BY DAN OLMSTED

In Alfred Hitchcock movies, there's a characteristic plot device that the great director called a "McGuffin": What looks like the central premise of the movie ultimately has nothing to do with story.

The classic example is "Psycho," in which Janet Leigh steals $40,000 and goes on the lam, but ends up dead for reasons having nothing to do with the theft—and everything to do with the Bates Motel she had the misfortune to check into. It's an ingenious bit of misdirection.

After spending the last couple of years looking at the natural history of autism, I'm convinced that ever since the original case series was published in 1943, most mainstream research has fallen for the McGuffin.

Let's start at the beginning. One day in November 1935, a mother brought her 3-year-old son, Alfred, to Johns Hopkins University in Baltimore. "He has gradually shown a marked tendency toward developing one special interest, which will completely dominate his day's activities," she told the Hopkins medical staff, "and it is difficult to get his attention because of his symptoms."

After observing Alfred closely and seeing 10 similar children over the next few years, famed Hopkins child psychiatrist Leo Kanner wrote them up in his landmark 1943 paper, "Autistic Disturbances of Affective Contact." He said their common behavioral syndrome differed "markedly and uniquely" from "anything reported so far."

Kanner also noted commonalities among the parents. Alfred's mother, Kanner reported, was "very obsessive and excitable" and the father "does not get along well with people, is suspicious, easily hurt..." Overall, he remarked, "In the whole group there are very few really warmhearted fathers and mothers," and he later used the term "refrigerator parent," leading to an ugly stain of blame and guilt that, thankfully, has been discarded.

Another observation: "They all come from highly intelligent families," Kanner wrote. He acknowledged that "it is not easy to evaluate the fact that all of our patients have come of highly intelligent families." But it's safe to say that this idea— along with a handful of twin studies that are far less definitive than generally realized—paved the way for today's near-exclusive focus on genetics.

Blaming parents' genes certainly beats blaming parents' behavior, and that may in part explain the rush to embrace an exclusively genetic approach. But what matters most is truth. Kanner, blinded by the psychoanalytic bent of the day and his specialized medical background, simply and sadly overlooked a much more likely link: Several of Kanner's kids came from families where toxic exposure plausibly occurred. Let's take the cases in the order he presented them:

- Case 1: Donald T. was the son of "a brilliant lawyer." But as I've found in my own research, he was born in Forest, Miss., around the time the town was being replanted as a national forest.
- Case 2: Frederick W. was the son of a plant pathologist.
- Case 3: Richard M. was the son of a forestry professor at a southern university.

Once I found Donald's hometown, the idea that the first three cases shared some affinity to trees, nurseries and commercial agriculture seemed quite compelling. Still, I had no idea of a possible connection until Mark Blaxill of SafeMinds suggested a link via novel chemical compounds, in particular fungicides. He noted that Morris Kharasch, the research chemist who invented the ethyl-mercury-based vaccine preservative thimerosal in the 1920s, also patented ethyl-mercury-based fungicides at the same time. A ludicrous leap? Perhaps, but let's play out a "toxic connection" in those first cases:

- Case 4 was the son of a mining engineer. Heavy metals—mercury chief among them—are known neurotoxins.
- That brings us to Alfred L., arbitrarily listed as Case 8 in the series, although clearly the first to be seen at Johns Hopkins. His father? Yet another lawyer. But just as something else may have been going on with Donald T., the son of the "brilliant lawyer" from Forest in Case 1, there may be more to Alfred's story.
- Alfred's father, significantly, had dual degrees: he was a chemist as well a lawyer, according to Kanner. And he combined those skills in a perfect job: working for the United States Patent Office.

It is interesting that the first case of a novel disorder was the son of a chemist in the patent office. Who knows what compounds Alfred's father had his hands on, but is there any simpler definition of patent-worthiness than something "markedly and uniquely" different—Kanner's observation about the disorder itself?

Perhaps autism was newly observed in the 1930s because whatever caused it was new too. Since I first wrote about the possibility of harmful exposures in Kanner's first 11 cases—a link that, as far as I know, had never been proposed—I've become aware of three studies that suggest a chemical connection in the subsequent rise of the age of autism.

In the 1976 book, *The Autistic Syndromes,* Dr. Mary Coleman described her study of 78 autistic children in which she noticed "an unusual exposure of parents to chemicals in the preconception period." Out of 78 autistic kids, 20 were from families with chemical exposure; four were from families where both parents had such exposures with seven out of the eight chemists. Still, Coleman worried that because the parents volunteered for the survey, they might have been scientifically inclined, skewing the results toward careers like chemistry.

To test that idea, one of Coleman's young associates, Thomas Felicetti, did his dissertation on a group of 60 children: 20 with autism, 20 with mental retardation and 20 "typicals," all enrolled at the Avalon School in Connecticut where he worked. The association held up: "The results did, in fact, suggest a chemical connection," Felicetti wrote in the journal *Milieu Therapy* in 1981.

"Eight of the 37 known parents of the autistic children had sustained occupational exposure to chemicals prior to conception. Five were chemists and three worked in related fields. The exposed parents represented 21 percent of the autistic group. This compared to 2.7 percent of the retardation controls and 10 percent of the normal controls. ...The data, subjected to statistical analysis, demonstrated a chemical connection."

In the 2002 book, *Impact of Hazardous Chemicals on Public Health, Policy, and Service,* the authors review those studies and cite another—an unpublished manuscript by Marcus and Broman: "They found a higher incidence of occupations involving exposure to chemicals among the parents of children with autism."

Let's review our story so far: The first autistic child to come to Leo Kanner's attention in 1935 was the son of a chemist-lawyer at the patent office. Signs of novel toxic exposures suggest themselves in other children in that first cohort. By the early 1980s, subsequent studies found, again and again, a striking proportion of parents with clear chemical exposures.

Genes certainly could play a susceptibility role in this scenario, "loading the gun," as the geneticists say. My point simply is that the fingerprints of harmful exposure are all over the trigger in many early cases.

So here's my question: Why has this alarmingly plausible hypothesis—evident as early as Alfred L.'s chemist father in 1935—all but disappeared from the research radar, while increasingly arcane gene studies get the attention and money?

Guess what. The parents didn't do it. It's time to spot the McGuffin in this mystery.

AUTHOR

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