Conspiracy theory versus tragic and ongoing reality? Is it possible to tell the difference?

William Parker, February 9, 2022

Conspiracy theories have become an important part of society, with some theories deceiving millions of people. University professors have studied conspiracy theories and how to detect them. For example, Timothy R. Tangherlini, Professor of Danish Literature and Culture, University of California, Berkeley, is one such professor. His work and the work of others tells us the best way we know now to detect a conspiracy theory. The table below provides some common features of conspiracy theories, and compares those with the view that early-life exposure to acetaminophen causes neurodevelopmental problems in susceptible children.

Characteristic	Typical features of a conspiracy theory	Early-life exposure to acetaminophen causes neurodevelopmental problems
Critical supporting evidence is based in reality.	**No**	**Yes**
The scientific method can be applied to test conclusions.	**No**	**Yes**
Supporting facts linked together in a fragile chain of logic.	**Yes**	**No**
Independent lines of supporting evidence point in the same direction.	**No**	**Yes**
Developed slowly over time as evidence emerges.	**No**	**Yes**
Fits Occam's Razor: the simplest explanation for the data.	**No**	**Yes**

Notes:

The number one difference between a conspiracy theory and reality, no matter how bad and shocking that reality may be, is that reality bears up under close examination. Key elements of data from conspiracy theories may be impossible to verify, may depend on "secret" knowledge, may contain distortions of actual evidence, or may even be completely false. In contrast, all available evidence points toward the view that early-life exposure to acetaminophen causes neurodevelopmental problems in susceptible children. Further, the scientific method has been used in the past and is being applied to test this view.

Conspiracy theories often involve linking a series of apparently unconnected factors together in a chain that creates the appearance of a conspiracy. If one link breaks, then the conspiracy theory falls apart. Fortunately, reality is subject to scientific evaluation, and multiple lines of independent evidence can generally be used to support reality. A wide range of evidence points independently toward the view that early-life exposure to acetaminophen causes neurodevelopmental problems in susceptible children. Even if one line of evidence turns out to be difficult to verify, it does not affect the other lines of evidence.

Conspiracy theories often appear fully formed overnight, connecting many complex ideas together. Our understanding of real-life problems can develop quickly if key pieces of evidence are suddenly uncovered. However, our understanding of complex, real-life problems tends to develop slowly over time as information becomes available. Our understanding that early-life exposure to acetaminophen causes neurodevelopmental problems in

susceptible children has progressed slowly over time, starting from the first study published in 2008 by Stephen Shultz. The story of how Stephen used the scientific method to first formulate the connection between early life exposure and autism is described below, taken from an article I wrote in 2015.

Conspiracy theories tend to offer horrific explanations for observations that have otherwise simple explanations. In contrast, no alternative explanation exists for the wide range of evidence pointing toward the view that early-life exposure to acetaminophen causes neurodevelopmental problems in susceptible children.

How it All Began:

The text below is an excerpt written by William Parker taken from an article originally posted on September 11, 2015 by SafeMinds on their website. The original article contained medical advice, which has not been included in this excerpt. William Parker and WPLab, Inc. do not provide medical advice, and none of the information presented in this article or on this website is intended to substitute for advice from your physician.

My research looks at what causes harmful inflammation in people in Western societies. The triggers of inflammation are recent developments in human history, appearing after the agricultural revolution only 10,000 years ago. Most did not appear until just a few decades ago, as we entered the postindustrial age [1]. My favorite example is the loss of biodiversity from the human body. Humans have always been bathed inside and out with bacteria, viruses, fungi, worms, and other organisms, but in recent decades our bodies' diverse, to our detriment.

Inflammation can be described simply as an aggressive immune response. It's not a bad thing, and in fact is necessary under certain circumstances. However, inflammation in Western society has got ten out of control, resulting in pandemics of allergies, autoimmune conditions , and increased rates of cancer. I'll list the main factors known to cause inflammation in humans living in Western society.

These are the "big five":
1. Loss of biodiversity (biome depletion)
2. Inflammatory diets
3. Sedentary lifestyles
4. Chronic psychological stress
5. Vitamin D deficiency

Scientists hypothesize that if we could eliminate these five factors, we would virtually eliminate allergies and autoimmune disorders [2 , 3]. We also think that the rate of cancer would drop profoundly [4 , 5].

Most importantly for this discussion, the rate of neuropsychiatric disorders is expected to plummet if we could control these five factors [6, 7]. The high rates of autism should drop dramatically [6, 7].

People need to make drastic lifestyle changes. Those might not happen overnight. But, fortunately, sometimes there is another way to avoid an inflammatory disease. If we can identify a "trigger," a necessary factor that interacts with inflammation to cause a specific disease, then we can avoid that specific disease. We can't avoid ragweed pollen to avoid hay fever, and we can't avoid food to avoid food allergies, but it looks like we may be in luck when it comes to avoiding autism. An apparent and easily avoidable trigger for autism has been found.

Steve Schultz is the scientist who identified what appears to be an important trigger for autism. He was a practicing dentist for 21 years before going back to school to earn a Ph.D. in an effort to determine why his son, Nathan, had regressed into autism. Based on his personal observations, Schultz at first thought that it was the MMR vaccine, but that had already been tested and it looked like a dead end. There was not going to be any way to get a Ph.D. working on that. However, as Schultz explained in his book, "Understanding Autism: My quest for Nathan":

> *"Then I remembered that Nathan had gotten so sick from the MMR vaccine and how I had given him so much acetaminophen. Perhaps there was something about acetaminophen."*

Schultz was thinking about the acetaminophen. My first thought would be that this makes no sense. Why would one of the most commonly used drugs on the planet, known to fight inflammation, lead to an inflammatory disease such as autism? My second and third thoughts would be the same: Schultz's thinking makes no sense. The presence of some sort of toxin might be the trigger, but acetaminophen?

But Schultz was running out of possible suspects.

With zero evidence, Schultz began to examine acetaminophen. As a detective investigates the last remaining suspect, Schultz started to work on acetaminophen not because he was suspicious of this much loved pain reliever, but because there was no other culprit to examine.

Quickly, he found plenty of reason to be suspicious.

Acetaminophen was the last remaining drug in a particular class of relatively toxic drugs that is derived from coal tar. It was, even at that time, the only drug left in its class not discontinued because of toxicity. It was already known to be associated with the development of asthma in children [8]. Oddly enough, it was not known if acetaminophen actually worked to alleviate fever in children [9].

Acetaminophen rapidly gained traction as a 9]. if great suspect. First, Schultz found that the rise in autism corresponded to the rise in acetaminophen use. The first cases of autism happened around the time when coaltar derived drugs, which give rise to acetaminophen upon metabolism, were introduced. T hen Schultz did what is, in my view, a marvelous thing. He remembered two cases of deadly poisonings in which drugs containing acetaminophen were tampered with, first in 1982 and then in 1986. These two events caused temporary declines in the use of acetam inophen. Schultz's initial test would be simple. If indeed acetaminophen was a possible culprit, then the steady, decades long rise in autism should be punctuated by two brief periods of relief. As Schultz explained:

> *"I saw that children born during both of these acetaminophen poisoning episodes were less likely to have autism. And I knew. Acetaminophen use was linked to the rise in autism rates."*

He went on to test this idea further. He performed and then published a retrospective survey in 2008 [10]. The results were clear:

> *"Children who used acetaminophen at age 12 to 18 months were more than eight times as likel y to be in the autism group when all children were considered and more than 20 times as likely to be in the autism group when limiting cases to children with regression."*

There was no similar autism effect with ibuprofen. In fact, as Schultz explained to me recently by email,

> *"I did not see a direct association with any of the vaccines themselves. It was only the combination of acetaminophen and the MMR vaccine which increased autism risk."*

Schultz had data that should have warned the world. The study pointed singularly at acetaminophen. Not a vaccine. A bad reaction to a vaccine plus ibuprofen was not associated with autism. A bad reaction to a vaccine plus acetaminophen was associated with autism.

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