



Mothers' Ideas About Causes of Autism Spectrum Disorder (ASD): Differences Over Time and by Household Experience with ASD

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Abstract

To understand trends in mothers' causal ideas about autism spectrum disorder (ASD) that may be important in targeting education efforts for parents and healthcare providers, as these may be associated with healthcare choices. Data from the Study to Explore Early Development (SEED) was used to examine the causal ideas mothers had, sociodemographic characteristics associated with causal ideas, and whether reported ideas differed over time. SEED included 8307 mothers of pre-school children, with and without ASD, who completed a maternal phone interview between 2007 and 2020. Less than half of mothers (39.3%) offered a causal idea. The most frequently offered causal ideas were genetics, vaccines, environment, parent lifestyle issues, pre/peri-pregnancy maternal medical issues, labor/delivery experiences, and child diet. Some aspects of sociodemographic characteristics, particularly ethno-cultural background, were significantly associated with reporting several causal ideas. Odds of reporting vaccines decreased over time while odds of reporting genetics, parent lifestyle, maternal medical, or labor/delivery issues increased over time. There were significant differences in odds of reporting several causal ideas between those with and those without a child with ASD in the household. Causal ideas

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reported were consistent with prior research but important differences between those with and those without household ASD as well as significant time trends for certain causes were noted. Implications for public health communication and future research are discussed.

Keywords

Autism etiology (cause); Parental perspectives; Lay beliefs; Ethnicity; Culture; Socioeconomic factors

Autism spectrum disorder (ASD) is a lifelong neurological and developmental disorder that affects how individuals communicate, behave, and/or interact with others. Scientific research on factors contributing to the occurrence of ASD or developmental problems currently focuses on genetics and interactions between some environmental factors and genes (Bolte et al., 2019; Carlsson et al., 2021; Sandin et al., 2014; Zhou et al., 2022). There is also evidence of some smaller contributions of parent age, low birth weight, birth defects, perinatal hypoxia, gestational age, maternal infection/inflammation, and stress (Beversdorf et al., 2018; Bolte et al., 2019; Croen et al., 2019).

Here, we use the phrase “causal ideas” to denote what parents think causes ASD or developmental problems. Causal ideas among the parents of children with ASD have been reported for decades (Chaidez et al., 2018; Dardennes et al., 2011; Felnhofer et al., 2020; Fombonne et al., 2020; Gray, 1994; Goin-Kochel & Myers, 2005; Harrington et al., 2006a, b; Mercer et al., 2006; McLeod & DiSabatino, 2019; Reiff et al., 2017; Zuckerman et al., 2016) with variation in the prevalence of specific ideas. For example, as low as 4% and as high as 40% of North American parents of children with ASD reported believing in no specific cause (Goin-Kochel et al., 2015; Zuckerman et al., 2016). In other studies in westernized countries, varying proportions of parents thought genetics alone caused ASD and low proportions thought external events or exposures were the cause (Al Anbar et al., 2010; Brewton et al., 2021; Goin-Kochel & Myers, 2005). A 2020 study among caregivers of children with ASD reported 58.4% of caregivers endorsed genetic causes, 21.9% endorsed environmental exposures, and 16.5% endorsed vaccines as a cause (Fombonne et al., 2020). Further, Fombonne et al. (2020) noted that caregivers of older children had a higher proportion endorsing vaccines as causal, suggesting a decreasing trend in thinking vaccines cause ASD; however, the nature of their sample did not allow them to test this trend specifically. Taken together, these findings suggest causal ideas of ASD vary and might differ over time.

Additionally, previous research suggests that several sociodemographic factors may influence parent ideas about causes. For example, race, ethnicity, or cultural background as well as education or income were associated with likelihood of stating specific causal ideas (Chaidez et al., 2018; Fombonne et al., 2020; Hebert & Koulouglioti, 2010; McLeod & DiSabatino, 2019; Zuckerman et al., 2015, 2016). Five small studies suggest that parental expectations of children’s behavior vary by cultural background and may affect reporting of behavior symptoms (Blacher et al., 2014; Glick et al., 2012; McLeod & DiSabatino, 2019; Sistler & Gottfried, 1990; Zachor et al., 2011) which, in turn, might influence parents’ ideas about causes of developmental problems.

Parental ideas about causes are often associated with treatment and/or healthcare choices (Harrington et al., 2006a, b; Levy & Hyman, 2003; Mire et al., 2017; Reyes et al., 2018; Zuckerman et al., 2015). For example, among parents in France, use of behavioral training was higher among those who thought there was a genetic cause of ASD (Al Anbar et al., 2010) while pharmacologic therapy was more likely among those who stated ‘illness during pregnancy’ as a cause (Dardennes et al., 2011). Among a sample of United States (US) families with ASD diagnosis in a child, adherence to recommended vaccination schedules for both the diagnosed child and younger siblings declined after the diagnosis (Zerbo et al., 2018). Therefore, understanding causal ideas of ASD among parents is particularly important for practitioners and healthcare professionals so they can educate parents and engage in shared decision making when developing plans to support their children with ASD. Additionally, understanding causal ideas may be important to practitioners in supporting the public in making well-informed, universal healthcare decisions such as adherence to vaccination schedules.

Only one study was found to report on causal ideas among a public sample. Mitchell and Locke (2015) surveyed about 800 North American individuals, approximately 12% of whom reported having a family member (self/sibling/child/other relationship) with ASD. Findings suggested that different causal ideas might exist between those with a family member with ASD versus those without. However, the study recruitment via crowdsourcing resulted in a predominantly White sample (77.8%) with low representation of Black (7.2%) and Hispanic (4.3%) respondents so findings may not be generalizable.

While prior studies report varying frequencies of specific causal ideas, none have explored changes in the frequency of specific causal ideas over a long period of time nor differences in ideas held by those with versus without a child with ASD. The Study to Explore Early Development (SEED) enrolled participants over a 13-year period making it uniquely poised to examine the following research questions (RQ): (RQ.1) what ideas about causes of ASD did mothers of preschool children report? (RQ.2) What sociodemographic characteristics were associated with the reported causal ideas? (RQ.3) How was enrollment date (i.e. time) associated with the frequency of reporting specific causal ideas? (RQ.4) Were there differences in the frequencies of reporting individual causal ideas between those with and those without ASD in a child in the household?

Methods

Overview

SEED was a US-based, multi-site, multi-wave, case–control study of contributing factors and correlates of ASD that took place between 2007 and 2020. Full details of study methods are published elsewhere (Schendel et al., 2012). Briefly, children with an ASD or another non-ASD developmental disorder were identified through healthcare and education providers. Population controls were identified from birth records. All families were invited by mail and/or phone and were informed by study recruiters during the enrollment screening call that the study intended to compare “similarities and differences between children with and without autism.” SEED enrolled preschool children, with and without a diagnosis of ASD, for participation in a complex protocol that included several questionnaires, a phone

interview with the primary caregiver (hereinafter referred to as “mother” since 99.4% of respondents self-identified as the biological mother), in-person developmental assessments with the child, and biologic sample collection from child and parents. Families were offered financial incentives for each study component and received results of the developmental assessments. IRB approval was received from each participating site and appropriate consent obtained from a parent of each participating child.

Participants and Procedures

Eligibility criteria included: (1) child’s birthdate in one of three ranges (September 2003–August 2006, January 2008–December 2011, or January 2014–December 2017); (2) age 2–5 years at enrollment; (3) residence at birth and during study participation in a study catchment area (regions in eight states: California, Colorado, Georgia, Maryland, Missouri, North Carolina, Pennsylvania, and Wisconsin); and (4) a consistent caregiver/mother since six months of age (or younger) who could communicate in English (all sites) or Spanish (two sites). Despite there being three waves of the study, recruitment and data collection were conducted continuously from December 2007 through August 2020 using a common study protocol.

A scripted, comprehensive phone interview inquired about the mother’s health and pregnancy experiences as well as her demographic information. Study interviewers asked about both parents’ dates of birth, the mother’s education, race, ethnicity, birth within or outside of the US and timing of immigration if non-US, and household income. For the purposes of this study, Hispanic ethno-cultural background was defined as those who affirmed that they considered themselves “of Hispanic or Latina origin” during the maternal phone interview. Interviewers also asked for details about all past pregnancies, including any developmental or medical diagnoses each live-born child might have received. ASD in the household was defined as report of an ASD diagnosis in any child born to the mother by the time of the phone interview. Because SEED data collection began when an ASD diagnosis included five separate disorders (i.e., Asperger syndrome, autistic disorder, pervasive developmental disorder-not otherwise specified, Rett syndrome, and childhood disintegrative disorder), any mother who had a child with any of these five diagnoses was considered to have ASD in the household.

At the end of the interview, interviewers asked, “*Is there anything, including some of the factors we’ve already talked about, that you think might cause autism or developmental problems?*” Respondents who said “yes” were then asked an open-ended question, “*Can you tell me about those factors?*” Interviewers entered these responses verbatim into an electronic data-capture system. Fathers’ ages (if not reported during the interview), as well as child sex, were obtained from birth records.

Ideas about causes of ASD reported on the phone interview were coded by two independent coders with consensus review. Text data was isolated from all other study data for coding. Consensus on discrepant coding was achieved through discussion between both coders. Coders used 15 deductive categories based on existing literature (Al Anbar et al., 2010; Brewton et al., 2021; Chaidez et al., 2018; Dardennes et al., 2011; Felnhofer et al., 2020; Fischbach et al., 2016; Fleury & Kemper, 2022; Fombonne et al., 2020; Goin-Kochel &

Myers, 2005; Goin-Kochel et al., 2015; Harrington et al., 2006a, b; Hebert & Kouloughlioti, 2010; McLeod & DiSabatino, 2019; Mercer et al., 2006; Mitchell & Locke, 2015; Reiff et al., 2017; Zakirova-Engstrand et al., 2020; Zuckerman et al., 2016). Descriptions and text examples of the causal idea categories are available in Supplemental Table 1. Causal idea categories were: **genetics**; **vaccines** given to the child; physical **environment** of the mother or child (pesticides, air pollution, occupational/industrial chemicals, household cleaners, allergens, etc.); pre-pregnancy/prenatal/perinatal events and experiences (separated into four categories to better describe the array of causal ideas falling into this global category—**lifestyle events or experiences** of either parent during pre-pregnancy or prenatal periods; **maternal medical issues** prior to or during the pregnancy; **labor and delivery** and/or immediate postpartum events or experiences; **peri-pregnancy issues-other** such as inter-pregnancy intervals or generic statements about pregnancy); **child's diet**; **child's medical issues** (postnatal injuries or illnesses, chronic or acute); **psychological stress** to the mother (occupational or personal); **parent age** at conception; **medications given to the child** (any type); **parenting**/caretaking behaviors and family dynamics (psychosocial stress experienced by the child); and **metaphysical/religious causal ideas**. A **miscellaneous** category was created consisting of mainly elective exposures such as television viewing or technology use and microwave/computer/cell phone exposure to separate them from more incidental environmental exposures related to job or residence. Finally, '**cannot categorize**' contained content that was, in its entirety, too general or too unclear to categorize (e.g., "diagnosis is better now," "varies," "a lot of things"). The categories of metaphysical/religious, initial miscellaneous category, and "cannot categorize" were combined into the final 'miscellaneous' category for analyses.

Statistical Analyses

Among those who provided at least one causal idea, we estimated the probability of stating each causal idea separately. For each causal idea, we fit logistic regression models with a logit link to quantify the association between the causal idea and several characteristics based on previous literature: child age at enrollment, child sex (male vs female), maternal and paternal age at child's birth, maternal education (collapsed into two groups: less than bachelor's degree and bachelor's degree or higher), maternal ethno-cultural background (collapsed into non-Hispanic Black, non-Hispanic Other, non-Hispanic White, and Hispanic), maternal birth location and timing of immigration (US-born or non-US, immigrating before age 13 or non-US, immigrating at or after age 13), income as a percent of federal poverty level (collapsed into four groups: $\leq 138\%$, $> 138\%$ to $\leq 250\%$, $> 250\%$ to $\leq 400\%$, $\geq 400\%$), SEED enrollment site, time as a continuous variable using the full date of enrollment, and presence/absence of a child with ASD in the household. All continuous characteristics (i.e., child age at enrollment, maternal and paternal age) were centered at their mean and scaled to their standard deviation using all data for participants that gave at least one causal idea. Time was coded as follows: each full enrollment date was coded as the number of days since the earliest enrollment date of any participant (i.e., the earliest enrollment date was time zero). We then scaled this continuous measure by dividing by 365.25. We assessed multi-collinearity among the covariates using the Generalized Variance Inflation Factor (GVIF). All covariates had a GVIF below 2.5, indicating no extreme multi-collinearity so all were retained in models. Additionally, a chi-square test for homogeneity

was conducted to evaluate if the proportion of participants who reported a causal idea differed by ASD in the household. Odds ratios and 95% Wald confidence intervals were estimated for all coefficients in the regression analyses. All analyses were conducted using R version 4.1.3 (R Core Team, 2021). Regression analyses were performed on those with complete data (92.4% of those that gave at least one causal idea). We used a significance level of 0.05 and performed no multiple testing corrections.

Results

Sample Characteristics

There were 8307 SEED mothers who completed the maternal phone interview, 3265 of whom stated they thought something causes ASD and 3018 who were not missing covariate data. Supplemental Fig. 1 displays the flow of SEED families from enrollment to inclusion in this analysis.

Table 1 describes the demographic characteristics of those who completed the maternal phone interview. On average, children were 4 years of age during participation. Mothers were mostly US-born (83.8%), with an additional 3.8% beginning residence in the US before age 13 and 11.8% beginning residence in the US at, or after, age 13. Around 58% of the mothers were non-Hispanic White; 20% were non-Hispanic Black; 12% were Hispanic, and the remaining groups were combined for analyses as non-Hispanic Other backgrounds. Of the mothers, 56.8% had a bachelor's or advanced degree, and 78.7% had income levels above 138% of the US federal poverty level. About a third of the families (32.3%) had at least one child with ASD. Overall, 56.5% of mothers replied "no" when asked if there was anything they think causes ASD while 39.3% of mothers stated "yes".

RQ.1: Ideas About Causes

Table 2 displays the frequencies of causal ideas among SEED mothers who reported one or more causal ideas ($n = 3265$) and the frequency with which each cause was reported alone (sole cause) or in combination with other causes. The most frequently stated causal ideas were genetics (30.9%), vaccines (21.2%), environment (20.5%), lifestyle issues of the parent(s) before or during pregnancy (18.2%), medical issues of the mother pre-pregnancy and/or during pregnancy (16.0%), labor and delivery experiences (9.8%), and child's diet (9.7%). Other causal ideas were stated by less than 9% of mothers who thought something causes ASD. Among those who reported at least one causal idea, 60.2% ($n = 1964$) offered a sole causal idea, 25.2% ($n = 824$) gave two causal ideas, and 14.6% ($n = 477$) gave three or more causal ideas. Although lifestyle issues was one of the five most reported overall, it was not one of the top five reported alone. Further, maternal medical issues and/or maternal stress were reported with similar frequency as a sole cause or as one of two or as one of three or more causal ideas. Child diet and child medication were more frequently reported as one of three or more ideas. Thus, those who thought something causes ASD most often suggested one main idea as causal but a few ideas were more frequently offered in conjunction with other ideas.

RQ.2: Associations Between Sociodemographic Characteristics and Causal Ideas

Tables 3 and 4 show the results of adjusted logistic regression models estimating the associations between sociodemographic characteristics and reported ideas about causes among those stating one or more causal ideas and with complete covariate data ($n = 3018$). Table 3 displays results for the seven most frequently reported causal ideas and Table 4 displays results for the seven least frequently reported ideas.

The older the child at study enrollment, the higher the odds that parent lifestyle (Odds Ratio (OR) = 1.13, 95% Confidence Interval (95%CI) [1.01, 1.26]), child diet (OR 1.23, 95%CI [1.06, 1.43]), or maternal stress (OR 1.24, 95%CI [1.01, 1.52]) were given as causal ideas. Mothers of older children had lower odds of reporting maternal medical issues as causal (OR 0.84, 95%CI [0.75, 0.95]). Those with greater maternal age had higher odds of stating environment as causal (OR 1.29, 95%CI [1.13, 1.47]). Additionally, greater maternal or paternal age was associated with higher odds of reporting parent age (respectively, OR 1.78, 95%CI [1.39, 2.27] and OR 1.37, 95%CI [1.10, 1.71]).

Some causal ideas varied by ethno-cultural background. Compared to those of non-Hispanic White background, mothers of non-Hispanic Black background had lower odds of reporting genetics (OR 0.64, 95%CI [0.50, 0.82]), environment (OR 0.63, 95%CI [0.47, 0.85]), or child medical issues (OR 0.63, 95%CI [0.40, 0.97]) as causal ideas. However, mothers of non-Hispanic Black background had higher odds of reporting vaccines as causal (OR 1.43, 95%CI [1.10, 1.86]). Mothers in the non-Hispanic Other group, compared to non-Hispanic White, had higher odds of stating parent lifestyle as causal (OR 1.50, 95%CI [1.08, 2.08]). Most notably, compared to non-Hispanic White mothers, mothers of all other ethno-cultural backgrounds had around twice the odds of reporting maternal stress as causal. Further, mothers beginning residence in the US at or after age 13 also had higher odds of citing maternal stress (OR 1.62, 95%CI [1.03, 2.54]) compared to those born in the US; they also had lower odds of stating genetics as causal (OR 0.68, 95%CI [0.52, 0.90]) and higher odds of citing parenting as causal (OR 1.95, 95%CI [1.14, 3.34]). Those coming to live in the US prior to age 13 had lower odds than those US-born of reporting parent lifestyle as causal (OR 0.54, 95%CI [0.30, 0.99]).

Few socioeconomic characteristics were significantly associated with ideas about causes. Those living above 250% of federal poverty had higher odds of stating child's diet as a causal idea than those living at or below 138% of poverty (OR 1.95, 95%CI [1.15, 3.31]) and those living at or above 400% of federal poverty had lower odds of reporting maternal stress (OR 0.53, 95%CI [0.31, 0.93]); otherwise poverty status was not significantly associated with reported causal ideas. Having a bachelor's degree or higher was associated with higher odds of reporting environment (OR 1.37, 95%CI [1.09, 1.73]) and with lower odds of stating child medication as causal (OR 0.48, 95%CI [0.30, 0.74]).

RQ.3: Association Between Time and Causal Ideas

After adjusting for other covariates, each additional year of enrollment time was significantly associated with lower odds of stating vaccines (OR 0.86, 95%CI [0.83, 0.88]) and higher odds of stating genetics (OR 1.03, 95%CI [1.01, 1.06]), parent lifestyle (OR 1.07,

95%CI [1.04, 1.10]), maternal medical issues (OR 1.08, 95%CI [1.05, 1.12]), and labor and delivery experiences (OR 1.07, 95%CI [1.03, 1.11]) as causal ideas (see Tables 3, 4).

RQ.4: Differences in Causal Ideas Between Those With or Without ASD in the Household

Mothers of children with ASD were more likely to report a causal idea (56.4%) than those without (30.8%) [$X^2(1, N = 8307) = 482, p < 0.0001$], and the frequencies of several ideas also differed between the two groups. After adjusting for other covariates, mothers of children with ASD had higher odds of reporting genetics (OR 1.69, 95%CI [1.43, 1.99]), vaccines (OR 1.35, 95%CI [1.12, 1.63]), or child medical issues (OR 1.47, 95%CI [1.12, 1.94]) and had lower odds of reporting parent lifestyle (OR 0.62, 95%CI [0.51, 0.76]), child diet (OR 0.67, 95%CI [0.51, 0.87]), miscellaneous (OR 0.64, 95%CI [0.45, 0.92]), or parenting (OR 0.46, 95%CI [0.30, 0.70]) when compared to those without a child with ASD in the household. Figure 1 presents the predicted probability over time from the regression models in Table 3 for the most frequently reported causal ideas, stratified by having/not having a child with ASD, among those who gave any idea and were not missing any covariate data. Supplemental Table 2 displays the causal idea categories and their frequencies of responses by having or not having at least one child with ASD.

Discussion

While causal ideas reported in SEED were very similar to those reported in previous studies, there are three key findings to discuss: (1) differences between mothers with and without a child with ASD; (2) associations between ethno-cultural background and causal ideas, particularly maternal stress; and (3) differences over time in causal ideas and persistence of causal ideas currently unsupported by scientific research.

While causes of ASD remain largely unknown, contemporary scientific research focuses on genetics, interactions between environment and genes, and contributions of parent age, pre-pregnancy and perinatal medical issues, and parent lifestyle as contributing to the development of ASD or developmental delays (Beversdorf et al., 2018; Bolte et al., 2019; Carlsson et al., 2021; Croen et al., 2019; Lyall et al., 2017; Sandin et al., 2014; Zhou et al., 2022). SEED mothers generally reported causal ideas that are similar to those reported in prior causal idea research and similar to those causes under scientific investigation (genetics, environment, parent lifestyle issues, and maternal medical issues) (Chaidez et al., 2018; Dardennes et al., 2011; Felnhofer et al., 2020; Fombonne et al., 2020; Gray, 1994; Goin-Kochel & Myers, 2005; Harrington et al., 2006a, b; Mercer et al., 2006; McLeod & DiSabatino, 2019; Reiff et al., 2017; Zuckerman et al., 2016). However, fewer than half of mothers participating in the SEED maternal interview reported they had ideas about causes of ASD. The overall proportion of SEED mothers of children with ASD who gave no ideas about causes is similar to prior research among families of children with ASD (4% to 40%) (Goin-Kochel et al., 2015; Harrington et al., 2006a, b; Zuckerman et al., 2016). SEED results show that fewer mothers in the general population report ideas about what causes ASD which supports Mitchell and Locke's (2015) suggestion that "public" perception of causes of ASD may be different from that of families experiencing ASD.

Of the mothers suggesting any causal ideas, nearly half had a child with ASD despite only one third of the sample having a child with ASD. Further, mothers of children with ASD were more likely than those without to offer medical or biological concerns such as genetics, vaccines, and child medical issues as causal and less likely to offer behavioral or daily-life concerns such as parent lifestyle, child diet, or parenting issues as causal. These differences also support Mitchell and Locke's (2015) idea that these groups hold differing ideologies.

Our findings further suggest differences between ethno-cultural subgroups. More than half of the causal ideas were associated with ethno-cultural background and/or immigration status: genetics, vaccines, environment, parent lifestyle, labor and delivery experiences, child medical issues, maternal stress, and parenting. These results might suggest differing ethno-cultural experiences with pregnancy, delivery, and/or child development and healthcare, and differences in sources of health information. One notable finding was that mothers of other ethno-cultural backgrounds compared to non-Hispanic White, as well as those immigrating to the US at or after age 13, compared to US-born, were much more likely to report maternal stress as causal. This finding holds even after controlling for timing of enrollment. It is possible that these mothers may experience higher levels of pre-and peri-pregnancy stress and may feel it could contribute to negative health outcomes for their children. Stress has been linked to maternal inflammatory responses or other physiologic responses which have been associated with ASD or developmental issues in offspring (Beverdort et al., 2018; Glover, 2011; King et al., 2012), suggesting stress may be an important factor to examine further in different ethno-cultural strata as neurodevelopmental research continues.

The probability of offering the ideas of environment, child's diet and/or medical issues, maternal stress, parent age, and/or parenting issues did not significantly differ over the 13 years of SEED data collection. The probability of stating genetics, parent lifestyle issues, maternal medical issues, and labor and delivery experiences as causal increased over time, albeit small shifts (see Fig. 1). Vaccines as a causal idea significantly decreased over time. The shifts that moved toward alignment with scientific research [i.e. parent lifestyle (increasing) and vaccines (decreasing)], reflect two areas where parents have some control or choice, suggesting that some progress has been made in educating parents about the association of these topics with neurodevelopment. The decreased reporting of vaccines over time supports Fombonne and colleagues' (2020) suggestion that a belief in vaccines causing ASD might be diminishing.

Although vaccines as a causal idea was diminishing, SEED mothers reported it with relatively high frequency across time despite a large body of research supporting the safety of vaccines for children (Geoghegan et al., 2020; Taylor et al., 2014). SEED mothers also reported a few other ideas not currently supported by the scientific community, including parenting issues and child's diet. Parenting strategies (or parent/family-child relationships) and child's diet may alter behaviors or improve outcomes for those with developmental challenges rather than act as a cause of ASD (Crowell et al., 2019; Morton et al., 2023). Although the odds of citing parenting/family relationship issues or child's diet as causal did not change significantly over the course of SEED, they were cited with low frequency. Knowing these causal ideas exist among caregivers is important for practitioners to note because these ideas might influence healthcare choices in general and/or treatment seeking

in those whose children exhibit ASD characteristics thus affecting long-term health or functioning of some children (Al Anbar et al., 2010; Chaidez et al., 2018; Mire et al., 2017; Zerbo et al., 2018).

Limitations

Limitations of this study include sample selectivity. SEED participants generally lived well above federal poverty level (61.3% above 250% of poverty level) and most were highly educated (56.8% had at least a bachelor's degree). Thus, socioeconomic associations may not be representative of the general population and must be interpreted with caution. Additionally, questions about causal ideas were asked at the end of a lengthy phone interview, possibly artificially inflating the frequency of stating 'no' to the initial question to hasten the end of the interview. Further, the question was a single, open-ended question and the interviewers did not probe for clarification when recording the answer from the respondent. Thus, coding interpretations might be inaccurate. It seems unlikely that the question itself influenced the causes offered because a) the study was described to participants as a study comparing similarities and differences between children with and without autism, and b) the causes provided (both by those with and without household ASD) were similar to ideas previously stated in other studies. Additionally, these data were collected over time periods that included ASD diagnoses using the DSM-IV and the DSM-5 and we could not evaluate potential influences of that change on perception of cause. Mothers' ideas were represented in this study and may not reflect the ideas that fathers or other primary caregivers had about causes. Lastly, our regression models have the potential for inflated Type I error (i.e. chance for false positives) due to multiple testing. This approach may lead to optimistic estimates of precision in our confidence intervals. However, given the exploratory nature of our study, we aimed to examine a broad range of covariates to identify potential associations worth further investigation.

While SEED offers the first long-term assessment of maternal ideas about causes of ASD or developmental problems, the 13 years of cross-sectional data collection may not be sufficient to characterize true shifts in mothers' ideas about causes. Additionally, there may be more complex patterns over time between mothers with and without children with ASD for a few causal ideas but these are not explored in depth in this analysis. Longitudinal studies or longer-term cross-sectional studies would be beneficial to monitor public knowledge and how that knowledge might impact healthcare decisions for children.

Conclusion

While these findings might simply reflect the complexity and uncertainty about causes of ASD among both families and the scientific community, mothers' ideas about causes of ASD and developmental problems in SEED were more often offered by those who had children with ASD. Those who had children with ASD were more likely to offer causal ideas reflecting medical or biological concerns and less likely to offer behavioral or daily-life concerns. Moreover, there were ethno-cultural differences in likelihood of reporting several causal ideas suggesting potential differences in information and perspectives among cultural groups. Few causes were different over time but, importantly, lifestyle issues

and vaccines seemed to be trending in directions consistent with peer-reviewed science. Because ideas about cause might influence health-seeking behaviors such as vaccination scheduling or use of applied behavioral therapy for children, education efforts that increase overall awareness about what is or is not supported within the scientific community as contributing to ASD have potential to optimize healthcare decision-making among all families. Additional long-term studies on causal ideas should monitor shifts over time, differences between ethno-cultural subgroups, as well as explore the connections between causal ideas and decisions related to healthcare and/or intervention services for children.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Data Availability

Data from the study described in this manuscript will be shared with National Database for Autism Research (NDAR) for those participants who gave explicit permission for data sharing. This sharing has not yet occurred.

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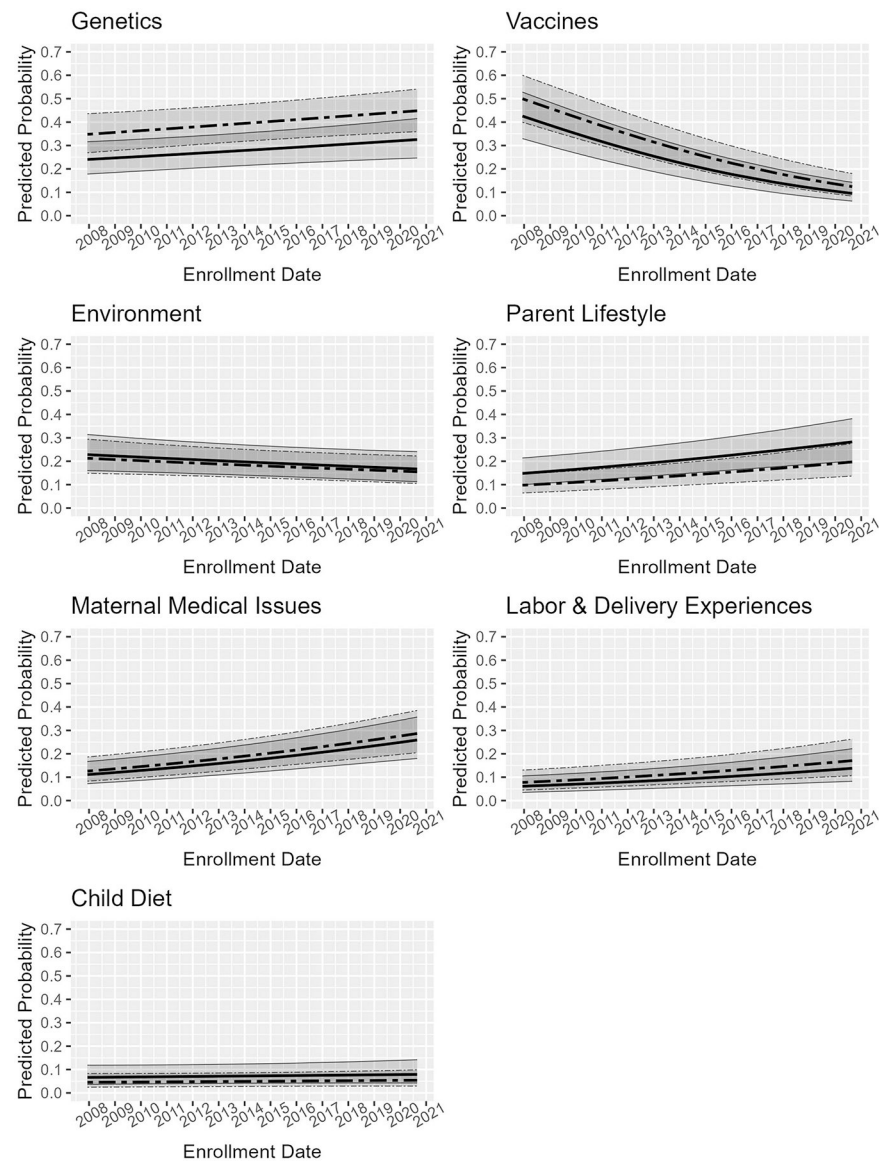
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**Fig. 1.**

Predicted probability for the top seven causal ideas over time and by ASD (Autism spectrum disorder) in the household ($n = 3020$). Note: the ribbons represent the 95% Confidence Intervals. The other covariates were held constant at their reference level for categorical variables and their mean for continuous variables. Dashed lines = with ASD in the household; solid lines = without ASD in the household

Table 1

Characteristics of participants in the Study to Explore Early Development (SEED) who completed the maternal interview, 2007–2020

	Total (N = 8307)
Child age at enrollment (years)	
Mean (SD)	4.3 (0.78)
Median [Min, Max]	4.4 [1.9, 5.7]
Child sex	n (%)
Male	5437 (65.5)
Female	2870 (34.5)
Maternal age at child's birth (years)	
< 30	3166 (38.1)
30–34	2884 (34.7)
35–39	1800 (21.7)
40+	455 (5.5)
Missing	2 (0)
Paternal age at child's birth (years)	
< 30	2202 (26.5)
30–34	2569 (30.9)
35–39	2015 (24.3)
40+	1286 (15.5)
Missing	235 (2.8)
Maternal ethno-cultural background, self-reported	
Non-Hispanic White	4842 (58.3)
Non-Hispanic Black	1672 (20.1)
Non-Hispanic American Indian/Alaska Native	19 (0.2)
Non-Hispanic Hawaiian/Pacific Islander/Asian	454 (5.5)
Non-Hispanic, > 1 race	284 (3.4)
Hispanic	1027 (12.4)
Missing	9 (0.1)
Maternal age at entry into US	
US-born	6958 (83.8)
Non-US born, entry < age13 years	313 (3.8)
Non-US born, entry ≥ age13 years	979 (11.8)
Missing	57 (0.7)
Maternal education	
Less than completed high school	377 (4.5)
High school graduate or equivalent	900 (10.8)
Some college	2257 (27.2)
Bachelor's degree	2551 (30.7)
Advanced degree(s)	2165 (26.1)
Missing	57 (0.7)
Income as percent of US federal poverty index	

	Total (N = 8307)
≤ 138%	1387 (16.7)
> 138%, ≤ 250%	1447 (17.4)
> 250%, < 400%	1821 (21.9)
≥ 400%	3274 (39.4)
Missing	378 (4.6)
Study site	
California	1007 (12.1)
Colorado	1424 (17.1)
Georgia	1552 (18.7)
Maryland	1176 (14.2)
Missouri	420 (5.1)
North Carolina	1420 (17.1)
Pennsylvania	899 (10.8)
Wisconsin	409 (4.9)
Children with ASD ^a	
No children with ASD ^a	5506 (66.3)
At least 1 child with ASD ^a	2681 (32.3)
Missing	120 (1.4)
Causal idea	
No, don't think there's anything that causes ASD ^a	4690 (56.5)
Don't know if there is any cause or what might cause	269 (3.2)
Missing	83 (1.0)
Yes, think there is a cause/causes ^b	3265 (39.3)

^a Autism spectrum disorder

^b Multiple causal ideas could be stated by each respondent

Table 2

Frequencies of ASD (Autism spectrum disorder) causal ideas (multiple causal ideas could be stated by each respondent) given by mothers who think there is a cause in the Study to Explore Early Development (SEED), 2007–2020 (n = 3265)

Cause	Overall n (%)	Given as sole causal idea n (%)	Given as one of 2 causal ideas n (%)	Given as one of 3–8 causal ideas n (%)
Genetics	1009 (30.9)	489 (48.5)	303 (30.0)	217 (21.5)
Vaccines	692 (21.2)	332 (48.0)	189 (27.3)	171 (24.7)
Environment	669 (20.5)	183 (27.4)	219 (32.7)	267 (39.9)
Parent lifestyle before/during pregnancy	594 (18.2)	141 (23.7)	192 (32.3)	261 (43.9)
Maternal medical issues	523 (16.0)	165 (31.5)	188 (35.9)	170 (32.5)
Labor and delivery experiences	319 (9.8)	157 (49.2)	104 (32.6)	58 (18.2)
Child Diet	318 (9.7)	30 (9.4)	99 (31.1)	189 (59.4)
Child medical issues	266 (8.1)	98 (36.8)	93 (35.0)	75 (28.2)
Maternal stress before/during pregnancy	176 (5.4)	61 (34.7)	57 (32.4)	58 (33.0)
Miscellaneous	162 (5.0)	101 (62.3)	38 (23.5)	23 (14.2)
Parent age	149 (4.6)	45 (30.2)	56 (37.6)	48 (32.2)
Child medication	131 (4.0)	17 (13.0)	47 (35.9)	67 (51.1)
Parenting/relationship issues during child's early life	132 (4.0)	60 (45.5)	41 (31.1)	31 (23.5)
Maternal other pre- or peri-pregnancy issues	63 (1.9)	24 (38.1)	22 (34.9)	17 (27.0)

Table 3

Adjusted odds ratios and 95% confidence intervals of characteristics associated with seven most frequently mother-reported ASD (Autism spectrum disorder) causal ideas in the Study to Explore Early Development (SEED), 2007–2020 (n = 3018)

	Gene	Vaccines	Env	Lifestyle	Mat. medical	Labor and deliv	Child diet
Child enroll age	0.98 (0.89, 1.07)	1.00 (0.9, 1.12)	1.07 (0.96, 1.19)	1.13 (1.01, 1.26)*	0.84 (0.75, 0.95)*	0.98 (0.84, 1.13)	1.23 (1.06, 1.43)*
Child sex							
Male (REF)							
Female	1.06 (0.89, 1.27)	0.92 (0.75, 1.13)	0.97 (0.80, 1.19)	1.08 (0.88, 1.32)	0.90 (0.72, 1.13)	0.76 (0.57, 1.01)	1.18 (0.91, 1.53)
Maternal age	1.05 (0.94, 1.18)	1.10 (0.97, 1.25)	1.29 (1.13, 1.47)*	0.99 (0.86, 1.12)	0.95 (0.83, 1.09)	0.93 (0.78, 1.10)	0.96 (0.81, 1.14)
Paternal age	0.96 (0.86, 1.07)	0.99 (0.88, 1.12)	0.92 (0.81, 1.04)	0.97 (0.85, 1.10)	1.04 (0.91, 1.18)	0.90 (0.76, 1.07)	1.03 (0.87, 1.23)
Maternal ethno-cultural background							
NH ^a , White (REF)							
NH ^a , Black	0.64 (0.50, 0.82)*	1.43 (1.10, 1.86)*	0.63 (0.47, 0.85)*	1.18 (0.90, 1.56)	1.08 (0.81, 1.44)	1.15 (0.81, 1.63)	0.75 (0.51, 1.11)
NH ^a , other	0.86 (0.64, 1.14)	1.13 (0.81, 1.56)	0.96 (0.70, 1.32)	1.50 (1.08, 2.08)*	1.03 (0.72, 1.46)	1.21 (0.80, 1.83)	1.05 (0.68, 1.63)
Hispanic	1.05 (0.78, 1.41)	0.91 (0.63, 1.30)	1.14 (0.81, 1.59)	1.20 (0.84, 1.71)	1.09 (0.76, 1.57)	0.55 (0.32, 0.94)*	1.12 (0.70, 1.79)
Maternal age at entry to US							
US born (REF)							
Non US born, < 13 years	1.18 (0.77, 1.79)	0.59 (0.32, 1.06)	1.18 (0.74, 1.89)	0.54 (0.30, 0.99)*	1.10 (0.65, 1.84)	1.75 (0.99, 3.07)	0.68 (0.32, 1.45)
Non US born, ≥ 13 years	0.68 (0.52, 0.90)*	1.07 (0.79, 1.45)	0.91 (0.67, 1.23)	0.99 (0.72, 1.35)	1.01 (0.72, 1.40)	0.96 (0.62, 1.47)	0.78 (0.50, 1.21)
Income as percent of poverty							
≤ 138% (REF)							
> 138% ≤ 250%	0.92 (0.68, 1.24)	1.09 (0.79, 1.51)	0.89 (0.62, 1.27)	1.16 (0.83, 1.63)	1.04 (0.73, 1.47)	1.26 (0.83, 1.91)	1.68 (0.99, 2.86)
> 250% < 400%	1.18 (0.87, 1.59)	0.97 (0.69, 1.35)	0.88 (0.62, 1.26)	1.20 (0.85, 1.71)	1.03 (0.71, 1.48)	0.77 (0.48, 1.22)	1.95 (1.15, 3.31)*
≥ 400%	1.21 (0.90, 1.63)	0.99 (0.71, 1.38)	0.93 (0.65, 1.32)	0.99 (0.69, 1.41)	1.01 (0.70, 1.46)	0.95 (0.60, 1.50)	1.70 (0.99, 2.91)
Maternal education							
Less than bachelor's (REF)							
Bachelor's or advanced degree	1.13 (0.92, 1.37)	0.84 (0.67, 1.04)	1.37 (1.09, 1.73)*	0.99 (0.78, 1.26)	1.11 (0.87, 1.43)	1.13 (0.83, 1.54)	1.17 (0.86, 1.59)
Time of enrollment	1.03 (1.01, 1.06)*	0.86 (0.83, 0.88)*	0.97 (0.94, 1.00)	1.07 (1.04, 1.10)*	1.08 (1.05, 1.12)*	1.07 (1.03, 1.11)*	1.01 (0.98, 1.06)
Children with ASD							
None (REF)							

	Gene	Vaccines	Env	Lifestyle	Mat. medical	Labor and deliv	Child diet
At least 1	1.69 (1.43, 1.99) [*]	1.35 (1.12, 1.63) [*]	0.91 (0.76, 1.1)	0.62 (0.51, 0.76) [*]	1.15 (0.94, 1.41)	1.29 (1.00, 1.66)	0.67 (0.51, 0.87) [*]

All models were also adjusted for study site and included an intercept. Child and parent ages were centered at their mean and scaled by their standard deviation. Shortened names of causal ideas were used due to table size

^{*} $p < 0.05$

^a Non-Hispanic

Table 4

Adjusted odds ratios and 95% confidence intervals of characteristics associated with seven least frequently mother-reported ASD (Autism spectrum disorder) causal ideas in the Study to Explore Early Development (SEED), 2007–2020 (n = 3018)

	Child medical	Mat. stress	Misc	Parent age	Child meds	Parenting	Mat. Other pre/perinatal
Child enroll age	1.06 (0.91, 1.24)	1.24 (1.01, 1.52)*	0.91 (0.75, 1.1)	0.99 (0.80, 1.21)	0.96 (0.77, 1.19)	1.05 (0.85, 1.31)	0.80 (0.59, 1.07)
Child sex							
Male (REF)							
Female	0.83 (0.61, 1.13)	0.97 (0.67, 1.41)	1.31 (0.92, 1.86)	1.00 (0.68, 1.47)	0.89 (0.58, 1.35)	1.11 (0.75, 1.66)	1.46 (0.84, 2.55)
Maternal age	0.96 (0.79, 1.16)	0.85 (0.68, 1.06)	1.04 (0.82, 1.31)	1.78 (1.39, 2.27)*	0.92 (0.72, 1.19)	1.01 (0.77, 1.31)	0.78 (0.53, 1.15)
Paternal age	0.90 (0.75, 1.09)	1.10 (0.89, 1.35)	1.03 (0.82, 1.29)	1.37 (1.10, 1.71)*	1.10 (0.87, 1.40)	1.03 (0.81, 1.33)	1.04 (0.73, 1.48)
Maternal ethno-cultural background							
NH ^a , White (REF)							
NH ^a , Black	0.63 (0.40, 0.97)*	1.89 (1.17, 3.06)*	0.63 (0.37, 1.09)	1.22 (0.73, 2.07)	0.62 (0.35, 1.11)	0.90 (0.49, 1.63)	0.93 (0.42, 2.03)
NH ^a , other	0.75 (0.46, 1.22)	2.11 (1.23, 3.60)*	0.79 (0.42, 1.47)	0.75 (0.38, 1.48)	1.42 (0.77, 2.64)	1.38 (0.74, 2.57)	0.42 (0.12, 1.50)
Hispanic	0.52 (0.30, 0.92)*	2.46 (1.43, 4.20)*	0.78 (0.41, 1.49)	1.16 (0.61, 2.23)	1.31 (0.68, 2.51)	1.28 (0.66, 2.51)	0.79 (0.27, 2.30)
Maternal age at entry to US							
US born (REF)							
Non US born, < 13 years	0.37 (0.11, 1.19)	0.74 (0.29, 1.92)	1.28 (0.53, 3.09)	0.90 (0.34, 2.38)	1.14 (0.44, 2.97)	0.52 (0.12, 2.19)	1.20 (0.28, 5.21)
Non US born, ≥ 13 years	1.26 (0.80, 1.97)	1.62 (1.03, 2.54)*	1.20 (0.68, 2.11)	0.90 (0.51, 1.59)	0.65 (0.33, 1.30)	1.95 (1.14, 3.34)*	1.19 (0.47, 3.04)
Income as percent of poverty							
≤ 138% (REF)							
> 138% ≤ 250%	1.16 (0.70, 1.92)	0.71 (0.43, 1.17)	0.68 (0.38, 1.21)	0.58 (0.28, 1.20)	0.96 (0.51, 1.79)	0.89 (0.47, 1.66)	0.54 (0.21, 1.43)
> 250% < 400%	1.08 (0.64, 1.81)	0.62 (0.35, 1.07)	0.83 (0.47, 1.48)	0.76 (0.38, 1.53)	1.09 (0.58, 2.08)	0.51 (0.25, 1.05)	0.83 (0.33, 2.11)
≥ 400%	1.20 (0.72, 2.02)	0.53 (0.31, 0.93)*	0.55 (0.30, 1.00)	1.03 (0.53, 1.99)	1.07 (0.55, 2.08)	0.72 (0.36, 1.42)	0.78 (0.30, 2.04)
Maternal education							
Less than bachelor's (REF)							
Bachelor's or advanced degree	1.06 (0.76, 1.48)	1.18 (0.78, 1.79)	0.85 (0.56, 1.28)	0.93 (0.60, 1.44)	0.48 (0.30, 0.74)*	1.12 (0.69, 1.83)	1.04 (0.53, 2.07)
Time of enrollment	1.00 (0.96, 1.04)	1.03 (0.98, 1.08)	1.03 (0.97, 1.08)	1.03 (0.98, 1.09)	1.01 (0.95, 1.07)	1.04 (0.98, 1.11)	1.05 (0.96, 1.14)
Children with ASD							
None (REF)							

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	Child medical	Mat. stress	Misc	Parent age	Child meds	Parenting	Mat. Other pre/perinatal
At least 1	1.47 (1.12, 1.94)*	1.26 (0.89, 1.77)	0.64 (0.45, 0.92)*	0.77 (0.53, 1.12)	1.01 (0.69, 1.48)	0.46 (0.30, 0.70)*	0.70 (0.39, 1.25)

All models were also adjusted for study site and included an intercept. Child and parent ages were centered at their mean and scaled by their standard deviation. Shortened names of causal ideas were used due to table size

* $p < 0.05$

^aNon-Hispanic