OPEN ACCESS

ISSN: 2835-6764

Milelli RJ (2026) 2643
American J Sci Edu Re, 2026; 5(5):100324
DOI: 10.71010/2835-6764/ajser-e324

Research Article

American Journal of Science Education Research

Maternal Age at Childbirth as a Demographic Correlate of Autism Spectrum Disorder Prevalence in the United States, 1992–2014: An Ecological Analysis with Diagnostic Era Stratification

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Citation: Milelli RJ (2026) Maternal Age at Childbirth as a Demographic Correlate of Autism Spectrum Disorder Prevalence in the United States, 1992–2014: An Ecological Analysis with Diagnostic Era Stratification. J Sci Edu Re: AJSER-324.

Received Date: 21 May, 2026: **Accepted Date:** 04 May, 2026: **Published Date:** 08 May, 2026

Abstract

Background: *Individual-level epidemiological evidence has established a monotonic dose-response relationship between advancing maternal age and autism spectrum disorder (ASD) risk, grounded in age-related increases in de novo chromosomal mutations, copy number variants, and epigenetic dysregulation. Over the same period that ASD prevalence has risen substantially in the United States, mean maternal age at childbirth has increased by 2.4 years. If the individual-level relationship is real, a sustained national shift toward later childbearing should produce a measurable demographic signal in population-level surveillance data. This study tests whether that signal is present and whether it persists across the transition from DSM-IV to DSM-5 diagnostic criteria.*

Methods: *ASD prevalence data were obtained from 12 CDC Autism and Developmental Disabilities Monitoring (ADDM) Network surveillance cycles (birth cohorts 1992–2014, surveillance years 2000–2022, approximately 50 million births). Mean maternal age data were drawn from the Human Fertility Database via Our World in Data. Three complementary analyses were conducted: (1) level-data Pearson and Spearman correlations with OLS regression and Newey-West autocorrelation-corrected standard errors; (2) first-differences analysis, correlating year-over-year changes in maternal age with year-over-year changes in ASD prevalence to remove the shared temporal trend; and (3) diagnostic era stratification, fitting separate regression models for the DSM-IV era (n = 7 cycles) and DSM-5 era (n = 5 cycles). A multiple regression model including both maternal age and a diagnostic era indicator assessed the independent contribution of each. A sensitivity analysis substituted mean age at first birth for mean age across all births.*

Results: *Level-data Pearson correlation was $r = .975$ ($p < 10^{-7}$), with OLS slope 10.74 (Newey-West SE = 0.955, $p < .001$) and $R^2 = .951$. First-differences analysis yielded $r = .635$ ($p = .036$), confirming the association persists after removing the shared temporal trend. The association held independently within both the DSM-IV era ($r = .985$, $p < .001$) and DSM-5 era ($r = .998$, $p < .001$), with slope increasing from 6.82 to 14.12 per year of maternal age. Multiple regression confirmed maternal age as the dominant predictor ($b = 9.40$, $SE = 1.22$, $p < .001$) after controlling for diagnostic era. Results were robust to the operationalization of maternal age (slope difference < 1%).*

Conclusions: *The association between maternal age and ASD prevalence is detectable beyond shared temporal trending, holds within each diagnostic era independently, and is consistent with the demographic translation of established individual-level risk relationships. These findings support the incorporation of population-level maternal age trends into epidemiological models of ASD prevalence and underscore the need for ADDM surveillance reporting to include maternal age as a core demographic variable. The ecological design precludes individual-level causal inference; findings should not be interpreted as implying individual reproductive decisions.*

Keywords: *maternal age; autism spectrum disorder; ASD prevalence; ecological epidemiology; ADDM; birth cohorts; DSM-IV; DSM-5; diagnostic era; demographic trends; de novo mutations.*

Background

ASD prevalence in the United States has risen from approximately 6.7 per 1,000 children in surveillance year 2000 to 32.2 per 1,000 in 2022, as documented by the CDC Autism and Developmental Disabilities Monitoring (ADDM) Network [1]. The drivers of this increase remain actively debated, with candidate explanations including expanded diagnostic criteria, improved case ascertainment, heightened clinical awareness, and genuine etiological change.

Over the same period, a parallel demographic shift has occurred: mean maternal age at childbirth in the United States rose from 26.6 years in 1992 to 29.0 years in 2014, an increase of 2.4 years driven by educational attainment, labor force participation, and delayed family formation [2]. These trends are not independent. Individual-level epidemiological evidence has established a clear, monotonic dose-response relationship between advancing maternal age and ASD risk.

Sandin et al. [3], in a meta-analysis of 16 epidemiological studies encompassing millions of births, confirmed that relative ASD risk increases monotonically with advancing maternal age — from 0.76 for mothers under 20 to 1.52 for mothers aged 35 and older relative to the 25–29 reference group — persisting after adjustment for paternal age and other potential confounders. A subsequent multinational cohort study of nearly 5.8 million children across five countries similarly found advancing maternal age to be independently associated with

increased ASD risk [4]. These individual-level findings are biologically grounded: advanced maternal age is associated with elevated rates of de novo chromosomal mutations and copy number variants directly implicated in ASD etiology, epigenetic dysregulation, and pregnancy complications such as gestational hypertension, all of which have established associations with neurodevelopmental outcomes [3,4].

The logical implication follows directly: if older maternal age increases individual ASD risk, then a sustained national shift toward later childbearing should produce a detectable demographic signal in population-level ASD surveillance data. Yet despite this straightforward prediction, maternal age is conspicuously absent from ADDM's core surveillance reporting, which emphasizes subgroup differences by sex, race/ethnicity, and geography. The population-level demographic translation of this well-established individual-level risk relationship has not been systematically quantified.

The present study addresses this gap through ecological epidemiology — a methodology appropriate when both the exposure (mean maternal age at childbirth) and the outcome (population-level ASD prevalence) are aggregate phenomena measured at the population level [5]. The ecological design is not incidental; it is appropriate to the research question, which concerns population-level demographic trends rather than individual-level risk. The study pursues three aims: (1) to quantify the association between mean maternal age and ASD prevalence across the full ADDM surveillance period, with autocorrelation-corrected standard errors; (2) to test whether the association persists after removing the shared temporal trend via first-differences analysis; and (3) to determine whether the association holds independently within each of the DSM-IV and DSM-5 diagnostic eras.

Methods

2.1 Data Sources

ASD prevalence. ASD prevalence data were obtained from the CDC ADDM Network [1], which provides the only official U.S. population-level surveillance estimates of ASD prevalence. ADDM employs active, records-based surveillance; trained clinicians review evaluation records to determine case status against standardized DSM criteria, without relying on parent or provider report. Data span surveillance years 2000–2022, corresponding to birth cohorts 1992–2014, with prevalence reported at age eight. The most recent ADDM release reports prevalence of 32.2 per 1,000 children for the 2014 birth cohort. The combined dataset encompasses approximately 50 million births and approximately two million ASD diagnoses.

Maternal age. Maternal age data were drawn from the Human Fertility Database as processed and made publicly available by Our World in Data [2]. The HFD was selected as the primary data source because it provides birth-order-specific maternal age data spanning the full 1992–2014 period corresponding to all 12 ADDM surveillance cycles. CDC National Vital Statistics data on maternal age by birth order (Mathews & Hamilton, 2016 [7]) are available for 2000–2014 only and therefore do not cover the complete ADDM surveillance period. As reported in Appendix A, the NCHS data independently corroborate the HFD birth-order trends over the overlapping 2000–2014 period, with consistent rank ordering across all birth orders in both sources. Two measures were obtained for each birth cohort year: (1) mean maternal age across all births, used as the primary exposure variable as it most directly represents the population-level demographic shift; and (2) mean maternal age at first birth, used in a pre-specified sensitivity analysis. Both measures are presented in Table 2.

Diagnostic era classification. The DSM-5, published May 2013, introduced substantive changes to ASD diagnostic criteria, including consolidation of Asperger's disorder and PDD-NOS under a single ASD umbrella. ADDM surveillance criteria transitioned accordingly. Surveillance years 2000–2012 (birth cohorts 1992–2004) are classified as the DSM-IV era (n = 7 cycles); surveillance years 2014–2022 (birth cohorts 2006–2014) are classified as the DSM-5 era (n = 5 cycles).

2.2 Statistical Analysis

All analyses used mean maternal age across all births as the primary exposure variable. Statistical analyses were conducted in Python using SciPy, NumPy, and Statsmodels libraries. All reported p-values are two-tailed; statistical significance was set at $p < .05$.

Level-data correlation and regression. Pearson's correlation coefficient (r) and Spearman's rank correlation (r_s) assessed the linear and monotonic associations between maternal age and ASD prevalence across the full dataset (n = 12). OLS linear regression was fitted with maternal age as the independent variable and ASD prevalence per 1,000 children as the dependent variable. Because time-series observations are not independent, the Durbin-Watson statistic was computed to assess autocorrelation in residuals; Newey-West heteroscedasticity and autocorrelation-consistent (HAC) standard errors (lag = 2, Bartlett kernel) were applied to all OLS models to provide valid inference in the presence of autocorrelation.

First-differences analysis. To test whether the association reflects a genuine relationship beyond two variables sharing a common upward trend, year-over-year changes in maternal age (Δ maternal age) were correlated with year-over-year changes in ASD prevalence (Δ ASD prevalence) across n = 11 consecutive pairs. First-differencing removes the shared temporal trend and provides a conservative test of whether short-term fluctuations in maternal age co-vary with short-term fluctuations in ASD prevalence.

Diagnostic era stratification. Pearson and Spearman correlations and OLS regression were computed separately for the DSM-IV era (n = 7) and DSM-5 era (n = 5). The change in OLS slope across eras was computed as a slope ratio. This stratification provides a within-era test of the association that is not vulnerable to the diagnostic artifact objection: if the association holds within a period of stable diagnostic criteria, it cannot be attributed to changing case definitions.

Multiple regression. A multiple regression model was fitted with both maternal age and a binary diagnostic era indicator (0 = DSM-IV, 1 = DSM-5) as predictors, to estimate the independent contribution of each after accounting for the other.
Sensitivity analysis. All primary analyses were repeated substituting mean maternal age at first birth for mean age across all births to assess robustness to the operationalization of the exposure variable.

Ethics. This study used publicly available, aggregated surveillance data and did not involve human subjects or

identifiable personal information; institutional review board approval was not required.

Results

3.1 Descriptive Data

Table 1 presents ASD prevalence estimates from each ADDM surveillance cycle. Prevalence increased from 6.7 per 1,000 children in surveillance year 2000 to 32.2 per 1,000 in 2022, a 4.8-fold increase over 22 years. Table 2 presents the two maternal age series. Mean age across all births rose from 26.6 years (birth cohort 1992) to 29.0 years (birth cohort 2014), an increase of 2.4 years. Mean age at first birth rose from 24.4 to 26.8 years over the same period — an identical magnitude, differing only in absolute scale by approximately 2.2 years.

Table 1: ASD Prevalence by ADDM Surveillance Cycle, 2000–2022.

Surveillance Year	Birth Cohort Year	Diagnostic Era	ASD Prevalence (per 1,000)	Approx. 1 in X Children
2000	1992	DSM-IV	6.7	150
2002	1994	DSM-IV	6.6	150
2004	1996	DSM-IV	8.0	125
2006	1998	DSM-IV	9.0	110
2008	2000	DSM-IV	11.3	88
2010	2002	DSM-IV	14.7	68
2012	2004	DSM-IV	14.5	69
2014	2006	*DSM-5*	16.8	59
2016	2008	*DSM-5*	18.5	54
2018	2010	*DSM-5*	23.0	44
2020	2012	*DSM-5*	27.6	36
2022	2014	*DSM-5*	32.2	31

Note. Shaded rows indicate DSM-5 era surveillance cycles. Prevalence estimates represent children aged 8 years identified through active records-based surveillance.

Table 2: Mean Maternal Age at Childbirth by Birth Cohort Year, United States, 1992–2014.

Birth Cohort Year	Mean Age at First Birth (years)	Mean Age All Births (years)
1992	24.4	26.6
1994	24.4	26.7
1996	24.7	26.9
1998	24.9	27.1
2000	25.2	27.4
2002	25.5	27.7
2004	25.7	27.9
2006	25.6	27.9
2008	25.7	28.0
2010	26.1	28.3
2012	26.4	28.6
2014	26.8	29.0

Note. Both measures are derived from the Human Fertility Database via Our World in Data [2]. Mean age at first birth was used in the sensitivity analysis; mean age across all births was the primary exposure variable.

3.2 Primary Analysis: Level-Data Association (n = 12)

Across the full dataset, Pearson correlation between mean maternal age across all births and ASD prevalence was $r = .975$ ($p < 10^{-7}$). Spearman rank correlation was $r_s = .981$ ($p < 10^{-7}$), confirming the association is robust to the assumption of linearity. OLS regression yielded a slope of 10.74 (SE = 0.769, $p < 10^{-7}$), indicating that each one-year increase in mean maternal age is associated with approximately 10.7 additional ASD cases per 1,000 children. The coefficient of determination was $R^2 = .951$.

The Durbin-Watson statistic of 0.507 confirms positive autocorrelation in OLS residuals, as expected with time-series data. Newey-West HAC-corrected standard errors (lag = 2) yielded SE = 0.955 — modestly larger than the OLS SE of 0.769 — and the slope remained highly significant ($t = 11.24$, $p < .001$). The autocorrelation does not invalidate the association; it requires the corrected standard errors reported here. Figure 1 displays the scatter plot with the OLS regression line.

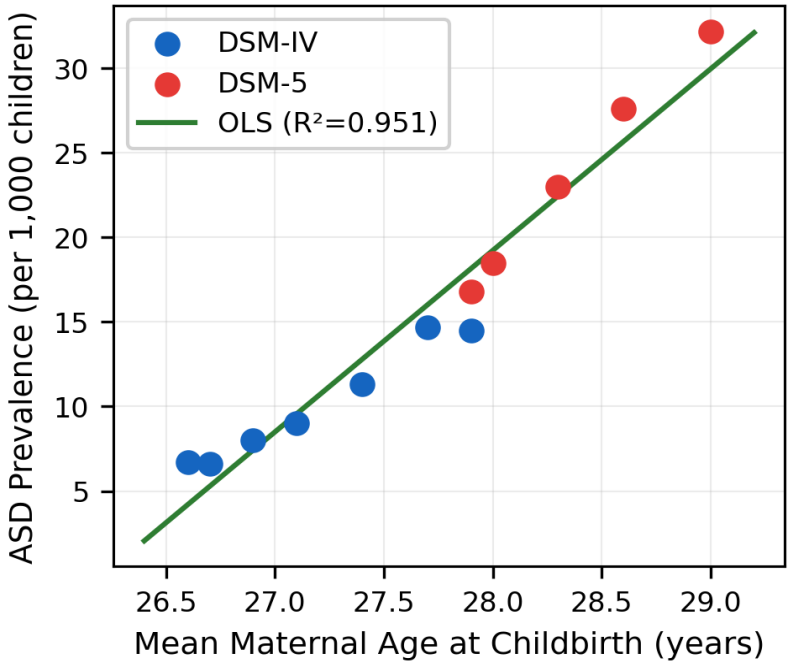

Figure 1: *Scatter plot of mean maternal age at childbirth and ASD prevalence per 1,000 children, United States 1992–2014 (n = 12 ADDM surveillance cycles). Blue circles = DSM-IV era; red circles = DSM-5 era. Green line = OLS regression (R^2 = .951, Newey-West corrected SE = 0.955, p < .001).*

3.3 First-Differences Analysis (n = 11)

To test whether the association reflects a genuine relationship beyond shared temporal trending, year-over-year changes in maternal age were correlated with year-over-year changes in ASD prevalence across n = 11 consecutive cohort pairs. First-differences Pearson correlation was r = .635 (p = .036); Spearman rank correlation was r_s = .725 (p = .012). OLS regression on first-differences yielded a slope of 9.65 (SE = 3.91, R^2 = .403, p = .036).

The R^2 of .403 in first-differences, compared to .951 in level data, reflects the honest decomposition of the level-data correlation: the shared temporal trend accounts for a substantial portion, but a statistically significant residual association remains after its removal. Year-on-year increases in maternal age predict year-on-year increases in ASD prevalence, independent of their common upward trend. Figure 2 displays the first-differences scatter plot.

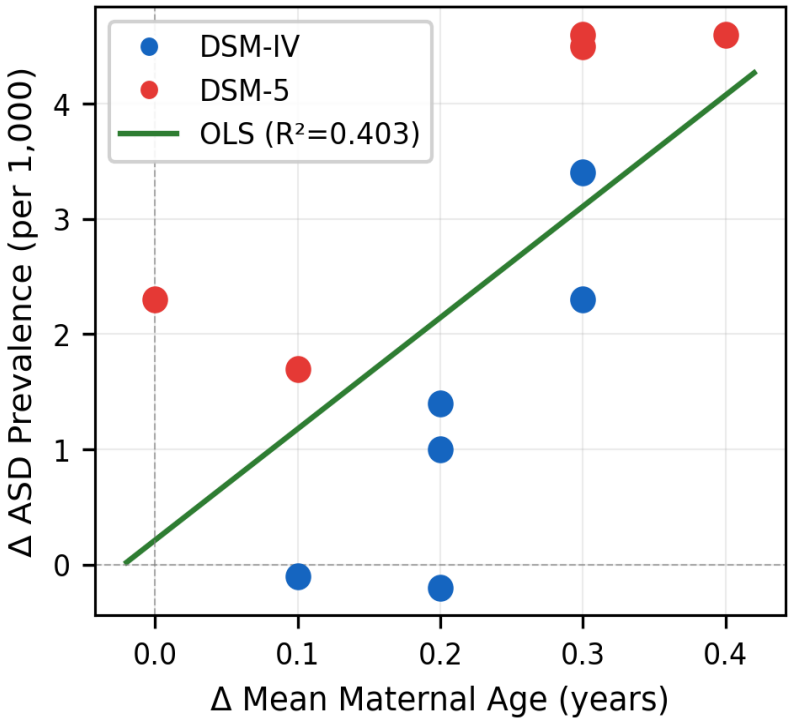

Figure 2: *First-differences scatter plot: year-over-year change in mean maternal age (Δ maternal age) vs year-over-year change in ASD prevalence (Δ ASD prevalence), n = 11 consecutive cohort pairs. Blue = DSM-IV era transitions; red = DSM-5 era transitions. OLS fit shown (R^2 = .403, p = .036). The association persists after removing the shared temporal trend.*

Table 3: First-Differences: Year-over-Year Changes in Maternal Age and ASD Prevalence.

Birth Year Pair	Δ Maternal Age (years)	Δ ASD Prevalence (per 1,000)
1992→1994	+0.10	−0.1
1994→1996	+0.20	+1.4
1996→1998	+0.20	+1.0
1998→2000	+0.30	+2.3
2000→2002	+0.30	+3.4
2002→2004	+0.20	−0.2
2004→2006	+0.00	+2.3
2006→2008	+0.10	+1.7
2008→2010	+0.30	+4.5
2010→2012	+0.30	+4.6
2012→2014	+0.40	+4.6

3.4 Sensitivity Analysis: Mean Age at First Birth

Substituting mean maternal age at first birth for mean age across all births produced virtually identical results: $r = .975$ ($p < 10^{-7}$), OLS slope = 10.64 (SE = 0.771, $R^2 = .950$), representing a slope difference of less than 1% from the primary analysis. First-differences with the first-birth series yielded $r = .593$ ($p = .054$), directionally consistent though marginally below the significance threshold, reflecting the slightly noisier year-over-year pattern of the first-birth series. The primary findings are robust to the operationalization of the exposure variable.

3.5 Diagnostic Era Stratification

Table 4 presents correlation and regression results stratified by diagnostic era. The association between maternal age and ASD prevalence was strong and statistically significant within both the DSM-IV era ($r = .985$, $p < .001$; $n = 7$) and the DSM-5 era ($r = .998$, $p < .001$; $n = 5$). The Spearman rank correlation under DSM-5 criteria was 1.000, indicating a perfect monotonic ordering with no rank inversions across all five surveillance cycles.

The OLS slope increased from 6.82 (SE = 0.539) in the DSM-IV era to 14.12 (SE = 0.542) in the DSM-5 era, a slope ratio of 2.07 — the DSM-5 era slope is approximately twice as steep. This is a critical finding: the association holds independently within each diagnostic regime. The maternal age signal does not depend on combining the two eras. Within a period of stable diagnostic criteria, year-by-year increases in maternal age track year-by-year increases in ASD prevalence with near-perfect consistency. Figure 3 presents the stratified regression.

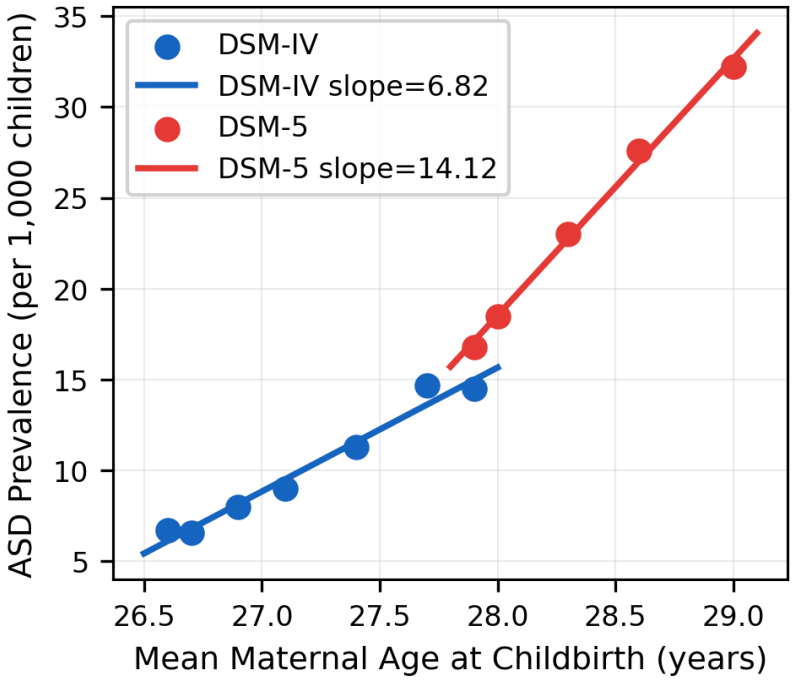

Figure 3: *OLS regression of ASD prevalence on mean maternal age, stratified by diagnostic era. DSM-IV era (blue, n = 7): slope = 6.82, $R^2 = .970$. DSM-5 era (red, n = 5): slope = 14.12, $R^2 = .996$. The association holds independently within each diagnostic regime; slope ratio DSM-5/DSM-IV = 2.07.*

Table 4: Correlation and Regression Results Stratified by Diagnostic Era.

Statistic	DSM-IV (n = 7)	DSM-5 (n = 5)	Combined (n = 12)
Pearson r	.985**	.998**	.975**
Spearman r_s	.929**	1.000**	.981**
OLS Slope	6.82**	14.12**	10.74**
OLS Intercept	−154.4	−313.5	−281.4
Standard Error (SE)	0.539	0.542	0.769
NW-Corrected SE	—	—	0.955
R^2	.970	.996	.951

*Note. ** p < .001 (two-tailed). OLS = ordinary least squares. NW = Newey-West HAC-corrected SE (lag = 2). Slope values in ASD cases per 1,000 children per one-year increase in mean maternal age.*

3.6 Multiple Regression: Maternal Age and Diagnostic Era

In the multiple regression model including both maternal age and the diagnostic era indicator, maternal age remained the dominant and statistically significant predictor (b = 9.40, SE = 1.22, t(9) = 7.70, p < .001). The diagnostic era indicator did not reach statistical significance after controlling for maternal age (b = 2.47, SE = 1.80, p = .202). The model R^2 = .960 (adjusted R^2 = .951). These results indicate that the maternal age effect is not an artifact of mixing the two diagnostic regimes: it accounts for the dominant share of variance independently.

Discussion

4.1 The Demographic Signal and Its Biological Foundation

The central finding of this study is that the association between mean maternal age at childbirth and ASD prevalence persists across multiple methodological tests: it survives detrending via first-differences analysis, holds independently within each of the two major diagnostic regimes, and remains the dominant predictor in multiple regression. This convergence of evidence is consistent with the hypothesis that demographic aging of the maternal population contributes a measurable signal to national ASD prevalence trends.

The biological foundation for this demographic translation is well established. Sandin et al. [3,4] demonstrated across millions of births in multiple countries that ASD risk increases monotonically with advancing maternal age, with a dose-response relationship that persists after adjustment for paternal age and other confounders. The proposed mechanisms are specific and biologically plausible: advanced maternal age is associated with increased rates of de novo chromosomal mutations and copy number variants directly implicated in ASD etiology, altered epigenetic regulation, and oxidative stress that may adversely affect fetal neurodevelopment [3,4]. This is not a general association with "older mothers"; it is a quantified, mechanism-grounded risk gradient.

What the present study contributes is the population-level translation of that gradient. If individual risk increases monotonically with maternal age, and if the national distribution of maternal age shifts upward by 2.4 years over two decades, then national ASD prevalence should rise — not because any individual etiology has changed, but because the population has aged into a higher-risk demographic configuration. The present findings are consistent with this prediction and quantify its magnitude. Figure 4 displays the parallel temporal trajectories of mean maternal age and ASD prevalence across all 12 ADDM surveillance cycles, illustrating the co-trending of both variables across the full study period.

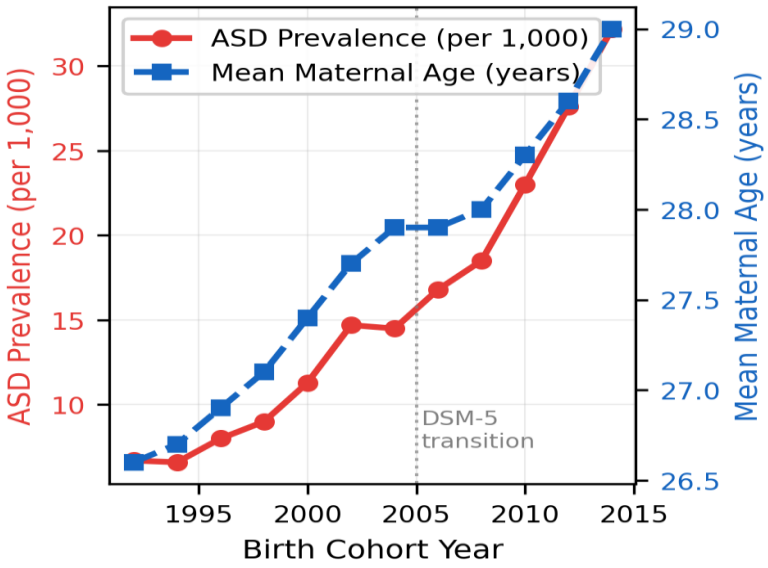

Figure 4: *Parallel temporal trends in mean maternal age at childbirth (blue dashed line, right axis) and ASD prevalence per 1,000 children (red solid line, left axis) across 12 ADDM surveillance cycles, birth cohorts 1992–2014. Both variables increase monotonically across the full study period. Dotted vertical line marks the approximate DSM-5 diagnostic transition (2013).*

4.2 The First-Differences Finding

The most rigorous test in the present analysis is the first-differences result. By correlating year-over-year changes rather than levels, first-differencing removes the shared upward trend that makes any two monotonically increasing time series appear correlated. The association survives this test ($r = .635$, $p = .036$), indicating that years in which maternal age increased more were also years in which ASD prevalence increased more. This is direct evidence of co-movement beyond shared trending.

The R^2 of .403 in first-differences, compared to .951 in level data, deserves explicit acknowledgment rather than minimization. It reflects the honest decomposition of the observed relationship: the shared temporal trend is real and accounts for a substantial portion of the level-data correlation. But the residual association — 40% of variance in year-over-year prevalence changes accounted for by year-over-year maternal age changes — represents a signal that is not reducible to shared trending alone.

4.3 The Diagnostic Era Stratification

The finding that the association holds independently within both the DSM-IV era ($r = .985$) and the DSM-5 era ($r = .998$) directly addresses the most common objection to ecological analyses of ASD prevalence trends: that the observed correlation is an artifact of changing diagnostic criteria rather than a meaningful demographic signal. Within a period of stable diagnostic criteria, the criteria cannot explain the trend. The maternal age association is present in both periods.

The near-doubling of the regression slope from the DSM-IV to DSM-5 era (6.82 to 14.12) has a straightforward interpretation: DSM-5's broader criteria capture milder ASD presentations that DSM-IV classified as Asperger's disorder or PDD-NOS. These milder presentations are likely to be disproportionately associated with the subtler biological effects of moderately advanced maternal age, compared to the more severe presentations captured by DSM-IV criteria. The steeper slope under DSM-5 is therefore consistent with, rather than contradictory to, the biological narrative: a broader diagnostic net captures more of the maternal age gradient.

The multiple regression result — maternal age remaining highly significant ($p < .001$) while the DSM-5 indicator does not reach significance ($p = .202$) after controlling for maternal age — further supports this interpretation. The diagnostic transition modulates the slope but does not explain the association.

4.4 Limitations

Several limitations must be considered. The ecological design, while appropriate for the population-level research question, precludes causal inference at the individual level. Findings must not be interpreted as implying that individual women should alter reproductive timing based on ASD risk, which remains low in absolute terms at the individual level. The ecological fallacy — drawing individual-level conclusions from aggregate data — is explicitly acknowledged.

The dataset consists of $n = 12$ aggregate surveillance observations, limiting statistical power for detecting interaction effects or non-linear relationships. Although the Newey-West correction addresses autocorrelation in standard errors, $n = 12$ time-series points represents a small sample for ecological time-series analysis, and results should be interpreted with corresponding caution. The analysis does not control for potential ecological-level confounders including temporal trends in paternal age, socioeconomic status, healthcare access, or geographic variation in diagnostic practices, beyond the diagnostic era covariate.

The ADDM Network sites do not constitute a nationally representative probability sample, which constrains direct generalization to the broader U.S. population. The most recent ADDM data available at time of analysis correspond to the 2014 birth cohort; replication with subsequent cohorts as they become available would strengthen the findings.

Notwithstanding these limitations, the convergence of evidence across three analytical approaches — level-data correlation with corrected standard errors, first-differences analysis, and within-era stratification — provides more robust support for the demographic signal than any single method alone. The underlying dataset encompasses approximately 50 million births and two million diagnoses, substantially mitigating concerns about measurement instability.

Conclusions

This study demonstrates that the association between mean maternal age at childbirth and ASD prevalence in the United States is detectable beyond shared temporal trending, holds independently within both major diagnostic eras, and is consistent with the demographic translation of a well-established individual-level biological risk relationship. The first-differences finding ($r = .635$, $p = .036$) establishes that year-over-year co-movement exists beyond shared trending. The diagnostic era stratification establishes that the signal is not a diagnostic artifact. The biological foundation established by Sandin et al. [3,4] provides the mechanism that makes the ecological correlation interpretable rather than merely statistical.

These findings support two practical conclusions. First, population-level maternal age trends should be incorporated into epidemiological models of ASD prevalence alongside diagnostic expansion and surveillance improvement as a quantifiable demographic contributor. Second, ADDM surveillance reporting should include maternal age as a core demographic variable; its current absence from ADDM's standard reporting leaves a systematic gap in the public health understanding of ASD prevalence drivers.

These findings do not establish individual-level causation, do not imply that maternal age is the sole or dominant cause of rising ASD prevalence, and should not be interpreted as guidance for individual reproductive decisions. They establish that a demographic signal consistent with established biology is present in national surveillance data and warrants integration into the broader scientific understanding of ASD prevalence trends.

Declarations

Availability of data and materials: All data used in this study are publicly available. ASD prevalence data were obtained from the CDC ADDM Network (https://www.cdc.gov/autism/data-research/index.html). Maternal age data were sourced from Our World in Data (https://ourworldindata.org/grapher/period-average-age-of-mothers-birth-order), derived from the Human Fertility Database.

Funding: No external funding was received for this research.

Acknowledgements: The author thanks the CDC ADDM Network for maintaining publicly accessible ASD surveillance data, and the Human Fertility Database and Our World in Data for providing accessible maternal age statistics.

Authors' contributions: RM: Conceptualization, data curation, formal analysis, visualization, validation, and writing. Statistical analyses were performed in Python (SciPy, NumPy, Statsmodels). All responsibility for interpretation and conclusions rests with the author.

Appendix A: Birth-Order Validation of the Aggregate Maternal Age Metric

The primary analysis uses mean maternal age across all births as the aggregate exposure variable. A potential concern is that this aggregate metric could reflect compositional change — for example, an increase in the proportion of higher-order births among older mothers — rather than a genuine population-wide shift toward later childbearing. This appendix addresses that concern directly.

Figure A.1: displays mean maternal age trends separately for birth orders 1 through 4, birth order 5 and higher, and all births combined, across the 12 birth cohort years from 1992 to 2014. The figure demonstrates that rising maternal age is not confined to any single birth order but reflects a consistent, monotonic upward trend across all parity groups. Table A.1 presents the OLS regression results for each series.

Table A.1: OLS Regression of Maternal Age on Birth Cohort Year, by Birth Order, United States 1992–2014.

Birth Order	1992 Age	2014 Age	HFD Change (1992–2014)	NCHS Change† (2000–2014)	OLS Slope	R^2
Order 1	24.4	26.8	+2.4 yrs	+1.4 yrs	0.104	.966
Order 2	27.2	29.4	+2.2 yrs	+1.0 yrs	0.094	.982
Order 3	28.6	30.6	+2.0 yrs	+0.8 yrs	0.083	.984
Order 4	29.8	31.7	+1.9 yrs	+0.8 yrs	0.080	.978
Order 5+	32.0	33.5	+1.5 yrs	+0.5 yrs	0.063	.962
All Births	**26.6**	**29.0**	**+2.4 yrs**	**+1.6 yrs***	**0.104**	**.979**

*Note. HFD = Human Fertility Database via Our World in Data (primary data source, full period 1992–2014). †NCHS = independent corroboration from Mathews & Hamilton (2016), NCHS Data Brief No. 232 (CDC National Vital Statistics, 2000–2014). Figure A.2 uses HFD values restricted to the same 2000–2014 window for a direct apples-to-apples comparison. *All Births NCHS value reflects the 2000–2014 sub-period only. All OLS models $p < .001$, $n = 12$ birth cohort years. The HFD values are systematically approximately 0.2–0.5 years larger than NCHS values across all birth orders. This consistent offset reflects two documented methodological differences between the two data systems. First, birth order definition: the HFD uses biological birth order, counting all prior pregnancies including fetal losses, whereas NCHS uses live-birth order, counting only prior live births. A mother whose first pregnancy ended in fetal loss would be classified as Order 2 in the HFD but Order 1 in NCHS, systematically shifting higher-order HFD births toward older mothers and producing higher mean ages at higher parities. Second, treatment of records with unknown birth order differs between the systems, each applying its own redistribution procedures. These are expected and documented features of the respective methodologies, not data quality concerns. The rank ordering of change magnitudes across birth orders is identical in both systems, confirming the demographic pattern is robust across independent data sources regardless of these definitional differences.*

Three findings from Table A.1 and the accompanying figures validate the use of the aggregate All Births metric in the primary analysis. First, all birth orders show consistent upward trends across the full 1992–2014 period with $R^2 \geq .962$ and $p < .001$ in every series, confirming the demographic shift is monotonic and statistically robust within each parity group independently.

Second, the real HFD data reveals meaningful and interpretable differentiation across birth orders. Order 1 shows the largest increase (+2.4 years, slope = 0.104) and Order 5+ the smallest (+1.5 years, slope = 0.063), with intermediate orders falling in between. This gradient directly reflects the well-documented societal drivers of delayed childbearing: first-birth postponement is the primary engine, consistent with women spending longer in education and the workforce before starting families, while higher-order births are already concentrated among older mothers and have less room to shift further. Crucially, all orders rise in the same direction across the entire study period, confirming the aggregate All Births metric captures a genuine population-wide trend rather than a compositional artifact driven by any single parity group.

Third, the HFD birth-order pattern is independently corroborated by official U.S. vital statistics using the same 2000–2014 time window. Mathews & Hamilton (2016) [7], reporting CDC National Vital Statistics data, found first births showing the greatest postponement (+1.4 years), followed by second (+1.0 years), third- and fourth-order (+0.8 years), and fifth-and-higher-order births (+0.5 years). Figure A.2 displays HFD and NCHS values side by side over the identical 2000–2014 period. The HFD values are consistently 0.2–0.5 years larger than the NCHS figures. This systematic offset has a straightforward methodological explanation: the HFD uses biological birth order, counting all prior pregnancies including fetal losses, whereas NCHS uses live-birth order, counting only prior live births. A mother whose first pregnancy ended in fetal

loss would be classified as Order 2 in the HFD but Order 1 in NCHS, shifting higher-order HFD categories toward older mothers and producing systematically higher mean ages. Differences in how each system redistributes records with unknown birth order contribute additionally. These are documented features of the respective methodologies, not data quality concerns. Critically, the rank ordering across birth orders is identical in both sources — Order 1 rises most, Order 5+ rises least — and all orders rise in both systems. The convergence of two independent data systems on the same directional pattern, with a fully explicable and consistent offset, provides strong corroboration that the rising maternal age trend documented in this study reflects genuine demographic change.

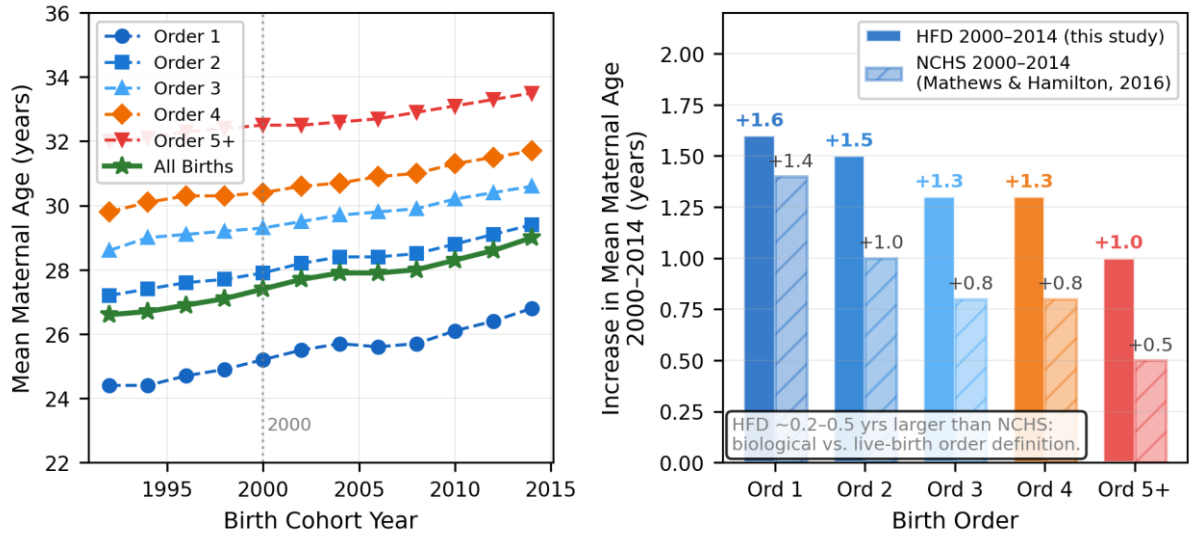

Figure A.1 (left panel). Mean maternal age by birth order, United States 1992–2014 (Human Fertility Database via Our World in Data). All series show monotonic upward trends (slopes 0.063–0.104 yr/calendar yr, $R^2 \geq .962$, $p < .001$). The All Births composite (bold solid line) is the primary exposure variable. Dotted vertical line marks 2000 (start of NCHS comparison period). Figure A.2 (right panel). Apples-to-apples comparison of maternal age increase by birth order, 2000–2014: HFD (solid bars, this study) vs CDC National Vital Statistics (hatched bars; Mathews & Hamilton, 2016, NCHS Data Brief No. 232). Identical rank ordering in both sources. Systematic ~0.2–0.5 yr HFD offset reflects biological vs live-birth order definition — a documented methodological difference.

$\geq .962$, $p < .001$ for all orders). The All Births composite (bold solid line) is the primary exposure variable used in the main analysis. The vertical dotted line marks 2000, the start of the NCHS comparison period. Figure A.2.

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