UNITED STATES DISTRICT COURT

NORTHERN DISTRICT OF CALIFORNIA

IN RE: BABY FOOD PRODUCTS LIABILITY LITIGATION

This document relates to:

ALL ACTIONS

Case No. 24-md-03101-JSC

ORDER RE DEFENDANTS' MOTION TO EXCLUDE PLAINTIFFS' EXPERT WITNESSES

Re: Dkt. Nos. 611, 612, 614

In this multidistrict litigation, Plaintiffs allege, through their guardians *ad litem*, that they developed autism spectrum disorder ("ASD") and attention-deficit hyperactivity disorder ("ADHD") from consuming certain of Defendants' baby food products. In particular, they contend the levels of lead and arsenic present in Defendants' baby food caused their injuries.[1] (Dkt. No. 451 ¶ 3.) Pursuant to the Court's management of the case, the parties have proceeded through discovery on the initial question of general causation. General causation expert discovery closed on August 29, 2025. (Dkt. No. 440.) Subsequently, the parties filed cross-motions to exclude expert witnesses on September 26, 2025. (*See* Dkt. Nos. 611, 612, 614, 615, 616.) Only Defendants' motions to exclude are the subject of this Order.

Beginning on December 8, 2025, the Court heard three days of testimony from Plaintiffs' experts, including epidemiologists Dr. Hannah Gardener, Dr. Howard Hu, and Dr. Beate Ritz, as well as clinical neurologist Dr. Kevin Shapiro. Then, on December 11, the Court heard oral

[1] Plaintiffs' Amended Master Complaint alleges the presence of lead, arsenic, mercury, and cadmium in Defendants' products caused their development of ASD and ADHD. (Dkt. No. 451 ¶ 1). But, they offer no expert testimony on the effects of mercury or cadmium, and thus have abandoned that theory of causation. (*See* Dkt. No. 697 at 5 (Plaintiffs' counsel noting that his clients were dismissing their claims as to those metals with prejudice).)

argument and closing presentations from the parties. Though the Court only heard testimony from the scientists and clinicians noted above, Defendants' motions to exclude extend to four additional experts put forward by Plaintiffs: infant dietician, Ms. Priscilla Barr; exposure scientist, Dr. Rachael Jones; and toxicologists, Drs. Michael Aschner and Tomas Guilarte. This Order resolves all motions to exclude Plaintiffs' experts.

Having considered the parties' submissions, and with the benefit of oral argument, the Court **GRANTS IN PART** and **DENIES IN PART** Defendants' motions to exclude. Plaintiffs have not identified any scientific studies of whether baby food, let alone Defendants' baby food, can cause ASD or ADHD. Indeed, they have not identified any studies of whether food of any kind can cause these conditions. So, Plaintiffs' causation theory is built upon a series of extrapolations from studies that do not look specifically at consumption of baby food. To that end, Plaintiffs attempt to meet their general causation burden by focusing on whether exposure to lead or arsenic from *any* source is capable of causing ASD or ADHD. Ultimately, they have failed to show, by a preponderance of the evidence, that their experts' causation opinions reliably extrapolate from such studies and pass muster under Federal Rule of Evidence 702.

LEGAL STANDARD

Two legal issues predominate in the instant motions. First, Plaintiffs must establish their proposed expert witnesses meet Federal Rule of Evidence 702's reliability requirements. Second, Plaintiffs must show their proposed experts' testimony fits—or is relevant to—the general causation question, as required under Subsection (a) of the Rule.

A. *Daubert* Standard

Federal Rule of Evidence 702 governs the admissibility of expert testimony. The Rule provides:

> A witness who is qualified as an expert by knowledge, skill, experience, training, or education may testify in the form of an opinion or otherwise if the proponent demonstrates to the court that it is more likely than not that:
>
> (a) the expert's scientific, technical, or other specialized knowledge will help the trier of fact to understand the evidence or to determine a fact in issue;

(b) the testimony is based on sufficient facts or data;

(c) the testimony is the product of reliable principles and methods; and

(d) the expert's opinion reflects a reliable application of the principles and methods to the facts of the case.

Fed. R. Evid. 702. Named after the Supreme Court's seminal case on this issue—*Daubert v. Merrell Dow Pharms., Inc.*, 509 U.S. 579, 582 (1993) (*Daubert I*)—a *Daubert* motion challenges whether the expert testimony's proponent has established, by a preponderance of the evidence, the testimony is admissible. *See Engilis v. Monsanto Co.*, 151 F.4th 1040, 1049 (9th Cir. 2025) (noting the "preponderance of the evidence" standard applies to Rule 702).

The *Daubert I* opinion, and subsequent caselaw, have raised additional factors beyond those expressly identified in Rule 702. Such factors include: "whether the theory or technique employed by the expert is generally accepted in the scientific community; whether it's been subjected to peer review and publication; whether it can be and has been tested; and whether the known or potential rate of error is acceptable." *Daubert v. Merrell Dow Pharms., Inc.*, 43 F.3d 1311, 1316 (9th Cir. 1995) (*Daubert II*) (citing *Daubert I*, 509 U.S. at 591-93). Further, the Ninth Circuit has weighed admissibility based on whether the proposed experts have developed their opinions "naturally and directly out of research they have conducted independent of the litigation, or whether they have developed their opinions expressly for purposes of testifying." *Id.* at 1317.

This list is not exhaustive. The Advisory Committee Notes to the 2000 Amendment to Rule 702 clarify, "[n]o attempt has been made to 'codify' these specific factors." Rather, "the Court has broad discretion to determine which factors are most informative in assessing reliability in the context of a given case." *In re Roundup Prods. Liab. Litig.*, 390 F. Supp. 3d 1102, 1112 (N.D. Cal. 2018), *aff'd sub nom. Hardeman v. Monsanto Co.*, 997 F.3d 941 (9th Cir. 2021) (citing *Kumho Tire Co., Ltd. v. Carmichael*, 526 U.S. 137, 141-42 (1999)). To guide this flexible inquiry, the Court centers two fundamental considerations: relevance and reliability. *See Primiano v. Cook*, 598 F.3d 558, 564 (9th Cir. 2010) ("In sum, the trial court must assure that the expert testimony 'both rests on a reliable foundation and is relevant to the task at hand.'"). "Expert opinion testimony is relevant if the knowledge underlying it has a valid connection to the pertinent

inquiry. In other words, the expert testimony must 'fit' the question the jury must answer. This bar is cleared where the evidence 'logically advances a material aspect of the proposing party's case.'" *Roundup*, 390 F. Supp. 3d at 1112 (internal citations omitted).

Such testimony "is reliable if the knowledge underlying it has a reliable basis in the knowledge and experience of the relevant discipline." *City of Pomona v. SQM N. Am. Corp.*, 750 F.3d 1036, 1044 (9th Cir. 2014). Typically, a party seeking admission of an expert can demonstrate reliability under Rule 702 by "[e]stablishing that an expert's proffered testimony grows out of pre-litigation research or that the expert's research has been subjected to peer review . . . ." *Daubert II*, 43 F.3d at 1318. If such evidence is unavailable, "the experts must explain precisely how they went about reaching their conclusions and point to some objective source—a learned treatise, the policy statement of a professional association, a published article in a reputable scientific journal or the like—to show that they have followed the scientific method, as it is practiced by (at least) a recognized minority of scientists in their field." *Id*. at 1319. Ultimately, "[t]he focus of the reliability inquiry is on the principles and methodology an expert uses in forming her opinions rather than the expert's conclusions. But in conducting the reliability analysis, the Court must also consider whether, for a given conclusion, 'there is simply too great an analytical gap between the data and the opinion proffered.'" *Roundup*, 390 F. Supp. 3d at 1112 (citing *General Electric Co. v. Joiner*, 522 U.S. 136, 146 (1997)).

When resolving *Daubert* motions, the Court assumes a "gatekeeping responsibility" but possesses neither "the obligation [n]or the authority to become [an] amateur scientist[] in order to perform that role." *Daubert I*, 509 U.S. at 600-01 (Rehnquist, C.J. concurring in part). "The district court is not tasked with deciding whether the expert is right or wrong, just whether his testimony has substance such that it would be helpful to a jury." *Alaska Rent-A-Car, Inc. v. Avis Budget Grp., Inc.*, 738 F.3d 960, 969-70 (9th Cir. 2013). Accordingly, the Court must "screen the jury from unreliable nonsense opinions, but not exclude opinions merely because they are impeachable." *Id*. at 969. "Vigorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible evidence." *Daubert I*, 509 U.S. at 595. That said, "[t]he district court

'cannot abdicate its role as gatekeeper,' nor 'delegat[e] that role to the jury.'" *Engilis*, 151 F.4th at 1050 (citation omitted) (alterations in original). Consequently, "there is no presumption in favor of admission," and it is the Court's role to ensure a party seeking admission of expert testimony shows, by a preponderance of the evidence, the testimony satisfies Rule 702. *Id*. at 1049.

B. General Causation

Plaintiffs seek admission of experts that opine whether the baby foods at issue here are capable of causing Plaintiffs' alleged injuries, namely, the development of ASD and/or ADHD.

"'Generic causation' has typically been understood to mean the capacity of a toxic agent . . . to cause the illnesses complained of by plaintiffs. If such capacity is established, 'individual causation' answers whether that toxic agent actually caused a particular plaintiff's illness." *In re Hanford Nuclear Rsrv. Litig.*, 292 F.3d 1124, 1129 (9th Cir. 2002) (citations omitted). For general causation, the relevant inquiry is "whether exposure to a substance for which a defendant is responsible, . . . at the level of exposure alleged by plaintiffs, is capable of causing a particular injury or condition in the general population." *Id*. at 1133. This differs from "individual causation," which "refers to whether a particular individual suffers from a particular ailment as a result of exposure to a substance." *Id*.; *see also Engilis*, 151 F.4th at 1045 ("In a 'toxic tort claim for physical injuries,' a plaintiff must 'show that he was exposed to chemicals that could have caused the physical injuries he complains about (general causation), and that his exposure did in fact result in those injuries (specific causation).'" (citations omitted)). Though general causation does not inquire as to any individual plaintiff in the MDL, Plaintiffs must establish a causal relationship between their injuries and the alleged toxic heavy metals in baby food, at "levels people realistically may have experienced." *Hardeman v. Monsanto Co.*, 997 F.3d 941, 963 (9th Cir. 2021).

So, the general causation question here is whether a child's consumption of each Defendant's baby food products is capable of causing ASD or ADHD. Further, it is not enough to say the baby food can cause generalized neurological injury; rather, it must be the specific neurological injury alleged here—namely, ASD and ADHD. *See, e.g.*, *Roundup*, 390 F. Supp. 3d at 1115 (noting it was not sufficient to show the pesticide at issue in the case caused cancer,

generally, but that the plaintiffs must show it caused the specific type of cancer alleged). This framing of the general causation question guides the Court's analysis of whether a proposed expert's testimony is relevant to this phase of the multidistrict litigation.

SCIENTIFIC BACKGROUND

Having framed the general causation inquiry, the Court reviews key terms and concepts raised throughout the proposed experts' reports. This case implicates three overlapping scientific disciplines: epidemiology, toxicology, and exposure science. The Federal Judicial Center's Reference Manual on Scientific Evidence (3d. ed.) provides a helpful primer on these subjects.

"Epidemiology is the field of public health and medicine that studies the incidence, distribution, and etiology of disease in human populations." Michael D. Green, D. Michal Freedman & Leon Cordis, *Reference Guide on Epidemiology*, *in* REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 551, 551 (3d ed. 2011). This scientific field is particularly relevant to the general causation question in toxic tort cases, since epidemiology asks whether an agent is capable of causing disease, rather than whether it did so in a particular individual. *Id*. at 552. Epidemiological studies identify associations between an agent and an outcome, which "may or may not be causal." *Id*. at 553. "Assessing whether an association is causal requires an understanding of the strengths and weaknesses of the study's design and implementation, as well as a judgment about how the study findings fit with other scientific knowledge." *Id.*

Toxicology "is the study of the adverse effects of chemical and physical agents on living organisms." Bernard D. Goldstein & Mary Sue Henifin, *Reference Guide on Toxicology*, *in* REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 635, 636 (3d ed. 2011). "For the most part, toxicological study begins with a chemical or physical agent and asks what impact it will have, while toxic tort cases begin with an individual or a group that has suffered an adverse impact and makes claims about its cause." *Id*. at 635. Even so, toxicology can "provide scientific information regarding the increased risk of contracting a disease at any given dose and help rule out other risk factors for the disease." *Id*. at 638. In this way, it complements epidemiological analysis and "contributes to the weight of evidence supporting causal inferences . . . ." *Id.* Further, when reviewing toxicological evidence, three fundamental tenets should be kept in mind:

> First, "the dose makes the poison"; this implies that all chemical agents are intrinsically hazardous—whether they cause harm is only a question of dose. Even water, if consumed in large quantities, can be toxic. Second, each chemical or physical agent tends to produce a specific pattern of biological effects that can be used to establish disease causation. Third, the toxic responses in laboratory animals are useful predictors of toxic responses in humans.

Id. at 636-37.

Lastly, exposure science "is the study of how people can come into contact with (are exposed to) chemicals that may be present in various environmental media (air, water, food, soil, consumer products of all types) and of the amounts of those chemicals that enter the body as a result of these contacts." Joseph V. Rodricks, *Reference Guide on Exposure Science*, *in* REFERENCE MANUAL ON SCIENTIFIC EVIDENCE 505, 507 (3d ed. 2011). "To evaluate whether individuals or populations exposed to a chemical are at risk of harm, or have actually been harmed, the information that arises from epidemiological and toxicological studies is needed, as is the information on the exposures incurred by those individuals or populations." *Id*. at 505. Indeed, "[u]nderstanding exposure is essential to understanding whether the toxic properties of chemicals have been or will be expressed. Thus, claims of toxic tort or product liability generally require expert testimony not only in medicine and in the sciences of epidemiology and toxicology, but also testimony concerning the nature and magnitude of the exposures incurred by those alleging harm." *Id*.

Plaintiffs submit experts in each of these fields to offer a general causation opinion that Defendants' baby food products can cause ASD and ADHD due to their lead and arsenic levels.

A. Varieties of Scientific Studies

Plaintiffs' proposed experts considered hundreds of scientific articles in the course of rendering their opinions, nearly all of which fall within three categories: case-control studies, cohort studies, and meta-analyses of those studies.[2] The Court broadly summarizes the features and limitations of these kinds of studies prior to reviewing the specific scientific evidence the

[2] Plaintiffs' proposed experts also consulted a limited number of cross-sectional and ecological studies. Those studies did not feature prominently either in the experts' reports or during the hearing on Defendants' motions. Therefore, the Court does not provide an additional gloss on the qualities of those studies.

proposed experts contemplated.

A case-control study compares two groups: individuals who have a disease (cases) and individuals who do not have the disease (controls). *Reference Guide on Epidemiology* at 559. The researcher then considers the past exposures of those groups to determine if exposure to a particular agent appears more frequently among the case population. *Id*. at 560. Case-control studies are more cost-effective than other kinds of studies and may be "particularly useful in the study of rare diseases," since gathering a sufficient sample size proves challenging. *Id*. That said, case-control studies can suffer from bias that limits a conclusion about causation. Two examples of such bias are "selection bias" and "information bias." "Selection bias refers to the error in an observed association that results from the method of selection of cases and controls (in a case-control study) or exposed and unexposed individuals (in a cohort study). The selection of an appropriate control group has been described as the Achilles' heel of a case-control study." *Id*. at 583-84. "Information bias is a result of inaccurate information about either the disease or the exposure status of the study participants or a result of confounding. In a case-control study, potential information bias is an important consideration because the researcher depends on information from the past to determine exposure and disease and their temporal relationship." *Id*. at 585. Such limitations must be accounted for in determining the reliability of a given case-control study.

Conversely, "[i]n cohort studies, researchers define a study population without regard to the participants' disease status." *Id*. at 557. Rather, the population is divided by exposure to the agent of interest, and then researchers observe whether the disease later develops. *Id*. at 557-58. "One advantage of the cohort study design is that the temporal relationship between exposure and disease can often be established more readily than in other study designs, especially a case-control design . . . ." *Id*. at 558. "This temporal relationship is critical to the question of causation, because ***exposure must precede disease onset*** if exposure caused the disease." *Id*. (emphasis added). Of course, cohort studies also suffer from certain limitations. One such limitation is the potential for confounding variables. Since "the investigator has no control over what other exposures a subject in the study may have had, an increased risk of disease among the exposed

group may be caused by agents other than the exposure of interest." *Id*. at 559. Accordingly, an expert should consider potential confounders in assessing the reliability of a cohort study.

Finally, "[m]eta-analysis is a method of pooling study results to arrive at a single figure to represent the totality of the studies reviewed." *Id*. at 607. "In a meta-analysis, studies are given different weights in proportion to the sizes of their study populations and other characteristics." *Id*. Such an analysis can be helpful in determining the relationship between an agent and its biological effect since it combines various studies that may have lacked statistical power to provide more meaningful results. *Id*. Ideally, a meta-analysis combines the results of various experimental studies that share similar methodology; however, oftentimes, researchers only have access to observational studies, which may vary more significantly in their approach. *Id*. This heterogeneity places limits on the conclusions to be drawn from a meta-analysis. For instance, differences in study design, population, or quality may undermine the validity of a meta-analysis' single estimate of effect. *Id*. Further, the authors of a meta-analysis must make decisions about which studies qualify for inclusion and which do not. This presents an opportunity for bias that can infect the results. So, an expert must carefully consider this potential for bias in a meta-analysis as well as the differences in methodology among the studies it comprises.

B. Summary of Scientific Evidence

With an understanding of the types of studies marshalled in support of the proposed experts' opinions, the Court now reviews a sampling of those publications. This list incorporates certain studies which featured prominently in the experts' reports and during the hearing. As explained at the outset of this Order, no expert has identified a study considering the relationship between consumption of baby food and development of ASD or ADHD. However, various studies have investigated the association between biological lead and arsenic levels and these two conditions.

1. Lead and ASD

Arora et al. (2017) is a case-control study involving sets of mono- and dizygotic twins, where one sibling had been diagnosed with ASD and the other had not. The researchers utilized a "validated tooth-matrix biomarker" to estimate lead exposure levels for each sibling in the twin

pair. Unlike other biomarkers, the tooth matrix presents more extensive and accurate historical exposure information—different parts of the baby teeth, and the lead present in those parts, correspond to different periods in the child's development. Therefore, the Arora et al. (2017) study differs from other case-control studies in that it presents more objective historical exposure information as compared to mere recall by study participants. The study authors ultimately found a statistically significant association between lead levels at ages 10 to 20 weeks and development of autistic behaviors.

Kim et al. (2016) is a cohort study involving Korean children between 7 and 8 years old, who had not been diagnosed with ASD. In selecting the cohort, the researchers controlled for sex, fetal and environmental tobacco smoke exposure, paternal and maternal education levels, monthly family income, low birth weight, breastfeeding, and gestational age at delivery. The researchers continued to observe the cohort and measured their blood lead levels at various intervals. Further, the study authors measured autistic behaviors of the cohort using the Autism Spectrum Screening Questionnaire ("ASSQ") and Social Responsiveness Scale ("SRS"). That said, the authors did not establish a baseline ASSQ or SRS score for any participant in the study. As for results, the study authors noted increased blood lead levels at ages 7 to 8 showed a statistically significant positive association with scores on the ASSQ and SRS.

Alampi et al. (2021) is a prospective cohort study from the Maternal-Infant Research on Environmental Chemicals (MIREC) Canadian pregnancy cohort. The researchers assessed maternal blood lead levels, and arsenic levels, during the prenatal period, between 6- and 13-weeks gestation, and then utilized the SRS to measure autistic behaviors in those children between 3 and 4 years old. In analyzing the results, the study authors determined there was an association between maternal blood lead levels and SRS scores. They further observed a stronger association at the higher end of SRS scores, which they suggest is indicative of high levels of lead susceptibility among those children demonstrating the most autistic behaviors. As for arsenic levels, the authors observed increasing effect estimates for SRS scores as arsenic levels increased in maternal samples.

2. Lead and ADHD

Ji et al. (2018) is a cohort study looking at the relationship between lead exposure and a diagnosis of ADHD among members of the Boston Birth Cohort. The study authors measured blood lead levels of participants and then subsequently compared those levels to electronic health records to discern whether there had been a physician diagnosis of ADHD. Ultimately, the authors observed a statistically significant positive association between blood lead levels and ADHD diagnosis. However, this statistically significant association was found only among boys—not girls—and only at blood lead levels exceeding 5 micrograms per deciliter. There was no statistically significant association at lower blood lead concentrations.

Rosenauer et al. (2024) is a meta-analysis of 14 case-control studies considering the relationship between lead exposure and ADHD. The authors' calculation of heterogeneity among the studies was high, indicating differences between the 14 studies may affect their comparability in a meta-analysis. Even so, the authors ultimately concluded there was a statistically significant positive association between lead exposure and diagnosis of ADHD.

3. Arsenic and ASD

Skogheim et al. (2021) is a Norwegian case-control study regarding the relationship between blood lead and arsenic levels and ASD. The study authors looked at stored maternal blood samples and measured blood lead levels of women who had children later diagnosed with ASD, according to national disease registers. From their analysis, the authors determined there was a statistically significant association between arsenic levels—within a particular range in pregnant women—and the child's later ASD diagnosis. The results were similar for observed blood lead levels.

Doherty et al. (2020) is a cohort study based on the New Hampshire Birth Cohort. The study considered the presence of various metals in maternal toenail samples at two points: 27-weeks gestation and 4-weeks postpartum. Infant toenail samples were also taken at 6 weeks old. The authors then tested the children at 3 years old to determine an SRS score. From this assessment, the study authors concluded there was an association between arsenic levels in the infant toenail samples and the later score on the SRS.

C. Bradford Hill Criteria

Following review of the various studies on lead and arsenic exposure as they relate to Plaintiffs' alleged injuries, many of the proposed experts deploy the Bradford Hill criteria to make a causal determination. In *In re Roundup Product Liability Litigation*, the court provided a summation of the constituent factors in the analysis:

> Epidemiology studies examine whether an association exists between an agent like [lead or arsenic in baby food] and an outcome like [ASD or ADHD]. Whether that agent causes the outcome, however, cannot be proven by epidemiological studies alone; an evaluation of causation requires epidemiologists to exercise judgment about the import of those studies and to consider them in context. Once epidemiologists have concluded from the studies that there is an association between an agent and an outcome, they often assess causation through a framework called the 'Bradford Hill criteria.' These criteria are named for Sir Austin Bradford Hill, who wrote a 1965 article that articulated nine 'viewpoints' now generally accepted to be relevant to assessing causation. Broadly, these factors are: (1) the strength of the association; (2) consistency; (3) specificity; (4) temporality; (5) biological gradient or dose response; (6) biological plausibility; (7) coherence with other scientific knowledge; (8) experimental evidence; and (9) analogy."

Roundup, 390 F. Supp. 3d at 1116 (citing Austin Bradford Hill, *The Environment and Disease: Association or Causation?*, 58 Proceedings of the Royal Society of Medicine 295 (1965)). These criteria connect the proposed experts' review of studies showing *associations* between an agent and a disease, to an opinion on whether the agent *caused* the disease.

DISCUSSION

As noted above, the relevant question during this phase of the MDL is: whether a child's consumption of each Defendant's baby food—at realistic doses—is capable of causing ASD or ADHD. Because Plaintiff's causation theory is premised on lead and arsenic in the baby food products, this general causation question implicates the fields of exposure science, toxicology, and epidemiology. Plaintiffs must ultimately present reliable evidence as to how much lead and arsenic children are realistically exposed to by consuming Defendants' baby food, what dose of those metals makes it into children's bodies, and once internalized, whether those metals can cause ASD and ADHD. So, the single general causation question in this MDL comprises four constituent questions: 1) What is a realistic baby food consumption pattern for children (i.e. how

much and what kinds of food) within the relevant postnatal feeding timeframe? 2) Based on that consumption, what quantity of lead and arsenic are the children exposed to through the food? 3) How much of the lead and arsenic consumed by the children is absorbed by the body? And 4) can a body burden of lead or arsenic at that level cause ASD or ADHD? Plaintiffs offer proposed expert opinions as to each of these questions.

The Court now turns to those opinions. Defendants' motions to exclude can be organized into three broad categories: 1) challenges to Plaintiffs' exposure experts, Ms. Barr and Dr. Jones; 2) challenges to Plaintiffs' biological plausibility expert, Dr. Shapiro; and 3) challenges to Plaintiffs' epidemiology and toxicology experts, Drs. Gardener, Hu, Ritz, Aschner, and Guilarte.

I. EXPOSURE EXPERTS

Plaintiffs' proposed exposure experts present opinions on the first three of the four questions that comprise the general causation inquiry. Ms. Priscilla Barr, infant dietician, contributed to the development of hypothetical menus—proposed consumption patterns for children within a certain age range (4 months to 3 years), that were specific to an individual defendant's products. (Dkt. No. 611-21.) Dr. Jones, exposure scientist, then utilized these hypothetical menus, and data from Defendants' heavy metal testing results, to estimate lead and arsenic exposure. She also employed additional methods to establish a blood lead level based on that exposure.

Defendants do not impugn, nor does the Court question, the academic credentials of these witnesses and their expertise in their respective fields. Accordingly, the Court finds Plaintiffs have met their burden to show qualification of their experts under Federal Rule of Evidence 702. The dispute lies in whether Ms. Barr and Dr. Jones have employed a reliable scientific method based on sufficient facts and data.

A. Ms. Barr's Expert Report

Ms. Barr offers two opinions related to Plaintiffs' hypothetical menus that estimate baby food consumption patterns for purposes of general causation:

> The diets described in the menus and consumption patterns of each Defendant's baby food products (Appendix D) are adequate to meet caloric and key nutrient requirements for each age group of children

> ages 0-3 in order to sustain adequate growth and development; however, I do not opine about whether any contamination in those products, i.e. heavy metals, would interfere with neurodevelopment.
>
> The potential diets described in the sample menus for each Defendant (Appendix D) reflect possible patterns of consumption for American children ages 0-3 during the time period relevant to this litigation.

(Dkt. No. 611-19 at 5.) Appendix D refers to a series of charts Plaintiffs created that present a consumption pattern from age 0 to 3 years old, divided into six time periods: 1) Less than 1 month; 2) 1 – 4 months; 3) 4 – 6 months; 4) 6 months – 1 year; 5) 1 year – 2 years; and 6) 2 years – 3 years. (*See* Dkt. No. 611-21.) Within each period of consumption, Plaintiffs include a list of product categories (e.g. jars, pouches, cereals, bars, etc.) and then indicate an average number of daily servings a child would consume of that product category. (*Id.*) Each chart contains only products manufactured by a single defendant within a certain range of years (e.g. 2019-21). (*Id.*) As to each product category, Plaintiffs provide a list of specific consumables from Appendix A to the Amended Master Complaint that fall within that category and could contribute to the average daily servings for the child. (*Id.*) That said, some of the consumption grids contain footnotes, which state that within certain product categories, at least one serving must be from a specific product (e.g. a jar of sweet potato puree), while the other servings may come from a list of various options. (*Id.*) Additionally, the consumption grids incorporate two further assumptions: first, the child does not contemporaneously breastfeed during the periods they consumed the food; and second, the child may contemporaneously consume table food, when age appropriate. (Dkt. No. 611-19 at 5.)

A sample of these consumption grids and product lists is provided below, illustrating a hypothetical consumption pattern for products manufactured by Defendant Beech-Nut:

//

//

//

//

//

//

BEECH-NUT
Consumption Grid

Product	< 1 Month	1- < 4 Mos.	4 - < 6 Mos.	6 Mos. - < 1 yr.	1 - < 2 yrs.	2 - < 3 yrs.
Jars	--	--	3 ser (339g)[1] (135-230 kcals.)	2 ser (226g)[2] (65-170 kcals.)	1 ser (113g) (60-100 kcals.)	--
Pouches	--	--		2 ser. (198g) (105-140 kcals.)	2 ser. (198g) (90-150 kcals.)	2 ser. (198g) (90-150 kcals.)
Cereals	--	--	1 ser (15g) (50 kcals.)	3 ser (45g) (150 kcals.)	3 ser (45g) (150 kcals.)	--
Bars	--	--	--	--	1 ser. (22g) (70-80 kcals.)	2 ser (44g) (150 kcals.)
Yogurt Melts (8+ months)	--	--	--	2 ser. (14g) (50 kcals.)	3 ser. (21g) (75 kcals.)	3 ser. (21g) (75 kcals.)
Baked Crisps	--	--	--	--	--	2 ser. (14g) (50 kcals.)
Subtotal Calories			185-280 kcals.	360-510 kcals.	445-555 kcals.	390-450 kcals.
Total Grams			354g	483g	399g	284g

(Dkt. No. 611-21 at 3.)

Beech-Nut Products (from Appendix A)

Time Window: 2019-2021

4- < 6 Months

Jars (Stage 1) [3]**:**
(1) Stage 1 Apple
(5) Stage 1 Carrots
(7) Stage 1 Green Beans
(11) Stage 1 Organics Sweet Potato
(12) Stage 1 Organics Prunes

Cereals:
(93) Rice Cereal

6 - < 1 yr

Jars[4]**:**
(7) Stage 1 Green Beans
(46) Stage 2 Organics Butternut Squash & Sweet Corn
(23) Stage 2 Apple, Cinnamon & Granola
(58) Stage 2 Sweet Carrots
(61) Stage 2 Sweet Potato
(67) Stage 3 Organics Sweet Potato & Barley

Pouches:
(69) Stage 2 Apple, Mango & Carrot
(73) Stage 2 Apple, Sweet Potato & Pineapple
(78) Stage 2 Banana, Pear & Sweet Potato
(79) Stage 2 Carrot Zucchini & Pear

Cereals:
(93) Rice Cereal

Yogurt Melts (8+ months):
(98) Apple & Pumpkin Fruit & Veggie

(*Id.* at 4.)

Plaintiffs have failed to show, by a preponderance of the evidence, Ms. Barr's opinions—and the menus she approves—are both relevant and reliable. *See Primiano*, 598 F.3d at 564 ("In sum, the trial court must assure that the expert testimony 'both rests on a reliable foundation and is relevant to the task at hand.'").

To start, the menus included as an appendix to Ms. Barr's report is not "the product of reliable principles and methods." Fed. R. Evid. 702(c). Ms. Barr did not construct any of the menus herself; rather, she was given the hypothetical menus by Plaintiffs' counsel and then performed her plausibility analysis. (Dkt. No. 611-19 at 4. ("These diets, which are set forth in Appendix D, were provided to me by Plaintiffs' counsel and include hypothetical menus of Defendants' baby food products and potential consumption patterns.).) Since Ms. Barr accepted the menus from Plaintiffs' counsel, her report does not address the basis for the assumptions included within those consumption patterns. For instance, Ms. Barr does not address how the products included on the hypothetical menus were selected. To use the Beech-Nut consumption grid as an example, Appendix A to the Amended Master Complaint contains 103 Beech-Nut products at issue in this MDL. (Dkt. No. 451 at 67-69.) Only 24 of those products are included among consumption options in the hypothetical menu. (Dkt. No. 611-21 at 7.) There is no explanation in her report, in her deposition testimony, or in the record at all, as to why these 24 products were chosen while others were omitted. Indeed, Ms. Barr testified that she does not know why these products—or any of the products on the seven hypothetical menus—were chosen. (Dkt. No. 611-40 at 25 ("**Q.** And do you have any understanding as to why these particular foods were selected? **A.** No. I was not told by [sic] how any particular food was selected.").)

Nor does Ms. Barr provide a reason for the requirement that a certain number of servings within a given product category must come from a specific product. Turning to the Beech-Nut grid once more, a child between 6 months and 1 year old is understood to consume two servings of jar products daily, (Dkt. No. 611-21 at 3); however, one of those jars must be sweet potatoes or sweet carrots, while the second jar can be any from a list of five options: "green beans; banana, mango & sweet potato; butternut squash & sweet corn; apple, cinnamon & granola; sweet potato & barley," (*id*. at 3 n.2). Ms. Barr does not mention this feature of the hypothetical menus in her

report, let alone its purpose, or what it is based upon. On these questions as well, she similarly testified she does not know the basis. (*See* Dkt. No. 611-40 at 36 ("**Q.** Are you aware, you yourself, are you independently aware of any scientific methodology or rationale as to why certain product types were included or excluded from Appendix D? **A.** I do not -- no, because as mentioned, I did not create Appendix D and was not involved in the creation of these menus. I was just asked to evaluate them.").)

Further, Ms. Barr does not identify any basis for the assumption that children consuming these hypothetical menus would be exclusively formula-fed and would not be contemporaneously breastfed. In a footnote within her report, Ms. Barr simply mentions she was told to make that assumption. (Dkt. No. 611-19 at 5 n.1; Dkt. No. 611-40 at 41 ("**Q.** The assumptions were provided to you? **A.** Yeah.").) The report's silence on these assumptions is emblematic of the broader problem here—Ms. Barr bases her opinion on menus created by Plaintiffs' counsel, and there is no way to assess the validity of that data, the method used to prepare it, or potential sources of bias in developing those consumption patterns. This absence of information on the hypothetical menus presents a reliability issue. *See, e.g.*, *GPNE Corp. v. Apple, Inc.*, No. 12-CV-02885-LHK, 2014 WL 1494247, at *4 (N.D. Cal. Apr. 16, 2014) ("Experts must follow some discernable methodology, and may not be 'a black box into which data is fed at one end and from which an answer emerges at the other.'" (citation omitted).). So, Plaintiffs have not met their burden to show an accepted, reliable scientific method was employed to construct the hypothetical menus.

As to the plausibility of those menus, Plaintiffs have also failed to show, by a preponderance of the evidence, that Ms. Barr's opinion comports with Rule 702. To begin, Ms. Barr's report operates from the understanding that a consumption pattern is "plausible" so long as it applies to at least one child in the United States. (Dkt. No. 611-40 at 34, 39.) This is a poor fit with the general causation standard, which requires evidence of causation from exposure at "levels people realistically may have experienced." *Hardeman*, 997 F.3d at 963. And because Ms. Barr's analysis focuses only on *possibility*, it often highlights the lack of realism in the hypothetical menus.

For example, Ms. Barr uses Feeding Infants and Toddler Study (FITS) data—as well as FDA data—to establish average levels of consumption for certain food groups (e.g. fruits, vegetables, and cereals). (Dkt. No. 611-19 at 23-24.) She then compares those averages to the quantities of those foods in the counsel-created hypothetical menus to determine plausibility. (*Id*.) For dry infant cereal, she cites the FDA 90th percentile consumption average for children 0-23 months old, which is 39 grams per day. (*Id*. at 24.) Though she notes the hypothetical menus for Beech-Nut and Hain exceed this 90th percentile average, she asserts it is "not a meaningful difference" because the quantity amounts to about half a teaspoon. (*Id*.) But Ms. Barr overlooks that the hypothetical menus for Nurture, Plum, Sprout, and Walmart contain ***no infant dry cereal products***, meaning they assume 0 grams consumed by the child per day. (*Id*.) This is not a "half teaspoon" difference, it is elimination of an entire food group, and the report offers no analysis as to whether this is a realistic consumption pattern. Such is the case for other food groups such as fruit-vegetable mixtures and single-ingredient root vegetable products—some menus exceed the 90th percentile of consumption and some menus do not include the food group at all. (*Id*. at 25.) For certain menus, these departures from realistic consumption patterns compound one another. The Sprout consumption grid illustrates this: based on Ms. Barr's report, the Sprout menu exceeds the 90th percentile of daily intake for fruits/vegetables, includes no infant dry cereal consumption, and no single-ingredient root vegetable consumption. (*Id*. at 24-25.) Ultimately, Ms. Barr fails to consider any of these idiosyncrasies in rendering her opinion.

Moreover, Ms. Barr does not consider that for certain menus, a food group may only have a single available product manufactured by the defendant. One such example is the Nurture consumption grid, which estimates two serving of "Yogis" per day from 6 months to 1 year of age, and then three servings per day from 1 year to 3 years of age. (Dkt. No. 611-21 at 16.) But there is only one product in the "Yogi" category: "Blueberry & Purple Carrot Yogis." (*Id*. at 20.) That means a child is expected to consume Blueberry & Purple Carrot Yogis every day for two and a half years. On this issue as well, Ms. Barr's report is silent. Of course, Ms. Barr's charge from Plaintiffs was not to create a menu that reflects a realistic consumption pattern for U.S. children; she was told what the menu was, and so her report functions as an ex-post analysis to justify its

plausibility. In this way, Ms. Barr's proffered opinions are "unduly results-driven," and lack a discernable, reliable method under Rule 702. *In re Viagra (Sildenafil Citrate) & Cialis (Tadalafil) Prods. Liab. Litig.*, 424 F. Supp. 3d 781, 796 (N.D. Cal. 2020). Therefore, the hypothetical menus, and her opinions as to their plausibility, are excluded.

Plaintiffs advance three arguments in response. First, they contend that counsel may supply experts with case-specific data to form the basis of an opinion, and so it is no issue that Plaintiffs' counsel developed the hypothetical menus. Second, they argue the menus are realistic given the plaintiffs in this MDL are not typical children and were exposed to the worst heavy metals. Last, they assert the menus need not be realistic, so long as the exposure to Defendants' products was "within 'the highest dose people might plausibly experience.'" (Dkt. No. 642 at 36 (citing *Roundup*, 390 F. Supp. 3d at 1113).) These arguments are insufficient to satisfy Plaintiffs' burden.

As to Plaintiffs' first point, counsel noted during the hearing that supplying the menu to Ms. Barr does not present a reliability issue because in any individual case, that exposure information would ordinarily be supplied by the plaintiff. So, general causation in this MDL is a stand-in for specific causation in an individual case. (Dkt. No. 700 at 23-25.) True, in a single-plaintiff toxic tort case, the causation question would be based on evidence of the plaintiff's actual exposure. But this is a consolidated action, and the parties have known since early days in this MDL that general causation would precede the specific causation phase. And, in any event, the general causation question is different from the specific causation question, and is not merely a stand-in. It requires Plaintiffs to put forth reliable evidence as to whether children who consume a realistic dose of Defendants' products could develop ASD or ADHD. Here, Plaintiffs' counsel has created hypothetical menus that purportedly represent a realistic dose of Defendants' products. Yet these menus are a black box; the record is silent as to how they were created. And, since Plaintiffs' expert was not involved in their creation, she cannot provide that information either. The Court would fail in its gatekeeping duty if it accepted these menus without an explanation of the method underlying their creation. *See United States v. Valencia-Lopez*, 971 F.3d 891, 900 (9th Cir. 2020) ("This 'prerequisite to making the Rule 702 determination that an expert's methods are

reliable' requires the district court to 'assure that the methods are adequately explained.'" (citation omitted)). Counsel's undisclosed strategy is not the same as a reliable scientific method, and so the menus fail to pass muster under Rule 702.

Regarding Plaintiffs' second point, at oral argument counsel insisted the menus are realistic because the plaintiffs in these cases are "not the typical U.S. child," and are "a very select group who ate the foods that tend to have the worst metals in them . . . ." (Dkt. No. 700 at 40.) But this is merely unsupported attorney argument. Nothing in the record explains how Plaintiffs' counsel selected the products on the hypothetical menus or why certain assumptions were provided to Ms. Barr. Moreover, Ms. Barr does not indicate in her report, or her deposition testimony, that the hypothetical menus were in any way representative of the plaintiffs in the MDL. Rather, she disclaimed any such knowledge. (Dkt. No. 611-40 at 85 ("**Q.** Did you assess whether the hypothetical menus in Appendix D are actually representative of any specific child who has filed a lawsuit in this litigation? **A.** Oh, no, I'm not aware of any specific child and their intake patterns from like these time periods.").) On this record, it is just as likely, if not more likely, counsel cherry-picked the products for their higher lead or arsenic content as it is counsel selected them based on any child's actual diet. Such cherry-picking would be impermissible under Rule 702. *See, e.g.*, *Waymo LLC v. Uber Techs., Inc.*, No. C 17-00939 WHA, 2017 WL 5148390, at *4 (N.D. Cal. Nov. 6, 2017) ("[An expert's] willingness to stitch together strategic fragments of contradictory evidence further indicates that he picked facts to suit his conclusions instead of drawing conclusions from reliable analysis of the facts."). There is zero evidence as to how counsel selected the products. And without such evidence, the Court is deprived of the opportunity to evaluate whether the seven hypothetical menus are reliable or realistic. Plaintiffs cannot meet their burden of showing admissibility by placing their "method" in an inaccessible black box.

Turning to Plaintiffs' third point, they misunderstand the standard discussed in *Roundup*, and later elaborated by the Ninth Circuit in *Hardeman*. In *In re Roundup*, the court distinguished the general causation inquiry from specific causation, noting the plaintiffs need only show the toxin (an herbicide known as "glyphosate") can cause the alleged disease "when people are

exposed to the highest dose people might plausibly experience." 390 F. Supp. 3d at 1113. The court then provided an example: "Picture, for instance, a professional gardener who has applied Roundup without using protective equipment several times per week, many hours per day, for decades." *Id.* From the *Roundup* court's analysis, it is clear that "the highest dose" is still constrained by a realistic hypothetical—the gardener scenario does not strain credulity. Plaintiffs appear to conflate this language with the idea that any "possible" dose suffices for general causation. But that is not the law. Indeed, in affirming the *Roundup* decision, the Ninth Circuit set out the standard binding on this Court: "To establish general causation, [Plaintiffs'] experts needed to show that [the toxin] can cause [the disease] at exposure levels people ***realistically*** may have experienced." *Hardeman*, 997 F.3d at 963 (emphasis added). For the reasons discussed in the Court's analysis of Ms. Barr's opinion, Plaintiffs have not established their counsel-created hypothetical menus represent a realistic consumption pattern for any children.

* * *

In sum, the hypothetical menus attached as Appendix D to Ms. Barr's report are not based upon a scientific method, let alone a reliable method. Further, they are not relevant, since they fail to fit the general causation requirement of a realistic exposure scenario. In this way, the menus do not "logically advance[] a material aspect of the proposing party's case." *Roundup*, 390 F. Supp. 3d at 1113 (citing *Messick v. Novartis Pharmaceuticals Corp.*, 747 F.3d 1193, 1196 (9th Cir. 2014)). Additionally, Ms. Barr's opinions as to the plausibility of those hypothetical menus are not the product of a reliable application of scientific methods or principles, and are unduly results-driven. So, Plaintiffs have not shown by a preponderance of the evidence that Ms. Barr's opinions and the hypothetical menus satisfy Rule 702. Therefore, the Court **GRANTS** Defendants' motion to exclude.

B. Dr. Jones' Expert Report

Dr. Jones performed an exposure assessment to determine "the exposure of infants and young children to arsenic and lead from Defendants' food products." (Dkt. No. 611-22 at 8.) To do so, she started from a "hypothetical consumption pattern provided by Plaintiffs' attorneys . . . ." (*Id.* at 7.) Then, using heavy metal testing results for Defendants' products, Dr. Jones

"calculate[d] daily intake of [arsenic] and [lead]" for a child consuming those products. (*Id*.) Once an exposure estimate had been calculated, Dr. Jones utilized the EPA's Integrated Exposure Uptake Biokinetic (IEUBK) Model to generate an expected blood lead level. (*Id*.) Since the IEUBK Model only estimates the body's uptake of lead, Dr. Jones did not supply an estimated blood arsenic level or other associated biomarker level beyond exposure.

Dr. Jones provides an extensive discussion on her methodology and her application of the "scenario evaluation approach" to conduct this exposure assessment. Included among the types of data she considered were:

> 1. Exposure factors that describe the rate at which baby food products are taken into the body by infants and young children at different life stages, specifically the food consumption rate for a specific menu of food items;
>
> 2. Frequency and duration of exposure that describe how often and for how long the exposure occurs, specifically the daily consumption of specific food products at each life stage; and
>
> 3. Concentrations of As and Pb in baby food products consumed by infants and young children.

(*Id*. at 10.) Further, Dr. Jones explains: her approach to calculating mean and max intake rates; her use of the IEUBK Model; her process for "cleaning" and organizing the data she received to conduct her analysis; and her process for interpreting "left-censored data" (test results that did not quantify a specific amount of heavy metals, but rather stated the amount was less than a certain threshold). (*Id*. at 11-17.)

Since her initial report, Dr. Jones has amended her calculations two times in response to critiques raised by Defendants and their proposed experts. (*See* Dkt. Nos. 611-24, 611-25.) As noted in her rebuttal report, the updated figures incorporate changes based on data entry errors and the addition of certain information that was not available prior to submission of her initial report. (Dkt. No. 611-24 at 8.) Though Defendants argue these repeated, significant adjustments to the majority of her calculations call into question their reliability, the issue of her revisions is more appropriately the subject of cross-examination, not exclusion. *See Daubert I*, 509 U.S. at 595 ("Vigorous cross-examination, presentation of contrary evidence, and careful instruction on the burden of proof are the traditional and appropriate means of attacking shaky but admissible

evidence."). Ultimately, Dr. Jones employs a reliable exposure assessment method that has been endorsed by scientific bodies like the EPA.

That said, Dr. Jones' report is based upon a hypothetical consumption pattern of Defendants' products that the Court has excluded for lacking reliability and relevance. *See* Section I.A., *supra*. These issues infect the core assumptions of Dr. Jones' opinions and render them inadmissible under Rule 702. Dr. Jones did not have any information about how the consumption patterns were constructed, nor did she question them, since it was "outside the scope of [her] task." (Dkt. No. 611-42 at 22-23.) This means two of the three data sources supporting her calculations come from Plaintiffs' counsel, without any scientific validation. An expert cannot blindly accept data without assessing its reliability prior to incorporating it into her analysis. *See, e.g., Baker v. Firstcom Music,* No. LA-16-CV-08931-VAP, 2018 WL 2676636, at *2 (C.D. Cal. May 8, 2018) ("First, experts are expected to verify the reliability of the data underlying their conclusions independently instead of simply adopting the representations of an interested party."); *Powell v. Anheuser-Busch Inc.*, No. CV 09-729-JFW, 2012 WL 12953439, at *7 (C.D. Cal. Sept. 24, 2012) ("Reliance on incomplete facts and data will make an expert's opinion unreliable because an expert must know 'of facts which enable him to express a reasonably accurate conclusion.'"). Indeed, Dr. Jones' colleague, Dr. Beate Ritz, testified she had assumed Dr. Jones would confirm the consumption patterns reflected realistic exposure scenarios. (Dkt. No. 699 at 184 ("**Q**. Okay. Did you do anything on your own to confirm whether the menus reflect realistic, real-world scenarios? **A.** I assume that Dr. Jones knows what she's doing and would have done that. **Q.** Okay. You assumed that Dr. Jones determined that the menus were realistic and plausible; isn't that true? **A.** I would assume that, yes.").) This failure to engage in any assessment of the consumption scenarios indicates Dr. Jones did not *reliably apply* the principles and methods of an exposure assessment in this case.[3] *See* Fed. R. Evid. 702(d).

[3] On reply, Defendants raised an additional argument regarding the reliability of Dr. Jones' use of the IEUBK model. Dr. Jones calculated blood lead levels by inputting her exposure calculations for food and then setting all other sources of exposure in the model to zero. (Dkt. No. 611-22 at 13.) Because the argument was only raised on reply, the Court does not consider this point as a basis for exclusion at this time. (Dkt. No. 700 at 27-28, 48.)

However, there is a serious question as to the reliability of this method. Dr. Hu testified he

Separately, Dr. Jones' use of these unrealistic consumption scenarios as the basis for her calculations undermines the relevance of her lead and arsenic exposure figures and estimated blood lead levels. "Federal judges must [] exclude proffered scientific evidence under Rules 702 and 403 unless they are convinced that it speaks clearly and directly to an issue in dispute in the case, and that it will not mislead the jury." *Daubert II*, 43 F.3d at 1321 n. 17. Though "[t]he relevancy bar is low," *Messick*, 747 F.3d at 1196, it still serves an important purpose, since expert evidence "can be both powerful and quite misleading because of the difficulty in evaluating it," *Daubert II*, 43 F.3d at 1321 n. 17 (citation omitted). Here, Dr. Jones' expert report has been offered in support of Plaintiffs' general causation position. That general causation inquiry requires Plaintiffs to present reliable evidence that realistic levels of exposure to Defendants' baby food can cause ASD and ADHD. Dr. Jones' opinion is not based on such a realistic exposure scenario.

Accordingly, the exposure calculations and blood lead levels presented in Dr. Jones' expert report lack both reliability and relevance. Plaintiffs have not shown, by a preponderance of the evidence, these calculations meet the criteria set out in Rule 702.

So, Defendants' motion to exclude is **GRANTED**.

II. BIOLOGICAL PLAUSIBILITY EXPERT

Plaintiffs also put forward Dr. Kevin Shapiro to opine on the biological plausibility of lead and arsenic influencing the development of ASD and ADHD symptoms. Dr. Shapiro is a clinical neurologist specializing in child neurology. (Dkt. No. 611-17 at 5.) He has been practicing for over 10 years and serves as the co-director of "the Cortica Innovation Network," where he researches "outcomes and novel therapies for children with autism." (*Id.*) Defendants do not contend Dr. Shapiro lacks sufficient qualifications to offer his opinions, and the Court is satisfied

---

had not used the IEUBK model in that way. (Dkt No. 705 at 84-85 (Dr. Hu testifying he typically makes an assumption about the value of other sources of exposure rather than zeroing them out).) Further, the reliability of the IEUBK model has been validated through actual testing of blood lead levels to confirm the accuracy of its projections. (Dkt. No. 611-22 at 12-13.) But Dr. Jones does not discuss the real-world validation of the model with respect to the assumptions underlying the hypothetical consumption patterns (e.g. the children are not breastfed, they consume a similar pattern of products every day, etc.). Nor does Dr. Jones cite to any peer-reviewed literature which has employed the IEUBK model while zeroing out various sources of exposure other than food. Indeed, during her deposition, she testified she herself could not recall seeing the model used this way in other studies. (Dkt. No. 611-42 at 76.)

he meets this threshold requirement of Rule 702.

A. Dr. Shapiro's Expert Report

Plaintiffs have made clear that Dr. Shapiro does not offer a general causation opinion—meaning he does not assert Defendants' products, when consumed at realistic levels, can cause ASD or ADHD in children. (*See* Dkt. No. 642 at 68.) Though Dr. Shapiro does not employ the Bradford Hill Criteria to reach a causal conclusion, his opinion goes to a specific component of that analysis: biological plausibility. *See Roundup*, 390 F. Supp. 3d at 1116 (listing "biological plausibility" among the nine factors included within the Bradford Hill Criteria). His expert report distills this analysis into nine opinions, which broadly describe: the pathogenesis of ASD and ADHD in the brain; the role of oxidative stress in this process; genetic and environmental factors that bear on ASD and ADHD symptoms; a summary of literature regarding the impact of heavy metals on neuron function, intelligence, and behavioral problems; as well as the role of nutrients in offsetting the neurotoxic effects of heavy metals in food. (Dkt. No. 611-17 at 8-9.)

To reach these conclusions, Dr. Shapiro drew upon his experience in the practice of neurology and conducted a literature review of relevant scientific publications. (*Id*. at 9-10.) An opinion based upon professional experience, supported by published scientific literature, has been recognized as a reliable scientific method for purposes of a *Daubert* motion. *See, e.g.*, *Wendell v. GlaxoSmithKline LLC*, 858 F.3d 1227, 1236 (9th Cir. 2017) (noting an expert's opinion was reliable when based on his "wealth of experience and additional literature"). So, the Court sees no reliability issue as to Dr. Shapiro's method of analysis.

Since he does not offer a general causation opinion, Defendants' arguments for exclusion are relatively narrow. To wit, Defendants assert Dr. Shapiro did not reliably apply his method to his expert report because: 1) he did not properly consider the potential for nutrients in food to offset absorption or the neurotoxic effects of lead/arsenic, and 2) he did not adequately address the dosage at which lead/arsenic can have neurotoxic effect such that a child would develop ASD or ADHD. Ultimately, neither argument warrants exclusion of Dr. Shapiro's opinions.

As to Defendants' first point, Dr. Shapiro's expert report does review literature considering the effect of certain nutrients on the uptake and mitigation of heavy metals' neurotoxic effects.

(*See* Dkt. No. 611-17 at 36-37.) In his analysis, which cites over a dozen publications, Dr. Shapiro reviews studies investigating the relationship between zinc, calcium, iron, vitamin B-12, and folate levels, and the neurotoxic effects of lead. (*Id*.) After weighing these findings, Dr. Shapiro concludes:

> In sum, the available data do not permit the conclusion that contemporaneous exposure to heavy metals and most beneficial nutrients via baby food consumption (or food consumption generally) will result in a clinically significant impact on the absorption or neurotoxicity of heavy metal in early life. The literature on beneficial minerals such as zinc and iron is limited and inconsistent, with few recent studies. B12 and folate do appear to be important for detoxification of heavy metals, but there is little evidence that neurodevelopmental toxicity can be prevented by typical dietary intake of these vitamins. If anything, much higher than normal intake of B12 and folate may be required even to partially offset neurodevelopmental effects of heavy metal toxicity.

(*Id*. at 37.) This opinion does not state definitively that nutrients have no relationship to the absorption and effect of lead. Indeed, elsewhere in the report, Dr. Shapiro notes nutrient deficiency has been implicated in the pathogenesis of ASD. (*Id*. at 26.) Rather, Dr. Shapiro notes the data on the subject is limited and inconsistent, and based on his scientific training, one cannot conclude cotemporaneous nutrient intake mitigates the effects of lead exposure. Defendants take issue with this conclusion, and observe Dr. Shapiro considered only single-nutrient studies, not studies of whole food consumption. (*See* Dkt. No. 614 at 22-27.) As Dr. Shapiro's testimony confirmed, he reviewed Ziegler et al. (1978) in his report, which looked at consumption of whole baby food and lead absorption. (Dkt. No. 699 at 106.) In that respect, he did not solely consider single-nutrient studies. Regardless, the Court's role per Rule 702 is not to decide whether the expert is right or wrong, it is to ensure his methods are reliable. *See Alaska Rent-A-Car, Inc.*, 738 F.3d at 969-70. Here, the Court does not find "there is simply too great an analytical gap between the data and the opinion proffered," *Joiner*, 522 U.S. at 146, especially given the limited nature of that opinion. Defendants are free to cross-examine Dr. Shapiro as to the studies he considered, how he weighed their results, as well as any inconsistencies therein, but the Court will not exclude his opinion entirely.

Regarding Defendants' second argument, they contend Dr. Shapiro offers an unreliable

opinion that exposure to heavy metals at any level can cause ASD and ADHD. (Dkt. No. 614 at 30-31.) In support, they rely on excerpts from Dr. Shapiro's deposition testimony when he addressed a hypothetical scenario in which a single carrot with 1 microgram of arsenic could cause ASD. (Dkt. No. 611-37 at 39.) Whatever his response to that line of questioning, that opinion is not offered in Dr. Shapiro's expert report. He does not assert a causation opinion as to Defendants' products and has no opinion about the heavy metal exposure figures calculated by Dr. Jones for those products. (*See* Dkt. No. 642 at 88 ("Dr. Shapiro has no opinions about Dr. Jones's numbers.").) Nor does his report discuss any threshold level of lead or arsenic exposure that would indicate a causal relationship to diagnosis of ASD or ADHD. The most Dr. Shapiro opines as to specific heavy metal exposure amounts is a summary of the evidence he reviewed: "Notably, both lead and arsenic have been repeatedly associated with autism, and lead has been repeatedly associated with ADHD at low doses in the scientific literature." (Dkt. No. 611-17 at 38.) He does not identify a dose, nor does he conclude that *any* dose could cause ASD or ADHD; the report merely indicates that studies have observed an association between the heavy metals and ASD/ADHD at "low levels." Defendants' concerns do not go to an opinion actually offered by Dr. Shapiro in this case, and thus pose no problem under Rule 702.

For the reasons discussed above, Plaintiffs have carried their burden to show Dr. Shapiro's proffered opinions comport with Rule 702. Therefore, the Court **DENIES** Defendants' motion to exclude.

III. EPIDEMIOLOGY & TOXICOLOGY EXPERTS

The final component question of general causation is whether the amount of lead or arsenic a child absorbs from each Defendant's baby food products can cause ASD or ADHD. To answer this question, Plaintiffs submit the opinions of three epidemiologists and two toxicologists: Dr. Hannah Gardener, a Research Associate Professor in the Miller School of Medicine at the University of Miami; Dr. Howard Hu, a professor of Preventive Medicine in the Keck School of Medicine at the University of Southern California, and an adjunct professor at the University of Michigan School of Public Health; Dr. Beate Ritz, a professor of epidemiology at the UCLA Fielding School of Public Health; Dr. Michael Aschner, a professor in the Departments of

Molecular Pharmacology, Neuroscience, and Pediatrics at Albert Einstein College of Medicine; and Dr. Tomas Guilarte, a professor in the Department of Environmental Health Sciences at Florida International University. No challenge has been made to the qualifications of these proposed experts, and the Court has no independent concern as to their expertise for purposes of Rule 702.

Defendants advance multiple arguments as to the reliability of the experts' reports. That said, Defendants do not organize these arguments by expert witness; rather, they identify reliability issues they contend apply across all five experts' opinions. The Court's analysis follows this same course. Ultimately, Plaintiffs have failed to show, by a preponderance of the evidence, that their epidemiology and toxicology experts' opinions comply with Rule 702. Two reliability issues undermine the experts' analyses.

First, the experts rely on Dr. Jones' lead and arsenic exposure calculations, as well as her blood lead estimates, to form their causation opinions. Namely, they assume Plaintiffs' lawyer-created menus represent realistic consumption patterns for children, and they accept Dr. Jones' blood lead calculations based on those unreliable menus. Since Ms. Barr's and Dr. Jones' reports are inadmissible, so, too, are the expert reports that rely upon them.

Second, Plaintiffs' epidemiology and toxicology experts fail to bridge the analytical gap between the data they consider and the causation conclusions they offer. *See Joiner*, 522 U.S. at 146 ("A court may conclude that there is simply too great an analytical gap between the data and the opinion proffered."). There are ***no studies*** considering whether consumption of baby food is associated with ASD or ADHD, and the experts fail to scientifically justify extrapolating from the studies they cite. For the epidemiology experts, this failure to close the gap between the studies and their opinions results in an unreliable application of the Bradford Hill Criteria. For the toxicology experts, their weight-of-the-evidence analysis fails to reliably extrapolate from the body of research, resulting in a similar *Joiner* problem. The Court addresses each of these grounds for exclusion further.

A. Reliance on Dr. Jones' Expert Report

Each of the five experts states they have reviewed Dr. Jones' calculations, and the

exposure levels indicated therein match or exceed the levels identified in the scientific literature such that they could cause ASD or ADHD in a child. (*See* Dkt. Nos. 611-2 at 71 (Ritz), 611-5 at 42 (Hu), 611-9 at 3 (Gardener), 611-11 at 76 (Aschner), 611-14 at 54 (Guilarte).) Indeed, Dr. Jones' analysis is a critical link in the general causation chain—her calculations connect general epidemiological studies on lead and arsenic to the causation issues in this MDL. As explained previously, there is no study investigating whether consumption of baby food is associated with ASD or ADHD; instead, the cited epidemiological literature considers whether lead or arsenic is associated with ASD or ADHD, and is agnostic as to the source of exposure. However, to satisfy general causation, it is not enough for Plaintiffs to merely present evidence on lead or arsenic. Plaintiffs must submit reliable evidence that the products at issue in this MDL can cause the alleged injury. *See, e.g., In re Zantac (Ranitidine) Litig.*, 342 A.3d 1131, 1152 (Del. 2025) ("A general causation expert's opinion must focus on the product at issue and must show that exposures examined in non-product studies on which the expert relied are reliably linked to the exposures caused by the product at issue."); *Chapman v. Procter & Gamble Distrib., LLC*, 766 F.3d 1296, 1304 (11th Cir. 2014) (holding general causation applied to the product at issue in the case, Fixodent, not a subcomponent of that product, i.e. zinc). So, the opinions of Plaintiffs' exposure experts form the foundation for the remaining experts' general causation opinions about Defendants' baby food products. Without Dr. Jones' calculations, Plaintiffs' remaining experts cannot opine that a study on lead or arsenic exposure from unidentified sources is a reliable basis for their causation opinions.

But, as discussed in the Court's analysis of Ms. Barr's and Dr. Jones' reports, the exposure experts provide an unreliable foundation. *See* Sections I.A.– I.B., *supra*. Plaintiffs have not shown the hypothetical consumption patterns from Ms. Barr's report are realistic or reliably constructed, and so they have also failed to show Dr. Jones' calculations based on those patterns are reliable or relevant to general causation. Consequently, the causation opinions of the epidemiology and toxicology experts are not sufficiently reliable because they are not based on a realistic exposure level to either lead or arsenic. Whether these experts opine that 1 microgram of lead absorption is enough to cause ASD/ADHD or 100 micrograms, they must be able to connect

that dosage to Defendants' products. For this reason alone, Plaintiffs have failed to carry their burden under Rule 702.

Plaintiffs respond their exposure experts' opinions are "not strictly necessary to general causation," and all five of the epidemiology and toxicology experts would still reach their conclusions even absent Dr. Jones' calculations. (Dkt. No. 642 at 35-36.) To support this position, Plaintiffs rely on the district court's analysis in *Roundup*. In admitting the plaintiffs' general causation experts' opinions, the court did not require the experts to define precisely the "human-relevant dose" of glyphosate. *Roundup*, 390 F. Supp. 3d at 1115, 1151. The court noted that "even at the end of this [general causation] ruling, precisely what the range of actual human exposure is will remain vague, a product of bifurcated proceedings where the hundreds of individual plaintiffs' experiences remain on the periphery for now." *Id*. at 1115. Plaintiffs transplant the reasoning from *Roundup* onto the instant case to suggest an exposure estimate is ultimately unnecessary. The Court disagrees. *Roundup* differed from the instant case in a critical respect—the toxin at issue. Glyphosate is an herbicide that Monsanto brought to market under the trade name "Roundup," and subsequently became commonplace across much of the United States. *Id*. at 1109-10. But glyphosate is not a naturally occurring substance, and so observational studies of the effect of glyphosate on humans were *necessarily* studies of the defendant's product at a "human-relevant dose." Here, the toxins at issue are lead and arsenic, which are ubiquitous in the environment. Consequently, studies investigating the association of lead or arsenic with the development of ASD/ADHD are *not necessarily* studies of Defendants' products; in fact, none of Plaintiffs' experts refers to a study specifically assessing whether consumption of baby food is associated with the development of ASD or ADHD. General causation in this MDL requires a realistic consumption model because Plaintiffs must distinguish baby food exposure from other environmental exposures.

As in *Roundup*, here too, Plaintiffs' exact range of exposure will not be known—that is a question for the specific causation phase. But unlike *Roundup*, the scientific evidence available to Plaintiffs' experts does not examine Defendants' products. For that reason, a realistic estimate of the heavy metal exposure caused by Defendants' baby food must be calculated to determine

whether the doses studied in the scientific literature are applicable here. Plaintiffs fail to demonstrate the hypothetical consumption patterns and exposure calculations offered by Ms. Barr and Dr. Jones produce a realistic and reliable exposure estimate. Therefore, they also fail to show the experts' causation opinions are reliable since they are based on those calculations. So, per Rule 702, the Court excludes those opinions.

Separately, even if Plaintiffs had shown Dr. Jones' calculations were reliable, the epidemiology and toxicology experts do not provide a scientific rationale for concluding those estimated exposure levels can cause ASD or ADHD. All five experts engage only briefly with Dr. Jones' calculations in their reports, and subsequent rebuttal reports, concluding the hypothetical numbers are consistent with peer-reviewed literature such that they could constitute a substantial contributing factor to development of these conditions. Plaintiffs' experts generally do not identify which studies they are considering when assessing the effect of Dr. Jones' exposure estimates, but rather gesture broadly to all the studies discussed previously in their reports. (*See, e.g.*, Dkt. No. 611-2 at 71 (Dr. Ritz noting the estimated "levels meet or exceed levels identified in the scientific literature"); Dkt. No. 611-14 at 54 (Dr. Guilarte noting the estimated "levels meet or exceed levels identified in the scientific literature").) Dr. Aschner's report takes a slightly different approach, stating he was "instructed that 'substantial' factor means a factor that is not trivial or remote," and "[s]uch legal concepts to [*sic*] not easily fit within standard toxicological concepts . . . ." (Dkt. No. 611-11 at 75.) He goes on to assert exposure levels "that exceed mean background levels" of lead or arsenic could cause ASD or ADHD and cites studies calculating those mean levels to be 1.3 micrograms per day of lead for 0-11 months, and 2.4 micrograms per day of lead for children aged 1-6 year olds. (*Id.*) As for background arsenic levels, Dr. Aschner's cited studies posit an average of 0.2 micrograms per kilogram per day of total arsenic exposure between 6-11 months of age and 0.4 micrograms per kilogram per day of total arsenic exposure at age 2. (*Id.*) Based on these figures, he concludes Dr. Jones' calculations indicate a causal relationship between exposure to Defendants' products and development of ASD/ADHD. (*Id.* at 76.)

That said, Dr. Jones amended her exposure calculations twice, which included precipitous

drops in certain exposure estimates and blood lead levels. For instance, Dr. Jones initially estimated the mean daily lead exposure from Hain products at 26.4 micrograms per day for children between the age of 6 months and 1 year old. (Dkt. No. 611-23 at 2.) After her second revision, that number dropped to 6.52 micrograms. (Dkt. No. 611-25 at 35.) Nearly all of Dr. Jones' mean and maximum calculations changed between her initial report and her second amended report. (*Compare* Dkt. No. 611-23 at 2-4 *with* Dkt. No. 611-25 at 35.) Despite these significant changes in the estimated values, Plaintiffs' causation experts did not further engage with the literature to update their opinions. Instead, they offered short amendments to their own reports stating they had reviewed the new calculations, and their causation opinion did not change. The lack of analysis in response to these significant changes in exposure estimates, alone, presents a reliability problem. In particular, Dr. Aschner—who identified specific background exposure levels underlying his opinion—did not change his causation conclusion when Dr. Jones' estimates for certain defendants dropped *below* his identified values. (*See* Dkt. No. 611-25 at 35 (showing daily mean lead exposure values for certain Sprout products below 1.3 micrograms per day and Gerber products below 2.4 micrograms per day during the relevant age range); Dkt. No. 611-13 at 3 (Dr. Aschner confirming the updated values do not alter his causation opinion).) Perhaps there is a scientific justification for maintaining the position that all Defendants' products can cause ASD and ADHD, even in the face of Dr. Jones' significant amendments. But the Court cannot evaluate Dr. Aschner's method, or its reliability, because he supplies none. This kind of perfunctory analysis to connect Dr. Jones' calculations to the scientific literature is common across Plaintiffs' experts. (*See, e.g.*, Dkt. No. 611-27 at 47 (Dr. Ritz noting she had not compared Dr. Jones' calculations to the mean and range levels of heavy metals in the studies cited in her report).) And their failure to thoroughly explain the basis for their causation opinions, as Dr. Jones' calculations shifted, indicates a lack of reliability. Here too, Plaintiffs have not shown, by a preponderance of the evidence, that their epidemiology and toxicology experts' opinions satisfy Rule 702.

B. Analytical Gap Between the Data and the Experts' Opinions

To reach their conclusion that Defendants' baby food products can cause ASD and ADHD

in children who consume them, Plaintiffs' causation experts review the available scientific evidence on: 1) the relationship between lead exposure and ASD; 2) the relationship between lead exposure and ADHD; and 3) the relationship between arsenic exposure and ASD. Given the absence of studies directly observing consumption of baby food, or consumption of any food, and an association with an ASD or ADHD diagnosis, all experts must extrapolate from existing data on lead and arsenic exposure that is non-specific to the route of exposure. However, the experts fail to identify sufficient data to support their extrapolated opinions, or put differently, they present a *Joiner* problem in that "there is simply too great an analytical gap between the data and the opinion offered." *Engilis*, 151 F. 4th at 1048 (quoting *Joiner*, 522 U.S. at 146).

For the epidemiology experts, this *Joiner* problem is paired with a failure to reliably apply the Bradford Hill Criteria, specifically the temporality criterion. For the toxicology experts, the *Joiner* problem manifests in their weighing of the available scientific evidence. The Court first addresses the epidemiology reports, discussing Plaintiffs' failure to establish their experts' reliable extrapolation from the three relevant bodies of evidence: 1) studies on the association between lead and ASD; 2) studies on the association between lead and ADHD; and 3) studies on the association between arsenic and ASD. Then the Court turns to the toxicology reports and the extrapolations deployed within their weight-of-the-evidence approach.

1. Epidemiology Expert Reports

a. Lead and ASD

Plaintiffs' epidemiologists apply the Bradford Hill Criteria to assess causation. Though "[t]here is no formula or algorithm that can be used to assess whether a causal inference is appropriate based on the[] [nine criteria]," one factor—temporality—must exist to establish causation. *Reference Guide on Epidemiology* at 600-01. This means "[a] temporal, or chronological, relationship must exist for causation to exist. If an exposure causes disease, the exposure must occur before the disease develops. If the exposure occurs after the disease develops, it cannot have caused the disease." *Id.* at 601; *see also Roundup*, 390 F. Supp. 3d at 1132 (noting an expert's description of the temporality criterion as "the only non-discretionary Bradford Hill factor"). Plaintiffs' experts agree temporality is essential to a causation conclusion.

(*See, e.g.*, Dkt. Nos. 611-2 at 15 (Dr. Ritz's expert report stating temporality "is the only component that is necessary for making a causal attribution."); 611-36 at 18 (Dr. Guilarte testifying temporality is "a required component" to make a causal conclusion); 705 at 110-11 (Dr. Hu agreeing it is a basic epidemiological principle that the exposure must precede the injury).) But, the studies these experts cite to establish temporality do not provide a sufficient basis to reliably draw such a conclusion.

Drs. Ritz, Hu, and Gardener rely principally on a few key studies: Kim et al. (2016), Skogheim et al. (2021), Alampi et al. (2021), and Arora et al. (2017).[4] Each of these studies was a prospective cohort study, with the exception of Arora et al. (2017). Even so, Arora et al. (2017) is rare among case-control studies in that the biomarker it considered (baby teeth) can provide long-term exposure data, since the presence of lead in certain parts of the teeth corresponds to lead exposure at different developmental periods. So, all four studies could provide a window into lead exposure and its potential developmental effects on children. But neither Alampi et al. (2021), nor Skogheim et al. (2021), made statistically significant findings related to the association between blood lead levels and ASD. (*See* Dkt. No. 699 at 214 (Dr. Ritz agreeing that of the studies that satisfy temporality, only two (Kim et al. (2016) and Arora et al. (2017)) have statistically significant results).) Further, both Skogheim et al. (2021) and Alampi et al. (2021) looked at maternal blood samples indicating *prenatal* exposure to lead, not *postnatal* exposure.

Though Plaintiffs' experts acknowledge both studies do not fit the relevant exposure period, they fail to consider why that is a significant limitation to their causation opinions. Critically, the Diagnostic and Statistical Manual of Mental Disorders ("DSM"), and various scientific publications, recognize certain *prenatal* factors that are associated with ASD. Dr.

[4] The experts refer to these studies at various times through their reports and during their testimony at the hearing on the parties' motions to exclude. (*See, e.g.*, Dkt. No. 697 at 119-23 (Dr. Gardener identifying Arora et al. (2017) and Kim et al. (2016) as two of the studies that significantly contributed to her analysis); Dkt. No. 705 at 115 (Dr. Hu confirming Kim et al. (2016), Alampi et al. (2021), and Arora et al. (2017) as three studies important to the temporality analysis); Dkt, No. 699 at 214 (Dr. Ritz agreeing Kim et al. (2016) and Arora et al. (2017) were the two studies she considered with statistically significant findings related to lead exposure and ASD); Dkt. No. 611-2 (Dr. Ritz's report referring to Skogheim et al. (2021) in the temporality analysis for lead and ASD).)

Shapiro noted as much in both his testimony and his report, where he identified advanced parental age, premature birth, and exposure to sodium valproate *in utero* among those factors. (*See* Dkt. No. 699 at 27-28, 65; Dkt. No. 611-17 at 24-29.) Alampi et al. (2021) and Skogheim et al. (2021), then, might support an assertion that *prenatal* exposure to lead can cause ASD. But Drs. Ritz, Hu, and Gardener offer no analysis on this alternative causal explanation—they seemingly treat all these studies the same and as "strong" evidence of temporality. (*See, e.g.*, Dkt. No. 611-2 at 58 (Dr. Ritz noting she weighed temporality and dose-response as the "most important items"); Dkt. No. 611-5 at 34 (Dr. Hu noting there was "strong support" for fulfillment of the temporality criterion).) Plaintiffs' experts downplay the limitations of these studies as to the most critical Bradford Hill factor, indicating their analysis is unduly results-driven. *See In re Viagra*, 424 F. Supp. 3d at 797 (finding an expert's attempt to downplay the weakness of one Bradford Hill criterion by conflating it with another indicated the analysis was unreliable).

To this, Plaintiffs counter that prenatal studies are useful for projecting postnatal effect because neurodevelopment "is a protracted process that starts about 2 weeks after conception and comprises several key stages that progress through the neonatal and infant period well into adolescence before the brain is fully mature." (Dkt. No. 642 at 100 (citing Dkt. No 611-11).) But Drs. Ritz, Hu, and Gardener do not explain the similarities or differences between the *in utero* environment and postnatal ingestion of lead through baby food. Nor do they articulate the overlap between neurodevelopment *in utero* and postnatally to justify a conclusion that the effect of lead on a fetus is the same as its effect on a child between the ages of 4 months and 3 years old. The mere fact that neurodevelopment is an ongoing process is not a sufficient basis to support the experts' opinion that the effect of lead is identical at all points *in utero* as well as postnatal, and regardless of the source. Accordingly, Plaintiffs have not demonstrated Alampi et al. (2021) and Skogheim et al. (2021) serve as data that permit Plaintiffs' experts to reliably extrapolate *postnatal* lead exposure can cause ASD, let alone that consumption of Defendants' baby food products can lead to such a diagnosis.

The remaining two studies underlying the experts' conclusions are Kim et al. (2016) and Arora et al. (2017). Kim et al. (2016), however, did not consider whether the children studied

were subsequently diagnosed with ASD. Instead, the study used the Autism Spectrum Screening Questionnaire ("ASSQ") and Social Responsiveness Scale ("SRS") to measure an increase in "autistic behaviors." Yet, Plaintiffs' experts do not opine Defendants' baby food can cause merely "autistic behaviors"; they opine Defendants' products can cause ASD. As Plaintiffs' expert Dr. Shapiro explains, ASD diagnosis "is based on the presence of a cluster of psychological and developmental symptoms, and not on any specific underlying neurobiological or genetic pathology." (Dkt. No. 611-17 at 12.) So, clinicians are mindful "the detection of early symptoms does not necessarily mean that a child will ultimately be diagnosed with autism. Some children exhibit early signs consistent with characteristics of autism or other neurodevelopmental conditions, but do not receive a clinical diagnosis because they do not go on to develop symptoms that meet established criteria for ASD." (*Id*. at 12-13.) In fact, the DSM has historically evinced overlapping behavioral criteria among ASD and other disorders. (*See id*. at 14 (referencing Asperger syndrome, pervasive developmental disorder, and childhood disintegrative disorder).) An ASD diagnosis requires a trained clinician to consider the behavioral presentation of the patient and make a professional judgment as to the proper diagnosis. Since Kim et al. (2016) did not consider diagnosed ASD as its endpoint, there is an analytical gap between the data it provides and Plaintiffs' experts' causation opinions. Indeed, Plaintiffs' expert Dr. Guilarte agreed that "to reach reliable opinions about causation of ASD, you need to look at diagnosed ASD." (Dkt. No. 611-36 at 20.) Even the Kim et al. (2016) study authors themselves cautioned: "Although lead concentration was associated with an ASSQ score greater than or equal to 19, this finding should be interpreted cautiously ***because the ASSQ is not a diagnostic tool***." (Dkt. No. 705 at 18 (emphasis added).) For these reasons, the "analytical gap" between Kim et al. (2016) and the experts' opinions is "simply too great." *Joiner*, 522 U.S. at 146.

Turning to the final study, Arora et al. (2017) was cited as critical support for the experts' causation opinions. (Dkt. No. at 697 at 119 (Dr. Gardner stating Arora et al. (2017) is the strongest study to support her opinion: "Arora would be my number one."); Dkt. No. 705 at 26 (Dr. Gardner again confirming Arora et al. (2017) was the most influential study for her opinion on temporality); Dkt. No. 705 at 186 (Dr. Hu identifying Arora et al. (2017) as the only study that

satisfied temporality with statistically significant results); Dkt. No. 699 at 214 (Dr. Ritz agreeing Arora et al. (2017), along with Kim et al. (2016), are the only studies satisfying temporality with statistically significant results).) However, after performing a statistical correction for multiple comparisons, the Arora et al. (2017) authors found ***there was no statistically significant association between lead and diagnosed ASD.*** (*See* Dkt. No. 705 at 199 (Dr. Hu confirming this summation of the findings in Arora et al. (2017)); Dkt. No. 611-36 at 61-62 (Dr. Guilarte interpreting the results similarly).) So, Plaintiffs' experts seize on a different finding in the study: between 10 and 20 weeks postnatal, there was a statistically significant association between lead exposure observed in baby teeth and autistic traits as measured by the SRS-2. As in Kim et al. (2016), this finding does not apply to the relevant outcome in this MDL—a diagnosis of ASD. So, the experts' heavy reliance on Arora et al. (2017), which found no statistically significant association between lead exposure and ASD, to support their opinions that Defendants' baby food products can cause ASD, is too far a leap to satisfy Rule 702.

In response, Plaintiffs assert studies looking at ASD behaviors are more useful than studies with an ASD diagnostic endpoint because they permit a researcher to understand gradations in severity based on exposure to a toxin. (Dkt. No. 642 at 94.) Moreover, studies assessing "autistic behaviors" fortify the causation opinion because they provide a distinct way to corroborate the association between lead exposure and diagnosed ASD. (*Id*. at 95.) The Court is not persuaded. While assessing gradations in behaviors can be useful clinically, this is a court of law, not a doctor's office or a research lab. For general causation, Plaintiffs bear the burden of presenting reliable expert evidence to show Defendants' products can cause ASD, not merely autistic behaviors. And Plaintiffs supply such opinions. Yet gradations in autistic behaviors that fall short of diagnosed ASD do not ultimately support those causation conclusions. Perhaps such studies could lend support to other factors in the Bradford Hill Criteria, but as to temporality—the only essential factor—the behavior studies with no confirmed ASD diagnosis do not support a reliable conclusion that lead exposure can cause ASD.

Finally, none of these four studies measure blood lead levels during periods when baby food is consumed. The precise period of exposure to lead is fundamental to any theory of

causation in this case. Plaintiffs have alleged that exposure to heavy metals in Defendants' baby food can cause ASD. Unlike other consumables, baby food products are only ingested within a certain window of development; so, it is not enough for Plaintiffs to present evidence that does not account for exposure during that period. As Plaintiffs' experts recognize, the biomarkers used to measure lead or arsenic in the body (e.g. blood, hair, urine, toenails, etc.) present only a snapshot of recent exposure, usually within the last 30 to 90 days. (*See* Dkt. No. 705 at 218.) Consequently, studies like Kim et al. (2016), which observed children from age 7 onward, do not provide objective data on what heavy metal exposure looked like beyond the 30- to 90-day window shown through blood lead measurement. Arora et al. (2017) utilized a unique biomarker, baby teeth, which show a much broader exposure history, but even then, the authors ***did not*** find a statistically significant association between lead and ASD at all, let alone during the relevant exposure window. Ultimately, this is not a case about whether lead or arsenic can cause ASD or ADHD—it is a case about whether consumption of certain baby food products can cause those conditions. And yet, Plaintiffs' experts consider only evidence of the former and simply assume the latter.

In sum, the reports of Drs. Ritz, Hu, and Gardener outline a reliable method in the Bradford Hill Criteria, but they do not apply that method reliably to the opinions offered in this MDL. They can point to no study satisfying temporality that demonstrates a statistically significant association between lead exposure and ASD diagnosis, let alone a study evincing a positive association between consuming baby foods and development of ASD. "[C]onclusions and methodology are not entirely distinct from one another. Trained experts commonly extrapolate from existing data. But nothing in either *Daubert* or the Federal Rules of Evidence requires a district court to admit opinion evidence that is connected to existing data only by the *ipse dixit* of the expert. A court may conclude that there is simply too great an analytical gap between the data and the opinion proffered." *Joiner*, 522 U.S. at 146. The absence of any study establishing a link between exposure to lead during the early postnatal period and a subsequent ASD diagnosis creates a similarly impassable gap here.

b. Lead and ADHD

Drs. Ritz, Hu, and Gardener also offer causation opinions as to lead exposure and the development of ADHD. Among the various publications they considered, the most informative to their temporality analyses were the studies Ji et al. (2018), Burns et al. (1999), Bellinger et al. (1994), and various meta-analyses. Of these studies, only Ji et al. (2018) investigated the relationship between lead exposure in the early postnatal period and development of diagnosed ADHD. (*See* Dkt. No. 705 at 205-06.) For the reasons set out in the Court's analysis of lead exposure and the development of ASD, *supra*, prospective studies which do not consider the relevant postnatal time period and diagnosed ADHD, alone, are not a reliable basis for concluding consumption of baby food can cause that condition. So, the Court looks to Ji et al. (2018) to assess the reliability of the experts' opinions.

The study authors in Ji et al. (2018) observed a statistically significant positive association between blood lead levels over 5 micrograms per deciliter and development of ADHD in boys. To start, Ji et al. (2018) did not look at the impact of baby food consumption, specifically. And even if the study could support a temporality conclusion as to boys, Plaintiffs' experts have no way to connect the identified blood lead levels from Ji et al. (2018) to a lead exposure estimate in this MDL. As the Court noted in Sections I.A., I.B., and III.A., *supra*, Dr. Jones' calculations are based upon an unrealistic hypothetical consumption pattern of Defendants' products. Still, even assuming those numbers were reliable or relevant, a blood lead level of 5 micrograms per deciliter is higher than all of Dr. Jones' calculated mean blood lead levels, and higher than most of her maximum estimated levels. (*See* Dkt. No. 611-25 at 35.) Plaintiffs' experts cannot connect the dosage studied in Ji et al. (2018) to a realistic exposure scenario in this case, and they do not explain how the studied dosage connects to even the unrealistic exposure scenario in this case. So, Plaintiffs have not established the experts reliably opine Defendants' products can cause ADHD in the children who consume them. Plaintiffs have thus failed to carry their burden under Rule 702 as to their experts' causation opinions for lead exposure and the development of ADHD.

c. Arsenic and ASD

Among Plaintiffs' epidemiology experts, only Drs. Ritz and Gardener offer an opinion as

to the causal relationship between exposure to arsenic in Defendants' products and the development of ASD. Both experts rely heavily on a few key studies: Skogheim et al. (2021), Alampi et al. (2021), Doherty et al. (2020), and Long et al. (2019). However, all of these studies involved maternal samples of heavy metal levels *in utero*. Neither Dr. Ritz nor Dr. Gardener grapples with the limitations of this body of literature when assessing causation for *postnatal* arsenic exposure. Indeed, as the Court previously noted, the existence of recognized prenatal risk factors suggests these prenatal studies might indicate *in utero* exposure to arsenic is associated with ASD. Given this alternative causal explanation, the experts do not provide sufficient support to conclude postnatal exposure, generally, causes ASD, let alone exposure based upon consumption of Defendants' baby food products.

Accordingly, the analytical gap between the existing data and the experts' opinions is too great. They do not explain how prenatal studies of arsenic and ASD (or, more precisely, one study finding an association between prenatal exposure to arsenic and a subsequent diagnosis of ASD) can be reliably extrapolated to support their litigation-created opinion that consumption of Defendant's baby food products can cause a child to develop diagnosed ASD. Absent more relevant data, these causation opinions are nothing more than the experts' *ipse dixit*. *Joiner*, 522 U.S. at 146. Therefore, they are excluded.

* * *

For all the reasons discussed above, Defendants' motion to exclude the causation opinions of Plaintiffs' epidemiology experts is **GRANTED**.

2. Toxicology Expert Reports

As toxicologists, Drs. Aschner and Guilarte employ a "weight of the evidence" approach, "in which the toxicological, mechanistic, and epidemiological data are rigorously assessed to form a judgment regarding the likelihood that the agent produces a specific effect." *Reference Guide on Toxicology* at 651. The opinions of Drs. Aschner and Guilarte contribute to the epidemiological evidence discussed above "by explaining how a chemical causes a specific disease through describing metabolic, cellular, and other physiological effects of exposure." *Id*. at 637. To that end, Drs. Aschner and Guilarte provide both an analysis of relevant epidemiological literature as

well as a mechanistic analysis common to the field of toxicology. From this analysis, they opine lead and arsenic play a causal role in the pathogenesis of ASD and ADHD, and consumption of baby food can result in these conditions. (*See* Dkt. No. 611-11 at 10-12; Dkt. No. 611-14 at 54-55.) Plaintiffs have not shown, by a preponderance of the evidence, the opinions of Drs. Aschner and Guilarte satisfy Rule 702.

Here, both experts rely on generally the same array of studies presented by the epidemiology experts, particularly for prospective cohort studies. For instance, Dr. Aschner reviews Skogheim et al. (2021), Doherty et al. (2020), Kim et al. (2016), Arora et al. (2017), and Ji et al. (2018). (*See* Dkt. No. 611-11 at 30-31, 38-39, 54.) Dr. Guilarte similarly considered those studies. (*See* Dkt. No. 611-14 at 21, 23-24, 26, 30.) But, as the Court has explained, the studies did not find a statistically significant association between lead or arsenic exposure and diagnosed ASD/ADHD during the relevant postnatal period. The only exception is Ji et al. (2018) for blood lead levels above 5 micrograms per deciliter and development of ADHD in boys. Yet even then, Plaintiffs have not connected the dosage from the literature to a realistic exposure scenario. Though Drs. Aschner and Guilarte weave a mechanistic analysis of lead's and arsenic's neurotoxicity into their reports, at base, they rely on the same epidemiological evidence to draw their conclusions as Plaintiffs' other experts. To reliably offer an opinion that Defendants' products can cause ASD and ADHD in the children who consume them, Plaintiffs' experts must show some scientific evidence that satisfies temporality and connects exposure to these metals during the relevant postnatal period to diagnosed ASD or ADHD. They have not done so. For the same reasons the epidemiologists' causation opinions have been excluded, these toxicology causation opinions are excluded.

During oral argument, Plaintiffs referred to a few studies in which they contend the study authors concluded lead or arsenic can cause ASD/ADHD. (*See* Dkt. No. 700 at 62-67.) The Court understands this list of studies to include: Grandjean & Landrigan (2014), Goel & Aschner (2021), Filon et al. (2020), and Mohamed et al. (2015). Though these studies did not feature during the evidentiary hearing, and occupied little, if any, space in the experts' reports, the Court reviewed them to assess whether they made causal assertions. Following that review, the Court understands

why the studies did not play a prominent role in the experts' testimony—they offer nothing new beyond what the Court has already considered at length. These case-control studies and literature reviews do not contribute any distinct points as to the question of temporality. Indeed, this laundry list of studies is representative of the broad reliability issue inherent in these reports and Plaintiffs' approach to general causation. Rather than confront the absence of evidence as to temporality, the reports downplay the issue by gesturing to the hundreds of other studies considered. But inadvertently, Plaintiffs have highlighted a critical point: even in an area of epidemiology marked by hundreds of studies, none has developed the data needed to support the causation conclusion Plaintiffs' experts assert in this MDL.

Therefore, Defendants' motion to exclude Plaintiffs' toxicology experts' causation opinions is **GRANTED**.

CONCLUSION

For the reasons stated above, Defendants' motion to exclude the hypothetical menus created by Plaintiffs' counsel, and Ms. Barr's opinions as to the plausibility of those menus, is **GRANTED**. The menus do not represent realistic consumption patterns and do not evince a reliable scientific basis. So, they are neither reliable nor relevant to the general causation phase.

Defendants' motion to exclude the exposure calculations and blood lead level calculations offered by Dr. Jones is similarly **GRANTED**. The calculations are based upon the hypothetical menus that lack reliability and relevance. Additionally, Dr. Jones did not reliably apply the principles and methods of an exposure assessment by failing to scientifically validate the exposure scenario presented to her by Plaintiffs' counsel.

Defendants' motion to exclude the opinions of Dr. Shapiro is **DENIED**. Plaintiffs have shown, by a preponderance of the evidence, that Dr. Shapiro applied a reliable scientific method in offering his opinions.

Lastly, Defendants' motion to exclude the causation opinions of Plaintiffs' epidemiology and toxicology experts is **GRANTED**. These experts rely on Dr. Jones' calculations to reach their causation opinions—calculations which the Court has excluded for lacking reliability and relevance to general causation. Separately, those opinions must also be excluded because "there is

simply too great an analytical gap between the data and the opinion [Plaintiffs' experts] offered." *Engilis*, 151 F. 4th at 1048 (quoting *Joiner*, 522 U.S. at 146).

The Court will hold a further case management conference on April 2, 2026 at 9:00 a.m. to discuss next steps in the litigation. An updated joint case management conference statement is due one week in advance.

This Order disposes of Docket Nos. 611, 612, and 614.

IT IS SO ORDERED.

Dated: February 27, 2026

JACQUELINE SCOTT CORLEY
United States District Judge