

# Neurolaw: Revisiting *Huberty v. McDonald's* through the Lens of Nutritional Criminology and Food Crime

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**Abstract:** Recent studies have illuminated the potential harms associated with ultra-processed foods, including poor mental health, aggression, and antisocial behavior. At the same time, the human gut microbiome has emerged as an important contributor to cognition and behavior, disrupting concepts of the biopsychosocial ‘self’ and raising questions related to free will. Since the microbiome is undeniably connected to dietary patterns and components, the topics of nutrition and microbes are of heightened interest to neuroscience and psychiatry. Research spanning epidemiology, mechanistic bench science, and human intervention trials has brought legitimacy to nutritional criminology and the idea that nutrition is of relevance to the criminal justice system. The individual and community-level relationships between nutrition and behavior are also salient to torts and the relatively new field of food crime—that which examines the vast harms, including grand-scale non-communicable diseases and behavioral outcomes, caused by the manufacturers, distributors, and marketers of ultra-processed food products. Here in this essay, we will synthesize various strands of research, reflecting this emergent science, using a notable case that straddled both neurolaw and food crime, *Huberty v. McDonald's* (1987). It is our contention that the legalome—microbiome and omics science applied in neurolaw and forensics—will play an increasing role in 21st-century courtroom discourse, policy, and decision-making.

**Keywords:** neurolaw; criminal justice; nutrition; mental health; diminished capacity; microbiome; aggression; psychiatry; biological criminology; ultra-processed foods



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## 1. Introduction

The American Bar Association describes neurolaw as the intersection of science and the law, a field that attempts to translate the rapid and voluminous advances in brain science into legal decisions and policy (Hayes 2017). Of course, ‘brain science’ is a broad term, as evidenced by the increasingly robust areas of research related to omics technologies and the human microbiome. Omics refers to rapid advances in our ability to simultaneously measure large numbers of biomolecules representing genes, gene expressions, proteins, and metabolites, as they relate to antisocial behavior (Pietrini et al. 2017; Hagenbeek et al. 2023), and the latter, the related science of microbiomes, includes research indicating that gut microbes and their metabolites make significant contributions to mental outlook and behavior, including aggression and antisocial behaviors (Gulledge et al. 2023; Mikami et al. 2023). Neurolaw is enveloped by exposome science, the study of total lived experiences (both positive and negative “exposures”) interacting with genes, over time; that is, exposome science, which is aided by omics and includes the microbiome, can help predict the biological responses of the “total organism to the total environment” throughout the life course (Prescott and Logan 2017; Liu et al. 2023). As renowned biologist Robert Sapolsky argues in his best-selling book, *Determined: A Science of Life Without Free Will* (Sapolsky

2023), when we examine the firing of neurons in the context of law and criminal justice, everything is embedded in what came before (Crockett 2023).

Although neurolaw is a complex, interdisciplinary field of research, the emergent research in the realm is already presenting fundamental questions to the courts, and the criminal justice system writ large: To what extent should the criminal justice system point to a biological rather than psychological source of criminal behavior? What is the ultimate upstream driver of behavior? Do criminals possess as much free will (or ‘choice’) as prosecutors, judges, juries, and others in the criminal justice system, think they do (Pernu and Elzein 2020; Maoz 2023; C. Wilson 2023)? The answers to these questions lead to many more, including whether or not corporations—such as those manufacturing/distributing ultra-processed food products—are liable for behavioral health outcomes (Robinson 2022a, 2022b). Here, we contend that the emerging science of nutritional criminology (Logan and Schoenthaler 2023), and its intersection with microbiome sciences (Gato et al. 2018), is of high relevance to neurolaw. Moreover, the individual-level outcomes are salient to the relatively new field of food crime—that which examines the vast harms, including grand-scale non-communicable diseases and behavioral outcomes, as caused by the manufacturers, distributors, and marketers of ultra-processed food products (Robinson 2023). We will synthesize various strands of emerging research, and reflect on them using a 1987 case that straddled both neurolaw and food crime, *Huberty v. McDonald’s*. To that end, using a Freedom of Information Act request, we obtained the roughly 900-page *Huberty v. McDonald’s* case file from the Stark County, Ohio, Clerk of Courts.

## 2. McNuggets and Mass Murder?

In autumn 1987, Stark County, Ohio judge James R. Unger pondered a wrongful death lawsuit in which the plaintiff, Etna Huberty, argued that the McDonald’s Corporation and an Ohio-based power company, Babcock & Wilcox, had contributed to the death of her husband, James Oliver Huberty (Associated Press 1987a). The case was notable because, at the time of the suit, the deceased was the perpetrator of the largest mass murder in United States history. Three years earlier, at approximately 4 pm on 18 July 1984, Huberty entered a McDonald’s outlet in suburban San Diego and murdered 21 people (injuring 19) before he was fatally wounded by responding police. Available evidence suggests there was very little planning involved; on the morning of the attack, James Huberty had a favorable outcome in a local court, where he had contested a minor traffic ticket, then had lunch with Etna and their daughter at McDonald’s, proceeded to visit the San Diego Zoo with them, and returned home. At approximately 3:45 pm, James Huberty rose from his sofa, changed into camouflage clothes, and departed the residence, saying “I’ll see you later.” When asked by Etna where he was going, Huberty responded that he was “going to hunt humans,” a comment that was not taken seriously (Sun-Sentinel Wire 1984).

In the suit, Etna Huberty claimed that her deceased husband had been consuming copious amounts of McDonald’s Chicken McNuggets in the days, weeks, and months leading up to the shooting, and that the monosodium glutamate (MSG) in the product contributed to his disordered mind. The widow further claimed that the heavy metals (lead and cadmium) found in perpetrator’s scalp hair samples (taken at autopsy) were a product of his work as a welder at Babcock & Wilcox, and that the toxic metals had further added to his vulnerability. An independent lab had found that the perpetrator’s hair cadmium levels were 30 times higher, and lead levels 8.4 times higher, than the reference normal. The chemist who conducted the hair analysis, William J. Walsh, PhD, later informed a science journalist that “He [Huberty] had the highest cadmium level we had ever seen in a human being” (Wilson 1998).

Responding to questioning by McDonald’s attorneys in sworn depositions, Etna Huberty stated that her husband ate McNuggets and French fries on a near-daily basis in the year before the shooting (*Huberty v. McDonald’s Civil Court Case. Stark County, Ohio, United States 1987*). She stated that until the end of 1982, when he lost his job via layoffs, the deceased was “extremely health conscious”, including attention to a healthy

diet, limitation of sugar, sweets, and salty foods, and that he used “wheat germ” and supplemental vitamins. It was during this time of stress that the perpetrator was introduced to McNuggets by his daughter. After this introduction, Etna testified that her husband abandoned his healthy diet routine and developed a desire for more McNuggets over time: “I’ve never seen anything like it, and it kept getting worse, and worse, and worse. He had an addiction to Chicken McNuggets” (p. 32). In the seven months leading up to the mass shooting, Etna testified that “I think it would be fairly safe to say that he was probably there [at McDonald’s] very close to once—at least once—every 24 hours. . . it was generally Chicken McNuggets, French fries and ice cream” (p. 54). Approximately four hours before the mass shooting, while eating lunch at McDonald’s, she witnessed him consume a ‘family’ sized box of 20 pieces, a soft drink, and an order of French fries.

The case received considerable media attention (Figure 1), and the dismissal of the case was not immediate. Judge Unger pondered over the facts of the case for a little over a month before he decided to dismiss the case (Associated Press 1987b). When Judge Unger was evaluating the merits of Etna Huberty’s claims, as articulated through attorney Thomas Lally, the scientific support was scant. Huberty’s primary supportive document was a 1984 hypothesis paper (published two months after the shooting) by psychologist Robert W. Hall. The author paired existing animal studies and clinical anecdotes related to MSG with the reported lifestyle behaviors of the deceased gunman (Hall 1984). Hall did not contend that MSG consumption was the sole factor in the case—instead, MSG was presented as a ‘tipping point’ factor that intersected with other biological and psychological vulnerabilities in the presence of stress (e.g., Huberty’s unemployment at the time of the shooting).



**Figure 1.** Associated Press (**top**) and United Press International (**bottom**) headlines are typical of global media covering the case in September, 1987.

Although Hall’s argument was decades before the field of neurolaw was formalized, he was wading into the fundamental questions of the discipline. In court depositions, Etna Huberty acknowledged that her husband was psychologically vulnerable—in the year preceding the incident, he had been experiencing visual and auditory hallucinations, and

in the months prior to the incident, he had a delusion that he was a war criminal (Huberty never served in the military), even going so far as attempting to turn himself over to local police for war crimes. Huberty was retrospectively diagnosed with schizophrenia by Phillip J. Resnick, the psychiatrist hired by McDonald's (Resnick 1987).

In his article, Hall was visualizing criminal culpability had the gunman survived, and even though *Huberty v. McDonald's* was about corporate liability, the civil case outcome depended on quality scientific studies linking, or at least potentially linking, MSG-containing products with violent criminal behavior. Judge Unger's job was made easier because in 1987, those studies did not exist—the case was “denied for want of knowledge”. Although there was, at that time, research linking refined food consumption with elevated hair cadmium, cognitive disturbances, and related electroencephalogram (EEG) abnormalities (Lester et al. 1982; Thatcher et al. 1984), Huberty's legal team focused only on the MSG in McNuggets, and left discussion of toxic metals to the industrial defendant, Babcock & Wilcox. Food-derived cadmium will be discussed in more detail below.

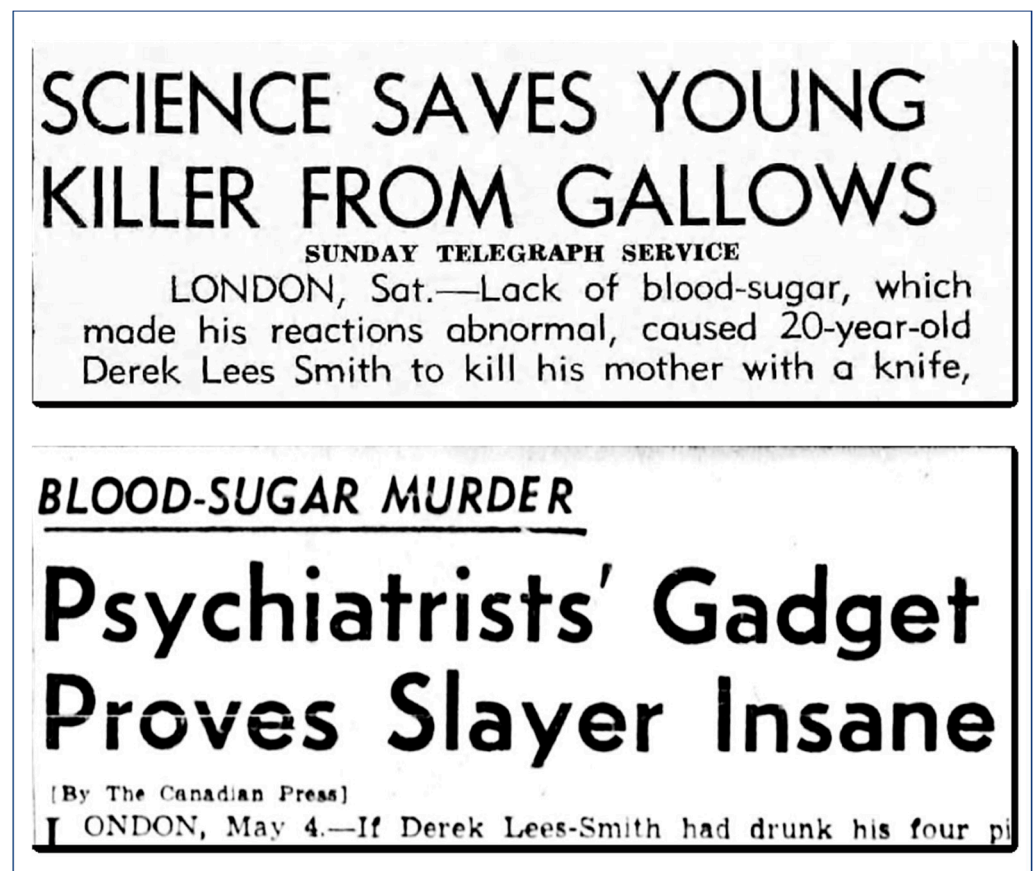
### 3. Nutritional Criminology and Food Crime

Links between dietary deficiencies, malnourishment, and criminal behavior emerged in the mid-20th century with a focus on juvenile delinquency. In the 1940s, it was recognized that deficiencies of niacin and other B vitamins are associated with anger and irritability (Cobb et al. 1943; Bell 1958). There were also observations that insulin-induced hypoglycemia causes irritability, which could be tempered with dietary changes reducing sugar and refined foods (Harris 1924). Joseph Wilder, a professor of neurology at New York Medical College, reported multiple cases of impulsivity, and antisocial, violent, and criminal behavior, in association with hypoglycemia (Wilder 1940, 1943). Wilder argued that hypoglycemia causes increased emotional reactivity and loss of central inhibitory control (Wilder 1947). This period also witnessed the origins of neurolaw—the first use of EEG in cases determining criminal liability (Anon 1939). In the 1940s, several studies suggested that EEG signatures could differentiate adults with aggressive/antisocial/violent tendencies from healthy, well-adjusted populations (Knott and Gottlieb 1944; Silverman 1944; Brill et al. 1942), and more specifically, that EEG abnormalities could differentiate impulsive, motiveless murderers from those who planned with motive (Staffordclark and Taylor 1949).

At this wellspring of neurolaw, diet emerged as a central feature: in the 1943 criminal trial of 20-year-old Derek Thayer Lees-Smith, who had fatally stabbed his mother in an impulsive act, defense psychiatrist (Sir) John D. Hill used EEG testing to show that the defendant had post-prandial hypoglycemia, which, in turn, led to disturbed brain functioning. Hill testified that Lees-Smith knew right from wrong, he knew what he was doing, yet due to the hypoglycemia-induced brain alterations (as evidenced by EEG recordings), the defendant was unable to control his violent impulse. The jury found Lees-Smith guilty but insane—not generally insane, but insane at the precise time of the matricide. Since the jury found that Lees-Smith was not responsible for his action, he was spared death by hanging and sent to a medical institution. Hill wrote the case up in the *Lancet* (Hill et al. 1943), emphasizing that Lees-Smith had eaten poorly on the day of the crime and stabbed his mother while en route to the refrigerator to quench a sudden desire for a sugary soft drink. International headlines emerged, such as “The Case of the Sugar Starved Murderer” (Anon 1943), “Blood Sugar Murder: Psychiatrists' Gadget Proves Slayer Insane” (Canadian Press International 1943), and “Science Saves Young Killer From Gallows” (Sunday Telegraph Press 1943) (Figure 2).

The mid-20th century also witnessed the first intervention studies targeting mental disorders with nutritional supplementation. In a single-blind study published in the *Journal of Psychology*, Dr. George Watson, a proponent of biological psychiatry and staunch critic of Freudian pseudoscience (Watson 1956), reported that that an oral nutrient–food supplement (vitamin/mineral formula with some added ingredients such as alfalfa, watercress, seaweed, parsley, etc.) could improve symptoms in persons with depression and

anxiety (Watson and Comrey 1954). Although Watson and others published additional studies (Watson and Currier 1960), the grip of Freudian pseudoscience on North American psychiatry held firm until the early 1970s (Lattey 1969; Rippere 1983).



**Figure 2.** Headlines in Australia (top) and Canada (bottom) typify global media coverage of the EEG and blood sugar evidence used in the 1943 Lees-Smith case.

Beginning in the 1980s, researchers began reporting on quasi-experimental dietary intervention trials in correctional settings—removing ultra-processed foods and replacing them with less-processed foods lower in sugar and higher in polyphenols and fiber—concluding that the changes were producing positive outcomes, including decreased antisocial behavior (Schoenthaler 1984; Schoenthaler and Bier 1985; Logan and Schoenthaler 2023). These were followed up with better-designed, but more reductionist, nutritional supplement intervention trials (vs. placebo), again demonstrating that nutrients can improve antisocial behavior, rule violations, aggression, and/or violence, in correctional or institutional settings (Gesch et al. 2002; Zaalberg et al. 2010; de Bles et al. 2022; S. Schoenthaler et al. 2023).

Today, the rapid growth of research in the transdisciplinary field of nutrition and behavior, supported by top-down nutritional epidemiology, bottom-up pre-clinical and microbiome studies (illuminating biophysiological mechanisms), and clinical intervention trials, has provided clear support for the idea that nutrition influences cognition and behavior. Advances in the field (often referred to interchangeably as nutritional neuroscience, nutritional psychology, nutritional psychiatry) are occurring in tandem with volumes of studies illustrating the potential harms associated with the consumption of what are termed ultra-processed foods (Lane et al. 2024). Consumption of these foods (use of the word ‘food’ in reference to ultra-processed products is debated (Schoenthaler and Logan 2023)) has been linked to cognitive difficulties, mental disorders, emotional distress, aggression, and antisocial activity. While the generalized Western dietary pattern of high-sugar/-

fat/-sodium foods has been linked to changes in hippocampal volume (Jacka et al. 2015; Stadterman et al. 2020), emerging research is showing that a more specifically identified ultra-processed dietary pattern is associated with lower grey matter volumes in mesocorticolimbic (i.e., reward processing) brain regions (Contreras-Rodriguez et al. 2023). Moreover, structural neuroimaging studies have linked processed food consumption in early through mid-childhood with differences in brain morphology, which may explain the observed relationships between dietary patterns and neurodevelopment in children (Mou et al. 2023).

Collectively, the potential intersection of this research with the criminal justice system, at both the individual and community levels, and in prevention and treatment efforts, is described as nutritional criminology (Prescott et al. 2024). The available research indicates that nutritional interventions, both dietary and supplemental, can influence outcomes of relevance to antisocial behavior and aggression. Related to this research is the emerging realm known as food crime, an area of inquiry that scrutinizes the tactics of the ultra-processed food industry, and potential culpability for harms associated with the manufacturing, marketing, and distribution, of such products (Robinson 2022a, 2022b). We will now turn our attention to food-based cadmium as a possible (if not probable) explanation for the cadmium in Huberty's hair.

#### 4. Cadmium and Antisocial Activity

The United States Environmental Protection Agency (EPA) recognizes the multiple health hazards associated with cadmium, and sets an enforceable Maximum Contaminant Level (MCL) of 5 parts per billion (ppb) in drinking water (United States Environmental Protection Agency (EPA) 2002). Although the refining of foods during processing can reduce heavy metals, in the case of cadmium, the levels are higher in refined vs. whole grains (Thielecke and Nugent 2018); currently, French fries served at fast-food outlets represent one of the most significant sources of dietary cadmium in North America, with samples containing cadmium at levels reported to be as much as 1158% higher than the EPA's MCL for safe drinking water (EIN Presswire 2023). Cadmium exposure via food consumption leads to higher levels of hair cadmium (Liu et al. 2024).

Rodent studies using an intruder model (i.e., a non-familiar rodent of the same or different species is introduced into a resident animal's cage) have demonstrated that cadmium exposure significantly increases the likelihood that the resident animal will make a lethally violent attack on the intruder (Heihachiro et al. 1981). In a recent intruder-model study involving cadmium exposure (at levels similar to those found in French fries), researchers reported that the total number of attacks, total duration of attack manifestations, and composite aggression scores (by the resident against the intruder) are significantly increased when the resident animal had just been subjected to a separate stress (Terçariol et al. 2011); that is, chronic exposure to cadmium, mixed in with existing psychological distress, is a toxic union—potentially lethal to the innocent. Animal studies also demonstrate that cadmium exposure promotes behaviors reflective of human anxiety, and that healthy dietary components, such as quercetin and other plant-based antioxidants (the sort absent in a dominant ultra-processed food diet), can help mitigate these behavioral disturbances (Abdalla et al. 2014; Adebisi et al. 2022).

Lead has long been associated with brain pathology, and the chronic, low-grade exposure to environmentally sourced lead as a path toward human cognitive and behavioral disturbances (including learning/developmental disorders, aggression, and violence) has been referred to as the "neurotoxicity hypothesis" (Needleman 1995). Epidemiological research demonstrates significant relationships between lead exposure and violent crime (Higney et al. 2022; Talayero et al. 2023). In any case, at the time of *Huberty v. McDonald's*, research had shown that the combination of elevated lead and cadmium (as measured in hair samples) was a predictor of lower verbal/nonverbal skills (Thatcher et al. 1982) and aggressive behavior in juveniles (Marlowe et al. 1985). In adults with a violent criminal history (vs. non-violent incarcerated adults), hair cadmium and lead levels were found to be higher (Pihl and Ervin 1990). While older hair analysis studies have been disputed due

to methodological issues, contemporary human studies have noted higher blood and/or urine cadmium levels in bipolar disorder (González-Estecha et al. 2011), depression (Berk et al. 2014; L. Yang et al. 2023), schizophrenia (Arinola et al. 2010), anxiety (Gui et al. 2023), and, of relevance to a retrospective analysis of *Huberty v. McDonald's*, “non-planning impulsiveness” (Comai et al. 2019).

Animals exposed to a stressor have more cadmium in the brain, which supports the theory that stress-induced blood–brain barrier (BBB) disruption can lead to cadmium distribution in areas governing aggression (Terçariol et al. 2011). Lack of dietary antioxidants also appears to be a factor in disturbances to the BBB (Kim et al. 2022). Thus, the presence of cadmium-rich French fries and the absence of polyphenol-rich foods are two sides of the same coin (Prescott and Logan 2017). Similar stress and dietary-related disruptions of the BBB are also thought to play a significant role in MSG-associated disturbances, discussed in more detail below.

### 5. Monosodium Glutamate and Neuropsychology

In *Huberty v. McDonald's*, the corporation provided a sworn affidavit from Michael J. Goldblatt, its director of nutrition. Goldblatt affirmed that during the (approximate) one year period prior to the mass shooting, over 400 million pieces of McNuggets were served in the United States, without a single report (other than Huberty's) of post-consumption psychosis or violent behavior (Goldblatt 1987). McDonald's also secured expert testimony from Andrew G. Ebert, a pharmacologist with long-term ties to the MSG industry. Ebert held a senior position at Robert H. Kellen, an overarching firm that represented clients such as RJ Reynolds Nabisco, the Coca-Cola Co., PepsiCo, General Mills, Kraft, the General Foods Co., the Glutamate Association, and the International Glutamate Technical Committee. Kellen was president of the Glutamate Association (Gill 1987), and Ebert was chair of the International Glutamate Technical Committee. In 1987, Kellen acknowledged to the *Atlanta Journal-Constitution* that his group (and trade organization sub-groups where he served as president) was responsible for pressuring Congress to thwart potential bans on food additives; he also acknowledged that it was lucrative work, supporting his collection of high-end art: “there is some suspicion that I opened our Washington [lobbying] office to have more wall space for my art” (Herndon 1987). In *Huberty v. McDonald's*, the court records note that Ebert was the author of a “Monosodium Glutamate Press Information Kit,” and a booklet called “The Remarkable Story of Glutamate,” both published by the “International Glutamate Technical Committee” out of its K Street address in Washington, DC.

In his testimony, Ebert stated that, given the extensive body of research on MSG safety, “if MSG induced violent behavior, such behavior would have become evident. . .ingested MSG does not represent a hazard in psychiatric or normal persons. . .nowhere, to my knowledge, has there been any claim that MSG induced or contributed to violent behavior.” Ebert then focused on the BBB, claiming that the barrier is protective against glutamate's entry into the brain, that the brain is well equipped to export glutamate out of the brain, and that there is no evidence that cadmium or lead would have caused a more porous barrier: “There is no scientific evidence to show that lead or cadmium in the blood would effect the restricting properties of the barrier” (Ebert 1987). Despite Ebert's claims, there was already research indicating that heavy metals can have a potentially toxic effect on the capillaries that make up the BBB (Goldstein 1984). As mentioned above, it is now clear that cadmium can damage the integrity of the normal BBB.

Ebert made no mention of the large body of animal studies, dating back to the late 1960s, indicating that MSG does have neurotoxic activity (Olney 1969, 1973, 1989). He made no mention of human studies from the 1940s showing that MSG was used as a brain stimulant to awaken persons with schizophrenia who had been subjected (i.e., “treated”) to insulin-induced coma, or so-called shock therapy (Mayer-Gross and Walker 1949). He made no reference to studies showing that glutamic acid increased spontaneous motor activity in persons with schizophrenia (Ewalt and Bruce 1948), and that the persons most likely to respond were catatonic; researchers reported that with increasing doses of glutamic

acid, subjects would experience distractibility or attention deficit, including “an increase in excitability, hostility, and lack of emotional control” (Kitzinger et al. 1949).

While there were no case reports that MSG caused completed violence per se, Ebert made no mention of a 1978 case published in the *New England Journal of Medicine*. In the case report, Arthur D. Colman, a physician and professor at the University of California, San Francisco, described post-MSG cognitive–behavioral changes in someone he knew very well—his spouse. Colman’s 36-year-old wife, who had no history of neuropsychiatric problems, experienced a collection of acute physical symptoms after MSG consumption (e.g., skin flushing, chest tightness, abdominal discomfort), and a longer-term psychologic effect that began after about 48 h, and lasted for approximately two weeks; included in the cognitive–behavioral reactions were “paranoia”, “gloomy fantasies”, “unanticipated outbursts of rage”, and the perception that other people were “strange and ominous”. When this period passed, there were no sequelae, and the experience was described as a “bad dream;” these reactions, subsequent to dining in restaurants where MSG was in use, were noticed by other friends and family members. During a symptom-free period, she intentionally consumed an MSG-containing soup, and on a separate occasion, a 99% pure MSG “seasoning” powder, and the psychosis-like symptoms emerged (Colman 1978). Ebert knew about the publication of Colman’s case because he responded to it in the press at the time, referring to it as unfounded and weak (Associated Press 1978).

MSG is not the only dietary excitotoxin in the fast-food arena. The artificial sweetener aspartame is known to lower the uptake of tryptophan into the mammalian brain, leading to reduced serotonin production (Sharma and Coulombe 1987), which might explain the observation of aspartame-induced aggression in rodents (Kring 1997). Animal studies have linked MSG to aggressive behavior (Shivasharan et al. 2013; Cammaerts and Cammaerts 2016; Swamy et al. 2013; M. Sharma et al. 2023). It is interesting to note that among the ultra-processed foods connected to depression, those containing artificial sweeteners appear to have the strongest relationship (Samuthpongton et al. 2023). Human research examining the acute effects of dietary excitotoxins on aggression and antisocial activity is wanting; again, much like hypoglycemia, a good place to start would be to examine dietary excitotoxin consumption in the context of post-prandial social-stress experiments where aggression is often observed (e.g., lab road-rage experiments).

There are numerous mechanisms by which glutamate, the primary component of MSG, might cause neuropsychiatric symptoms, although the most obvious is that the chemical, and related dietary excitotoxins such as aspartame, cause overexcitation of neurons (Olney 1990; Rycerz and Jaworska-Adamu 2013); a growing number of animal studies indicate that oral MSG and aspartame can lead to abnormal behaviors in animals, including those that mimic depression and/or anxiety (Chakraborty 2019; Kraal et al. 2020; Brant et al. 2023; Ashok et al. 2014; Choudhary and Lee 2018; Erbaş et al. 2018; Jones et al. 2022; Fowler et al. 2023; O.J. Onalapo et al. 2012). MSG reduces brain-derived neurotrophic factor (BDNF), a natural brain chemical that otherwise supports the differentiation, maturation, and survival of neurons (Rosa et al. 2016; Gürgen et al. 2021); BDNF has been linked to aggression, impulsivity, and violence, in multiple animal and human studies (Maynard et al. 2016; Yochum et al. 2014; Ito et al. 2011; Martinotti et al. 2015; Y. S. Wu et al. 2017). Recent human studies have found that the elimination/low intake of excitotoxin additives, including aspartame and MSG, or MSG-like chemicals, can improve symptoms of depression, anxiety, post-traumatic stress disorder (PTSD) (Murray and Holton 2022; Brandley et al. 2022), and fibromyalgia (Holton et al. 2012); this includes improved depression, anxiety, cognitive function and reduced pain sensitivity in veterans with Gulf War illness (Kirkland et al. 2022; Langan et al. 2022; Holton et al. 2020).

There is little doubt that for most adults, modest amounts of MSG and related dietary excitotoxins can be consumed without obvious neuropsychiatric consequences. What individual differences could explain why dietary excitotoxins provoke symptoms in a relative minority? As mentioned earlier, stress-induced BBB permeability could account for increased access to the brain; disturbances to normal blood–brain barrier structure



and function can be influenced by psychological trauma and acute and chronic stress, and may have a bidirectional relationship with mental illness (Dion-Albert et al. 2022). In animal studies, MSG administration increases the burden of inflammation and oxidative stress, and lowers the amount of serotonin and gamma-aminobutyric acid (GABA) in the brain (El-Hashash et al. 2023; Albrakati 2023; Ankul et al. 2023). GABA is the primary inhibitory neurotransmitter in the central nervous system, and low levels have been linked to schizophrenia, major depression, and anxiety disorders (Allen et al. 2023).

Individuals with chronic inflammatory illnesses have been reported to have higher sensitivities to MSG, and this has been theorized to be a product of both increased intestinal permeability (so-called 'leaky gut') allowing increased glutamate into the blood, and increased BBB permeability—a 'double-hit' against normal regulatory mechanisms (Logan 2003). This double hit of increased intestinal and blood–brain barrier permeability can be compounded by a high-fat, ultra-processed food diet, which disturbs the normal gut microbiota ecosystem (dysbiosis), with resultant changes to the expression of tight junction proteins that otherwise control barrier function (Wu et al. 2023; Braniste et al. 2014; Noble et al. 2017; Dion-Albert et al. 2022). Indeed, intestinal permeability increases the likelihood that gut microbial breakdown products, such as lipopolysaccharide endotoxin (LPS), enter circulation. When this occurs, LPS can work in synergy with dietary chemicals such as MSG to promote neuroinflammation and dysfunction of neurotransmission (Asejeje et al. 2024). Differential levels of intestinal and/or BBB permeability can help explain why subsets of the population might be more vulnerable to the consumption of dietary excitotoxins. We next turn our attention to the microbiome.

## 6. Microbiome and the Legalome

Research emerging from the transdisciplinary field of microbiome sciences, especially that related to the gut microbe–brain axis, is forcing hard questions into the social sciences, humanities, and law. The available evidence indicates that human cognition and behavior can influence the microbes that live on and within each of us, yet at the same time, our own cognitions and behaviors are influenced by microbes. For example, human intervention studies using oral non-pathogenic microbes (e.g., probiotics, postbiotics) and/or agents that can positively influence the gut microbial ecosystem (e.g., prebiotics) have been shown to lower anxiety, depression, and distress, and improve sleep (Musazadeh et al. 2023; Z. Zhao et al. 2023; Mutoh et al. 2023; Nishida et al. 2019; Chan et al. 2023). Researchers are actively trying to determine whether certain gut microbial signatures are associated with temperament (Sumich et al. 2022), violent tendencies (Chen et al. 2021), and regulation of emotions (Ke et al. 2023). Emerging human studies using specific strains of probiotics indicate that targeting the gut microbiome might lower aggressive thoughts (Steenbergen et al. 2015; Walden et al. 2023) and impulsivity (Arteaga-Henriquez et al. 2020). It is already known that manipulating the microbiome with orally consumed probiotics can influence human mental outlook (S. Zhao et al. 2024), and objective neuroimaging studies continue to link gut microbes with brain structure and function (Bagga et al. 2019; Zhang et al. 2023).

One harbinger of the legalome—microbiome and omics science applied in neuro-law and the larger legal system—is the growing number of fecal transplant (aka, fecal transfer) studies that demonstrate that gut microbes influence physiology and behavior. When the fecal material of animals with diet- or stress-induced dysbiosis is transplanted into otherwise healthy animals, the recipients have similar observable neuropsychiatric disturbances to those found in the dysbiotic donors (Bruce-Keller et al. 2015; N. Li et al. 2019). The reverse also appears to be the case—transplant of fecal material from healthy animals has been found to improve behavioral signs of depression and alcohol-seeking in an animal model of alcohol dependence; the potential mechanisms include improved intestinal barrier function and changes to brain serotonin turnover (Li et al. 2023). Behavioral changes via microbiota transfer have also been observed when the fecal material originated from human donors with behavioral disorders; for example, microbiota from alcohol-dependent patients induced the behavioral alterations associated with alcohol

dependence in recipient lab animals, including increased anxiety- and depression-like behaviors, reduced exploratory and recognition memory, and higher alcohol preference; these behavioral changes were accompanied by objective brain-related signals known to be associated with alcohol dependence (C. Wang et al. 2023).

When fecal material from human donors with social anxiety disorder is transplanted into otherwise healthy recipient animals, the recipients develop a heightened sensitivity to social fear (Ritz et al. 2024); similar designs using fecal material from human adults with schizophrenia (vs. healthy adults) show that recipient animal behaviors are disturbed, metabolic pathways are altered, and brain GABA is lowered (Zheng et al. 2019; Zhu et al. 2020). Remarkably, the transfer of fecal material from human infants with disruptions to normal microbiome development (via administration of antibiotics) leads to aggressive-like behavior in recipient lab animals, observations not seen with the transfer of microbiota from healthy infants (Uzan-Yulzari et al. 2023). These animal studies are supported by a small but growing number of human studies indicating that fecal transplants can improve psychiatric symptoms (Vasiliu 2023). Taken as a whole, and when understood in the context that dietary patterns and components (both nutritive and non-nutritive) are primary drivers of dysbiosis, the relevance to neurolaw is obvious.

These microbiome studies allow us to revisit the central factors in *Huberty v. McDonald's*—dietary excitotoxins and cadmium—through a new lens. It is now known that cadmium causes gut dysbiosis and disturbances to the intestinal barrier (Liu et al. 2014; Liu et al. 2020). Fecal transplant research shows that cadmium-included gut dysbiosis can be transferred to healthy recipient animals, with resultant metabolic disturbances in the recipients (Yang et al. 2021). MSG has also been shown to alter the gut microbiome (Naimi et al. 2021; Feng et al. 2015; Nahok et al. 2021; Kyaw et al. 2022) and dysbiosis appears to increase blood glutamate levels (Liu et al. 2017). Even a single fast-food meal (inclusive of McDonald's fries, chicken tenders, soft drink, and milk shake) is enough to reshape the gut microbial community in rodents, yielding a unique signature of food-derived microbial metabolites (Osborn et al. 2021). Fried (vs. boiled/steamed) chicken has been shown to promote dysbiosis, with resultant disturbances in glucose homeostasis and increased systemic inflammation (Gao et al. 2021). Fried foods, including French fries, contain significant amounts of acrylamide, a chemical formed when foods are cooked with high heat in the absence of water; in a familiar theme, acrylamide has been shown to cause gut dysbiosis and inflammation (Yue et al. 2022; Z. Wang et al. 2021). This might help explain why fried food consumption by humans predicts neuroinflammation and anxiety and/or depression (Wang et al. 2023). In addition, a steady diet of fried chicken and ice cream is notable for its absence of omega-3 essential fatty acids (Marriott et al. 2014; Young et al. 2017); this is important because a lack of dietary omega-3 fatty acids has been linked to human aggression (Raine et al. 2020) and gut dysbiosis (Kerman et al. 2023). Indeed, a consistent intake of high-fat fast foods is associated with higher body levels of an industrial chemical class known as phthalates (Zota et al. 2016); this class of chemicals is known to provoke dysbiosis (Goyal and Saravanan 2023) and has been linked to emotional reactivity and aggression (Hliseníková et al. 2021).

More broadly, dietary patterns dominated by ultra-processed foods (with relative absence of minimally processed, fiber- and polyphenol-rich plant foods) are associated with gut dysbiosis. This takes us to the question of addiction, and why so many consumers of hyperpalatable ultra-processed foods meet food addiction criteria (LaFata and Gearhardt 2022; Delgado-Rodríguez et al. 2023). As many as 14% of adults meet ultra-processed food addiction criteria (Gearhardt et al. 2023), and persons living with socioeconomic vulnerabilities appear particularly susceptible to ultra-processed food addiction (Leung et al. 2023). If Etna Huberty's claims of 'addiction' are taken at face value, why would her husband have such strong cravings for McDonald's McNuggets, fries, and ice cream, and soups containing MSG? Emerging evidence suggests that the combination of refined carbohydrate and fat can influence the endogenous opioid and mesolimbic dopaminergic pathways, while enhancing somatosensory reward (Gearhardt and Schulte 2021). Although

dietary excitotoxins are often overlooked in discussions of ultra-processed food addiction, animal studies indicate that MSG can stimulate the brain's reward system (Onaolapo et al. 2017) and encourage food addiction (Buzescu et al. 2013).

Recent research involving young adults shows that the craving associated with binge drinking is strongly linked with alterations in microbiome composition and social cognition over time (Carbia et al. 2023). Since a normal, diverse gut microbiome appears to suppress palatable food cravings in animals, it is possible that once diet and/or stress-induced dysbiosis is set in place, the disturbed microbiome actively contributes to further cravings for highly palatable, but unhealthy, foods (Ousey et al. 2023; Fan et al. 2023). The consumption of flavor enhancers such as MSG might also influence behavior depending on the specific food to which it is added, such as meat. It is worth noting that emerging human research has linked higher levels of meat consumption with increased aggression toward an intimate partner (Taft et al. 2023). In animal studies, a high-fat, high-chicken-meat diet is associated with disturbances to the gut microbiota (Shi et al. 2020); if this research is replicated and extended, researchers should query the types of meat (highly processed, inclusive of dietary excitotoxins?), the types of foods that surround meat consumption (phytochemical- and fiber-rich fruits and vegetables, or ultra-processed foods?), and potential overlaps with addiction and microbiome sciences.

## 7. Conclusions

The origins of neurolaw can be traced to the well-publicized criminal case of Derek Thayer Lees-Smith and the use of EEG to link dietary sugar and behavior. Just over 40 years later, the civil case of *Huberty v. McDonald's* attempted to once again link diet and violent behavior. Now, another 40 years later, advances in a variety of branches of science are converging to give legitimacy to links between diet and criminology. Emergent microbiome sciences are disrupting concepts of the biopsychosocial 'self' and what it means to be a human with free will (Rees et al. 2018; Ironstone 2019). The rapid evolution of microbiome science, as it intersects with nutritional components and numerous environmental variables, illuminated by omics technologies, has further expanded the boundaries of neuropsychiatry. Looking back on *Huberty v. McDonald's*, while holding cutting-edge research in hand, we can see that the legalome will take on increased relevancy to both criminal and civil law. The Huberty case was dismissed for want of knowledge. Four decades later, a significant body of scientific knowledge is in place, and although questions remain, it is very likely, given the existing evidence, that Judge Unger would move the case forward if it was presented in 2024. The implications for neurolaw are already obvious, and if research continues on its current trajectory, there seems little doubt that the legalome will be part of 21st-century courtroom discourse, policy, and decision-making.

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