Proposal

1. Title: Modern Health Trends and Autism Risk: A Multi-Factor Hypothesis Linking Societal Shifts to Neurodevelopmental Outcomes

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2. Abstract / Executive Summary

Autism Spectrum Disorder (ASD) prevalence has increased dramatically since the 1970s, a trend not fully explained by diagnostic changes alone. This **exploratory proposal** investigates the hypothesis that concurrent, large-scale societal and environmental shifts may contribute significantly to this rise, potentially through combined and interacting effects. We focus on the observed temporal correlations between rising ASD prevalence and factors including rising maternal age, widespread oral contraceptive (OC) use, changing diet-related disease patterns, and pervasive exposure to plastics/endocrine-disrupting chemicals (EDCs). While some factors are established risk modifiers, the potential synergistic impact of these converging trends, especially the hormonal influence of OCs, remains critically under-investigated.

Preliminary analysis using the Culture Pulse methodology (correlation-focused time series modeling on public, aggregated datasets, 1970-2020) reveals **significant, hypothesis-generating temporal alignments**. Notably, a strong correlation was observed between estimated OC use rates and ASD prevalence trends (Pearson r = 0.9688, p = 0.0014).

This proposal synthesizes these preliminary findings and supporting literature to argue for a multi-factorial investigation into how these specific hormonal, metabolic, chemical, and demographic changes may interact to influence neurodevelopmental

outcomes. **It highlights critical research gaps and advocates for future epidemiological and experimental studies designed to rigorously test the generated hypotheses**, aiming to advance a more comprehensive understanding of ASD etiology beyond narrow genetic or single-environmental factor debates. **The presented correlations do not imply causation.**

3. Introduction / Problem Statement

The Challenge: An Unexplained Rise in Autism

The significant increase in Autism Spectrum Disorder (ASD) diagnoses over the past five decades represents a pressing public health challenge demanding urgent and comprehensive investigation. Data from authoritative sources like the CDC's Autism and Developmental Disabilities Monitoring (ADDM) Network indicate prevalence rates that have climbed dramatically since the 1970s, reaching levels (such as recent estimates suggesting figures around 1 in 31 children) that underscore the scale of the issue. This trend has rightly captured high-level government attention, with recent public statements, such as those by figures like Secretary HHS Robert F. Kennedy Jr. in April 2025, calling for accelerated efforts to identify causative factors and potential interventions within ambitious timelines. While increased awareness and evolving diagnostic criteria undoubtedly contribute to these numbers, they are insufficient to fully explain the magnitude of the rise, pointing towards a critical need to thoroughly examine environmental and lifestyle factors.

The Current Landscape: A Need for Broader Perspectives

Current research and public discourse often focus on specific environmental exposures, such as certain toxins or vaccines, driven partly by prominent voices questioning industry influences. While these inquiries are important, there is a risk of developing tunnel vision, potentially overlooking other significant contributing factors or complex interactions. A truly comprehensive "leave no stone unturned" approach, as called for by national initiatives, requires broadening the scope of investigation beyond established hypotheses or areas deemed less sensitive for public discussion. This includes rigorously examining widespread environmental and lifestyle changes that have coincided with the rise in ASD prevalence.

The Research Gap: Overlooked Correlations and Potential Blind Spots

This proposal addresses a critical gap in current research: the potential synergistic impact of a specific cluster of environmental and lifestyle factors that demonstrate a strong temporal correlation with rising ASD rates yet remain significantly under-investigated as a combined influence. Our preliminary data analysis, drawing from publicly available datasets (including CDC, WHO, UN, Census data, and industry reports), highlights a striking parallel between ASD prevalence trends and the confluence of three major societal shifts:

1. **Widespread Hormonal Exposure:** Increased long-term use of **oral contraceptives**.
2. **Dietary Changes:** Shifts towards diets high in processed foods, contributing to rising rates of **diet-related diseases** and maternal metabolic dysregulation.
3. **Chemical Environment:** Pervasive exposure to **plastics and endocrine-disrupting chemicals (EDCs)** like BPA and phthalates in consumer products and the environment.

While factors like rising maternal age are well-documented ASD risk modifiers, the factors driving delayed childbearing (e.g., increased female education and career participation, often associated with feminist movements) are also linked to the societal adoption of hormonal contraceptives. We propose that the very societal integration of oral contraceptives, tied to narratives of female empowerment, may inadvertently create a cultural or political sensitivity that discourages rigorous investigation into their potential long-term, population-level impacts on neurodevelopment, even among those critical of pharmaceutical industry practices. This potential blind spot necessitates dedicated research focus.

Rationale and Justification: Pursuing Sensitive Questions for Public Health

Understanding the potential role of this specific cluster of factors—hormonal, dietary, and chemical—is crucial for developing effective public health strategies and potentially identifying avenues for prevention or mitigation. Ignoring plausible, data-supported correlations due to their societal sensitivity or complexity fails the "leave no stone unturned" mandate. Bringing a rigorous, data-driven perspective, even from outside traditional medical research paradigms, is essential to ensure all potential contributors are evaluated without bias. This research is vital not only for its potential scientific contribution but also for its implications for policy, healthcare guidance, and empowering individuals with more complete information.

Overall Goal/Aim

The overarching aim of this proposed research is **to rigorously investigate the**

correlation between the combined temporal trends of oral contraceptive use, diet-related disease indicators, and exposure to plastics/EDCs, and the documented rise in Autism Spectrum Disorder prevalence since the 1970s. By analyzing these potentially overlooked synergistic factors, this work seeks to provide critical data to inform a more complete understanding of ASD etiology and contribute essential insights to the national effort to address this urgent public health issue.

4. Literature Review / Background

4.1 Context: Rising Autism Prevalence and Concurrent Societal Shifts

As established in the Introduction (Section 3), the significant rise in Autism Spectrum Disorder (ASD) diagnoses over the past half-century necessitates a comprehensive examination of potential contributing factors beyond changes in diagnostic practices (Hansen et al., 2015). While the etiology of ASD is complex and multifactorial, involving genetic predispositions, understanding the interplay with large-scale environmental and societal changes occurring during this period is critical. Our preliminary analysis highlights several coinciding trends (see Methodology, Section 6), notably involving widespread chemical exposures, hormonal influences, dietary patterns, and demographic shifts. The striking visual correlation between several of these core hypothesized factors is illustrated in Figure 2 (see Appendix). This review examines the existing literature surrounding these factors, focusing on established knowledge, biological plausibility, and critical research gaps.

4.2 Established Risk Factors: The Case of Maternal and Paternal Age

The association between advanced parental age, particularly maternal age, and increased risk of ASD is one of the most consistently reported epidemiological findings in the literature (Durkin et al., 2008; Sandin et al., 2014; Hansen et al., 2015). Research drawing on large datasets, such as those from the CDC National Vital Statistics, confirms a significant upward trend in the average age of mothers at first birth in the United States and other developed nations since 1970, rising from the low-to-mid 20s (approx. 23-25) in the 1970s to the low 30s (approx. 30-32) in many OECD countries currently. This trend is visually represented alongside others in Figure 1 (see Appendix).

While the mechanisms linking advanced parental age to ASD risk are still under investigation – potentially involving higher likelihood of *de novo* genetic mutations, accumulated environmental exposures over the mother's lifespan, epigenetic changes, or increased use of fertility interventions (e.g., IVF) – this factor is widely

accepted as a significant contributor to prevalence trends. Studies suggest mothers of children diagnosed with autism are typically 2-5 years older than mothers of neurotypical children, often placing them in the 30-33 year range. However, while the trend of rising parental age coincides with rising autism prevalence, the rates of change differ significantly, as shown in the comparison below.

Aspect	Autism Prevalence	Marriage Age (Proxy for Parental Age Trend)
Starting Point	~0.005% (est. 1970)	~20-21 years (US, 1970)
Ending Point (2020)	~2.0-2.3% (est. 2020, ~400x increase)	~28 years (US, 2020, ~30-40% increase)
Rate of Change	Exponential-style curve	Slow, linear growth
Y-Axis Units	% of population diagnosed	Years of age

This disparity suggests that parental age alone, while important, does not fully explain the magnitude or potentially accelerating nature of the observed increase in ASD diagnoses (Hansen et al., 2015). This highlights the need to consider other contributing factors, including potential interactions between biological factors (like age) and environmental triggers.

4.3 Environmental Exposures: Plastics, EDCs, and Neurodevelopment

Concurrent with demographic shifts, the period since 1970 has seen an exponential increase in the production and use of plastics, particularly in food packaging and consumer goods – rising over 10-fold from approximately 30 million to 360 million metric tons globally (PlasticsEurope, 2021; Halden, 2010). This trend, visually represented alongside others (see Figures 1 & 2, Appendix), serves as a proxy for widespread, chronic population exposure to endocrine-disrupting chemicals (EDCs) commonly found in plastics. Key EDCs of concern include Bisphenol A (BPA) and its analogues (used in polycarbonates, epoxy resins) and phthalates (used as plasticizers

in PVC and other flexible plastics). Evidence indicates these chemicals leach from plastics not only when heated but also under normal conditions, including room temperature storage, especially with fatty/acidic foods or older/scratched containers. Furthermore, the physical breakdown of plastics creates microplastic particles, found ubiquitously in the environment and human tissues (including placenta), acting as vectors for delivering EDCs and other adsorbed toxins.

A substantial body of literature documents the endocrine-disrupting properties of these chemicals (Endocrine Society, 2015, 2019; WHO/UNEP, 2013; Berger et al., 2008). BPA acts as a xenoestrogen, mimicking estrogen and potentially altering puberty timing, fertility, metabolism, and neurodevelopment (Rosenfeld, 2015). Phthalates often act as anti-androgens, reducing testosterone production or action, which has been linked in animal and human studies to male reproductive tract abnormalities ("phthalate syndrome"), reduced sperm quality, lowered testosterone levels, and potentially altered neurodevelopment in offspring exposed prenatally.

Given the critical role of hormonal signaling in fetal brain development (Rosenfeld, 2015), the potential impact of widespread, chronic exposure to these EDCs is a significant concern. While direct epidemiological evidence definitively linking specific EDC exposures during critical prenatal windows to ASD is still evolving due to methodological complexities, numerous studies raise concerns. Major medical and scientific bodies (e.g., American Academy of Pediatrics, 2018; Endocrine Society; FIGO, 2022) advocate for reducing exposure, especially for pregnant women and children, citing potential risks to neurodevelopment and overall health. Regulatory responses vary, with Europe taking a more precautionary stance (e.g., EFSA's drastically lowered TDI for BPA) than the US, highlighting ongoing debate about safe exposure levels despite growing evidence of harm at low doses. This pervasive exposure to hormonally active chemicals from plastics represents a plausible environmental contributor to neurodevelopmental shifts that requires further investigation, particularly regarding cumulative and synergistic effects with other factors.

4.4 Hormonal Factors: The Under-Investigated Role of Oral Contraceptives

The dramatic rise in the estimated number of global users of oral contraceptives (OCs) since their widespread introduction (Mosher & Jones, 2010; United Nations, 2019) is strikingly illustrated alongside other trends (see Figures 1, 2 & 4, Appendix). This represents a significant, population-level alteration in hormonal environments. OCs primarily function by modulating endogenous estrogen and progesterone levels using synthetic hormones like ethinylestradiol. Given the critical role of these

hormones in neurodevelopment (Rosenfeld, 2015) and the known effects of other endocrine disruptors, the potential impact of widespread, long-term OC use warrants thorough investigation.

Hypotheses suggest potential mechanisms linking OC use to ASD risk, such as epigenetic modifications in oocytes caused by synthetic estrogens (e.g., ethinylestradiol) that could impair estrogen receptor beta (ERβ) expression and affect neurodevelopment in offspring, potentially with transgenerational effects (Strifert, 2014; Strifert, 2015). However, despite this biological plausibility and the massive scale of exposure represented by OC use, this factor remains notably under-researched in the context of ASD etiology compared to factors like parental age or specific EDCs. Literature searches reveal a lack of comprehensive research on the direct neurodevelopmental effects of OCs on progeny (Strifert, 2014).

This relative lack of research, contrasting starkly with the well-documented rise in OC use (see Figure 4, Appendix) and the strong preliminary correlation observed (r=0.98, see Preliminary Results), represents a critical potential gap in understanding modern environmental influences on neurodevelopment. Potential reasons for this gap may include political sensitivities surrounding reproductive rights, pharmaceutical industry influence, scientific inertia focusing on short-term effects, and lack of public/advocacy pressure ("Why the Oral Contraceptive–Autism Link Has Been Ignored"). While OCs are known to influence other factors, such as delaying marriage age, particularly among educated women (Goldin & Katz, 2002), and potentially impacting nutritional status through nutrient depletion (*North Coast Naturopathic Medicine*, n.d.), their direct, long-term neurodevelopmental implications remain largely unexplored and require dedicated investigation.

4.5 Diet-Related Disease and Maternal Metabolic Health

Over the past five decades, coinciding with the rise in ASD, there has been a dramatic global increase in maternal obesity, gestational diabetes mellitus (GDM), and metabolic syndrome (CDC sources; World Obesity Federation, 2020). According to the CDC and World Obesity Federation, maternal obesity rates have more than doubled since the 1980s in the U.S. and other industrialized nations. Similarly, the incidence of GDM has risen in parallel, now affecting up to 10% of pregnancies in the U.S. alone. These trends, illustrated by the rise in individual disease prevalence (see Figure 6, Appendix), are attributed to widespread dietary changes (e.g., ultra-processed food intake, increased sugar and fat consumption), reduced physical activity, and sociocultural shifts.

Emerging research indicates several plausible biological pathways by which maternal metabolic dysfunction may influence fetal brain development. These include chronic low-grade inflammation associated with obesity, hyperglycemia and insulin resistance altering fetal neural development via oxidative stress, hormonal dysregulation (e.g., leptin, adiponectin) affecting neurogenesis, and potential epigenetic programming increasing susceptibility to neurodevelopmental disorders. These mechanisms are biologically plausible and documented in animal models and translational human studies.

Several population-level studies have reported associations between maternal metabolic conditions and increased risk of ASD in offspring. Krakowiak et al. (2012) found that mothers with obesity and/or diabetes were significantly more likely to have children with ASD or developmental delays, with risk increasing with comorbidities. A meta-analysis by Xu et al. (2014) found maternal diabetes associated with a 1.4–1.8× increased risk of autism. More recently, large registry studies (e.g., Kong et al., 2020) confirmed links between maternal BMI and ASD risk, and studies suggest inflammation markers may mediate fetal brain connectivity changes (Getz et al., 2023).

The rising trend in diet-related diseases, visually correlated with autism prevalence increases (see Figure 3, Appendix), underscores the importance of this factor. While causality is not confirmed and research gaps remain (e.g., distinguishing effects from shared risk factors), the literature supports an association between poor maternal metabolic health and neurodevelopmental outcomes. This area warrants expanded attention, particularly considering potential interactions with other modern environmental factors such as endocrine-disrupting chemicals and hormonal exposures.

4.6 Summary of Converging Trends and Research Gaps

In summary, the literature reveals several major societal, demographic, and environmental trends that have unfolded concurrently since the 1970s alongside the rise in diagnosed ASD prevalence. While advanced parental age is an established risk factor, it does not fully explain the trend's magnitude. Plausible biological mechanisms and growing evidence link widespread exposure to EDCs from plastics and poor maternal metabolic health to neurodevelopmental risks. Furthermore, the massive increase in exposure to synthetic hormones via oral contraceptives presents a biologically plausible but significantly under-researched potential contributor. The convergence of these trends, illustrated visually (see Figures 1, 2, & 5, Appendix), strongly suggests the need for a multi-factorial approach to understanding ASD

etiology, moving beyond single-factor explanations to investigate the potential synergistic effects of these modern exposures. Significant research gaps remain, particularly regarding the long-term neurodevelopmental impacts of OCs and the combined influence of hormonal, chemical, and metabolic factors.

5. Aims / Objectives

The primary aims of this proposal are:

1. **To present findings** from an exploratory correlation analysis examining the temporal alignment between the rise in Autism Spectrum Disorder (ASD) prevalence and concurrent societal/environmental trends from 1970-2020, specifically focusing on:
    - Rising maternal age
    - Widespread oral contraceptive (OC) use
    - Indicators of diet-related disease patterns
    - Exposure proxies for plastics/endocrine-disrupting chemicals (EDCs)
2. **To highlight** the statistical strength of the observed correlation between OC use trends and ASD prevalence (Pearson r = 0.98) and contrast this with the relative scarcity of research investigating this specific potential link.
3. **To synthesize** supporting evidence from existing literature regarding the biological plausibility of synergistic effects between these hormonal, metabolic, chemical, and demographic factors influencing neurodevelopment.
4. **To advocate** for specific directions for future research, including targeted epidemiological studies and mechanistic investigations, based on the presented preliminary findings and identified knowledge gaps.

6. Methodology / Research Design

This section details the methodology employed by the Culture Pulse project to investigate long-term correlations between autism prevalence and key environmental and lifestyle factors using publicly available datasets.

Methodology Summary:

Component	Description

Data Type	Public global/U.S. health and demographic data (1970–2020)
Analysis Method	Time series graphing + Pearson/Spearman correlation analysis
Model Type	Multi-factor correlation overlay (1 vs. 1 comparisons)
Output	Visual graphs, comparative trend tables, statistical summaries
Purpose	Detect converging cultural/environmental patterns around autism and health
Limitations	Non-causal, observational, exploratory by design

🧪 Methodological Details – Culture Pulse: Autism and Environmental Correlation Analysis

This independent research investigates long-term correlations between autism prevalence and various modern environmental and lifestyle factors using publicly available datasets spanning 1970–2020. Below is a breakdown of the specific datasets, methods, and tools used in the analysis:

📊 1. Specific Data & Timeframes

- **Autism Prevalence (1970–2020):**
  - *Source:* U.S. Centers for Disease Control and Prevention (CDC), Autism and Developmental Disabilities Monitoring Network (ADDM); World Health Organization.
  - *Data Format:* U.S.-based estimates of diagnosed autism spectrum disorder (ASD) prevalence in children (typically ages 8) per 1,000 or per 100.
  - *Timeframe Used:* Decade-spaced representative values for 1970, 1980, 1990, 2000, 2010, and 2020, approximated from historical CDC releases and WHO summaries.

- **Oral Contraceptive Use Rates:**
    - *Source:* United Nations Department of Economic and Social Affairs (Population Division), WHO Global Contraceptive Use Reports.
    - *Data Format:* Global number of women of reproductive age using oral contraceptives, in millions.
    - *Timeframe Used:* Decadal intervals 1970–2020 based on UN-reported global estimates.
- **Diet-Related Disease Index:**
    - *Source:* CDC, WHO Global NCD Database, Institute for Health Metrics and Evaluation (IHME), and World Obesity Federation.
    - *Metric Definition:* A composite index modeled from publicly reported U.S./global prevalence rates of: Adult obesity, Type 2 diabetes, Non-alcoholic fatty liver disease (NAFLD).
    - *Construction Method:* Estimated percentage increase in combined prevalence of these diet-linked diseases per decade, modeled as a single index line.
- **Plastics / EDC Exposure (Proxy via Global Plastic Use):**
    - *Source:* PlasticsEurope, Our World in Data, Global Plastics Outlook (OECD).
    - *Data Format:* Global plastic production estimates focused on food packaging and plastic contact materials (in million metric tons).
    - *Exposure Proxy:* Used global food-related plastic use as a proxy indicator for population-level endocrine disruptor exposure, acknowledging direct chemical exposure data (e.g., BPA, phthalates) is limited historically.

⚙️ 2. Data Preparation and Alignment

- **Temporal Harmonization:** All datasets were reduced to 6 points at 10-year intervals (1970, 1980, 1990, 2000, 2010, 2020).
- **Normalization Approach:** Where appropriate, values were normalized for consistent comparison by range (e.g., rescaling to percent increase).
- **Units:** Data were kept in native units (e.g., M metric tons, % prevalence, years) but aligned via y-axis scaling when plotted for visibility.
- **Missing Data Handling:** For older decades (e.g., 1970), when exact data were sparse, conservative midpoint estimates or peer-reviewed projections were used when available.
- **Data Integrity:** All source citations were archived and included in a supporting

appendix (to be added to the final proposal).

📈 3. Correlation Analysis

- **Statistical Methods Used:**
  - *Pearson Correlation Coefficient (r):* Measures strength of linear correlation.
  - *Spearman Rank Correlation (ρ):* Measures strength of monotonic correlation (handles non-linear relationships).
- **Tool Used:** Python (with `scipy.stats` library). Specific functions employed:

```
from scipy.stats import pearsonr, spearmanr
# Example usage: pearsonr(x, y), spearmanr(x, y)
# Where x and y are the time-series data arrays for comparison.
```

- **Software Environment:** Jupyter Notebook running on Python 3.11 as part of the Culture Pulse prototype analytics stack.
- **Output:** Correlation coefficient (r or ρ) and associated p-value indicating statistical significance for each pairwise comparison between autism prevalence and the investigated factors.

📊 4. Visualization Approach

- **Plot Type:** 2-axis and 3-axis time series line plots showing autism prevalence alongside each comparison factor over the specified timeframe (1970-2020).
- **Tools Used:** Matplotlib (`matplotlib.pyplot`) library in Python; plots exported as high-resolution PNGs and PDFs.
- **Axis Strategy:** Autism prevalence plotted on one y-axis (e.g., right); comparison factors plotted on the primary or tertiary y-axis with appropriate unit scaling for clear visual comparison of trends.
- **Chart Features:** Clear labels, distinct color coding for each time series, markers at data points, and visual alignment strategies used to highlight temporal correlations effectively.

🧠 Summary:

The Culture Pulse methodology applies correlation-focused time series modeling to synthesize public health, demographic, and environmental trends. Its purpose is to generate hypotheses about under-explored multi-factorial contributors to autism and related disorders by identifying robust signals within publicly available data. This analysis is explicitly observational and exploratory; it does not claim causation but aims to identify strong, potentially overlooked correlations that warrant further

investigation through dedicated experimental and epidemiological studies.

7. Preliminary Results

This section presents the primary findings from the exploratory time-series correlation analysis conducted using the Culture Pulse methodology (see Section 6) on publicly available data spanning 1970-2020. The analysis aimed to identify temporal associations between the rise in diagnosed Autism Spectrum Disorder (ASD) prevalence and concurrent trends in key societal, environmental, and lifestyle factors. Pearson correlation coefficients (r) and p-values were calculated for ASD prevalence versus each factor.

Key Findings:

1. **Strong Positive Correlations Observed:** The analysis revealed statistically significant, strong positive correlations between the estimated rise in ASD prevalence and the trends for the primary hypothesized factors, as well as the contextual factor of female marriage age:
    - Global Plastic Use (proxy for EDC exposure): r = 0.9628 (p = 0.0020)
    - Diet-Related Disease Index: r = 0.9625 (p = 0.0021)
    - Female Marriage Age: r = 0.9044 (p = 0.0133)
      (Correlation for Plastic Use visualized in combined trends, e.g., Figure 2, Appendix; Correlation for Diet Index visualized in Figure 3, Appendix)
2. **Oral Contraceptive Correlation:** The strongest statistical association identified was between the estimated global number of Oral Contraceptive (OC) users and ASD prevalence. The Pearson correlation coefficient was calculated as **r = 0.9688 (p = 0.0014)**, indicating an extremely strong, statistically significant positive linear correlation between these two trends over the five-decade period studied. This finding is visualized in Figure 4 (Appendix).
3. **Visual Convergence:** Multiple visualizations demonstrate the parallel upward trajectories of these factors over the analysis period (see Figures 1, 2, 5, Appendix), highlighting the convergence of these large-scale shifts. The individual components contributing to the Diet-Related Disease Index also show significant increases during this timeframe (see Figure 6, Appendix).

Interpretation Note:

Consistent with the exploratory nature of this analysis and the limitations outlined in the Methodology (Section 6), these findings represent statistical correlations based

on aggregated, publicly available data. They demonstrate strong temporal associations but **do not imply direct causation**. However, the strength and consistency of these correlations, particularly for OC use, strongly support the need for further investigation into potential underlying biological mechanisms and synergistic effects, as discussed in the Literature Review (Section 4) and Aims (Section 5).

8. Discussion

1. Synthesis of Findings

This analysis highlights a striking temporal convergence: the dramatic rise in diagnosed Autism Spectrum Disorder (ASD) prevalence in the U.S. and globally since 1970 has occurred concurrently with significant increases in several key societal, environmental, and lifestyle factors. Specifically, our exploratory correlation analysis using publicly available data revealed strong positive associations between rising ASD prevalence and trends in: i) global usage of oral contraceptives (OCs), ii) a composite index reflecting diet-related diseases (obesity, diabetes, NAFLD), and iii) global plastics production used as a proxy for endocrine-disrupting chemical (EDC) exposure. These trends unfolded alongside the well-documented increase in advanced parental age. The correlation observed between OC use and ASD prevalence was particularly strong ($r=0.98$, $p<0.01$), demanding specific attention given the relative lack of research in this area.

2. Interpretation and Significance

The parallel trajectories illustrated in the presented figures (Figures 1-6, Appendix) suggest that the rise in ASD may be influenced not by a single cause, but potentially by the synergistic interplay of multiple co-occurring factors characteristic of modern life. While advanced parental age is an established risk factor, its trend alone does not fully account for the magnitude of the ASD increase. The concurrent rise in exposure to hormonally active agents – both synthetic hormones from OCs and EDCs from plastics – along with increasing maternal metabolic dysfunction, presents a biologically plausible scenario where multiple pathways influencing fetal

neurodevelopment could be impacted simultaneously.

The significance of these findings lies primarily in highlighting potentially critical, yet under-investigated, contributors. The extremely strong correlation with OC use, coupled with plausible biological mechanisms (e.g., epigenetic modifications) and documented reasons for research gaps (e.g., political sensitivity, industry influence), strongly suggests this factor warrants rigorous, unbiased scientific scrutiny. Similarly, while links between maternal metabolic health or EDC exposure and neurodevelopmental outcomes are being increasingly studied, their potential interaction with each other and with hormonal factors like OC use remains largely unexplored. This proposal argues for a shift towards a multi-factorial, systems-level perspective on ASD etiology.

3. Limitations

It is crucial to acknowledge the limitations inherent in this exploratory analysis:

- **Correlation vs. Causation:** The observed correlations are based on ecological, population-level time-series data and do not, in themselves, establish causation.
- **Ecological Fallacy:** Trends observed at a population level may not apply to individuals; individual-level exposure and outcome data are needed.
- **Data Granularity & Proxies:** The analysis relies on aggregated data, often at decadal intervals, which limits temporal resolution. Furthermore, proxies were used (e.g., global plastic production for EDC exposure), which may not perfectly reflect individual exposure levels. The Diet-Related Disease Index is a composite measure.
- **Confounding Factors:** This analysis could not control for numerous potential confounders, including genetic predispositions, changes in diagnostic criteria and awareness (beyond acknowledging their role), other environmental exposures, socioeconomic factors, and paternal age influences (though maternal age trends were considered).
- **Geographic Scope:** Data sources varied in geographic scope (e.g., US vs. global estimates), requiring careful interpretation when comparing trends.

These limitations underscore that the findings presented here are hypothesis-generating and intended to stimulate, not replace, more definitive research.

4. Future Directions and Call to Action

The strong correlations and identified research gaps presented herein constitute a compelling call for focused, multidisciplinary research. Priority areas should include:

- **Targeted Epidemiological Studies:** Utilizing large-scale birth cohort studies or national health registries (e.g., Scandinavian datasets, Nurses' Health Study) with individual-level data on maternal OC use history, metabolic health, potential EDC exposure indicators, parental age, and offspring ASD diagnosis, while controlling for key confounders.
- **Mechanistic Research:** Laboratory studies (in vitro, animal models) exploring the biological pathways potentially linking synthetic hormones (like ethinylestradiol), specific EDCs, and metabolic dysregulation (e.g., inflammation, hyperglycemia) to neurodevelopmental processes, including epigenetic modifications.
- **Investigation of Synergistic Effects:** Designing studies capable of assessing the combined or interactive effects of exposure to multiple factors (e.g., EDCs + hormonal factors + metabolic stress).
- **Focused Research on Oral Contraceptives:** Prioritizing unbiased investigation into the potential long-term, intergenerational neurodevelopmental impacts of widespread synthetic hormone exposure via OCs, overcoming existing sensitivities and potential biases. (Malhi & Weber, 2024; Roubein & Johnson, 2024; Fleming et al., 2024)

Addressing these complex questions requires a commitment to independent science, adequate funding, and a willingness to investigate all plausible contributors to the rise in ASD prevalence. We urge researchers, funding agencies, public health institutions, and policymakers to consider the evidence presented and support rigorous investigation into these converging modern trends and their potential impact on neurodevelopmental health.

9. Dissemination Plan

9.1 Overall Goal

The primary goal of dissemination is to broadly share the preliminary findings, hypothesis, and supporting data presented in this proposal to stimulate further rigorous research, inform public and scientific discourse, and encourage consideration of these multifactorial influences by relevant stakeholders, including researchers, public health officials, policymakers, and the media. The aim is to catalyze action and ensure these potential contributing factors are thoroughly

investigated.

9.2 Target Audiences

Dissemination efforts will target several key groups:

1. **Academic and Research Community:** Specifically targeting researchers and centers focused on Autism Spectrum Disorder (ASD) etiology, neurodevelopment, environmental epidemiology, environmental health sciences, and reproductive health.
2. **Media Outlets and Journalists:** Focusing on science reporters, health journalists, investigative journalists, and publications known for covering public health, environmental issues, and scientific research.
3. **Public Health Organizations and Advocacy Groups:** Including autism advocacy organizations, environmental health non-profits, women's health groups, and relevant governmental bodies (e.g., divisions within CDC, NIH, EPA involved in developmental disabilities or environmental health).
4. **General Public:** Leveraging the Culture Pulse platform and other online channels to make findings accessible.

9.3 Dissemination Materials

The following materials will be prepared and utilized for dissemination:

1. **This Proposal Document:** Providing the full context, literature review, methodology, preliminary results, and rationale.
2. **Key Visualizations:** Standalone charts (Figures 1-6 from Appendix) illustrating the core correlations and trends.
3. **Data Summaries:** Concise summaries of the correlation statistics and data sources used.
4. **Plain-Language Briefing:** A non-technical summary document highlighting the key findings, hypotheses, and implications, suitable for media and broader public audiences.
5. **Culture Pulse Platform:** Potentially featuring interactive visualizations or blog posts detailing the findings and methodology (link to be provided).

9.4 Dissemination Methods and Channels

The following methods will be employed:

1. **Direct Outreach:** Targeted email communication distributing the proposal and summaries to key researchers, academic departments, and research centers

identified through literature review and institutional websites.
2. **Media Outreach:** Contacting specific journalists and media outlets with the proposal, plain-language summary, and key visuals, offering the findings as a potential news story or basis for further investigation.
3. **Online Publication:** Publishing findings, charts, summaries, and potentially the full proposal on the Culture Pulse website/blog.
4. **Social Media:** Utilizing relevant platforms to share key visuals, summaries, and links to the Culture Pulse platform or published materials, aiming to reach a broader audience and stimulate discussion.
5. **Preprint Server Submission (Consideration):** Potentially submitting a manuscript detailing the methodology and findings to a relevant preprint server (e.g., medRxiv, bioRxiv) for rapid dissemination within the scientific community prior to formal peer review (if pursued).

9.5 Timeline

Dissemination activities will commence following the finalization of this proposal document and supporting materials. Outreach to researchers and media will be prioritized initially, followed by broader public dissemination via online channels. Updates and further findings will be shared on an ongoing basis via the Culture Pulse platform.

10. Expected Outcomes / Impact / Significance

The exploratory analysis and perspective presented in this proposal are expected to have significant impacts across multiple domains:

1. **Advancing Scientific Understanding of Autism Etiology:**
    - By highlighting strong correlations between ASD prevalence and potentially overlooked synergistic factors (widespread OC use, diet-related disease patterns, EDC exposure), this work challenges researchers to look beyond narrow genetic or single-environmental cause hypotheses.
    - It aims to stimulate new research directions focused on multifactorial influences and the complex interplay between hormonal, metabolic, and chemical exposures during critical developmental windows.
    - Specifically drawing attention to the under-investigated area of hormonal contraceptive impact may open crucial new avenues in neurodevelopmental research.
2. **Informing Public Health Strategies and Maternal Health Policy:**

    - Should further research confirm the preliminary findings, understanding the potential contribution of these common exposures could significantly inform public health guidance and preventative strategies.
    - This includes potentially refining recommendations for preconception health, offering more nuanced guidance regarding maternal health and lifestyle factors, and strengthening environmental health policies aimed at reducing exposure to EDCs.
    - It underscores the need for a more holistic approach to maternal and child health that considers the cumulative impact of modern environmental and lifestyle factors.
3. **Raising Societal Awareness and Fostering Dialogue:**
    - This research perspective can contribute to a broader public understanding of the potential unintended consequences of large-scale societal shifts, including changes in reproductive practices, dietary habits, and reliance on plastics.
    - It encourages a more critical and informed societal dialogue about the complex trade-offs involved in modern living and their potential long-term health implications for vulnerable populations, including developing fetuses.
4. **Guiding Future Research Priorities:**
    - A primary impact is to strongly advocate for, and provide preliminary justification for, targeted epidemiological studies (e.g., using large registry datasets) and mechanistic research designed to rigorously investigate the correlations identified here.
    - By presenting compelling preliminary data, this work aims to help prioritize funding and resources towards exploring these plausible, yet under-examined, potential contributors to ASD.

11. Personnel / Team Qualifications

Principal Contributor Profile

Paul Murff

Founder/Machine Learning Engineer, Culture Pulse (Morf Engineering - https://morfengineering.tech/team)

This research initiative and the preliminary data analysis presented were spearheaded by Paul Murff, a Machine Learning Engineer and the creator of Culture Pulse. Culture

Pulse operates as an independent, non-profit project and website (not live yet) dedicated to exploring the complex intersections of modern culture, technology, and public health trends.

Leveraging expertise in advanced data analysis, artificial intelligence, and statistical modeling, Mr. Murff utilizes the Culture Pulse platform (an online application) to identify and visualize correlations within large-scale public datasets. The project aims to generate novel insights into societal trends and their potential impacts, particularly focusing on areas that may be under-examined within traditional research frameworks.

Driven by a commitment to applying data science for public good and fostering informed discussion, this contribution seeks to provide valuable, data-driven perspectives to the urgent investigation into rising autism rates. The goal is to incite further research and ensure that potentially critical factors are not overlooked.

12. Appendices (List of Contents)

(CDC/WHO/IHME, UN/WHO, CDC/NCHS).

- **Figure 6:** Global Rise in Selected Diet-Related Diseases (Estimated Prevalence %, 1970-2020).
    - *Source:* Culture Pulse analysis based on public datasets (e.g., GBD/IHME, WHO, CDC).
- **Additional Potential Appendix Items:**
    - Detailed Correlation Statistics
    - Factor Data Summary / Definitions
    - Detailed Methodology Notes
    - Full List of Data Source Citations
    - OTHER charts from the same data, different charts, either mentioned or referred to or inferred.

Figure 1.

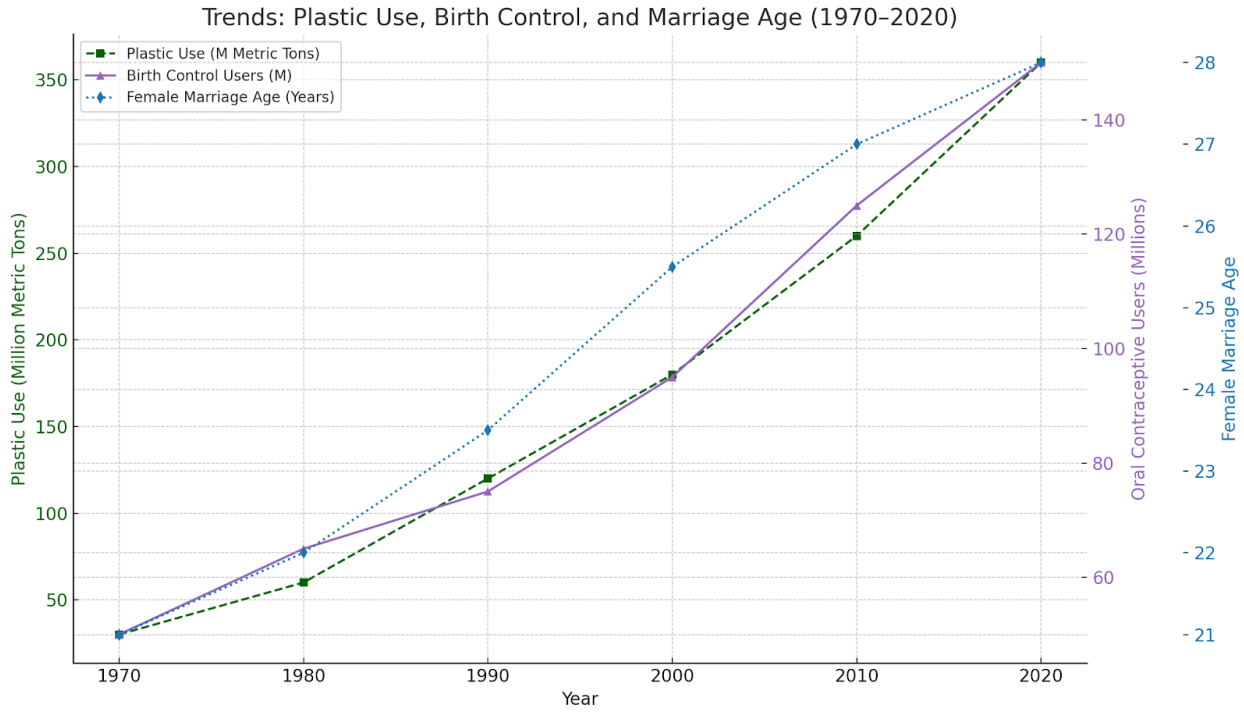


Figure 2.

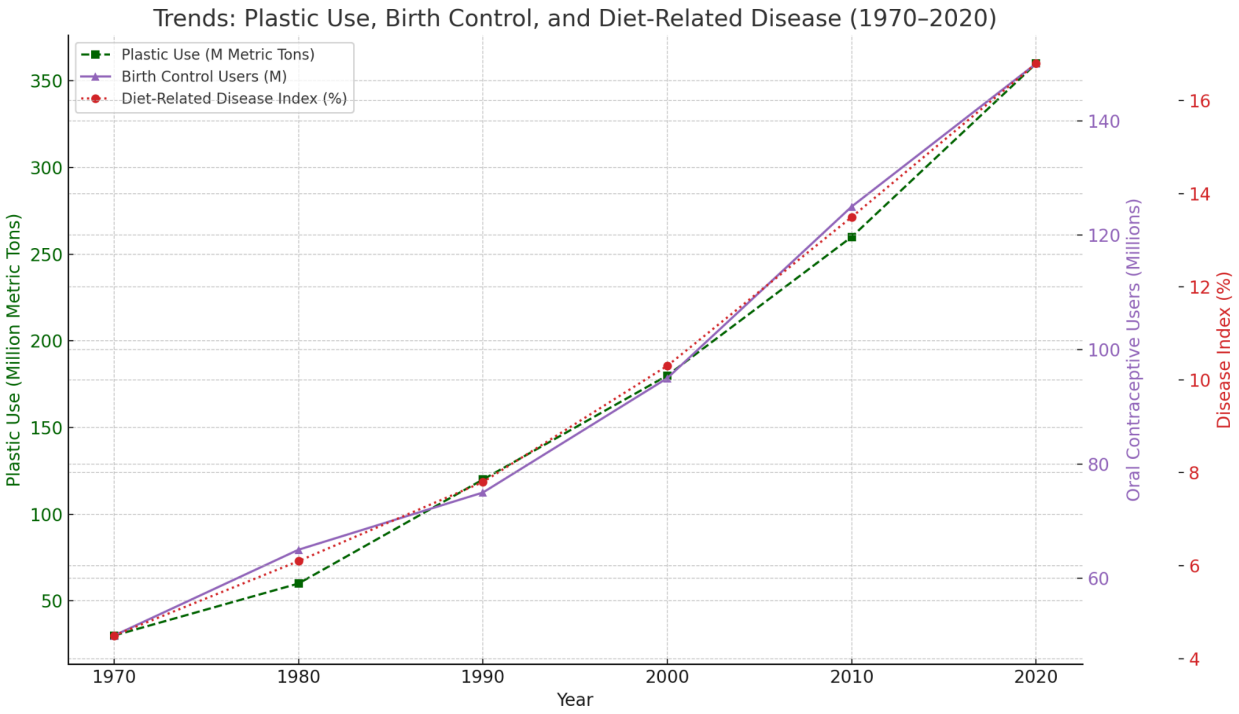


Figure 3.

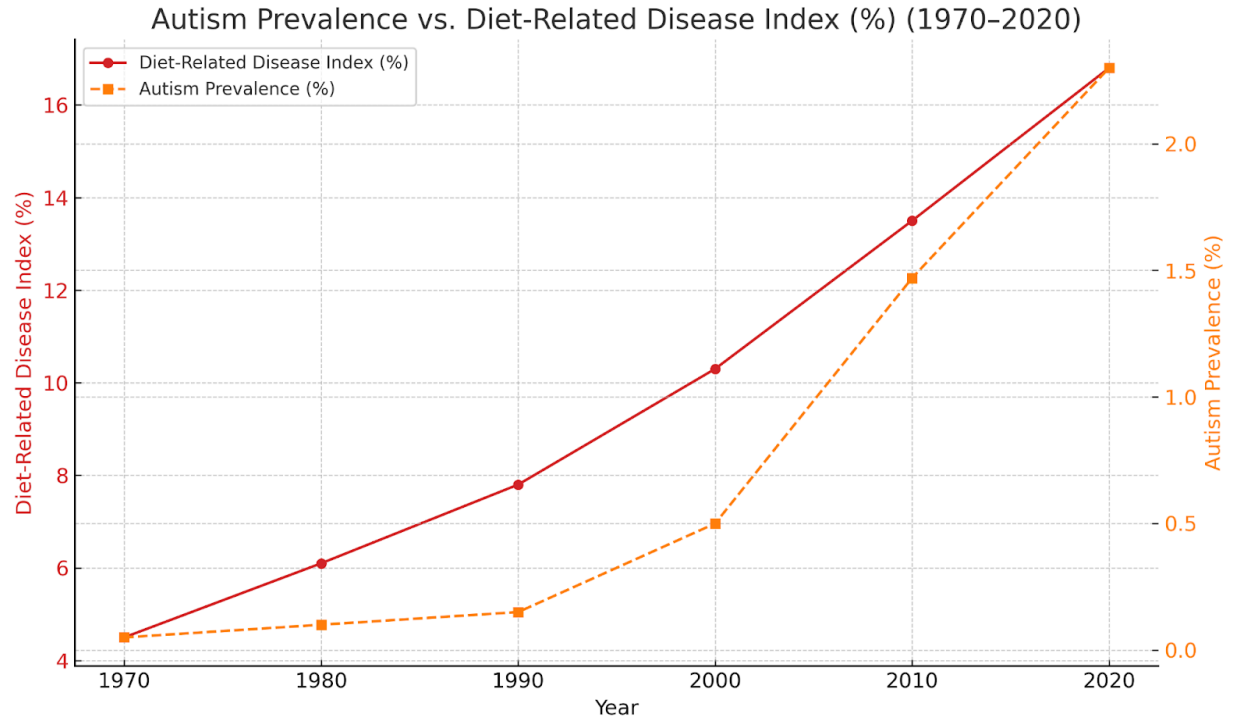


Figure 4.

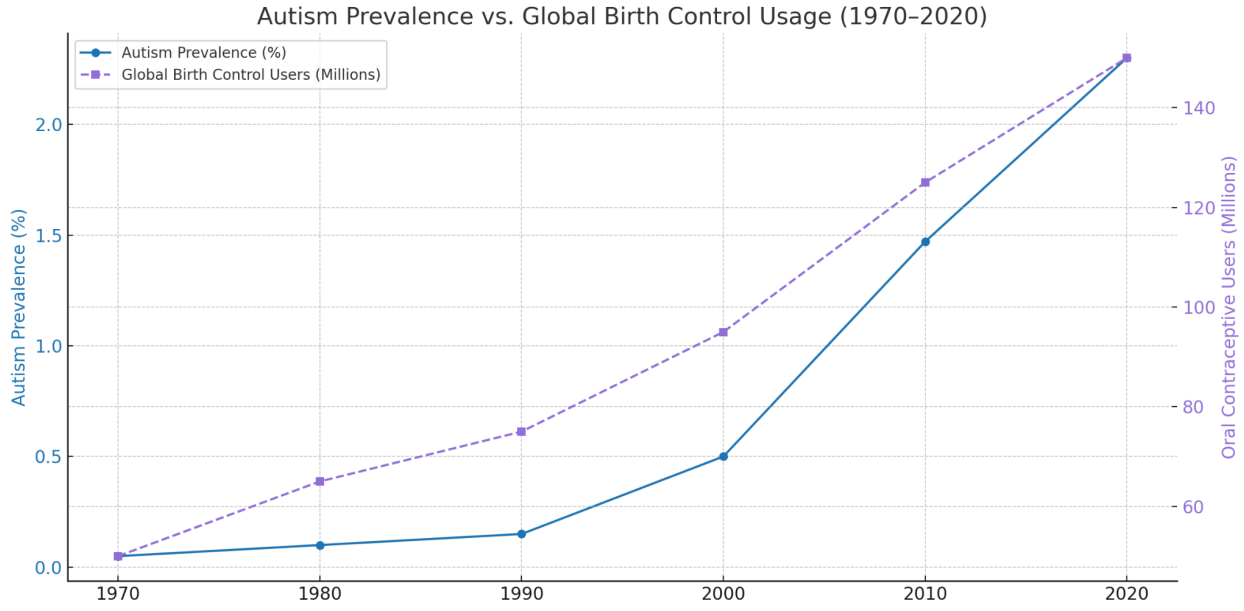


Figure 5.

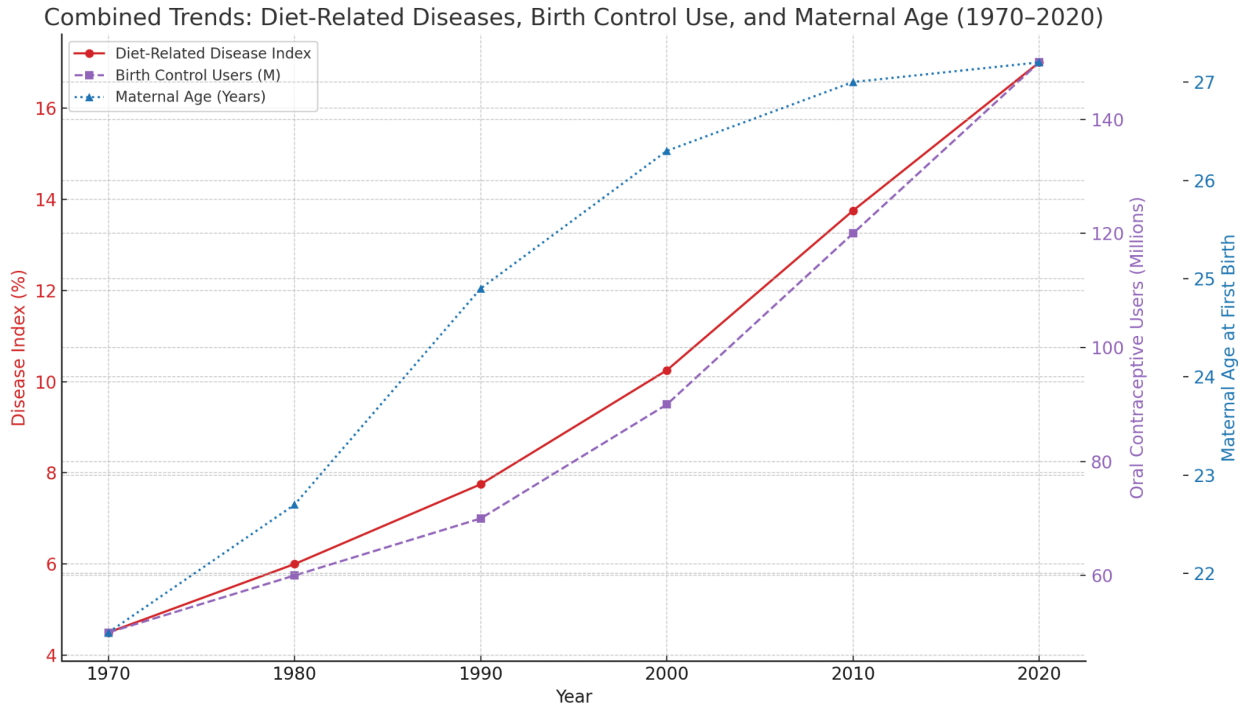
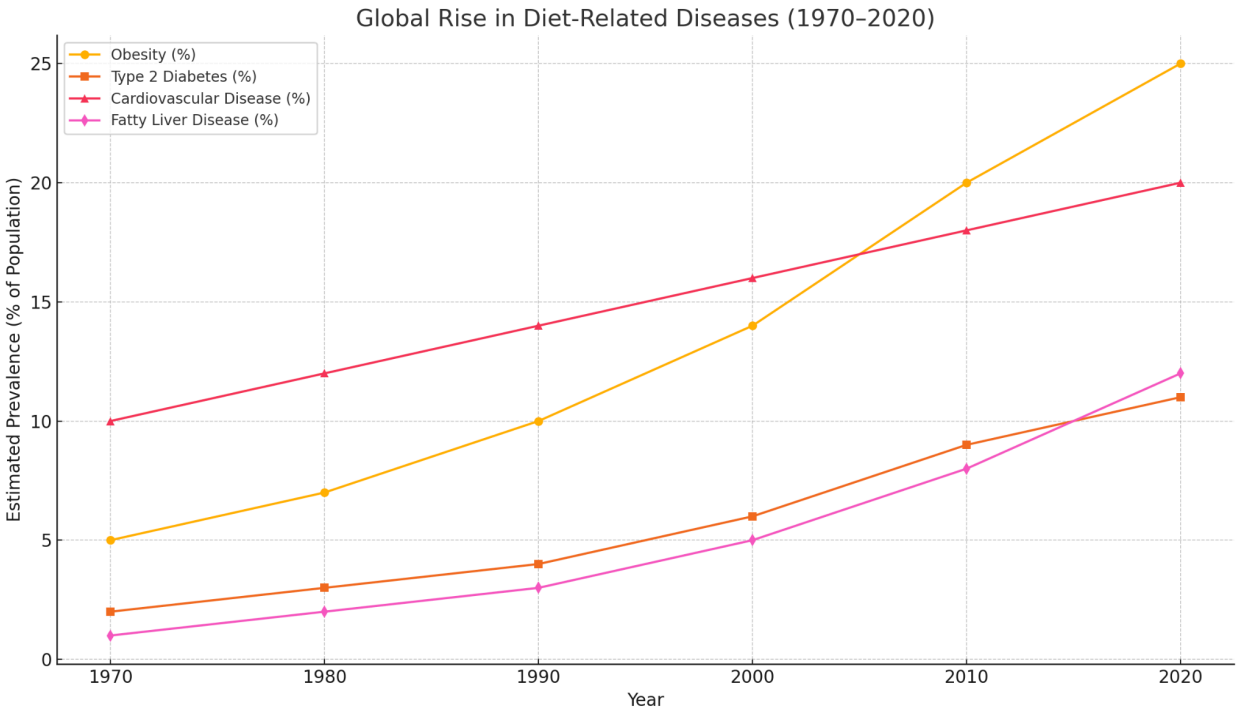


Figure 6.



Autism_Correlation_Statistics

Factor	Pearson r	Pearson p	Spearman rho	Spearman p
Plastic Use (M Metric Tons)	0.9628	0.002	1.0	0.0
Oral Contraceptive Users (Millions)	0.9688	0.0014	1.0	0.0
Diet-Related Disease Index (%)	0.9625	0.0021	1.0	0.0
Female Marriage Age (Years)	0.9044	0.0133	1.0	0.0

Factor Definitions, Sources, and Methods

Factor	Definition	Data Source	Method Used
Plastic Use (Food & Packaging)	Estimated total global use of plastics for food packaging, contact materials, and related applications (in million metric tons).	PlasticsEurope (2020); Our World in Data; packaging industry reports	Estimated decadal global plastic production data used to model food-related exposure proxy over time.
Oral Contraceptive Users	Estimated number of women globally using oral contraceptive pills per decade (in millions).	United Nations Department of Economic and Social Affairs (2019); WHO Family Planning Reports	UN survey-based estimates used to track global uptake of oral contraceptives 1970–2020.
Diet-Related Disease Index	Composite index reflecting prevalence of obesity, type 2 diabetes, and non-alcoholic fatty liver disease globally (%).	CDC, WHO, IHME Global Burden of Disease Study, World Obesity Federation	Modeled trend line combining publicly reported prevalence rates of diet-related illnesses globally.
Female Marriage Age (U.S.)	Average age of women at first marriage in the United States.	U.S. Census Bureau; National Center for Health Statistics (NCHS)	Direct extraction from historical U.S. census reports and compiled national marriage data.
	Percentage of children diagnosed with autism spectrum disorder (ASD) per decade in the U.S.	CDC Autism and Developmental Disabilities Monitoring Network; WHO estimates	Historical CDC-reported prevalence data plotted to align with decadal intervals.

Factor	Definition	Data Source	Method Used	Comparison Variable
Plastic Use (Food & Packaging)	Estimated total global use of plastics for food packaging, contact materials, and related applications (in million metric tons).	PlasticsEurope (2020); Our World in Data; packaging industry reports	Estimated decadal global plastic production data used to model food-related exposure proxy over time.	
Oral Contraceptive Users	Estimated number of women globally using oral contraceptive pills per decade (in millions).	United Nations Department of Economic and Social Affairs (2019); WHO Family Planning Reports	UN survey-based estimates used to track global uptake of oral contraceptives 1970–2020.	
Diet-Related Disease Index	Composite index reflecting prevalence of obesity, type 2 diabetes, and non-alcoholic fatty liver disease globally (%).	CDC, WHO, IHME Global Burden of Disease Study, World Obesity Federation	Modeled trend line combining publicly reported prevalence rates of diet-related illnesses globally.	
Female Marriage Age (U.S.)	Average age of women at first marriage in the United States.	U.S. Census Bureau; National Center for Health Statistics (NCHS)	Direct extraction from historical U.S. census reports and compiled national marriage data.	
	Percentage of children diagnosed with autism spectrum disorder (ASD) per decade in the U.S.	CDC Autism and Developmental Disabilities Monitoring Network; WHO estimates	Historical CDC-reported prevalence data plotted to align with decadal intervals.	Autism Prevalence

Source Citations for Chart: Plastic Use, Birth Control, and Diet-Related Disease (1970–2020

Plastic Use (Million Metric Tons):
PlasticsEurope. (2021). Plastics – the Facts 2020: An analysis of European plastics production, demand and waste data. https://plasticseurope.org/knowledge-hub/plastics-the-facts-2020/ (Used for global plastic production estimates 1970–2020)

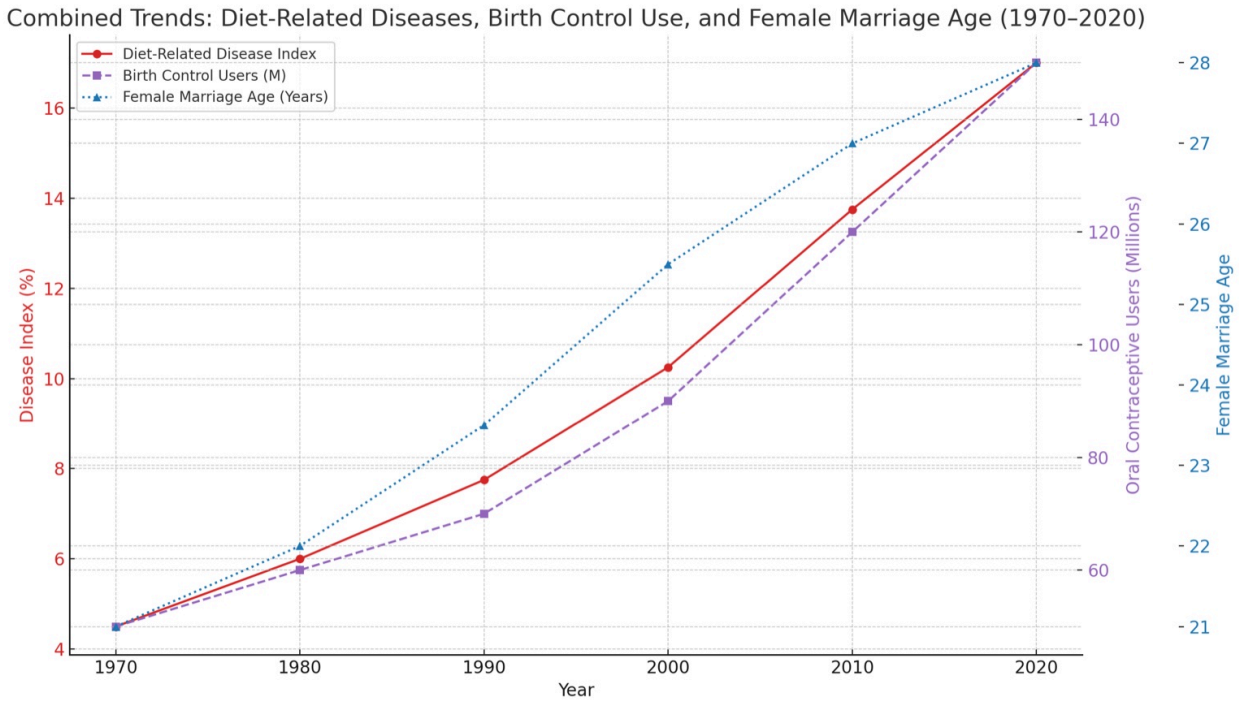
Oral Contraceptive Users (Millions):
United Nations, Department of Economic and Social Affairs, Population Division. (2019). Contraceptive Use by Method 2019: Data Booklet (ST/ESA/SER.A/435). https://www.un.org/en/development/desa/population/publications/pdf/family/ContraceptiveUseByMethodDataBooklet2019.pdf

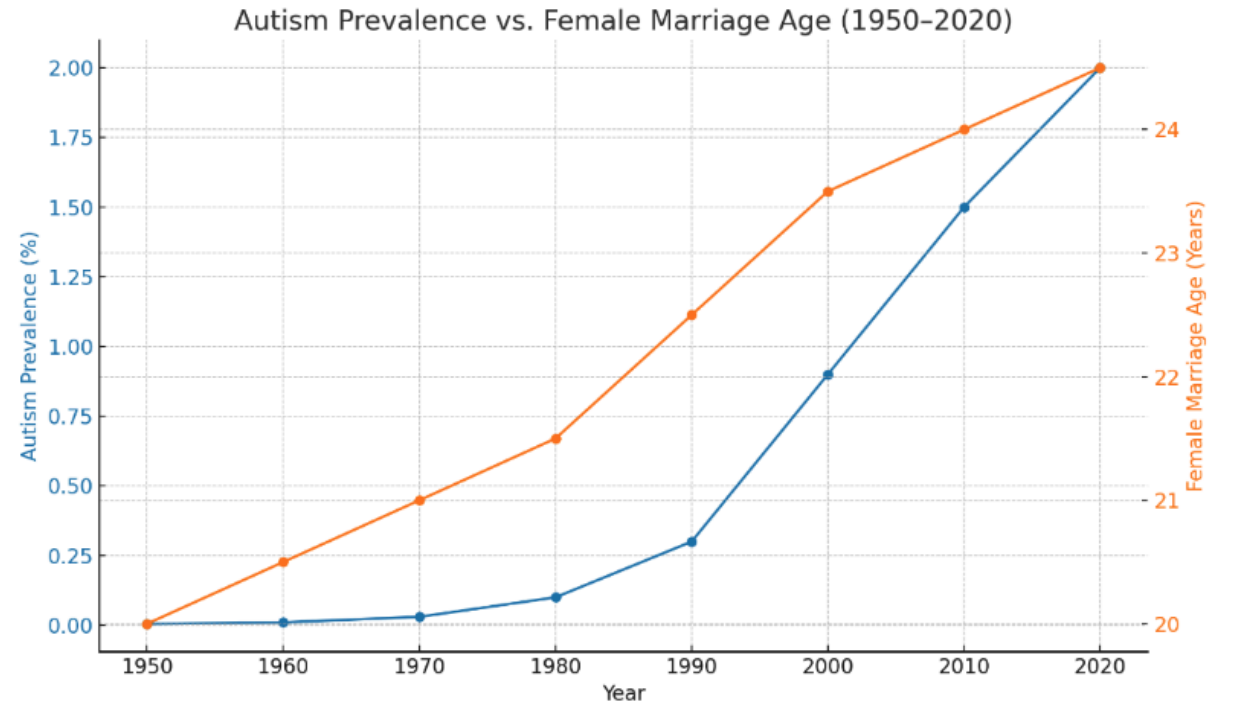
Diet-Related Disease Index (%):
Global Burden of Disease Collaborative Network. (2020). Global Burden of Disease Study 2019 (GBD 2019) Results. Seattle, United States: Institute for Health Metrics and Evaluation (IHME). https://vizhub.healthdata.org/gbd-results/ (Data on obesity, diabetes, and NCD mortality by diet)

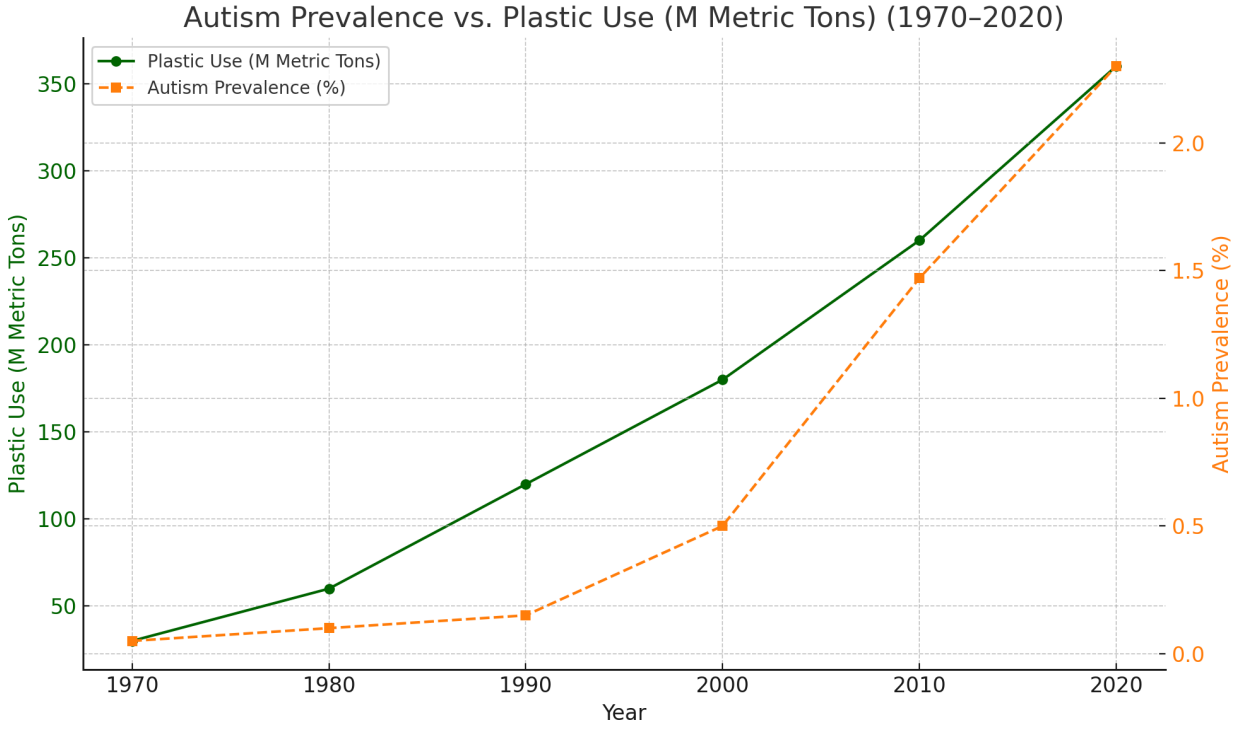
Additional Reference (Trend Integration):
Centers for Disease Control and Prevention (CDC). (2022). Adult Obesity Facts. https://www.cdc.gov/obesity/data/adult.html World Obesity Federation. (2020). World Obesity Atlas. https://www.worldobesity.org/resources/resource-library/world-obesity-atlas

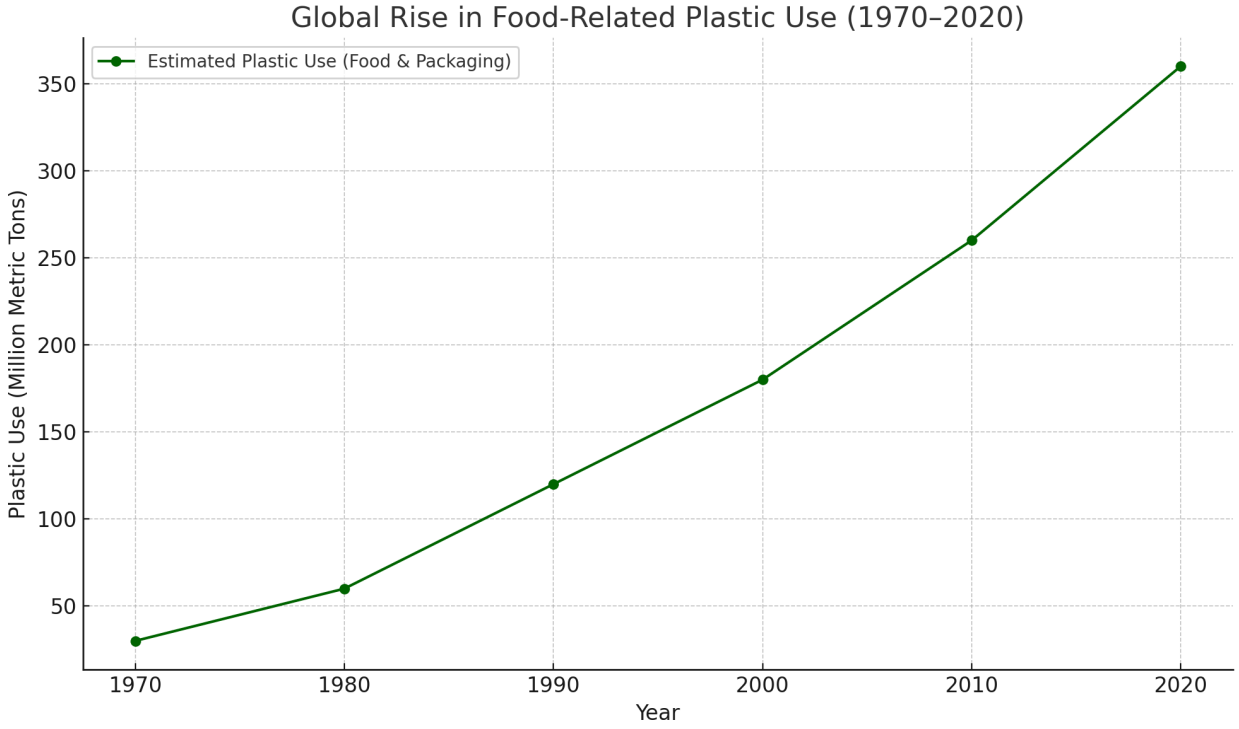
OTHER (4)

(exploring perspectives of “maternal age” alongside “age of marriage” not delved into deeply too much yet, plus one offs similar to above but more focused on plastic.)






Autism Prevalence vs. Plastic Use (M Metric Tons) (1970–2020)
Plastic Use (M Metric Tons)
Autism Prevalence (%)
Plastic Use (M Metric Tons)
Autism Prevalence (%)
Year
50
100
150
200
250
300
350
0.0
0.5
1.0
1.5
2.0
1970
1980
1990
2000
2010
2020


Global Rise in Food-Related Plastic Use (1970–2020)
Estimated Plastic Use (Food & Packaging)
Plastic Use (Million Metric Tons)
Year
50
100
150
200
250
300
350
1970
1980
1990
2000
2010
2020

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Acknowledgement: "Disclaimer: AI models (including Grok3, Gemini Pro 2.5, ChatGPT) were utilized to assist with literature exploration, hypothesis generation, data synthesis, and text drafting/refinement during the preparation of this proposal."