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Opinion

Homicide or Happiness: Did Folate Fortification and Public Health Campaigns Influence Homicide Rates and the Great American Crime Decline?

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Abstract: The last several years have witnessed a remarkable growth in research directed at nutrition and behavior, with increased interest in the field of nutritional criminology. It is becoming clear that dietary patterns and specific nutrients play an important role in cognition and behavior, including those related to aggression, violence, and antisocial activity. Included in this expanding knowledge base is the recognition that folate, through multiple pathways, including enzymatic reactions and gut microbiome ecology, plays a critical role in central nervous system functioning. These mechanistic advances allow for a retrospective analysis of a topic that remains unexplained—the sudden and unpredicted drop in homicide and other violent crime rates in the United States and other nations in the 1990s. Here, we revisit this marked reduction in homicide rates through the lens of the coincident public health campaign (and subsequent mandatory fortification) to increase folic acid intake. Based on objectively measured blood folate levels through the National Health and Nutrition Examination Surveys, there is little doubt that tissue folate witnessed a dramatic rise at the national level from 1988 through 2000. Drawing from accumulated and emerging research on the neurobehavioral aspects of folate, it is our contention that this relatively sudden and massive increase in tissue folate levels may have contributed to reductions in violent crime in the United States.

Keywords: folate; behavior; criminal justice; nutritional criminology; mental health; microbiome; aggression; biological criminology; ultra-processed foods; violence

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1. Introduction

Recent years have witnessed a growing interest in the area of nutrition, mental health, and behavior. Most often, these studies have examined dietary factors in relation to depression, anxiety, and psychosis [1,2]. However, investigators have also examined the role of dietary patterns and select nutrients on outcomes such as impulsivity, aggression, violence, and antisocial behavior [3–6]. Research in the area of attention deficit hyperactivity disorder (ADHD) has helped to shed light on the role of nutrients and dietary patterns in impulsivity [7]. As a result, there is increased scrutiny of the intersection between nutrition and the criminal justice system, in terms of both treatment and prevention [8,9]. For example, omega-3 fatty acids, or a lack thereof, have been linked to impulsivity, suicide by violent means, and national homicide rates [10]. Although folate, part of the B vitamin family, has long-since been understood to play a critical role in supporting normal central nervous system function [11], and folic acid and other related compounds have been reported to improve depression and other mental disorders [12,13], its role in antisocial behavior and aggression is less often discussed.

Here, we suggest that the increased marketing of folic acid supplementation and subsequent food fortification, as a means to reduce neural tube defects, played a part in the relatively sudden and unexpected decrease in homicide rates and other violent crimes in the 1990s. This drop, often referred to as the Great American Crime Decline [14], witnessed a 43% reduction in homicide rates between 1991–2001, and a 34% reduction in violent crime over the same period [15] (Figures 1 and 2). While various social theories have been proposed for the rapid crime decline, no single theory emerges as definitive [16,17]. Also, of relevance to our later discussions on folate and behavior, suicide rates also decreased during this time period, especially among young adults [18] who are historically in the highest risk categories for the commission of homicide and violent crime.

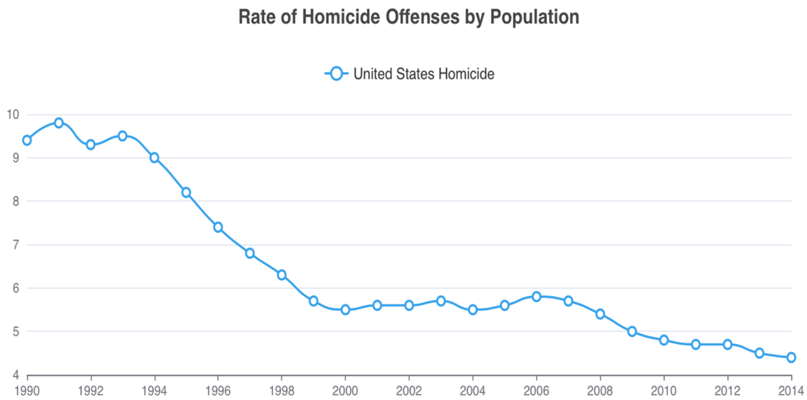

Figure 1. United States homicide rates per 100,000 from 1990 to 2014. From Federal Bureau of Investigation Crime Data Explorer https://cde.ucr.cjis.gov/.

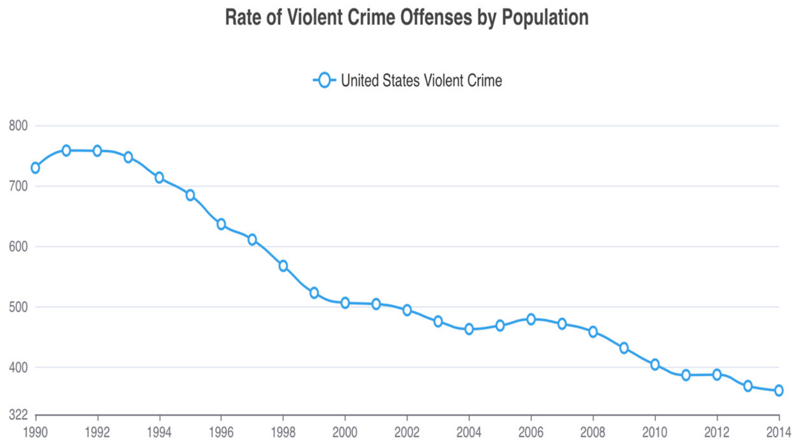

Figure 2. United States violent crime rates per 100,000 from 1990 to 2014. From Federal Bureau of Investigation Crime Data Explorer https://cde.ucr.cjis.gov/.

2. Folic Acid Recommendations

Beginning in the early to mid-1980s, there was increased recognition that prenatal folic acid supplementation appeared to cut the risk of neural tube defects [19]; in 1989, the results of a large cohort study (n = 23,491) showed that folic acid supplementation during the first six weeks of pregnancy resulted in neural tube defects at a rate of 0.9 per 100,000 births, compared to 3.5 per 100,000 for women who did not take a multivitamin or folic acid supplement [20]. The results, published in the Journal of the American Medical Association (JAMA), were highly publicized in front page newspaper articles throughout the United States, with highly regarded experts recommending that all women who are planning a pregnancy take a daily multivitamin inclusive of folic acid [21,22]. In the year following the JAMA article, 1990, Associated Press articles ran in nationwide newspapers, with quotations from experts supporting the use of perinatal multivitamins inclusive of folic acid [23]. This advice filtered into the clinical domain, as exemplified by one notable public health physician who maintained a regular newspaper column: *"If one of my daughters decides to have a child, I hope she will hedge her bets by taking multivitamins containing folic acid even before she becomes pregnant and throughout her pregnancy"* [24].

The point here is that even before the Centers for Disease Control formally recommended folic acid supplementation (400 mcg) for all women of childbearing age, in 1992 [25], there was already heightened public awareness. It seems safe to assume that although the media-driven awareness of folic acid was directed at women, it appeared to influence folate levels in both genders. We can base this assumption off the National Health and Nutrition Examination Survey (NHANES) evidence showing that the red blood cell folate levels for the entire US population, both men and women, increased significantly from 1988 through the 1990s. By 1999, the total number of persons in the US with what is considered to be "low" red blood cell folate dropped to 2.8%, whereas in 1988, over 30% of the population had low red blood cell folate. However, this average belies disparities, and the fact that almost 60% of Black females (aged 15 to 45) experienced low levels of blood folate by CDC standards; by 2000, the percentage of low folate among Black women had dropped to 12.1%. The median red blood cell folate levels of the entire US population aged 4 and older increased by over 60% from 1988 through 1999 [26] (Figures 3 and 4).

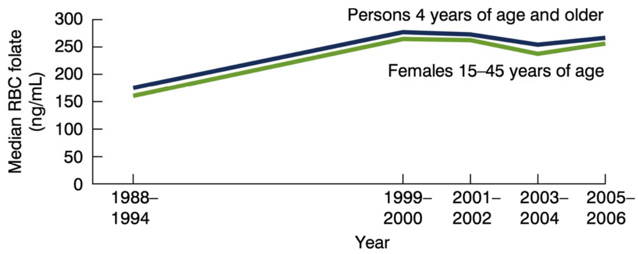

Figure 3. Red blood cell folate trends in the United States. Source: Source: United States Department of Health and Human Services Data Brief #6, May 2008.

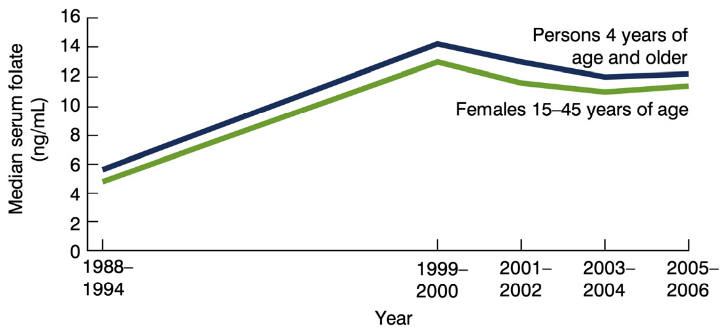

Figure 4. Serum folate trends in the United States. Source: United States Department of Health and Human Services Data Brief #6, May 2008.

The first attempts at evaluating red blood cell folate levels at the national level, the NHANES II study (1978–1980), revealed that Black males had significantly lower levels of folate than White males [27]. As mentioned, the period between 1988 and 2000 witnessed significant elevations in blood folate among Black adults (although as discussed below, disparities were, and still are, significant) [26]. Notwithstanding the difficulties with comparing disparate national surveys, in 1986 it was reported that 18.4% of all Black male adults consumed dietary supplements [28], while in the period 1988–1994, 26.7% of Black male adults consumed dietary supplements [29], and like most demographic groups, supplement use continued to increase, more recently noted to be 39.3% [30]. Of course, this does not indicate an increased intake of folic acid per se, but does reflect nutritional awareness and a potential increase in large-scale nutrient intake that predates mandatory folate food fortification. Although the mandatory fortification of most flours was effective from 1 January 1998, the Food and Drug Administration (FDA) had already announced mandatory fortification enforcement almost two years earlier, on 29 February 1996 [31].

The idea that nutrients such as folic acid could influence brain and behavior was certainly ongoing when the fields of nutritional neuroscience and nutritional psychiatry emerged in the 1980s, yet were only at the periphery of criminology and the criminal justice system. It is only with advances in research related to nutritional criminology and food crime [8,9] that it is possible to retrospectively examine cases and epidemiological trends that previously escaped explanatory (biological) discourse within the realm of criminal justice [32]. We now turn our attention toward such evidence, including preclinical work, population studies, and mechanistic pathways.

3. Folate, Brain, and Behavior

Links between deficiencies of various B vitamins and mental disturbances, including depressive symptoms, anxiety, and irritability, date back to the 1940s [33]. Reports of clinical improvement in mental disorders (including irritability reductions) with B vitamin complex supplements (inclusive of 1500 mcg of folic acid) vs. placebo were first reported in 1954 [34]. In the decades that followed, researchers reported that individuals with mental disorders—ranging from anxiety to schizophrenia, and depression to substance abuse—are more likely to have low blood folate compared to other healthy populations [35,36]. These findings led to the pursuit of possible mechanistic pathways that might explain why folate deficiencies are associated with wide-ranging mental and behavioral symptoms, and why folic acid supplements seem to improve clinical outcomes [35,37]. Folate is at the cornerstone of the one-carbon metabolism. It is involved in numerous enzymatic reactions and metabolic pathways, including mitochondrial function [38] and neurotransmitter production and enzymatic breakdown via S-adenosylmethionine and other methyl reactions. In animal studies, it has been observed that folate deficiencies can lead to alterations in serotonin, dopamine, norepinephrine, and acetylcholine [39–41]. Serotonin, for example, appears to promote prosocial behavior by enhancing aversion to harming others [42]. These alterations to serotonergic function and other functional changes in the central nervous system can help explain the loss of impulse control and increased aggression in folate-deficient animals [43,44] (Figures 3 and 4).

Although folic acid has not been studied as a stand-alone intervention in justice-involved individuals, several controlled studies in correctional facilities have examined multivitamin–mineral formulas, inclusive of folic acid. These studies have found reductions in antisocial behavior, rule-breaking, and violence [45–48]. Combined, this work supports older human studies indicating that the correction of low blood folate with folic acid supplements can improve neurobehavioral outcomes [37].

While folate levels and human violence and aggression have received little epidemiological and clinical attention, such is not the case with suicide. For example, epidemiological research shows that low serum folate is associated with various suicidal behaviors, including previous suicide attempts, suicidal severity, and fatal/non-fatal suicide attempts [49]. In a general population study in the United States, involving prescription claims of over 866,000 adults, a prescription for folic acid was associated with a 44% reduction in suicidal events [50]. These positive results at the general population level were extended to the narrower group of individuals with known psychiatric diagnosis, a history of prior suicidal behavior, and/or receiving a prescription for psychotropic medication; once again, folic acid prescription is robustly associated with lower suicidal event rates in higher suicide risk populations [51]. In addition to calls for controlled trials involving the inclusion of folic acid administration in high-risk psychiatric populations, there is a need to closely examine relationships between folate levels and/or folic acid prescriptions and the means by which completed suicide occurred. Violent methods of suicide (e.g., use of firearms, hanging, jump from high places, immolation, as compared to drug overdose, gas/suffocation, poison) are associated with higher levels of lifetime aggression and impulsivity [52] and distinct neurobiological underpinnings [53]. Included in those biological distinctions related to suicide by violent means are alterations in purinergic signaling and mitochondrial metabolism [54], both of which can be influenced by folate [55].

As mentioned earlier, inadequate omega-3 fatty acid intake (or deficiencies) has been linked to aggression, impulsivity, and suicide risk [56,57]; however, it is worth noting that folate, by way of its influence on methylation reactions, is responsible for the transport of omega-3 fatty acids to target tissue [58]. In animals, the administration of folate can increase plasma and red blood cell levels of omega-3 fatty acids [59], and diets deficient in folate lead to reductions in blood and nervous system tissue omega-3 levels [60,61]. This supports human research involving adults with aggression and hostility, wherein there is a positive association between blood folate levels and higher tissue omega-3 levels [62], and separate research showing that medication-naïve first-episode psychotic patients have a tandem of low folate and omega-3 levels [63]. Thus, in many ways, discussions of omega-3 fatty acids in the context of criminology, including their influence on cell signaling, neuroinflammation, and mitochondrial membrane functioning, are discussions of folate. Taken together, the available research suggests a need for folate (single-nutrient) intervention trials involving vulnerable populations, including justice-involved persons and others at-risk. Only well-designed intervention trials can overcome some of the biases and confounders that have plagued older studies. Recent evidence-based clinical guidelines issued by the World Federation of Societies of Biological Psychiatry and the Canadian Network for Mood and Anxiety Treatments Taskforce have recommended methylfolate for adjunctive use in depression and schizophrenia [64]. Given the potential value, it is surprising that there has been so little research within correctional settings.

4. Gut-Brain-Microbiome Links

One exciting area of research that unites discussions of folate and omega-3 fatty acids is the relationship between gut microbes and mental health. Since the development of the first scientific frameworks linking non-pathogenic microbes with mental health and cognition, in the early 2000s [65,66], it has become increasingly clear that the gut microbiome plays an important role in neuropsychiatry [67,68]. There are multiple mechanisms by which gut microbes can influence brain and behavior. These include, but are not limited to, direct communication to the brain via gut-innervating vagus and spinal nerves [69], indirect pathways via influences on systemic immune chemicals (e.g., cytokines) and neuropeptides/hormonal messengers that are well-known to impact mood and behavior [70], influencing the absorption of nutrients [71], and maintaining the integrity of the intestinal barrier (i.e., preventing intestinal permeability or so-called "leaky gut") [72]. It is worth noting that a compromised intestinal barrier has been linked to increased aggression [73]. Emerging evidence is connecting specific gut microbes and/or microbial signatures with personality features, including temperament [74], propensity for violence [75], and the regulation of emotions [76]. In addition, human intervention studies suggest that targeting the gut microbiome with beneficial microbes might lower aggressive thoughts [77] and impulsivity [78].

Since dietary patterns and nutrients play a critical role in shaping the human microbiome, the status of the gut microbial ecosystem has been postulated to play an important mechanistic role in behavioral outcomes, including those relevant to violence and aggression [3,79]. Strong support for a microbiome-behavior link can be found in fecal transplant studies; that is, when fecal material from animals with nutrient-related or stress-induced dysbiosis (or from human donors with mental disorders) is transplanted into otherwise healthy animals, the recipients have observable neuropsychiatric disturbances, as found in the dysbiotic donors [80–83]. For example, the transfer of fecal material from human infants with dysbiosis (vs. the transfer of microbiota from healthy animals) to recipient lab animals leads to aggressive-like behavior in recipients lab animals [84]. Mechanistic pathways have been illuminated—recipients of dysbiotic microbiota are noted to display alterations to metabolic pathways, disturbances of the intestinal barrier, changes in neurotransmitter levels (e.g., serotonin and gamma-aminobutyric acid), and micro-RNA alterations in the frontal cortex [85–88]. While the topic of dietary patterns and nutrition in relation to the gut microbiome is complex, several studies have shown that folate deficiency is associated with disturbances to the gut microbiome in animals [89–91], and that folic acid supplementation can prevent dysbiosis in various experimental chronic disease models [92,93]. In sum, there are certainly plausible mechanisms whereby folate deficiency/folic acid supplementation can influence irritability, aggression, impulsivity, and mental outlook.

5. Violent Suicide and Homicide—Complexity

It is understood that violent/non-violent suicide and homicide are complex subjects, and rates of both can be influenced by many environmental and socioeconomic factors, [94–97], regardless of optimal folic acid intakes. The same complexity applies to neural tube defects. It is worth noting that the levels of neural tube birth defects in the United States were already dropping before folic acid food fortification began in 1998, and this is to be expected based on the aforementioned public health campaigns dating back to the 1980s. However, in the two decades following mandatory fortification, rates of (folate-related) birth defects have not declined according to expected trajectories [98]. Despite significant gains in objectively measured serum and red blood cell folate across the United States population from 1988 through 2000, more recent measurements, in the years following fortification, have witnessed a small but meaningful decline (7% for serum and 12% for RBC) in folate levels [99].

The success of nutritional campaigns to lower folate-related birth defects may be dependent upon methyl donors and other nutrients that are involved in folate metabolism. These include nutrients such as vitamins B6 and B12, thiamine, riboflavin, zinc, choline, betaine, and methionine [100]. There are still large segments of the United States population with subpar intake of folic acid [101], including persons living with food insecurity [102,103], and research shows that neighborhood socioeconomic deprivation predicts neural tube defects [104].

It is also important to point out that our discourse is focused on homicide, suicide, and violent crime trends in the United States. In this context, the United States stands as an outlier. For example, the rates of homicide in the United States were, and continue to be, much higher than other high-income countries—in studies based on 2010 and 2015 statistics, homicide in the US was 7 to 8 times higher than in other high-income countries, and rates of violent suicide by firearm were 8 to 10 times higher [105,106]. The extent to which folic acid supplementation influences large-scale outcomes is, of course, dependent upon shifting environmental factors. For example, the less-than-expected declines in post-fortification neural tube defects may be due, at least in part, to rising obesity rates [98]. Overweight and obese individuals have lower serum folate concentrations when compared with individuals with normal weight; this difference may be mediated, at least in part, by the microbiome and changes in intestinal absorption [107]. The intake of energy from low-nutrient, hyper-palatable, and obesogenic ultra-processed foods has increased among adults in the United States in the years following fortification [108,109]. The dominant foods in supermarkets throughout the United States are now ultra-processed—approximately 58% of staples in U.S. leading supermarkets are ultra-processed, which is 41% more than supermarkets in Europe [110]. Moreover, hyper-palatable and ultra-processed foods have been heavily marketed in marginalized communities [111,112] wherein socioeconomic risk factors for violent offending and victimization are already elevated. At the biological level, these foods, and the presence/absence of nutrients, intersect with biopsychosocial vulnerabilities [113]. In the context of nutritional criminology, the increasing dominance of these foods, and their relationship to central nervous system structure and function, may have a detrimental effect on behavior, one that can paste over the potential benefits of folic acid supplementation.

6. Conclusions

Multiple theories have been used to explain the sudden and unpredicted drop in homicide and violent crimes through the 1990s. These include, but are not limited to, changes in the illicit drug markets, aggressive policing policies (especially those directed at illegal guns), incapacitation through the expansion of prison populations, and improved economic conditions [15,114]. It is not our contention that these intersecting explanations are without merit. We are simply presenting an additional input. It is our view that an unprecedented chemical experiment—one that produced dramatic shifts in tissue levels of a nutrient chemical at the mass population level—is now supported by biophysiological plausibility as a determinant of behavior. Of course, our observations of coincident increases in tissue folate and decreases in violent crime, throughout the 1990s, are merely associations and not proof of causation. However, the idea that a relatively sudden and significant rise in a blood chemical is without any neurobehavioral consequence (beyond those associated with neural tube defects) seems to be the more implausible consideration. At this stage, controlled intervention trials using folic acid, and other nutrients involved in folate metabolism, seem overdue [8,9].

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References

1. Kris-Etherton, P.M.; Petersen, K.S.; Hibbeln, J.R.; Hurley, D.; Kolick, V.; Peoples, S.; Rodriguez, N.; Woodward-Lopez, G. Nutrition and behavioral health disorders: Depression and anxiety. *Nutr. Rev.* **2021**, *79*, 247–260. [CrossRef] [PubMed]
2. Teasdale, S.; Morkl, S.; Muller-Stierlin, A.S. Nutritional psychiatry in the treatment of psychotic disorders: Current hypotheses and research challenges. *Brain Behav. Immun. Health* **2020**, *5*, 100070. [CrossRef] [PubMed]
3. Tcherni-Buzzeo, M. Dietary interventions, the gut microbiome, and aggressive behavior: Review of research evidence and potential next steps. *Aggress. Behav.* **2023**, *49*, 15–32. [CrossRef] [PubMed]
4. Choy, O. Nutritional factors associated with aggression. *Front. Psychiatry* **2023**, *14*, 1176061. [CrossRef] [PubMed]
5. Xie, T.; Schweren, L.J.S.; Larsson, H.; Li, L.; Du Rietz, E.; Haavik, J.; Lifelines Cohort, S.; Grimstvedt Kvalvik, L.; Solberg, B.S.; Klungsoyr, K.; et al. Do Poor Diet and Lifestyle Behaviors Modify the Genetic Susceptibility to Impulsivity in the General Population? *Nutrients* **2023**, *15*, 1625. [CrossRef] [PubMed]
6. Zaalberg, A. The effects of nutrients and neurotoxicants on aggressive behavior. *J. Crim. Just.* **2019**, *65*, 101592. [CrossRef]
7. Faraone, S.V.; Banaschewski, T.; Coghill, D.; Zheng, Y.; Biederman, J.; Bellgrove, M.A.; Newcorn, J.H.; Gignac, M.; Al Saud, N.M.; Manor, I.; et al. The World Federation of ADHD International Consensus Statement: 208 Evidence-based conclusions about the disorder. *Neurosci. Biobehav. Rev.* **2021**, *128*, 789–818. [CrossRef]
8. Prescott, S.L.; Logan, A.C.; D'Adamo, C.R.; Holton, K.F.; Lowry, C.A.; Marks, J.; Moodie, R.; Polland, B. Nutritional Criminology: Why the Emerging Research on Ultra- Processed Food Matters to Health and Justice. *Int. J. Environ. Res. Public Health* **2024**, *21*, 120. [CrossRef]
9. Robinson, M. Eating ourselves to death: How food is a drug and what food abuse costs. *Drug Sci. Policy Law* **2022**, *8*, 20503245221112577. [CrossRef]
10. Hibbeln, J.R. From homicide to happiness—A commentary on omega-3 fatty acids in human society. Cleave Award Lecture. *Nutr. Health* **2007**, *19*, 9–19. [CrossRef]
11. Grenell, R.G.; Gabay, S. (Eds.) *Biological Foundations of Psychiatry*; Raven Press: New York, NY, USA, 1976.
12. Liwinski, T.; Lang, U.E. Folate and Its Significance in Depressive Disorders and Suicidality: A Comprehensive Narrative Review. *Nutrients* **2023**, *15*, 3859. [CrossRef] [PubMed]
13. Falade, J.; Onaolapo, A.Y.; Onaolapo, O.J. The Role of Folate-supplementation in Depression: A Narrative Review. *Curr. Psychopharmacol.* **2021**, *10*, 115–122. [CrossRef]
14. Zimring, F.E. *The Great American Crime Decline*; Oxford University Press: New York, NY, USA, 2006.

15. Levitt, S.D. Understanding why crime fell in the 1990s: Four factors that explain the decline and six that do not. *J. Econ. Perspect.* **2004**, *18*, 163–190. [CrossRef]
16. Rosenfeld, R. The case of the unsolved crime decline. *Sci. Am.* **2004**, *290*, 82–89. [CrossRef]
17. Parker, K.F. *Unequal Crime Decline*; New York University Press: New York, NY, USA, 2008.
18. McKeown, R.E.; Cuffe, S.P.; Schulz, R.M. US suicide rates by age group, 1970-2002: An examination of recent trends. *Am. J. Public Health* **2006**, *96*, 1744–1751. [CrossRef] [PubMed]
19. Wald, N.J.; Polani, P.E. Neural-tube defects and vitamins: The need for a randomized clinical trial. *Br. J. Obstet. Gynaecol.* **1984**, *91*, 516–523. [CrossRef] [PubMed]
20. Milunsky, A.; Jick, H.; Jick, S.S.; Bruell, C.L.; MacLaughlin, D.S.; Rothman, K.J.; Willett, W. Multivitamin/folic acid supplementation in early pregnancy reduces the prevalence of neural tube defects. *JAMA* **1989**, *262*, 2847–2852. [CrossRef] [PubMed]
21. Kolata, G. Sharp Cut in Serious Birth Defect Is Tied to Vitamins in Pregnancy. *The New York Times*, 24 November 1989; p. A-1.
22. Anon. Early Multivitamin Use Found to Reduce Risk of Birth Defect. *The Atlanta Journal Constitution*, 24 November 1989; p. 1.
23. The Associated Press. Taking B vitamin prenatally can reduce spina bifida risk. *Waco Tribune-Herald*, 15 April 1990; p. 32.
24. MacCorquodale, D.W. Nutrition knowledge equals food for thought. *Richmond Times-Dispatch*, 7 February 1990; p. 6.
25. Centers for Disease Control. Recommendations for the Use of Folic Acid to Reduce the Number of Cases of Spina Bifida and Other Neural Tube Defects. *MMWR Recomm Rep.* **1992**, *41*, 1–7. Available online: https://www.cdc.gov/mmwr/preview/mmwrhtml/00019479.htm (accessed on 15 February 2024).
26. McDowell, M.A.; Lacher, D.A.; Pfeiffer, C.M.; Mulinare, J.; Picciano, M.F.; Rader, J.I.; Yetley, E.A.; Kennedy-Stephenson, J.; Johnson, C.L. *Blood Folate Levels: The Latest NHANES Results*; NCHS Data Briefs, No. 6; National Center for Health Statistics, Centers for Disease Control: Hyattsville, MD, USA, 2008. Available online: https://www.cdc.gov/nchs/products/databriefs/db06.htm#:~:text=Median%20RBC%20folate%20levels%20of%20persons%204%20years%20of%20age,-1994%20and%201999-2000 (accessed on 15 February 2024).
27. Senti, F.R.; Pilch, S.M. Analysis of folate data from the second National Health and Nutrition Examination Survey (NHANES II). *J. Nutr.* **1985**, *115*, 1398–1402. [CrossRef]
28. Moss, A.J. *Use of Vitamin and Mineral Supplements in the United States: Current Users, Types of Products, and Nutrients*; US Department of Health and Human Services, Public Health Service, Centers for Disease Control, National Center for Health Statistics: Hyattsville, MD, USA, 1989.
29. Ervin, R.B.; Wright, J.D.; Kennedy-Stephenson, J. Use of dietary supplements in the United States, 1988–1994. *Natl. Cent. Health Stat. Vital Health Stat.* **1999**, *11*, 244.
30. Mishra, S.; Gahche, J.J.; Ogden, C.L.; Dimeler, M.; Potischman, N.; Ahluwalia, N. *Dietary Supplement Use in the United States: National Health and Nutrition Examination Survey, 2017–March 2020. National Health Statistics Reports*; No 183; National Center for Health Statistics: Hyattsville, MD, USA, 2023. [CrossRef]
31. Bowman, L. *Folic Acid Ordered Added to Foods*; The San Francisco Examiner: San Francisco, CA, USA, 1996; p. 2.
32. Logan, A.C.; Nicholson, J.J.; Schoenthaler, S.J.; Prescott, S.L. Neurolaw: Revisiting Huberty v. McDonald's through the Lens of Nutritional Criminology and Food Crime. *Laws* **2024**, *13*, 17. [CrossRef]
33. Clarke, A.G.; Prescott, F. Studies in Vitamin B Deficiency. *Br. Med. J.* **1943**, *2*, 503–504. [CrossRef] [PubMed]
34. Watson, G.; Comrey, A.L. Nutritional replacement for mental illness. *J. Psychol.* **1954**, *38*, 251–264. [CrossRef]
35. Young, S.N.; Ghadirian, A.M. Folic-Acid and Psychopathology. *Prog. Neuro-Psychopharmacol.* **1989**, *13*, 841–863. [CrossRef] [PubMed]
36. Kariks, J.; Perry, S.W. Folic-Acid Deficiency in Psychiatric Patients. *Med. J. Aust.* **1970**, *1*, 1192–1195. [CrossRef] [PubMed]
37. Crellin, R.; Bottiglieri, T.; Reynolds, E.H. Folates and Psychiatric-Disorders—Clinical Potential. *Drugs* **1993**, *45*, 623–636. [CrossRef] [PubMed]
38. Morris, G.; Walder, K.R.; Berk, M.; Marx, W.; Walker, A.J.; Maes, M.; Puri, B.K. The interplay between oxidative stress and bioenergetic failure in neuropsychiatric illnesses: Can we explain it and can we treat it? *Mol. Biol. Rep.* **2020**, *47*, 5587–5620. [CrossRef] [PubMed]
39. Brocardo, P.S.; Budni, J.; Kaster, M.P.; Santos, A.R.; Rodrigues, A.L. Folic acid administration produces an antidepressant-like effect in mice: Evidence for the involvement of the serotonergic and noradrenergic systems. *Neuropharmacology* **2008**, *54*, 464–473. [CrossRef]
40. Ferguson, S.A.; Berry, K.J.; Hansen, D.K.; Wall, K.S.; White, G.; Antony, A.C. Behavioral effects of prenatal folate deficiency in mice. *Birth Defects Res. A Clin. Mol. Teratol.* **2005**, *73*, 249–252. [CrossRef]
41. Altaf, R.; Gonzalez, I.; Rubino, K.; Nemec, E.C., 2nd. Folate as adjunct therapy to SSRI/SNRI for major depressive disorder: Systematic review & meta-analysis. *Complement. Ther. Med.* **2021**, *61*, 102770.
42. Crockett, M.J.; Clark, L.; Hauser, M.D.; Robbins, T.W. Serotonin selectively influences moral judgment and behavior through effects on harm aversion. *Proc. Natl. Acad. Sci. USA* **2010**, *107*, 17433–17438. [CrossRef] [PubMed]
43. Ash, J.A.; Jiang, X.; Malysheva, O.V.; Fiorenza, C.G.; Bisogni, A.J.; Levitsky, D.A.; Strawderman, M.S.; Caudill, M.A.; Stover, P.J.; Strupp, B.J. Dietary and genetic manipulations of folate metabolism differentially affect neocortical functions in mice. *Neurotoxicol. Teratol.* **2013**, *38*, 79–91. [CrossRef] [PubMed]

44. Chan, A.; Tchantchou, F.; Graves, V.; Rozen, R.; Shea, T.B. Dietary and genetic compromise in folate availability reduces acetylcholine, cognitive performance and increases aggression: Critical role of S-adenosyl methionine. *J. Nutr. Health Aging* **2008**, *12*, 252–261. [CrossRef] [PubMed]
45. Gesch, C.B.; Hammond, S.M.; Hampson, S.E.; Eves, A.; Crowder, M.J. Influence of supplementary vitamins, minerals and essential fatty acids on the antisocial behaviour of young adult prisoners. Randomised, placebo-controlled trial. *Br. J. Psychiatry* **2002**, *181*, 22–28. [CrossRef] [PubMed]
46. Zaalberg, A.; Nijman, H.; Bulten, E.; Stroosma, L.; van der Staak, C. Effects of nutritional supplements on aggression, rule-breaking, and psychopathology among young adult prisoners. *Aggress. Behav.* **2010**, *36*, 117–126. [CrossRef] [PubMed]
47. Schoenthaler, S.J.; Amos, W.; Doraz, M.A.; Kelly, G.; Muedeking, J.; Wakefield, J. The effect of randomized vitamin-mineral supplementation on violent and non-violent antisocial behavior among incarcerated juveniles. *J. Nutr. Environ. Med.* **1997**, *7*, 343–352. [CrossRef]
48. Schoenthaler, S.; Gast, D.; Giltay, E.J.; Amos, S. The effects of vitamin-mineral supplements on serious rule violations in correctional facilities for young adult male inmates: A ran-domized controlled trial. *Crime Delinq.* **2023**, *69*, 822–840. [CrossRef]
49. Kim, J.M.; Kim, H.Y.; Lee, H.J.; Kim, J.W.; Kang, H.J.; Kim, S.W.; Shin, I.S.; Chun, B.J.; Stewart, R. Prediction of Suicidality According to Serum Folate Levels in Depressive Patients Receiving Stepwise Pharmacotherapy. *Front. Psychiatry* **2021**, *12*, 747228. [CrossRef] [PubMed]
50. Gibbons, R.D.; Hur, K.; Lavigne, J.E.; Mann, J.J. Association Between Folic Acid Prescription Fills and Suicide Attempts and Intentional Self-harm Among Privately Insured US Adults. *JAMA Psychiatry* **2022**, *79*, 1118–1123. [CrossRef]
51. Mann, J.J.; Hur, K.; Lavigne, J.E.; Gibbons, R.D. Folic acid prescription and suicide attempt prevention: Effect of past suicidal behaviour, psychiatric diagnosis and psychotropic medication. *BJPsych Open* **2023**, *9*, e159. [CrossRef]
52. Dumais, A.; Lesage, A.D.; Lalovic, A.; Seguin, M.; Tousignant, M.; Chawky, N.; Turecki, G. Is violent method of suicide a behavioral marker of lifetime aggression? *Am. J. Psychiatry* **2005**, *162*, 1375–1378. [CrossRef] [PubMed]
53. Almeida, D.; Turecki, G. Is Violent Suicide Molecularly Distinct? *Am. J. Psychiatry* **2022**, *179*, 180–181. [CrossRef] [PubMed]
54. Punzi, G.; Ursini, G.; Chen, Q.; Radulescu, E.; Tao, R.; Huuki, L.A.; Di Carlo, P.; Collado-Torres, L.; Shin, J.H.; Catanesi, R.; et al. Genetics and Brain Transcriptomics of Completed Suicide. *Am. J. Psychiatry* **2022**, *179*, 226–241. [CrossRef] [PubMed]
55. Zhang, L.; Verwer, R.W.; van Heerikhuizen, J.; Balesar, R.; Correa-da-Silva, F.; Slabe, Z.; Lucassen, P.J.; Swaab, D.F. Stress-associated purinergic receptors code for fatal suicidality in the hippocampal-hypothalamic-prefrontal circuit. *bioRxiv* **2022**. [CrossRef]
56. Gajos, J.M.; Beaver, K.M. The effect of omega-3 fatty acids on aggression: A meta-analysis. *Neurosci. Biobehav. Rev.* **2016**, *69*, 147–158. [CrossRef] [PubMed]
57. Sublette, M.E.; Daray, F.M.; Gananca, L.; Shaikh, S.R. The role of polyunsaturated fatty acids in the neurobiology of major depressive disorder and suicide risk. *Mol. Psychiatry* **2023**. [CrossRef] [PubMed]
58. Ued, F.V.; Mathias, M.G.; Toffano, R.B.D.; Barros, T.T.; Almada, M.; Salomao, R.G.; Coelho-Landell, C.A.; Hillesheim, E.; Camarneiro, J.M.; Camelo-Junior, J.S.; et al. Vitamin B2 and Folate Concentrations are Associated with ARA, EPA and DHA Fatty Acids in Red Blood Cells of Brazilian Children and Adolescents. *Nutrients* **2019**, *11*, 2918. [CrossRef] [PubMed]
59. Pita, M.L.; Delgado, M.J. Folate administration increases n-3 polyunsaturated fatty acids in rat plasma and tissue lipids. *Thromb. Haemost.* **2000**, *84*, 420–423. [PubMed]
60. Durand, P.; Prost, M.; Blache, D. Pro-thrombotic effects of a folic acid deficient diet in rat platelets and macrophages related to elevated homocysteine and decreased n-3 polyunsaturated fatty acids. *Atherosclerosis* **1996**, *121*, 231–243. [CrossRef]
61. Hirono, H.; Wada, Y. Effects of dietary folate deficiency on developmental increase of myelin lipids in rat brain. *J. Nutr.* **1978**, *108*, 766–772. [CrossRef]
62. Umhau, J.C.; Dauphinais, K.M.; Patel, S.H.; Nahrwold, D.A.; Hibbeln, J.R.; Rawlings, R.R.; George, D.T. The relationship between folate and docosahexaenoic acid in men. *Eur. J. Clin. Nutr.* **2006**, *60*, 352–357. [CrossRef] [PubMed]
63. Kale, A.; Naphade, N.; Sapkale, S.; Kamaraju, M.; Pillai, A.; Joshi, S.; Mahadik, S. Reduced folic acid, vitamin B12 and docosahexaenoic acid and increased homocysteine and cortisol in never-medicated schizophrenia patients: Implications for altered one-carbon metabolism. *Psychiatry Res.* **2010**, *175*, 47–53. [CrossRef] [PubMed]
64. Sarris, J.; Ravindran, A.; Yatham, L.N.; Marx, W.; Rucklidge, J.J.; McIntyre, R.S.; Akhondzadeh, S.; Benedetti, F.; Caneo, C.; Cramer, H.; et al. Clinician guidelines for the treatment of psychiatric disorders with nutraceuticals and phytoceuticals: The World Federation of Societies of Biological Psychiatry (WFSBP) and Canadian Network for Mood and Anxiety Treatments (CANMAT) Taskforce. *World J. Biol. Psychiatry* **2022**, *23*, 424–455. [CrossRef] [PubMed]
65. Logan, A.C.; Venket Rao, A.; Irani, D. Chronic fatigue syndrome: Lactic acid bacteria may be of therapeutic value. *Med. Hypotheses* **2003**, *60*, 915–923. [CrossRef] [PubMed]
66. Logan, A.C.; Katzman, M. Major depressive disorder: Probiotics may be an adjuvant therapy. *Med. Hypotheses* **2005**, *64*, 533–538. [CrossRef] [PubMed]
67. Merlo, G.; Bachtel, G.; Sugden, S.G. Gut microbiota, nutrition, and mental health. *Front. Nutr.* **2024**, *11*, 1337889. [CrossRef]
68. Martin, S.E.; Kraft, C.S.; Ziegler, T.R.; Millson, E.C.; Rishishwar, L.; Martin, G.S. The Role of Diet on the Gut Microbiome, Mood and Happiness. *medRxiv* **2023**. [CrossRef]
69. Bonaz, B.; Bazin, T.; Pellissier, S. The Vagus Nerve at the Interface of the Microbiota-Gut-Brain Axis. *Front. Neurosci.* **2018**, *12*, 49. [CrossRef]

70. Donoso, F.; Cryan, J.F.; Olavarria-Ramirez, L.; Nolan, Y.M.; Clarke, G. Inflammation, Lifestyle Factors, and the Microbiome-Gut-Brain Axis: Relevance to Depression and Antidepressant Action. *Clin. Pharmacol. Ther.* **2023**, *113*, 246–259. [CrossRef]
71. Barone, M.; D'Amico, F.; Brigidi, P.; Turroni, S. Gut microbiome-micronutrient interaction: The key to controlling the bioavailability of minerals and vitamins? *Biofactors* **2022**, *48*, 307–314. [CrossRef]
72. Aleman, R.S.; Moncada, M.; Aryana, K.J. Leaky Gut and the Ingredients That Help Treat It: A Review. *Molecules* **2023**, *28*, 619. [CrossRef] [PubMed]
73. Wang, C.; Zhang, T.; He, L.; Fu, J.Y.; Deng, H.X.; Xue, X.L.; Chen, B.T. Bacterial Translocation Associates With Aggression in Schizophrenia Inpatients. *Front. Syst. Neurosci.* **2021**, *15*, 704069. [CrossRef] [PubMed]
74. Fan, X.; Zang, T.; Liu, J.; Wu, N.; Dai, J.; Bai, J.; Liu, Y. Changes in the gut microbiome in the first two years of life predicted the temperament in toddlers. *J. Affect. Disord.* **2023**, *333*, 342–352. [CrossRef]
75. Chen, X.; Xu, J.; Wang, H.; Luo, J.; Wang, Z.; Chen, G.; Jiang, D.; Cao, R.; Huang, H.; Luo, D.; et al. Profiling the differences of gut microbial structure between schizophrenia patients with and without violent behaviors based on 16S rRNA gene sequencing. *Int. J. Legal Med.* **2021**, *135*, 131–141. [CrossRef] [PubMed]
76. Ke, S.; Guimond, A.J.; Tworoger, S.S.; Huang, T.; Chan, A.T.; Liu, Y.Y.; Kubzansky, L.D. Gut feelings: Associations of emotions and emotion regulation with the gut microbiome in women. *Psychol. Med.* **2023**, *53*, 7151–7160. [CrossRef] [PubMed]
77. Steenbergen, L.; Sellaro, R.; van Hemert, S.; Bosch, J.A.; Colzato, L.S. A randomized controlled trial to test the effect of multispecies probiotics on cognitive reactivity to sad mood. *Brain Behav. Immun.* **2015**, *48*, 258–264. [CrossRef] [PubMed]
78. Arteaga-Henriquez, G.; Rosales-Ortiz, S.K.; Arias-Vasquez, A.; Bitter, I.; Ginsberg, Y.; Ibanez-Jimenez, P.; Kilencz, T.; Lavebratt, C.; Matura, S.; Reif, A.; et al. Treating impulsivity with probiotics in adults (PROBIA): Study protocol of a multicenter, double-blind, randomized, placebo-controlled trial. *Trials* **2020**, *21*, 161. [CrossRef] [PubMed]
79. Gulledge, L.; Oyebode, D.; Donaldson, J.R. The influence of the microbiome on aggressive behavior: An insight into age-related aggression. *FEMS Microbiol. Lett.* **2023**, *370*, fnac114. [CrossRef]
80. Bruce-Keller, A.J.; Salbaum, J.M.; Luo, M.; Blanchard, E.T.; Taylor, C.M.; Welsh, D.A.; Berthoud, H.R. Obese-type gut microbiota induce neurobehavioral changes in the absence of obesity. *Biol. Psychiatry* **2015**, *77*, 607–615. [CrossRef]
81. Li, N.; Wang, Q.; Wang, Y.; Sun, A.; Lin, Y.; Jin, Y.; Li, X. Fecal microbiota transplantation from chronic unpredictable mild stress mice donors affects anxiety-like and depression-like behavior in recipient mice via the gut microbiota-inflammation-brain axis. *Stress* **2019**, *22*, 592–602. [CrossRef]
82. Wang, C.; Yan, J.; Du, K.; Liu, S.; Wang, J.; Wang, Q.; Zhao, H.; Li, M.; Yan, D.; Zhang, R.; et al. Intestinal microbiome dysbiosis in alcohol-dependent patients and its effect on rat behaviors. *mBio* **2023**, *14*, e0239223. [CrossRef] [PubMed]
83. Ritz, N.L.; Brocka, M.; Butler, M.I.; Cowan, C.S.M.; Barrera-Bugueno, C.; Turkington, C.J.R.; Draper, L.A.; Bastiaanssen, T.F.S.; Turpin, V.; Morales, L.; et al. Social anxiety disorder-associated gut microbiota increases social fear. *Proc. Natl. Acad. Sci. USA* **2024**, *121*, e2308706120. [CrossRef]
84. Uzan-Yulzari, A.; Turjeman, S.; Getselter, D.; Rautava, S.; Isolauri, E.; Khatib, S.; Elliott, E.; Koren, O. Aggression: A gut reaction? The effects of gut microbiome on aggression. *bioRxiv* **2023**. [CrossRef]
85. Zheng, P.; Zeng, B.; Liu, M.; Chen, J.; Pan, J.; Han, Y.; Liu, Y.; Cheng, K.; Zhou, C.; Wang, H.; et al. The gut microbiome from patients with schizophrenia modulates the glutamate-glutamine-GABA cycle and schizophrenia-relevant behaviors in mice. *Sci. Adv.* **2019**, *5*, eaau8317. [CrossRef] [PubMed]
86. Zhu, F.; Guo, R.; Wang, W.; Ju, Y.; Wang, Q.; Ma, Q.; Sun, Q.; Fan, Y.; Xie, Y.; Yang, Z.; et al. Transplantation of microbiota from drug-free patients with schizophrenia causes schizophrenia-like abnormal behaviors and dysregulated kynurenine metabolism in mice. *Mol. Psychiatry* **2020**, *25*, 2905–2918. [CrossRef]
87. Li, D.; Liang, W.; Zhang, W.; Huang, Z.; Liang, H.; Liu, Q. Fecal microbiota transplantation repairs intestinal permeability and regulates the expression of 5-HT to influence alcohol-induced depression-like behaviors in C57BL/6J mice. *Front. Microbiol.* **2023**, *14*, 1241309. [CrossRef] [PubMed]
88. Chen, S.; Li, M.; Tong, C.; Wang, Y.; He, J.; Shao, Q.; Liu, Y.; Wu, Y.; Song, Y. Regulation of miRNA expression in the prefrontal cortex by fecal microbiota transplantation in anxiety-like mice. *Front. Psychiatry* **2024**, *15*, 1323801. [CrossRef] [PubMed]
89. Park, S.; Kang, S.; Sol Kim, D. Folate and vitamin B-12 deficiencies additively impaire memory function and disturb the gut microbiota in amyloid-beta infused rats. *Int. J. Vitam. Nutr. Res.* **2022**, *92*, 169–181. [CrossRef]
90. Cheng, C.K.; Wang, C.; Shang, W.; Lau, C.W.; Luo, J.Y.; Wang, L.; Huang, Y. A high methionine and low folate diet alters glucose homeostasis and gut microbiome. *Biochem. Biophys. Rep.* **2021**, *25*, 100921. [CrossRef]
91. Chen, S.; Yang, M.; Wang, R.; Fan, X.; Tang, T.; Li, P.; Zhou, X.; Qi, K. Suppression of high-fat-diet-induced obesity in mice by dietary folic acid supplementation is linked to changes in gut microbiota. *Eur. J. Nutr.* **2022**, *61*, 2015–2031. [CrossRef]
92. Zhang, H.; Zuo, Y.; Zhao, H.; Zhao, H.; Wang, Y.; Zhang, X.; Zhang, J.; Wang, P.; Sun, L.; Zhang, H.; et al. Folic acid ameliorates alcohol-induced liver injury via gut-liver axis homeostasis. *Front. Nutr.* **2022**, *9*, 989311. [CrossRef] [PubMed]
93. Liu, Y.; Yang, J.; Liu, X.; Liu, R.; Wang, Y.; Huang, X.; Li, Y.; Liu, R.; Yang, X. Dietary folic acid addition reduces abdominal fat deposition mediated by alterations in gut microbiota and SCFA production in broilers. *Anim. Nutr.* **2023**, *12*, 54–62. [CrossRef] [PubMed]
94. Gobaud, A.N.; Mehranbod, C.A.; Dong, B.; Dodington, J.; Morrison, C.N. Absolute versus relative socioeconomic disadvantage and homicide: A spatial ecological case-control study of US zip codes. *Inj. Epidemiol.* **2022**, *9*, 7. [CrossRef] [PubMed]

95. Stack, S. Contributing factors to suicide: Political, social, cultural and economic. *Prev. Med.* **2021**, *152 Pt 1*, 106498. [CrossRef] [PubMed]
96. Martinez-Ales, G.; Jiang, T.; Keyes, K.M.; Gradus, J.L. The Recent Rise of Suicide Mortality in the United States. *Annu. Rev. Public Health* **2022**, *43*, 99–116. [CrossRef]
97. Go, T.H.; Kim, M.H.; Choi, Y.Y.; Han, J.; Kim, C.; Kang, D.R. The short-term effect of ambient particulate matter on suicide death. *Environ. Health* **2024**, *23*, 3. [CrossRef] [PubMed]
98. Yang, W.; Carmichael, S.L.; Shaw, G.M. Folic acid fortification and prevalences of neural tube defects, orofacial clefts, and gastroschisis in California, 1989 to 2010. *Birth Defects Res. A Clin. Mol. Teratol.* **2016**, *106*, 1032–1041. [CrossRef] [PubMed]
99. Pfeiffer, C.M.; Hughes, J.P.; Lacher, D.A.; Bailey, R.L.; Berry, R.J.; Zhang, M.; Yetley, E.A.; Rader, J.I.; Sempos, C.T.; Johnson, C.L. Estimation of trends in serum and RBC folate in the U.S. population from pre- to postfortification using assay-adjusted data from the NHANES 1988–2010. *J. Nutr.* **2012**, *142*, 886–893. [CrossRef]
100. Petersen, J.M.; Smith-Webb, R.S.; Shaw, G.M.; Carmichael, S.L.; Desrosiers, T.A.; Nestoridi, E.; Darling, A.M.; Parker, S.E.; Politis, M.D.; Yazdy, M.M.; et al. Periconceptional intakes of methyl donors and other micronutrients involved in one-carbon metabolism may further reduce the risk of neural tube defects in offspring: A United States population-based case-control study of women meeting the folic acid recommendations. *Am. J. Clin. Nutr.* **2023**, *118*, 720–728.
101. Wang, A.; Rose, C.E.; Qi, Y.P.; Williams, J.L.; Pfeiffer, C.M.; Crider, K.S. Impact of Voluntary Folic Acid Fortification of Corn Masa Flour on RBC Folate Concentrations in the U.S. (NHANES 2011–2018). *Nutrients* **2021**, *13*, 1325. [CrossRef]
102. Murphy, R.; Marshall, K.; Zagorin, S.; Devarshi, P.P.; Hazels Mitmesser, S. Socioeconomic Inequalities Impact the Ability of Pregnant Women and Women of Childbearing Age to Consume Nutrients Needed for Neurodevelopment: An Analysis of NHANES 2007–2018. *Nutrients* **2022**, *14*, 3823. [CrossRef] [PubMed]
103. Carmichael, S.L.; Yang, W.; Herring, A.; Abrams, B.; Shaw, G.M. Maternal food insecurity is associated with increased risk of certain birth defects. *J. Nutr.* **2007**, *137*, 2087–2092. [CrossRef]
104. Pruitt Evans, S.; Ailes, E.C.; Kramer, M.R.; Shumate, C.J.; Reefhuis, J.; Insaf, T.Z.; Yazdy, M.M.; Carmichael, S.L.; Romitti, P.A.; Feldkamp, M.L.; et al. Neighborhood Deprivation and Neural Tube Defects. *Epidemiology* **2023**, *34*, 774–785. [CrossRef] [PubMed]
105. Grinshteyn, E.; Hemenway, D. Violent Death Rates: The US Compared with Other High-income OECD Countries, 2010. *Am. J. Med.* **2016**, *129*, 266–273. [CrossRef]
106. Grinshteyn, E.; Hemenway, D. Violent death rates in the US compared to those of the other high-income countries, 2015. *Prev. Med.* **2019**, *123*, 20–26. [CrossRef]
107. Kose, S.; Sozlu, S.; Bolukbasi, H.; Unsal, N.; Gezmen-Karadag, M. Obesity is associated with folate metabolism. *Int. J. Vitam. Nutr. Res.* **2020**, *90*, 353–364. [CrossRef]
108. Sutton, C.A.; Stratton, M.; L'Insalata, A.M.; Fazzino, T.L. Ultraprocessed, hyper-palatable, and high energy density foods: Prevalence and distinction across 30 years in the United States. *Obesity* **2024**, *32*, 166–175. [CrossRef] [PubMed]
109. Juul, F.; Parekh, N.; Martinez-Steele, E.; Monteiro, C.A.; Chang, V.W. Ultra-processed food consumption among US adults from 2001 to 2018. *Am. J. Clin. Nutr.* **2022**, *115*, 211–221. [CrossRef] [PubMed]
110. Amaraggi, B.; Wood, W.; Guinovart Martin, L.; Gimenez Sanchez, J.; Fleta Sanchez, Y.; de la Garza Puentes, A. Ultra-processed food staples dominate mainstream US supermarkets. Americans more than Europeans forced to choose between health and cost. *medRxiv* **2024**. [CrossRef]
111. Leung, C.W.; Parnarouskis, L.; Slotnick, M.J.; Gearhardt, A.N. Food Insecurity and Food Addiction in a Large, National Sample of Lower-Income Adults. *Curr. Dev. Nutr.* **2023**, *7*, 102036. [CrossRef]
112. Fazzino, T.L.; Jun, D.; Chollet-Hinton, L.; Bjorlie, K. US tobacco companies selectively disseminated hyper-palatable foods into the US food system: Empirical evidence and current implications. *Addiction* **2024**, *119*, 62–71. [CrossRef] [PubMed]
113. Prescott, S.L.; Logan, A.C. Each meal matters in the exposome: Biological and community considerations in fast-food-socioeconomic associations. *Econ. Hum. Biol.* **2017**, *27 Pt B*, 328–335. [CrossRef]
114. Blumstein, A.; Rivara, F.P.; Rosenfeld, R. The rise and decline of homicide—And why. *Annu. Rev. Public Health* **2000**, *21*, 505–541. [CrossRef] [PubMed]