guard our safety from environmental toxins. But how do we detect which of the thousands of chemicals we release into the environment are harmful, and who is doing the detecting? It should be simple right? Almost every day we see a study on something, so there must be thousands of studies. Run studies to find out which chemicals are toxic, ban those chemicals, and use the harmless chemicals to build a better world. What could be simpler? But wait!!! It's not that easy. To determine which chemicals are harmful we have to ask many questions that aren't obvious. 1) Does everyone suffer the same risk from a chemical? Men? Women? Children? Fetuses? 2) how long does the chemical persist in the human body? Readily eliminated or does it accumulate? 3) Are some risks long term risks, that we might not ever notice? 4) Will the risk be passed on to the next generation rather than us? 5) If a chemical is not harmful to test animals, is it also safe for humans? 6) When we test for toxicity, are we looking for the right indicators? 7) When a chemical gets broken down to other chemicals in the environment do we test those as well? 8) Can we trust those doing the testing to put the public's interests above the interests of the businesses producing the chemicals?

To put our discussion into perspective, It might be instructive to look at the long history of lead. It was first mined about 8,000 years ago and valued for its density, lack of corrosion, and its malleability. It was first recognized as toxic about 2500 years ago! You read that correctly. It has been used to line pipes (Roman pipes led to 100 times the normal amount of lead in water). Romans lined their liquid containers with lead. It has been used in distilling liquor, and even sweetening wine. Its use in wine has led to epidemics of abdominal pain (see monks with colic), and epidemics of gout. These lead toxicities have occurred throughout history, including medieval and more recent. Because lead was so useful it was used in paints in the 1800's. In 1897 (yes over a hundred years ago) it was discovered that leaded paint caused toxic symptoms, and it was banned in Australia. In 1922, the League of Nations banned leaded paint. The US passed a ban on leaded paint in 1971! The ban wasn't fully implemented until 1978. Around the 1950's it became apparent that chronic lead exposure to lower levels of lead were also toxic. In the 1920's we began adding lead to gasoline to help the gas burn better. Leaded gas was phased out in the 1980's. Currently lead levels in dust and buildings are still elevated from past combusted gasoline, but declining. In the 1980's a scientist trying to prove the significant toxicity of chronic low level lead exposure was falsely accused of scientific misconduct by the lead industry. Today we know that NO lead level is safe. Children are particularly sensitive to chronic lead toxicity, and it affects their IQ. The lower your lead level the better off you are. It wasn't until 1986 that the use of lead products in water pipes was banned. Today lead levels in humans are gradually lowering, but are still over 100 times higher than in pre-industrial times. As you know, recently municipal water in Flint Michigan was found to be exposing children to dangerously high lead levels.

Closer to home, we continue to put lead in our environment in Forest Lake. Almost all fishing jigs are weighted with lead (dense, cheap). When we hook a fish and the line breaks, or the fish dies and the hook is still in the fish, that lead falls to the bottom to possibly be ingested by a duck or loon, or eaten by an eagle. These birds have very elevated lead levels, and some eagles and loons die of lead toxicity. Lead is slowly working its way through the biomass of Forest.

So what can we learn by the story of lead? I'll let you draw your own conclusions, but it seems to me that even when toxicity was known, humans have continued to use lead whenever it was cheapest or worked the best despite toxicity. It is much easier to ignore a toxin whose effects might just be a lowered IQ and nothing more obvious. Who's out there was protecting our children or even us? Why is lead even allowed as a weight for fish hooks? (I've tried to find small jigs for pan fishing on Forest that were not lead weighted. They make some hooks that

are considerably more expensive, but I can't find any small enough for fishing panfish! So clearly there is no demand) My own conclusion to the lead story is that even if there is an obvious toxin being released into the environment, it is difficult to prove, and difficult to change. Lead toxicity was easy to prove. In contrast, however, it is often very difficult to prove that a chemical is toxic.

Every day we are putting more things into the environment. And it isn't just industrial chemicals. We are urinating many of the drugs we take such as antidepressants, and they are now in our water supplies. We are taking natural plant products and modifying them for our uses. (pesticides) We are modifying plants with genetic engineering (GMO's or Crispr modified), Currently nobody I know would like to ingest Roundup, a herbicide, but actually unless you are eating organic only, anytime you eat wheat or oat products you are eating Roundup. (They spray these crops with Roundup 1 week before harvest, as this makes the harvest more profitable.) I don't know many people who have the means or the dedication to only eat organically. What are we to do? Who do we trust? These are difficult questions, and there are no guaranteed answers. Let's look first at how we determine if a substance is harmful to humans

It is very important to realize that when it comes to the thousands of things we are putting into the environment, very few things are as straight forward as lead. In this paper I would like to try to explain how difficult it is for scientists to come to reliable conclusions. The first problem we have is that there are so many chemicals being released into the environment, how do we sort through all the noise with countless chemical exposures, smoking, unhealthy diets, and other unhealthy lifestyles? In 2020 we do have suspicions that there are problems with something or things in our environment. Male sperm counts are down over 50% over the last century. Women are reaching menses earlier than ever before. Breast cancer rates continue to rise for unclear reasons. Autism rates continue to rise dramatically even when we account for looser diagnostic criteria. ADHD seems to be increasing. Crohn's disease and ulcerative colitis cases are rapidly increasing. There is a suspicion that at least some of these things are related to environmental toxins but without careful science, we will be powerless to make a difference.

A powerful tool to identify unknown toxins or side effects is the observational study. It is frequent when I pick up the paper and there is an article that states that scientists have found in a study that the use of a medication is associated with a previously unknown side effect. These are almost always known as observational studies, and it is VERY IMPORTANT to understand their limitations. When doing an observational study we do not randomize people, but we try to pick a suitable control group to compare. For example, when a drug is approved for market, it is usually after several thousand patients have been in a trial. It appears safe, but it is the FDA's responsibility to make sure that there are no serious but more rare side effects after a larger number (Millions?) of users. This is very prudent. For example, look at Medicare data, and compare patients that are on the medication with those that are not. Do the patients on the medication have any increase in health problems we hadn't noticed before? A big problem with observational studies is that there is no randomized control group. This means that we might find an association between a drug and a side effect, but it is not caused by the drug.

Let me give you an example. I prescribe Nexium (a PPI) for heartburn. This class of meds were found to be so safe that they were approved for over the counter. Later, an observational study noted that people on PPI's had more hip fractures than people not on PPI's. Newspapers and laypeople immediately concluded that the PPI's weakened bones and led to hip fractures. HOWEVER, there were what we call confounding variables. People that are overweight get more heartburn. In addition, people that are overweight have more hip fractures. Therefore the reason the PPI patients had more hip fractures was that as a group compared to the controls, they were more overweight. The hip fractures were not because of the PPI. Being overweight

was the confounding variable. This was proven when a randomized study was performed and PPI's did not cause hip fractures.

It is important to do observational studies, as they are much cheaper, and it is very difficult to do a randomized study, so most studies are observational. Because there are so many confounding variables (smoking, overweight, bad diet, other meds etc) that an observational study has to be confirmed with other studies. As another example, other observational studies have shown that people with higher Vit D levels are healthier than those with lower levels, but were confounded by the fact that people with higher Vit D levels tend to eat more fish, exercise more outdoors and smoke less. So they were healthier because of other factors than Vit D. In other words, observational studies are a primary source for raising suspicions, but they don't PROVE anything and usually we find confounding variables that were the culprit. That doesn't mean that we don't do observational studies, but we have to interpret them carefully. Randomized studies would be much more exact (where we divide our subjects into 2 groups, those who receive the drug/toxin being tested, and those who receive a placebo) Could we do randomized studies of industrial chemicals? Not likely. Who would volunteer for a study in which they had to take a potentially toxic substance? Who would pay the billions of dollars to study all of the chemicals we currently produce? So observational studies may detect the canary in a coal mine, and they are very important, but they can only be relied on to be suspicious of a possible toxin. They will never prove that something is definitively toxic.

So I have explained the difficulty of finding reliable answers in observational studies. However, given the fact that we are left with few options for discovering which of the chemicals weren't tested or slipped through testing with a safe rating, these studies remain very important. Ideally if we were suspicious of chemical "B" we would do a randomized study of "B". But this is almost impossible

As a substitute for randomized human studies, we do randomized animal studies. These can be very valuable. Unfortunately, it is very difficult to correlate the results in animals with results in humans. How do we measure IQ in a monkey? If a drug is safe in a rat will it be safe in a human which lives about 50 times longer? As a gastroenterologist I have learned that animal studies do not translate well into humans. For example, we can transplant feces from obese mice into lean mice and make them obese. When we do fecal transplant for incurable C difficile infections in humans, we don't make people obese or lean. Certainly animal studies can be helpful at predicting toxicity in humans, but these studies must always be interpreted cautiously. In other words animal studies can be worrying or reassuring, but again they do not provide absolute answer proof in either case

Another substitute for randomized human studies is looking at living human cells. For example, scientists will use living human cells and expose them to a chemical that is being studied. They will then examine the cells after a suitable period and look at the DNA. If the DNA has been harmed then it raises the suspicion the chemical may predispose to cancer. Intuitively this approach seems to be sound and thorough. Human cells are certainly more representative of us than monkeys or lab rats. Unfortunately, once again this type of study intuitively seems sound, but humans are more complicated. There are many different cell types in the human body, and humans develop many different types of cancer. I mentioned that breast cancers are becoming more common. It is suspected that one of the reasons is that we are releasing "false estrogens" into the environment. In other words, chemicals that are not estrogen, but attach to the human estrogen receptors and fool the body into thinking that estrogen levels are high. When a common ingredient that is used in many sunscreens (and is suspected to become a false estrogen) is tested against normal human cells, it does not harm the DNA and so the US considers the chemical safe. Recently however, the sunscreen chemical was tested against cells with estrogen receptors (breast, uterus etc) and they find damage to the DNA. This raises concern for promoting breast cancer in humans. Unfortunately,

in the US we do not consider different cell types and the sunscreen products pass the safety test. Could sunscreen be increasing female cancer rates? We don't know, but among scientists there is growing concern.