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Screen Exposure and Family History in Relation to Autism Spectrum Disorder in Romanian Children Aged 2–6 Years: An Age-Matched Case–Control Study

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Highlights

What are the main findings?

- In age-matched Romanian preschoolers, daily screen use of ≥2 h was reported in 54.5% of children with confirmed ASD versus 15.2% of controls (aOR 6.51, 95% CI 2.44–17.33), with higher odds per duration band (aOR 1.88 per band, 95% CI 1.26–2.82), and the excess concentrated above 2 h/day.
- A family history of ASD (aOR 5.41, 95% CI 1.19–24.51) and parent-reported birth complications (aOR 4.70, 95% CI 1.40–15.75) were also more frequent among children with ASD. Completion of the national vaccination schedule showed no clear evidence of an association with ASD (aOR 0.58, 95% CI 0.19–1.75).

What are the implications of the main findings?

- Asking about daily screen time and family history is already part of a routine developmental history: whether these questions add anything to developmental surveillance has not been tested and needs prospective evaluation.
- The cross-sectional design cannot show whether screen use precedes or follows the development of autistic traits, so screens should not be presented to families as a cause of autism.

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Abstract

Background/Objectives: Autism spectrum disorder is usually first recognised in the preschool years, when early identification can still change access to assessment and support. Screen exposure is now almost universal in this age group and is often discussed with families as a possible risk factor, yet much of that discussion rests on studies that mix age, diagnosis and screen time. Whether children with confirmed ASD truly have higher

daily screen use after accounting for age—and how this corresponds to family history and perinatal factors—remains poorly documented in Romania and Eastern Europe. **Methods:** All 33 children aged 24–72 months with a documented ASD diagnosis seen at a private paediatric clinic in Arad, Romania (2024–2026), were age-matched 1:2 to 66 attendees of the same clinic. Parent-reported screen time, family history, perinatal factors and selected child-health variables were analysed using age- and sex-adjusted logistic regression. To check the results, each case was also compared only with its own matched controls, a method that corrects for small-sample bias was applied, and caregiver education and income were added to the models. **Results:** Matching balanced age (median 43 versus 45 months). Daily screen use of ≥2 h was reported for 18 of 33 cases (54.5%) and 10 of 66 controls (15.2%, aOR 6.51, 95% CI 2.44–17.33), with a gradient across five duration bands (aOR 1.88 per band, 95% CI 1.26–2.82), concentrated in the 2–4 h/day bands. A family history of ASD (aOR 5.41, 95% CI 1.19–24.51) and parent-reported birth complications (aOR 4.70, 95% CI 1.40–15.75) were also associated with ASD, but both findings are inconclusive: family history rested on nine positive reports and did not meet the false-discovery-rate threshold within the main family of tests ($q = 0.057$); the birth-complication estimate depended on how missing answers were handled; completion of the national vaccination schedule showed no association (aOR 0.58, 95% CI 0.19–1.75). The screen-time and family history associations persisted in conditional and penalized models and after adding education or income; the birth complication association lost significance in the conditional analysis (OR 2.81, 95% CI 0.81–9.82). **Conclusions:** Heavy daily screen use was associated with ASD in this clinic sample, with wide confidence intervals. The design cannot establish direction, as screen use may follow rather than precede the development of autistic traits, and these results require confirmation in prospective studies.

Keywords: autism spectrum disorder; screen time; family history; perinatal factors; birth complications; case–control study; preschool children; Romania

1. Introduction

Among the potentially modifiable correlates of autism spectrum disorder (ASD) in early childhood, screen exposure has attracted particular attention. The World Health Organization advises against sedentary screen time before the age of 2 years [1], and the Canadian Paediatric Society recommends no screen time before 2 years of age, apart from video-chatting with caring adults [2]. Prospective cohort studies have linked higher screen time in early childhood to poorer performance on developmental screening tests [3], weaker language skills [4], and delays in the communication and problem-solving domains [5,6]. These studies measured developmental delay, not autism, using screening tools or language tests. ASD is diagnosed clinically and requires the presence of ongoing difficulties in social communication, together with restricted and repetitive behaviour [7]. A child may have language or communication delay without having ASD, so these findings do not show a link between screen time and autism itself. One proposed mechanism is the displacement of reciprocal interaction: home audio recordings show that more screen time at 12–36 months is associated with fewer adult words, child vocalizations and conversational turns, with the largest per-minute reductions at 36 months [8]. Evidence relating screen exposure specifically to autism outcomes is more contested. The Japan Environment and Children's Study (JECS) found that screen time at 1 year predicted an ASD diagnosis at 3 years in boys but not in girls [9]. A systematic review and meta-analysis found a positive pooled association that became non-significant after correction for publication bias and cautioned that the literature is dominated by cross-sectional designs [10]. Similarly, in an Australian

cohort, the association between screen time at 2 years and ASD at 12 years did not persist in instrumental-variable analysis [11]. The direction of the relationship also remains uncertain: in the JECS, poorer communication at 1–2 years predicted more subsequent screen time, as well as the converse [6], whereas a Singaporean cohort found that screen time predicted later social behavioural traits but not the reverse [12].

Three explanations, which are not mutually exclusive, could produce an association between screen use and ASD. The first is displacement. Young children learn to communicate through everyday talk and play with the adults around them, and time spent in front of a screen is time taken away from those exchanges. Children with more screen time hear fewer adult words and take part in fewer conversations [8]. If this is the main route to learning, screen use would be shaping development. The second is shared liability. Autism runs strongly in families, largely for genetic reasons [13,14]. The same family background that raises a child's likelihood of autism could also shape home routines, including screen use, so screen time and autism could appear together without one causing the other [15]. The third is reverse causation, where the direction is reversed, and early autistic traits lead to more screen use. Parents of a child who is hard to settle may turn to an electronic device to calm them, a pattern sometimes called compensatory soothing [16]. Children with ASD also tend to be drawn to screens: they spend more time on video games than do their brothers and sisters, as well as more time on television and video games than on all non-screen activities taken together [17]. In that case, heavy screen use would be a result of differences already present, not their cause. A study like the present one, which records screen use and diagnosis at the same point in time, cannot tell these explanations apart. We therefore treat screen exposure as a marker associated with ASD, and what it means depends on which of these routes is at work.

Data from Romania and Eastern Europe are scarce. Few, if any, case–control studies from the region have examined parent-reported screen use together with familial and perinatal factors in children with clinically confirmed ASD. We looked at these factors together for two reasons. Family history and perinatal adversity are among the best-documented correlates of ASD [13,18,19], so they give a reference point for judging the size of any screen-time association. Family history also gives an indication of shared liability, meaning the inherited and family-level factors that make autism more likely. If screen time were linked to ASD only because both tend to run in the same families, the screen-time association should become weaker once family history is considered. Putting both in the same model shows how much the screen-time estimate changes once our family-history question is considered. It cannot tell us whether a shared family background explains the link because one yes/no question cannot capture the wider genetic and family influences involved. Interpretation is further complicated by age: diagnosed children tend to be older, and older children usually have more screen time. We therefore conducted an age-matched case–control analysis nested in a consecutive series of 212 children, aged 24–72 months, attending a paediatric outpatient clinic in Arad, Romania. The primary objective was to estimate the association between parent-reported daily screen time (≥2 h/day and across five ordered duration bands) and clinically confirmed ASD. The 2 h/day cut-off was fixed before analysis, and is a value supported on three grounds. It corresponds to the upper limit of 1–2 h of daily screen time recommended by the American Academy of Paediatrics [20]; it is the lower limit of the 2 to <4 h/day category used in the JECS analysis of screen time and ASD [9]; and it coincides with the boundary between the second and third response options of our questionnaire. The value exceeds current guidance for children aged 2–5 years, and it was therefore used as a marker of high use rather than as a clinical threshold [1,2,21]. Secondary objectives were to examine family history of ASD and sociodemographic, perinatal and child-health factors, including completion of the national vaccination schedule. We hypoth-

esised that children with ASD would have higher daily screen time and a greater incidence of family history of ASD than did the age-matched controls, while recognizing that a cross-sectional design cannot establish direction. The analysis was exploratory and intended to generate hypotheses.

2. Materials and Methods

2.1. Study Design and Participants

Children aged 2 to 6 years (24–72 months) attending a paediatric outpatient private clinic in Arad, Romania, between 20 March 2024 and 27 April 2026 were eligible. Whatever the referral routes, a clinic series is not a population sample. Children brought in because of developmental or behavioural concerns are likely to be over-represented, and this can distort comparisons between cases and controls. This age window was chosen a priori because it is the period in which autism features become most apparent to parents and in which a diagnosis is typically first made. Parents completed an online questionnaire covering pregnancy and perinatal history, feeding, developmental milestones, lifestyle factors, infectious exposures and screen use.

The outcome was a single parent-report item: "Does your child display characteristics associated with autism spectrum disorders, or has your child been diagnosed with a disorder within this spectrum?", answered yes or no. Each affirmative answer was subsequently verified by the attending paediatrician against the child's medical records, including evaluations issued by specialist physicians who diagnose ASD. A child was counted as a case only when the parent's answer was confirmed by a medical letter, signed and stamped by a paediatric neuropsychiatrist, stating the ASD diagnosis. The diagnoses were made by several paediatric neuropsychiatrists in routine clinical practice. No common diagnostic protocol was used, and the criteria and instruments each specialist relied on were not recorded. These specialists had no access to the study questionnaire and did not know the parents' answers. Children whose affirmative answer could not be corroborated were not counted as cases. In practice, every affirmative answer was confirmed by such a letter, so all 33 children whose parents answered yes comprised the cases, and the parents of all 66 controls answered no. The paediatrician who checked the records had not seen the screen-time answers, and the check concerned only whether a signed specialist letter was present. Thirty-three children met this definition. Controls were children whose parents answered No and who had no ASD diagnosis in their clinic records. No further check for developmental concerns was documented for controls, and no standardised assessment was carried out in them, so the absence of a recorded diagnosis does not prove the absence of ASD, and some children with undiagnosed autism, particularly the youngest, may have been included as controls. Because children with ASD were older than other children attending the clinic (median 43 versus 36 months), the analysis set was restricted to an age-matched comparison. Every confirmed case was retained. For each case, two controls without ASD were selected without replacement from the same 24–72-month series by nearest age in months, preferring the same sex, within a range of ±6 months. All 66 matches met the range; 58 of 66 controls were the same sex as their matched case. The analysis sample therefore comprised 99 children (33 with confirmed ASD and 66 controls; Figure 1).

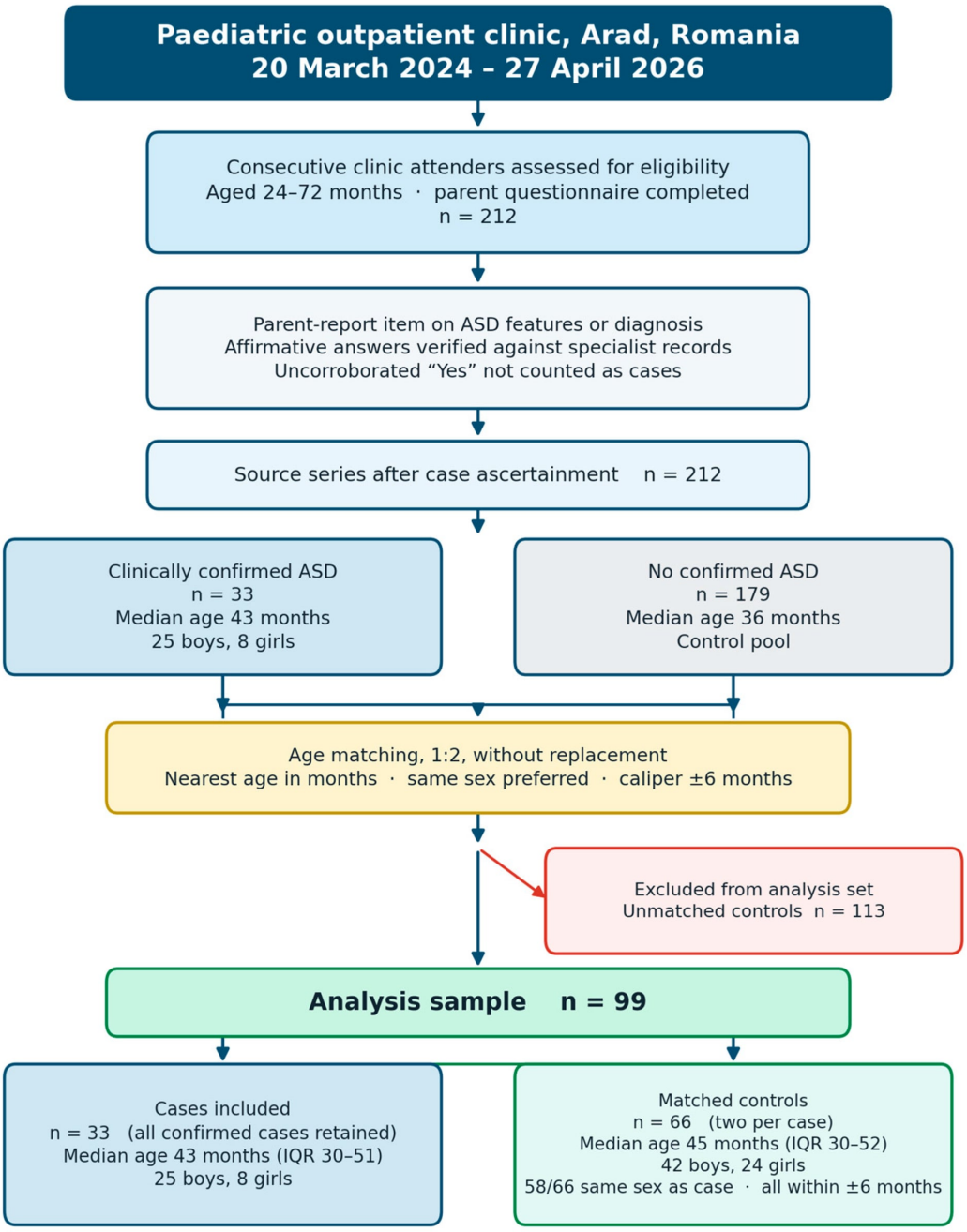


Figure 1. Flow diagram of participant selection and age–sex matching in a paediatric outpatient clinic, Arad, Romania. ASD, autism spectrum disorder; IQR, interquartile range.

Sociodemographic variables comprised child age in months, child sex, residence, respondent sex, respondent age at survey completion, respondent education collapsed into three levels, marital status, perceived income adequacy, health problems in the respondent (caregiver ill health), number of children in the family and firstborn status. Family history was captured by a separate item asking whether anyone in the family had been diagnosed with autism or an autism spectrum condition. The item did not ask about the degree of relationship, so it combines first-degree relatives (parents and siblings) with more distant relatives and could not be split by degree. Perinatal variables comprised birth weight, birth length, gestational age, preterm birth defined as below 37 weeks, the Apgar score at 5 min, reported birth complications, twin pregnancy and medically assisted conception. Birth complications were recorded as a single parent-reported yes/no item with no list of specific complications, and answers were not checked against obstetric records. Missing answers

were kept separate from "no complications". The item was missing for 36 of the 99 children (15 of 33 cases and 21 of 66 controls); all 36 missing answers came from questionnaires completed between 20 March and 7 August 2024, a period in which 17 of 53 respondents did answer it, and every respondent after that date answer it. Maternal variables comprised body mass index and gestational weight gain in five ordered categories. Pregnancy exposures comprised smoking, urinary tract infection, planned pregnancy, disturbed sleep and SARS-CoV-2 infection; child-level health variables comprised SARS-CoV-2 infection, adherence to the national vaccination schedule and the presence of a chronic disease.

Screen exposure was reported as any exposure (yes or no); as daily duration in five ordered categories (less than 1 h, 1–2 h, 2–3 h, 3–4 h, more than 4 h per day), modelled as an ordinal variable scored 1 to 5 and additionally dichotomized at 2 h per day; and as total duration of exposure. The response options are listed in Table A1. The dataset does not specify the reference period of the daily-duration question, so the answers are interpreted as typical current use. The options were whole-hour bands ("1–2 h", "2–3 h"), so a parent of a child watching exactly 2 h could have chosen either; the ≥2 h/day variable was defined as the "2–3 h" band or higher. The seven children reported as having no screen exposure (all controls) still had a daily-duration answer (five "<1 h", two "1–2 h") and were analysed in the band chosen. The questionnaire did not ask about content, device, purpose (for example, educational or therapeutic use) or co-viewing. Screen use was reported at the time of the survey, when every case already had a diagnosis, and the questionnaire did not ask about use before diagnosis, so it cannot show whether heavy use preceded the diagnosis or followed it. Age at first screen exposure was derived by subtracting the reported total duration from the child's current age in months. This derived variable has a basic problem. Total duration was answered with five options (1, 2, 6 or 12 months, or more than 12 months), and 54 of the 97 parents who answered chose "more than 12 months", which was coded as 12; for these children, the derived age is simply current age minus 12 months. The variable therefore reflects the current pattern of use, not the age at which screen use began, and thus, it was dropped from the inferential analyses. It is shown in Table 1. Free-text numeric fields were parsed, and values outside physiologically plausible ranges were set to missing. The Modified Checklist for Autism in Toddlers, revised (M-CHAT-R) screening score, available for part of the database, was excluded because the instrument is validated only for ages 16 to 30 months.

Table 1. Characteristics of the age-matched sample, children aged 24–72 months.

Characteristic	Confirmed ASD (n = 33)	Age-Matched Controls (n = 66)	p
Child age, months, median (IQR)	43 (30–51)	45 (30–52)	0.99
Male sex	25 (75.8%)	42 (63.6%)	0.26
Urban residence	19 (57.6%)	39 (59.1%)	1.00
Respondent age, years, median (IQR)	36 (33–39)	35 (31–37)	0.12
Tertiary education of respondent	23 (69.7%)	56 (84.8%)	0.054
Married	25 (75.8%)	59 (89.4%)	0.13
Perceived adequate income	10 (30.3%)	34 (51.5%)	0.055
Family history of ASD	6 (18.2%)	3 (4.5%)	0.057
Health problems in the respondent	11 (33.3%)	12 (18.2%)	0.13

Table 1. *Cont.*

Characteristic	Confirmed ASD ($n = 33$)	Age-Matched Controls ($n = 66$)	p
Birth weight, g, median (IQR)	3230 (2945–3600)	3235 (2900–3495)	0.42
Gestational age, weeks, median (IQR)	38 (37–39)	38 (38–39)	0.66
Preterm birth < 37 weeks	7/31 (22.6%)	9/63 (14.3%)	0.38
Apgar at 5 min, median (IQR)	9 (8–9)	9 (9–10)	0.036
Reported birth complications	9/18 (50.0%)	8/45 (17.8%)	0.014
Vaccination schedule completed	24/31 (77.4%)	55/64 (85.9%)	0.38
Any screen exposure	33 (100%)	59 (89.4%)	0.093
Daily screen time			
<1 h/day	8 (24.2%)	28 (42.4%)	
1–2 h/day	7 (21.2%)	28 (42.4%)	
2–3 h/day	9 (27.3%)	5 (7.6%)	
3–4 h/day	7 (21.2%)	3 (4.5%)	
>4 h/day	2 (6.1%)	2 (3.0%)	<0.001 †
Screen time ≥ 2 h/day	18 (54.5%)	10 (15.2%)	<0.001
Age at first screen exposure, months, median (IQR) ‡	31.5 (21.8–40.5) (n= 32)	36.0 (24.0–42.5) (n= 59)	0.88

Values are n (%) unless stated. Percentages use non-missing denominators. p from the Mann–Whitney U test or Fisher's exact/χ^2 test. † Overall test across the five screen-time categories. ASD, autism spectrum disorder; IQR, interquartile range. The seven children with no screen exposure are included in the band reported by their parents (five < 1 h/day, two 1–2 h/day). ‡ Derived variable that reflects the current pattern of use, not true onset (see Section 2.1); shown for description only. Missing data by variable are given in Table A2.

2.2. Statistical Analysis

Continuous variables were summarised as the median (interquartile range, IQR). Between-group comparisons used the Welch's t test when both distributions were consistent with normality on the Shapiro–Wilk test, and employed the Mann–Whitney U test otherwise. Categorical variables were compared using the χ^2 test or Fisher's exact test when any expected cell count was <5. Matching quality was assessed by the residual difference in age (months) and by the proportion of same-sex pairs.

The measure of association was the age- and sex-adjusted odds ratio (OR) with its 95% confidence interval (CI). In a case–control sample, the proportion of cases is set by the design, which is here one third because two controls were chosen per case. The proportions of children with ASD among exposed and unexposed children, and any risk differences or risk ratios built from them, therefore depend on that ratio and do not estimate risks in any population, whereas the odds ratio does not depend on it. Exposure frequencies are shown separately for cases and controls, and risk differences and risk ratios are not reported.

Adjusted ORs were obtained from ordinary logistic regression of case status on the exposure plus child age in months, along with child sex. Matching was treated as a design device to remove the age imbalance between children with and without ASD rather than as a matched-set analysis; ordinary logistic models with residual age were therefore used as the primary specification. When matching is performed using a variable such as age that can be adjusted for directly, an unconditional analysis that includes the matching variable is valid and can be more efficient than a conditional variable, especially when missing data would break up matched sets. Three sets of sensitivity analyses were run. First, the

Table 2 exposures, other than sex, were re-estimated with conditional logistic regression stratified on the matched set and adjusted for sex. For each model, we report the number of informative matched sets, meaning sets in which exposure differed between the case and at least one control; sets left without a case or without any control because of missing data dropped out, which mainly affected birth complications. Second, because some exposures were rare (family history in nine children) and the joint model had about eight cases per parameter (about eleven in the single-exposure models and six in the birth-complication model), the main models were refitted with Firth's penalised likelihood, which reduces small-sample bias, using profile penalised-likelihood confidence intervals [22]. Penalisation is one recognised remedy for sparse-data bias, although Firth's version does not remove it entirely [23]. Third, we added two family factors to the screen-time and family-history models, first one at a time and then together, i.e., whether the respondent had a university education and whether they felt their income was adequate. We chose these factors before running the analysis because they could plausibly affect both how much time a child spends on screens and how quickly the child reaches a diagnosis, not because they happened to differ between the groups [24]. We kept the number of added factors small so that the models would not be overloaded for the small number of cases [25]. All sensitivity models are exploratory. Multiple testing was handled using the Benjamini–Hochberg false-discovery-rate (FDR) procedure at q = 0.05 [26]. The primary family comprised the eight age- and sex-adjusted tests in Table 2 (screen time ≥ 2 h/day, screen time per band, family history, birth complications, caregiver ill health, male sex, vaccination and preterm birth). For a stricter check, we repeated the procedure across all 21 adjusted tests in the study. We left out the derived age at first exposure because it does not show when screen use began, and it was not used for inference. *p*-values before and after FDR adjustment are given in Table A4.

Table 2. Associations with confirmed ASD in the age-matched sample.

Exposure	Exposed Cases, *n*/N (%)	Exposed Controls, *n*/N (%)	Adjusted OR (95% CI)
Screen ≥ 2 h/day	18/33 (54.5%)	10/66 (15.2%)	6.51 (2.44–17.33)
Screen time, category	—	—	1.88 (1.26–2.82)
Family history of ASD	6/33 (18.2%)	3/66 (4.5%)	5.41 (1.19–24.51)
Birth complications	9/18 (50.0%)	8/45 (17.8%)	4.70 (1.40–15.75)
Caregiver ill health	11/33 (33.3%)	12/66 (18.2%)	2.24 (0.85–5.89)
Male sex	25/33 (75.8%)	42/66 (63.6%)	1.78 (0.70–4.57)
Completed vaccination	24/31 (77.4%)	55/64 (85.9%)	0.58 (0.19–1.75)
Preterm birth	7/31 (22.6%)	9/63 (14.3%)	1.78 (0.58–5.48)

Percentages are calculated only among children with data for that variable. Risk differences and risk ratios are no longer shown because, with two controls chosen for every case, they reflect the study design rather than real risk. Results from the matched-set, small-sample-corrected and education- and income-adjusted models are given in Table 3. Adjusted OR from a logistic model including child age, in months, and sex. Screen-time category is scored 1–5. Any screen exposure is not modelled (complete separation). ASD, autism spectrum disorder; CI, confidence interval.

Table 3. Sensitivity analyses for the main associations with confirmed ASD.

Exposure	Main Model, aOR (95% CI)	Conditional Logistic, OR (95% CI); Informative Sets	Firth Penalised, aOR (95% CI)	+Education, aOR (95% CI)	+Income, aOR (95% CI)	+Both, aOR (95% CI)	FDR q (Table 2 Family)
Screen ≥ 2 h/day	6.51 (2.44–17.33)	4.49 (1.59–12.64); 23	5.97 (2.36–15.93)	6.55 (2.41–17.84)	5.65 (2.07–15.41)	5.75 (2.07–16.01)	0.001
Screen time, per band	1.88 (1.26–2.82)	1.66 (1.10–2.51); 30	1.82 (1.25–2.75)	1.92 (1.27–2.89)	1.76 (1.16–2.65)	1.80 (1.18–2.74)	0.009
Family history of ASD	5.41 (1.19–24.51)	6.00 (1.21–29.74); 9	4.74 (1.22–22.12)	4.87 (1.04–22.77)	5.88 (1.26–27.38)	5.28 (1.12–24.84)	0.057
Birth complications ($n = 63$)	4.70 (1.40–15.75)	2.81 (0.81–9.82); 12	4.26 (1.36–13.93)	5.04 (1.47–17.29)	9.54 (2.01–45.40)	9.64 (2.02–46.03)	0.032

Main model: ordinary logistic regression with child age, in months, and sex. Conditional logistic regression is stratified on the matched set and adjusted for sex; informative sets are those in which exposure differed between the case and at least one control. Firth estimates use profile penalised-likelihood confidence intervals. Education is respondent tertiary education; income is perceived income adequacy; both are added to the main model. FDR q from the Benjamini–Hochberg procedure across the eight tests in Table 2. aOR, adjusted odds ratio; CI, confidence interval; FDR, false discovery rate.

Daily screen time was analysed using three complementary codings: any versus none (descriptive only, because all 33 cases were exposed, and the logistic model exhibited complete separation); a binary cut at ≥2 h/day, chosen a priori as a marker of high daily use; and an ordinal score of 1–5 across the five reported duration bands, entered as a linear term. Treating the five bands as a single score assumes that the odds of ASD increase by the same amount with each step up from one band to the next, including the last step into the open-ended ">4 h/day" band. To check this, we also entered each band separately, with <1 h/day as the reference, and compared the two models using a likelihood-ratio test. Age at first exposure was not used in any model, for the reasons given in Section 2.1. We also wanted to know whether the screen-time association changed once the recorded family-history item was added. We therefore placed screen time ≥ 2 h/day and family history into the same model, together with age and sex (four parameters for 33 cases). To show how well this model fits, we report McFadden's pseudo-R^2 (how much of the variation the model explains), a likelihood-ratio test against a model with no predictors, the Hosmer–Lemeshow test (whether predicted and observed numbers of cases agree across tenths of predicted risk) and the area under the ROC curve (how well the model separates cases from controls in this sample). All four were calculated in the same 99 children used to fit the model. They are in-sample summaries of fit, which tend to be optimistic, and they do not show how well the model would identify children with ASD in other samples; the screening performance of the model was not evaluated [27,28].

Analyses used complete cases for each model; denominators are reported in the tables. No imputation was performed. Missing data by variable and group are given in Table A2. Multiple imputation was not used: the incomplete item used in the main analyses, birth complications, was missing almost entirely in the first months of data collection, and with 33 cases, an imputation model would have had little information to draw on. For birth complications we compared children with and without data and ran three simple sensitivity analyses, with missing answers coded as "no", coded as "yes", and entered as a separate missing-indicator category. All tests were two-sided. Analyses were conducted in Python 3 using pandas, SciPy 1.17, and statsmodels 0.14. The sample size was set by the number of confirmed cases seen at the clinic during the study period, rather than by a calculation made

in advance. To get a sense of what the study could detect, we ran a simulation (Fisher's exact test, two-sided $\alpha = 0.05$). With 33 cases and 66 controls, the study had about a 77% chance of detecting an odds ratio of 4, and about a 90% chance for an odds ratio of 5, for an exposure found in 15% of the controls. For a rarer exposure present in about 5% of controls, as was family history, that chance fell below 50%, even for an odds ratio of 4. Firth's method was programmed directly in NumPy 2.5.5.

2.3. Ethics

The study was approved by the Research Ethics Committee of the Victor Babeș University of Medicine and Pharmacy, Timișoara (approval code 17, 29 February 2024). Parents or legal guardians provided informed consent before completing the survey. All data were analysed in anonymised form, and the study was conducted in accordance with the Declaration of Helsinki.

3. Results

The age-matched sample comprised 99 children: 33 with clinically confirmed ASD and 66 controls. The median age was 43 months (IQR 30–51) in cases and 45 months (IQR 30–52) in the controls ($p = 0.99$). Boys accounted for 25 of 33 cases (75.8%) and 42 of 66 controls (63.6%, $p = 0.26$), a case sex ratio of 3.1 to 1. Residence and most perinatal measures did not differ. Respondents in the case group were less often university educated (69.7% versus 84.8%; $p = 0.054$ across the three education levels) and less often reported adequate income (30.3% versus 51.5%, $p = 0.055$). Differences of this size matter, whether or not they cross $p = 0.05$, and their influence on the main estimates is examined below. A family history of ASD was reported for 6 of 33 cases (18.2%) and 3 of 66 controls (4.5%). Every child with ASD had some screen exposure, compared with 59 of 66 controls (89.4%). Daily exposure of at least 2 h was reported for 18 of 33 cases (54.5%) and 10 of 66 controls (15.2%) (Table 1).

Children with confirmed ASD spent more time in front of screens than did their age-matched controls. In the control group ($n = 66$), most children were concentrated in the lowest exposure categories: 28 (42.4%) had <1 h/day, and 28 (42.4%) had 1–2 h/day, together accounting for 84.8% of controls. Only 5 (7.6%) reported 2–3 h/day, 3 (4.5%) 3–4 h/day, and 2 (3.0%) > 4 h/day.

Among children with confirmed ASD ($n = 33$), the distribution was shifted toward higher daily use. Eight children (24.2%) had <1 h/day, and 7 (21.2%) had 1–2 h/day (45.5% combined). Nine (27.3%) used screens for 2–3 h/day and 7 (21.2%) for 3–4 h/day; 2 (6.1%) exceeded 4 h/day. Thus, mid-to-high screen-time categories (2–4 h/day) were more frequent in the ASD group than in the matched controls, whereas very high use (>4 h/day) remained uncommon in both groups (Figure 2).

Once residual age and sex were accounted for, three exposures were associated with case status, all with wide confidence intervals (Table 2, Figure 3). Daily screen use of 2 h or more was reported for 54.5% of cases and 15.2% of controls (adjusted OR 6.51, 95% CI 2.44–17.33). Treating daily screen time as an ordered variable yielded an adjusted OR of 1.88 per band (95% CI 1.26–2.82). Band-specific estimates did not show an even gradient (Table A5). Compared with the <1 h/day results, the adjusted OR was 0.89 (95% CI 0.28–2.80) for 1–2 h/day, 6.21 (1.60–24.06) for 2–3 h/day, 7.97 (1.58–40.22) for 3–4 h/day and 3.65 (0.43–30.62) for >4 h/day, with the last based on four children. The excess was concentrated above 2 h/day. A formal test comparing the two approaches found no clear difference between them ($\chi^2 = 5.17$, 3 df, $p = 0.16$). With so few children in some bands, i.e., only four in the highest, this test could easily miss a real difference, and a significant result for the single score does not by itself show that the odds rise steadily from band to band.

Family history of ASD yielded an adjusted OR of 5.41 (95% CI 1.19–24.51); the interval is wide because only nine children had a positive family history (6 cases, 3 controls). Reported birth complications yielded an adjusted OR of 4.70 (95% CI 1.40–15.75) using a reduced denominator (63 of 99 children with data).

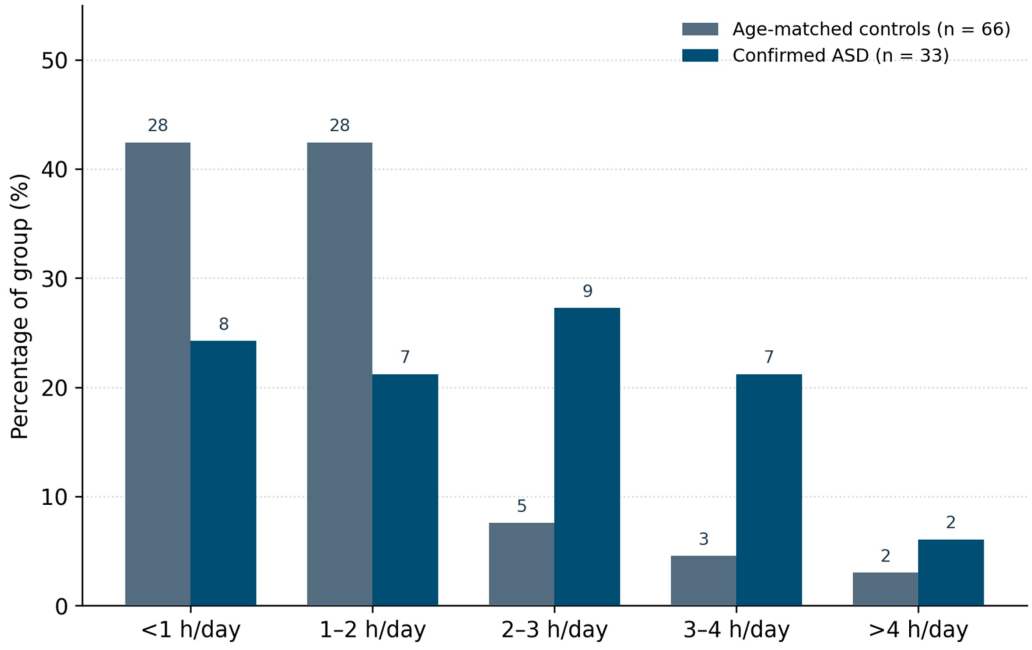


Figure 2. Daily screen time in children with confirmed ASD and age-matched controls. Bars show the percentage of each group; numbers above the bars are counts. ASD, autism spectrum disorder.

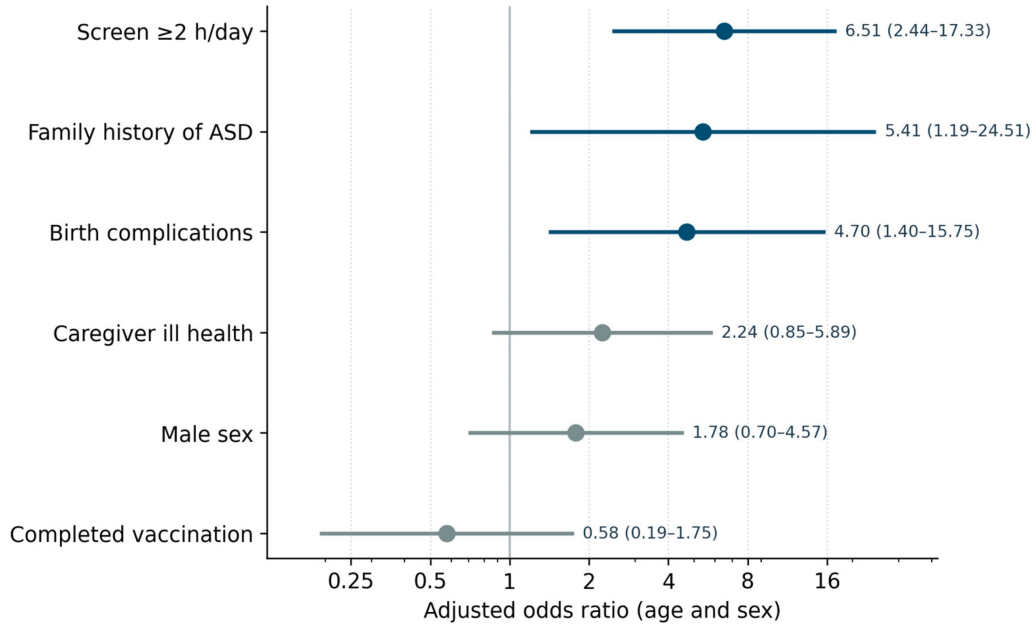


Figure 3. Age- and sex-adjusted associations with confirmed ASD. Points are adjusted odds ratios from separate logistic models including child age and sex; horizontal bars are 95% confidence intervals (CIs) on a logarithmic scale. ASD, autism spectrum disorder.

Caregiver ill health (adjusted OR 2.24, 95% CI 0.85–5.89) and male sex (adjusted OR 1.78, 95% CI 0.70–4.57) results were in the expected direction, but their confidence intervals included 1. Completion of the national vaccination schedule showed no clear evidence of an association in this sample (adjusted OR 0.58, 95% CI 0.19–1.75). Birth weight, gestational age, preterm delivery, and residence were also not clearly associated with case status. For these variables, the confidence intervals were wide enough to include modest associations in either direction, so the results show insufficient evidence, not evidence of no association.

In a joint model with age, sex, screen exposure ≥ 2 h/day and family history ($n = 99$, McFadden pseudo-$R^2 = 0.18$), screen exposure (OR 7.56, 95% CI 2.71–21.06) and family history (OR 7.42, 95% CI 1.52–36.18) were retained. Residual age was not (OR 1.00 per month, 95% CI 0.97–1.03), which is the expected consequence of a successful match. Male sex was not associated with case status in this model (OR 1.46, 95% CI 0.49–4.34). The model fitted adequately (likelihood-ratio $\chi^2 = 23.2$, 4 df, $p < 0.001$; Hosmer–Lemeshow $\chi^2 = 3.25$, 8 df, $p = 0.92$; area under the ROC curve 0.77). The two exposures were essentially uncorrelated ($r = -0.04$), and the model had about eight cases per parameter. With Firth's method, the ORs were 6.79 (95% CI 2.59–19.00) for screen time and 6.36 (1.50–31.17) for family history. Full results are shown in Table A6.

After Benjamini–Hochberg correction for multiple testing, the results of three of the eight tests in Table 2 stayed below the 0.05 threshold: screen time ≥ 2 h/day ($q = 0.001$), screen time per band ($q = 0.009$) and birth complications ($q = 0.032$). Family history fell just short ($q = 0.057$). When all 22 adjusted tests were corrected together, only the two screen-time measures stayed below 0.05 ($q = 0.004$ and $q = 0.024$; Table A4).

Table 3 shows how the main results held up when the analysis was performed in other ways. When each child with ASD was compared only with their own matched controls (conditional logistic regression), the OR was 4.49 (95% CI 1.59–12.64; 23 informative sets) for screen time ≥ 2 h/day, 1.66 (1.10–2.51; 30 sets) per screen-time band and 6.00 (1.21–29.74; 9 sets) for family history. Birth complications fared worse: because of missing answers, only 18 matched sets still retained one case and at least one control with data (49 children, 12 informative sets), and the OR dropped to 2.81 (0.81–9.82). For caregiver ill health, vaccination and preterm birth, the conditional ORs were 1.78 (0.63–5.08), 0.49 (0.15–1.65) and 2.06 (0.56–7.60). The screen-time estimates were somewhat smaller in this analysis than in the main investigation, but they pointed in the same direction.

Correcting for small-sample bias using Firth's method pulled the estimates down only slightly: 5.97 (2.36–15.93) for screen time ≥ 2 h/day, 1.82 (1.25–2.75) per band, 4.74 (1.22–22.12) for family history and 4.26 (1.36–13.93) for birth complications.

Taking the respondent's education into account barely changed the screen-time OR (6.55, 2.41–17.84). Taking income into account lowered it to 5.65 (2.07–15.41), and including both gave 5.75 (2.07–16.01). The family-history OR stayed between 4.87 and 5.88 across these models (Table 3). The birth-complication OR did not weaken, but it almost doubled when income was added (9.54, 2.01–45.40). A jump this large in a subset of 63 children points to an unstable rather than a robust estimate.

Children whose parents did not answer the birth-complication question were older than those with an answer (median 48 versus 36 months, $p < 0.001$), which fits the date pattern described in Section 2.1. They did not differ clearly in case status, sex, residence, education, income, family history or screen time (Table A3). How the missing answers were handled mattered: counting them as "no" gave an adjusted OR of 2.93 (0.97–8.90), counting them as "yes" gave 3.63 (1.41–9.37), and treating them as a separate category gave 4.58 (1.36–15.38). The size of the birth-complication association therefore depends partly on that choice.

4. Discussion

In this age-matched comparison of 33 children with clinically confirmed ASD and 66 controls aged 24–72 months, drawn from children attending a private paediatric outpatient clinic in western Romania, three characteristics were associated with the case status after adjustment for residual age and sex: daily screen use of at least 2 h (adjusted OR 6.51, 95% CI 2.44–17.33), a family history of ASD (adjusted OR 5.41, 95% CI 1.19–24.51) and parent-reported birth complications (adjusted OR 4.70, 95% CI 1.40–15.75). The screen-time association increased with duration band (OR 1.88 per band, 95% CI 1.26–2.82), rather than rising evenly, although band-specific estimates placed the excess above 2 h/day, and screen exposure and family history were both retained when entered jointly. Birth weight, gestational age, preterm birth, residence and completion of the national vaccination schedule were not associated with case status. Because matching removed the age difference present between children with and without ASD at the clinic (median 43 versus 36 months) and residual age carried no weight in the joint model (OR 1.00 per month), the contrasts cannot be attributed to children with a diagnosis simply being older and therefore, allowed more screen time. Matching dealt with age only. It does not address other differences between the groups, such as education and income, which are discussed below. Two design features distinguish this analysis from much of the questionnaire literature: case status was corroborated against specialist documentation rather than resting on the caregiver report alone, and the main estimates were checked with conditional, penalised and socioeconomically adjusted models. These findings are exploratory. Only the two screen-time measures survived correction across all tests, family history fell just short of significance after correction within the main family ($q = 0.057$), and the birth-complication association lost significance in the conditional analysis.

The direction of the screen-time association agrees with most of the published literature. A systematic review of 16 studies concluded that children with ASD are exposed to more screen time than typically developing peers or other clinical groups and that their exposure starts at a younger age [29]. In a Thai case–control study of somewhat younger children (mean age about 2.5 years), those with ASD watched television for 4.60 h/day, compared with 2.06 h/day among typically developing controls [30]. Prospective data are broadly concordant. In the JECS, boys exposed to 2 to <4 h/day of screen time at 1 year had 3.48-fold higher odds of an ASD diagnosis at 3 years than did unexposed boys, whereas no association was seen in girls [9]; in the US National Children's Study, television or video viewing at 12 months was associated with more ASD-like symptoms at 2 years, although not with a positive screening on the Modified Checklist for Autism in Toddlers (M-CHAT) [31]. In our sample, the excess among cases lay mainly in the 2–4 h/day rather than in very heavy use (>4 h/day: 6.1% versus 3.0%) bands.

The magnitude of our estimate is, however, considerably larger than the pooled figures. A systematic review and meta-analysis of 46 studies obtained a summary log OR of 0.54 (OR ≈ 1.7), which fell to a non-significant 0.22 (OR ≈ 1.25) after trim-and-fill correction for publication bias; the association was confined to studies of general screen use and was the largest in studies of children, and the authors concluded that the proposed link is not sufficiently supported by the existing literature, noting that 41 of the 46 included studies were cross-sectional [10]. In an Australian birth cohort, more than 14 h/week of screen time at 2 years was associated with ASD at 12 years (adjusted OR 1.79), but the association was no longer statistically significant when maternal education and family income were treated as instrumental variables [11]. Several features of our design plausibly inflate the estimate. First, exposure was reported after the diagnosis had been made, so current screen habits may partly reflect adaptations that followed it, e.g., the use of tablets or videos for calming, as a reward, or within home-based therapy, as well as differential recall by parents

who have reflected on possible causes. Second, the models were adjusted only for age and sex. Respondents in the case group were less often university educated and less often reported adequate income, and lower maternal education and lower family income were both associated with more screen time in the Australian cohort [11]; residual socioeconomic confounding is therefore likely. When we took income into account, the screen-time OR fell from 6.51 to 5.65; taking education into account barely changed it. The family circumstances we could measure, therefore, explain only a small part of the link. Those measures are rough, however. Four in five respondents had a university education, income was judged by a single question about whether it felt adequate, and neither tells us about parents' working hours, how crowded the home is, how many adults live there or whether the child has access to early intervention. Each of these can influence both how much a child uses screens and how a diagnosis is reached. Some confounding probably remains. In our data, families with less education and lower income revealed both more screen time and more children with ASD, so any remaining confounding would most likely make the screen-time OR look larger than it really is. Third, with 33 cases, the confidence interval is wide (2.44–17.33), and its lower limit approaches the effect sizes reported in cohort studies. Treating the bands as a single score gave a significant result, but looking at each band separately showed a jump above 2 h/day rather than a steady rise. Even a steady rise would not prove that screens cause autism. Because parents answered after their children had been diagnosed, a pattern like this could just as well run the other way or reflect how families adapt: parents may reach for a phone or tablet more often to calm a child whose social and communication difficulties are more severe.

Reverse causation is at least as plausible as a causal effect of screens. Children with emerging social-communication difficulties may find screens more predictable and rewarding than social interaction, and the authors of the meta-analysis noted explicitly that their findings do not rule out the hypothesis that children with ASD prioritise screen activities to avoid social challenges [10]. Several autistic characteristics could draw a child toward screens, including a preference for predictable, repetitive visual material, intense interests that screens can feed on demand, sensory sensitivities that make busy social settings uncomfortable, and communication difficulties that make screen-based activity easier than conversation. Most young people with ASD spend most of their free time on television and video games and do so in a higher proportion than in other disability groups [32], and children with ASD spend more time on video games than do their siblings [17]. Caregivers facing challenging behaviour may also rely on devices for calming, a practice associated with weaker emotion regulation in preschoolers [33], and in boys and children high in temperamental surgency, with later emotional reactivity [16]. Longitudinal evidence on the direction of the effect is mixed: in the JECS, lower communication scores at 1 and 2 years predicted more subsequent television and DVD time, as well as the converse [6], whereas in a Singaporean cohort, screen time predicted later social and non-social behavioural traits, but earlier social traits did not predict later screen time [12]. A cross-sectional study cannot adjudicate between these possibilities. Screen use in autistic children is also not all of one kind. Some families use tablets for speech and language apps, visual schedules or augmentative communication, and technology-based interventions are part of current autism support [32]; others use screens to manage transitions or distress. Our questionnaire recorded only duration, so it cannot separate these uses from entertainment viewing, and part of the higher use among cases may be therapeutic or supportive use that began after diagnosis. What our data show is that children with ASD in this setting were more heavily exposed to screens than were their age-matched peers, not that screen exposure contributes to ASD.

If screen exposure does contribute to the developmental profile of these children, the most plausible pathway is displacement of reciprocal caregiver–child interaction rather than a specific effect on the aetiology of autism. This pathway was not tested here. We did not measure caregiver–child interaction, language exchange or emotion regulation, so the explanation is a hypothesis drawn from other studies. Objective home audio recordings show that more screen time between 12 and 36 months is associated with fewer adult words, child vocalisations and conversational turns, with the largest per-minute reductions at 36 months [8]; heavier screen use in early childhood predicts poorer performance on developmental screening tests [3], weaker language skills [4] and delays in the communication and problem-solving domains [5,6]; moreover, daily caregiver–child play has been associated with fewer ASD-like symptoms at 2 years [31]. Such effects would be expected to accentuate social-communication difficulties in children already at elevated familial or genetic likelihood rather than to produce ASD de novo. The timing of exposure could not be examined adequately. The derived age at first exposure reflects current use rather than onset (Section 2.1) and cannot address this question. The earliest years of life, in which exposure was measured prospectively at 1 year in the JECS and at 2 years in the Australian cohort [9,11], are therefore poorly captured by our data.

A family history of ASD was reported for 18.2% of cases and 4.5% of controls, comprising the finding with the clearest biological rationale. It is, however, based on only nine children, it narrowly missed significance once we corrected for multiple testing ($q = 0.057$), and its confidence interval is wide, so the estimate is imprecise. Population-based family and twin studies attribute approximately 50% to 80% of the variance in ASD liability to additive genetic factors [13,14]. In the Swedish national cohort, the relative recurrence risk was 10.3 for full siblings, about 3 for half-siblings and 2.0 for cousins [13], and prospective infant-sibling studies found that 18.7% and, more recently, 20.2% of younger siblings of autistic children themselves received an ASD classification by the age of 3 years [34,35]. Because our questionnaire did not specify the degree of relationship, the item combines first-degree relatives, among whom recurrence is highest, with more distant relatives, among whom it is modest; an odds ratio of about 5 is compatible with such a mixture, although the interval is wide (1.19–24.51). Two non-genetic mechanisms may also contribute. Families already familiar with autism may recognise early signs sooner and pursue assessment more insistently so that an affected child is more likely to have been diagnosed by preschool age, and parents of a diagnosed child may be more likely to know of, or disclose, ASD in relatives. Both processes would inflate the estimate. That screen exposure and family history were both retained in the joint model means only that the screen-time association was not explained by this one item. A single yes/no question that mixes close and distant relatives and depends on what parents know and choose to disclose cannot stand in for genetic susceptibility or for the wider family influences on both autism and media use, so the joint model does not rule out shared familial liability. The high recurrence documented in sibling studies supports routine enquiry about family history and close developmental surveillance of younger siblings of autistic children [35].

Parent-reported birth complications were more frequent among cases (50.0% versus 17.8%; adjusted OR 4.70), and the 5-min Apgar score was slightly lower (median 9, IQR 8–9 versus median 9, IQR 9–10; $p = 0.036$). This pattern is consistent with meta-analytic evidence. Across 40 studies, abnormal presentation, umbilical-cord complications, foetal distress, birth injury or trauma, a low 5-min Apgar score and several neonatal conditions were associated with autism, and the authors concluded that a broad class of conditions reflecting general compromise of perinatal and neonatal health, rather than any single factor, may increase risk [18]. An umbrella review likewise found that birth complications associated with trauma, ischaemia or hypoxia showed strong links to ASD [19]. Our item,

however, did not specify the nature of the complication, was answered by only 63 of the 99 families, and was not verified against obstetric records. The missing answers all came from questionnaires completed in the first months of the study, and the association weakened when they were recoded (OR 2.93 to 4.58) and in the conditional analysis (OR 2.81, 95% CI 0.81–9.82). A broad, unverified yes/no item is not comparable with the specific obstetric and neonatal measures used in the studies cited above, so this is the least secure of the three findings and should be regarded as exploratory. Mothers of a child with a developmental diagnosis may review their obstetric history more thoroughly than other mothers, which would inflate the association, and perinatal adversity may in part be a marker of pre-existing atypical foetal development rather than a cause.

Birth weight, gestational age and preterm birth data showed no clear evidence of a difference between groups in this sample. Preterm birth was somewhat more frequent among cases (22.6% versus 14.3%; adjusted OR 1.78, 95% CI 0.58–5.48), but the study had little power to detect associations of the modest size typical of individual perinatal and prenatal factors, and meta-analyses have found insufficient evidence to implicate any single prenatal or perinatal exposure in autism aetiology [18,36].

Boys made up 75.8% of cases, with a male-to-female ratio of 3.1:1. This is close to the ratio of about 3:1 estimated by Loomes et al. for high-quality studies (3.32) and for studies that screened the general population (3.25), and lower than the ratio of 4.56, found when only children with a pre-existing diagnosis were ascertained [37], and the median of 4.2 reported in a global systematic review of prevalence studies [38]. With only eight girls and no assessment of undiagnosed children, these data cannot show whether girls were adequately identified in this clinic. Male sex showed no clear evidence of an association with case status (adjusted OR 1.78, 95% CI 0.70–4.57), as expected by design: controls were preferentially matched by sex, so the control group was itself predominantly male (63.6%). For the same reasons, our data cannot address the male-specific association between early screen time and ASD reported in the JECS [9].

Age at diagnosis was not recorded. Current age in children who already have a diagnosis is not comparable with age at diagnosis in other studies, so we draw no conclusion about how early children in this setting are diagnosed. Some controls may nonetheless be children whose autism will be recognized later. The direction of bias from this misclassification cannot be assumed. Non-differential error in a binary outcome tends to pull associations toward the null only on average [39], and here, recognition and referral may themselves depend on family history, socioeconomic circumstances or developmental concerns, any of which could push estimates in either direction.

Completion of the national vaccination schedule showed no clear evidence of an association with case status (77.4% of cases versus 85.9% of controls; adjusted OR 0.58, 95% CI 0.19–1.75). The point estimate lies on the protective side, and the interval is wide, so our data alone cannot exclude modest associations in either direction. They are, however, consistent with the Danish nationwide cohort of 657,461 children, in which measles–mumps–rubella (MMR) vaccination was not associated with autism (fully adjusted hazard ratio [HR] 0.93, 95% CI 0.85–1.02), including among children with a sibling history of autism [40], and with an umbrella review concluding that vaccination is unrelated to the risk of ASD [19]. The slightly lower completion among cases may reflect deferral of vaccination once developmental concerns had arisen, a process that would, if anything, generate a spurious inverse association. Given the small sample and wide interval, this secondary analysis adds little to the large cohort evidence and supports no conclusion of its own.

Several practical implications follow, provided that the cross-sectional nature of the data is kept in view. First, screen use was common in both groups: only 42.4% of controls and 24.2% of cases fell in the lowest category (<1 h/day), whereas the World Health

Organization recommends no more than 1 h of sedentary screen time per day for children aged 2 to 4 years, with less being better, and the Canadian Paediatric Society recommends limiting routine screen time to about 1 h or less per day between 2 and 5 years of age [1,2]. The American Academy of Paediatrics 2026 policy statement has moved away from fixed limits towards content, context and family routines, but suggests less than 1 h/day for toddlers and preschoolers when families want a specific limit [33]. The ≥2 h/day threshold used in our analysis is therefore more permissive than current guidance, and the contrast between groups arose largely at levels that guidance already discourages. Second, asking about daily screen time and about a family history of ASD costs little in a routine consultation, and both fit naturally into routine developmental history-taking. Using them to screen for ASD is a different matter. We did not test how well these questions identify autistic children, and any role in developmental surveillance or referral is a proposal that would need prospective evaluation against standard surveillance and screening [41]. Whether such enquiry aids in earlier recognition is a hypothesis that would need prospective evaluation because we measured screen use after diagnosis and did not test whether asking about it improves identification or referral; nothing in our data indicates that limiting screen time prevents autism, and advice to families should say so plainly. Third, for children who already have a diagnosis, heavy screen use may displace reciprocal parent–child talk [8], so a discussion of media habits is a reasonable part of family guidance, whatever the direction of the association. That discussion should start by determining what screens are used for. For some autistic children, apps and videos support communication, learning or self-regulation [42], and advice that treats all screen time as harmful risks implying that parents caused their child's autism, which nothing in these data supports. Finally, epidemiological data on ASD in Romania remain scarce, and the only Romanian prevalence study we could identify was published as a conference abstract [43]. Community-based studies with prospectively recorded exposure and standardized diagnostic assessment are needed before local figures can inform policy.

Strengths and Limitations

The main strengths of this study are the verification of every case against specialist documentation, the use of a consecutive clinic series as the sampling frame, age matching that removed the age imbalance between children with and without ASD, and a set of sensitivity analyses on the main estimates. Several limitations must be acknowledged. First, exposure and outcome were measured at the same time, and exposure was reported after the diagnosis, so the temporal sequence cannot be established, and differential reporting by parents of diagnosed children cannot be excluded. Second, no standardised diagnostic instrument was administered within the study, the records consulted varied in detail, and children whose autism had not yet come to clinical attention would have been classified as controls; the direction of the resulting bias cannot be assumed. Third, because two controls were selected for each case, the proportion of cases in the analysis sample (one third) is fixed by design; for this reason, we report odds ratios and not risk differences or risk ratios. Fourth, with 33 cases, the confidence intervals are wide; no more than two covariates could be added to age and sex in multivariable models; socioeconomic indicators could be added only in small exploratory models; power was low for rare exposures such as family history; and the female stratum contained only eight cases. Fifth, screen time was parent-reported in coarse duration bands without information on content, context or co-viewing, which may modify the associations of screen media with child behaviour [12], and without objective measurement of the kind now possible with audio-recording technology [8]; age at first exposure was derived indirectly, is imprecise, and was not used for inference; the family-history item did not record the degree of relationship; and the birth-complication item was

unverified and missing for 36 of 99 children. Sixth, the sample was drawn from a single outpatient clinic; respondents were almost exclusively mothers; 79.8% of respondents in the analysis sample had tertiary education; recruitment extended over 768 days, during which referral patterns may have changed; and the findings cannot be extrapolated to infants or to school-age children. Several details were not recorded in the study dataset, including which diagnostic criteria and tools the specialists used, how children were referred to the clinic, and the exact wording of the screen-time questions. Because only the matched children were kept for analysis, we could not describe the children who were not chosen as controls or compare them with those who were. Recruiting from a single clinic may also have shaped the comparison in ways we cannot measure. We do not know why children came to the clinic or how its controls differ from children in the wider community, so the findings may apply only to similar clinic populations.

These different limitations do not push the results the same way, and their combined effect cannot be measured. Parents reported after diagnosis, some socioeconomic confounding probably remains, and the family-history and birth-complication questions were imprecise; each of these could have made the estimates too high. Missing data made the birth-complication estimate less precise, and children with undiagnosed autism among the controls could have moved the estimates in either direction. The results should therefore be read as a starting point for new hypotheses, not as firm conclusions.

5. Conclusions

In age-matched Romanian children aged 2–6 years attending a paediatric outpatient clinic, daily screen use of 2 h or more and a family history of ASD were associated with a documented ASD diagnosis, and parent-reported birth complications were also more frequent among cases. These are exploratory associations from a small clinic sample: family history did not survive correction for multiple testing within the main family, and birth complications lost significance when the matched sets were analysed conditionally. The screen-time association was concentrated above 2 h/day and was considerably larger than pooled international estimates, a discrepancy that probably reflects the cross-sectional, clinic-based design, post-diagnostic reporting and unmeasured socioeconomic confounding as much as any true effect. Its direction cannot be determined from these data, and screen exposure should not be presented to families as a cause of autism. Completion of the national vaccination schedule, birth weight, gestational age and preterm birth showed no clear evidence of an association, with wide confidence intervals. Prospective, community-based studies with objective exposure measurement and standardized diagnostic assessment are needed to establish whether early screen exposure contributes to, or results from, emerging autistic traits.

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Institutional Review Board Statement: The study was conducted in accordance with the Declaration of Helsinki and approved by the Research Ethics Committee of Victor Babeș University of Medicine and Pharmacy, Timișoara (protocol code 17, approved 29 February 2024).

Informed Consent Statement: Informed consent was obtained from the parent or legal guardian of every participating child before completion of the survey.

Data Availability Statement: The anonymised dataset and analysis scripts supporting the findings of this study are available from the corresponding author on reasonable request.

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Conflicts of Interest: The authors declare no conflicts of interest.

Appendix A

Tables A1–A7 give the questionnaire items and supplementary analyses.

Table A1. Summary of the questionnaire items used in the analysis and their recorded responses.

Domain	Item	Response Options
Outcome	"Does your child display characteristics associated with autism spectrum disorders, or has your child been diagnosed with a disorder within this spectrum?"	Yes/No
Family history	Whether anyone in the family has been diagnosed with autism or an autism spectrum condition	Yes/No; degree of relationship not asked
Birth complications	Whether there were complications at birth	Yes/No
Any screen exposure	Whether the child is exposed to screens	Yes/No
Daily screen time	Average daily screen time (reference period not specified)	<1 h/day; 1–2 h/day; 2–3 h/day; 3–4 h/day; >4 h/day
Total duration of screen exposure	How long the child has been exposed to screens	1 month; 2 months; 6 months; 12 months; >12 months

The questionnaire was completed online in Romanian. The outcome item is quoted in an English translation; the other items are summarised from the study dataset, and response options are given as recorded.

Table A2. Missing data by variable in cases and controls.

Variable	Missing in Cases ($n = 33$)	Missing in Controls ($n = 66$)	p
Respondent age	3 (9.1%)	4 (6.1%)	0.683
Gestational age/preterm birth	2 (6.1%)	3 (4.5%)	1.000
Apgar score at 5 min	0 (0.0%)	3 (4.5%)	0.549

Table A2. *Cont.*

Variable	Missing in Cases ($n = 33$)	Missing in Controls ($n = 66$)	p
Birth complications	15 (45.5%)	21 (31.8%)	0.193
Twin pregnancy	17 (51.5%)	24 (36.4%)	0.195
Smoking in pregnancy	13 (39.4%)	28 (42.4%)	0.831
Urinary tract infection in pregnancy	13 (39.4%)	28 (42.4%)	0.831
Planned pregnancy	10 (30.3%)	15 (22.7%)	0.466
Disturbed sleep in pregnancy	11 (33.3%)	27 (40.9%)	0.517
SARS-CoV-2 in pregnancy	0 (0.0%)	1 (1.5%)	1.000
SARS-CoV-2 in the child	0 (0.0%)	1 (1.5%)	1.000
Vaccination schedule	2 (6.1%)	2 (3.0%)	0.599
Chronic disease	2 (6.1%)	2 (3.0%)	0.599
Maternal body mass index	1 (3.0%)	2 (3.0%)	1.000
Total duration of screen exposure	1 (3.0%)	1 (1.5%)	1.000
Derived age at first screen exposure	1 (3.0%)	7 (10.6%)	0.263

p from the Fisher's exact test comparing the proportion missing. Gestational age and preterm birth share the same missing values. No data were missing for child age and sex, residence, respondent sex, education, marital status, income adequacy, number of children, firstborn status, family history, caregiver ill health, birth weight, birth length, medically assisted conception, gestational weight gain, any screen exposure or daily screen time. The seven children with no screen exposure have no derived age at first exposure.

Table A3. Children with and without birth-complication data and sensitivity analyses for the birth-complication association.

Characteristic	Birth-Complication Item Missing ($n = 36$)	Item Answered ($n = 63$)	p
Confirmed ASD	15/36 (41.7%)	18/63 (28.6%)	0.193
Male sex	27/36 (75.0%)	40/63 (63.5%)	0.271
Urban residence	19/36 (52.8%)	39/63 (61.9%)	0.403
Tertiary education of respondent	27/36 (75.0%)	52/63 (82.5%)	0.438
Perceived adequate income	16/36 (44.4%)	28/63 (44.4%)	1.000
Family history of ASD	3/36 (8.3%)	6/63 (9.5%)	1.000
Screen time ≥ 2 h/day	12/36 (33.3%)	16/63 (25.4%)	0.488
Preterm birth	3/34 (8.8%)	13/60 (21.7%)	0.155
Married	32/36 (88.9%)	52/63 (82.5%)	0.562
Firstborn	30/36 (83.3%)	43/63 (68.3%)	0.154
Child age, months, median (IQR)	48 (43–60)	36 (26–48)	<0.001

Table A3. *Cont.*

Characteristic	Birth-Complication Item Missing ($n = 36$)	Item Answered ($n = 63$)	p
Birth weight, g, median (IQR)	3325 (3098–3638)	3200 (2900–3500)	0.058
Questionnaire completed	20 March–7 August 2024 (all 36)	21 March 2024–April 2026	—
Sensitivity analyses for birth complications, aOR (95% CI)			
Complete cases (main analysis, $n = 63$)	4.70 (1.40–15.75)		
Missing coded as "no" ($n = 99$)	2.93 (0.97–8.90)		
Missing coded as "yes" ($n = 99$)	3.63 (1.41–9.37)		
Missing-indicator model ($n = 99$)	4.58 (1.36–15.38); missing category 3.10 (1.06–9.10)		
Conditional logistic regression (49 children, 12 informative sets)	2.81 (0.81–9.82)		

p from the Fisher's exact test or Mann–Whitney U test. Sensitivity models are adjusted for child age and sex (conditional model: sex, within matched sets).

Table A4. p values before and after false-discovery-rate adjustment for all age- and sex-adjusted tests.

Exposure	n	aOR (95% CI)	p	q, Table 2 Family (8 Tests)	q, All Tests (22)
Screen time ≥ 2 h/day	99	6.51 (2.44–17.33)	<0.001	0.001	0.004
Screen time, per band	99	1.88 (1.26–2.82)	0.002	0.009	0.024
Family history of ASD	99	5.41 (1.19–24.51)	0.029	0.057	0.157
Birth complications	63	4.70 (1.40–15.75)	0.012	0.032	0.089
Caregiver ill health	99	2.24 (0.85–5.89)	0.101	0.162	0.202
Male sex	99	1.78 (0.70–4.57)	0.229	0.305	0.406
Completed vaccination schedule	95	0.58 (0.19–1.75)	0.331	0.331	0.455
Preterm birth	94	1.78 (0.58–5.48)	0.313	0.331	0.455
Urban residence	99	0.92 (0.39–2.16)	0.847	—	0.887
Married respondent	99	0.36 (0.12–1.12)	0.078	—	0.175
Perceived adequate income	99	0.39 (0.16–0.95)	0.039	—	0.171
Firstborn	99	0.57 (0.22–1.45)	0.240	—	0.406
Smoking in pregnancy	58	1.33 (0.41–4.37)	0.637	—	0.779
Urinary tract infection in pregnancy	58	2.76 (0.89–8.60)	0.080	—	0.175
SARS-CoV-2 in pregnancy	98	1.01 (0.27–3.82)	0.984	—	0.984

Table A4. *Cont.*

Exposure	*n*	aOR (95% CI)	*p*	q, Table 2 Family (8 Tests)	q, All Tests (22)
SARS-CoV-2 in the child	98	0.41 (0.15–1.09)	0.074	—	0.175
Tertiary education of respondent	99	0.37 (0.13–1.05)	0.062	—	0.175
Birth weight < 2500 g	99	0.72 (0.13–3.84)	0.696	—	0.785
Apgar score < 8 at 5 min	96	7.66 (0.80–73.49)	0.078	—	0.175
Birth weight, per g	99	1.0005 (0.9996–1.0013)	0.292	—	0.455
Gestational age, per week	94	0.95 (0.74–1.23)	0.714	—	0.785

All models include child age in months and sex (the male-sex model includes age only). q values from the Benjamini–Hochberg procedure. Exposures with complete or quasi-complete separation (any screen exposure, chronic disease) could not be modelled and are not included. The derived age at first exposure was not included because it does not reflect the age at which screen use began (see Section 2.1).

Table A5. Band-specific associations between daily screen time and confirmed ASD.

Daily Screen Time	Cases, *n*	Controls, *n*	aOR (95% CI)
<1 h/day	8	28	Reference
1–2 h/day	7	28	0.89 (0.28–2.80)
2–3 h/day	9	5	6.21 (1.60–24.06)
3–4 h/day	7	3	7.97 (1.58–40.22)
>4 h/day	2	2	3.65 (0.43–30.62)
Linear term, per band	—	—	1.88 (1.26–2.82)

Adjusted for child age, in months, and sex. Likelihood-ratio test of the linear (ordinal) coding against the five-category coding: $\chi^2 = 5.17$, 3 df, $p = 0.16$. The >4 h/day estimate rests on four children.

Table A6. Joint logistic model for confirmed ASD ($n = 99$; 33 cases).

Term	OR (95% CI)	*p*	Firth OR (95% CI)
Screen time ≥ 2 h/day	7.56 (2.71–21.06)	<0.001	6.79 (2.59–19.00)
Family history of ASD	7.42 (1.52–36.18)	0.013	6.36 (1.50–31.17)
Child age, per month	1.00 (0.97–1.03)	0.895	—
Male sex	1.46 (0.49–4.34)	0.499	—

Model fit: log-likelihood −51.40 (null −63.01); likelihood-ratio $\chi^2 = 23.2$, 4 df, $p < 0.001$; McFadden pseudo-$R^2 = 0.18$; AIC 112.8; Hosmer–Lemeshow $\chi^2 = 3.25$, 8 df, $p = 0.92$; area under the ROC curve 0.77. Correlation between the two exposures: r = −0.04. Events per parameter: 8.2.

Table A7. Screen time ≥ 2 h/day and confirmed ASD by respondent education and perceived income adequacy ($n = 99$).

Group	*n*	Screen ≥ 2 h/day, *n* (%)	Confirmed ASD, *n* (%)
Respondent with tertiary education	79	21 (26.6%)	23 (29.1%)
Respondent without tertiary education	20	7 (35.0%)	10 (50.0%)
p (Fisher's exact test)		0.58	0.11
Perceived adequate income	44	7 (15.9%)	10 (22.7%)

Table A7. *Cont.*

Group	*n*	Screen ≥ 2 h/day, *n* (%)	Confirmed ASD, *n* (%)
Perceived inadequate income	55	21 (38.2%)	23 (41.8%)
p (Fisher's exact test)		0.024	0.055

Counts from the 99 children in the age-matched sample; no missing data for education or income. These are unadjusted descriptive comparisons.

References

1. Guidelines on Physical Activity, Sedentary Behaviour and Sleep for Children Under 5 Years of Age. Available online: https://iris.who.int/items/677e3562-1deb-48ec-bb1e-4bc8aad3ea09 (accessed on 26 September 2026).
2. Ponti, M. Screen Time and Preschool Children: Promoting Health and Development in a Digital World. *Paediatr. Child Health* **2023**, *28*, 184–202. [CrossRef] [PubMed]
3. Madigan, S.; Browne, D.; Racine, N.; Mori, C.; Tough, S. Association Between Screen Time and Children's Performance on a Developmental Screening Test. *JAMA Pediatr.* **2019**, *173*, 244–250. [CrossRef] [PubMed]
4. Gath, M.; Horwood, L.J.; Gillon, G.; McNeill, B.; Woodward, L.J. Longitudinal Associations between Screen Time and Children's Language, Early Educational Skills, and Peer Social Functioning. *Dev. Psychol.* **2026**, *62*, 638–652. [CrossRef] [PubMed]
5. Takahashi, I.; Obara, T.; Ishikuro, M.; Murakami, K.; Ueno, F.; Noda, A.; Onuma, T.; Shinoda, G.; Nishimura, T.; Tsuchiya, K.J.; et al. Screen Time at Age 1 Year and Communication and Problem-Solving Developmental Delay at 2 and 4 Years. *JAMA Pediatr.* **2023**, *177*, 1039–1046. [CrossRef] [PubMed]
6. Yamamoto, M.; Mezawa, H.; Sakurai, K.; Mori, C.; Japan Environment and Children's Study Group. Screen Time and Developmental Performance Among Children at 1–3 Years of Age in the Japan Environment and Children's Study. *JAMA Pediatr.* **2023**, *177*, 1168–1175. [CrossRef] [PubMed]
7. Lord, C.; Elsabbagh, M.; Baird, G.; Veenstra-Vanderweele, J. Autism Spectrum Disorder. *Lancet* **2018**, *392*, 508–520. [CrossRef] [PubMed]
8. Brushe, M.E.; Haag, D.G.; Melhuish, E.C.; Reilly, S.; Gregory, T. Screen Time and Parent-Child Talk When Children Are Aged 12 to 36 Months. *JAMA Pediatr.* **2024**, *178*, 369. [CrossRef] [PubMed]
9. Kushima, M.; Kojima, R.; Shinohara, R.; Horiuchi, S.; Otawa, S.; Ooka, T.; Akiyama, Y.; Miyake, K.; Yokomichi, H.; Yamagata, Z.; et al. Association Between Screen Time Exposure in Children at 1 Year of Age and Autism Spectrum Disorder at 3 Years of Age: The Japan Environment and Children's Study. *JAMA Pediatr.* **2022**, *176*, 384–391. [CrossRef] [PubMed]
10. Ophir, Y.; Rosenberg, H.; Tikochinski, R.; Dalyot, S.; Lipshits-Braziler, Y. Screen Time and Autism Spectrum Disorder: A Systematic Review and Meta-Analysis. *JAMA Netw. Open* **2023**, *6*, e2346775. [CrossRef] [PubMed]
11. Lin, P.-I.; Wu, W.T.; Guo, Y.-L.L. Screen Time Before 2 Years of Age and Risk of Autism at 12 Years of Age. *JAMA Pediatr.* **2025**, *179*, 90–91. [CrossRef] [PubMed]
12. Aishworiya, R.; Magiati, I.; Phua, D.; Daniel, L.M.; Shek, L.P.; Chong, Y.S.; Gluckman, P.D.; Meaney, M.J.; Law, E.C. Are There Bidirectional Influences Between Screen Time Exposure and Social Behavioral Traits in Young Children? *J. Dev. Behav. Pediatr. JDBP* **2022**, *43*, 362–369. [CrossRef] [PubMed]
13. Sandin, S.; Lichtenstein, P.; Kuja-Halkola, R.; Larsson, H.; Hultman, C.M.; Reichenberg, A. The Familial Risk of Autism. *JAMA* **2014**, *311*, 1770–1777. [CrossRef] [PubMed]
14. Bai, D.; Yip, B.H.K.; Windham, G.C.; Sourander, A.; Francis, R.; Yoffe, R.; Glasson, E.; Mahjani, B.; Suominen, A.; Leonard, H.; et al. Association of Genetic and Environmental Factors with Autism in a 5-Country Cohort. *JAMA Psychiatry* **2019**, *76*, 1035–1043. [CrossRef] [PubMed]
15. La Monica, I.; Di Iorio, M.R.; Sica, A.; Rufino, F.; Sotira, C.; Pastore, L.; Lombardo, B. Autism Spectrum Disorder: Genetic Mechanisms and Inheritance Patterns. *Genes* **2025**, *16*, 478. [CrossRef] [PubMed]
16. Radesky, J.S.; Kaciroti, N.; Weeks, H.M.; Schaller, A.; Miller, A.L. Longitudinal Associations Between Use of Mobile Devices for Calming and Emotional Reactivity and Executive Functioning in Children Aged 3 to 5 Years. *JAMA Pediatr.* **2023**, *177*, 62. [CrossRef] [PubMed]
17. Mazurek, M.O.; Wenstrup, C. Television, Video Game and Social Media Use among Children with ASD and Typically Developing Siblings. *J. Autism Dev. Disord.* **2013**, *43*, 1258–1271. [CrossRef] [PubMed]
18. Gardener, H.; Spiegelman, D.; Buka, S.L. Perinatal and Neonatal Risk Factors for Autism: A Comprehensive Meta-Analysis. *Pediatrics* **2011**, *128*, 344–355. [CrossRef] [PubMed]
19. Modabbernia, A.; Velthorst, E.; Reichenberg, A. Environmental Risk Factors for Autism: An Evidence-Based Review of Systematic Reviews and Meta-Analyses. *Mol. Autism* **2017**, *8*, 13. [CrossRef] [PubMed]
20. Committee on Public Education. Children, Adolescents, and Television. *Pediatrics* **2001**, *107*, 423–426. [CrossRef] [PubMed]

21. Dilshad, A.; Khan, M.H.; Sai Sujana, C.; Ahsan, A.; Mehta, F.; Meshram, Y.; Singh, P.K.; Verma, A.; Singh, A.K.; Akbar, A. The Relationship between Autism Spectrum Disorder and Screen Time in Children: A Literature Review. *Ann. Med. Surg.* **2025**, *87*, 4184–4193. [CrossRef] [PubMed]
22. Firth, D. Bias Reduction of Maximum Likelihood Estimates. *Biometrika* **1993**, *80*, 27–38. [CrossRef]
23. Greenland, S.; Mansournia, M.A.; Altman, D.G. Sparse Data Bias: A Problem Hiding in Plain Sight. *BMJ* **2016**, *352*, i1981. [CrossRef] [PubMed]
24. VanderWeele, T.J. Principles of Confounder Selection. *Eur. J. Epidemiol.* **2019**, *34*, 211–219. [CrossRef] [PubMed]
25. Peduzzi, P.; Concato, J.; Kemper, E.; Holford, T.R.; Feinstein, A.R. A Simulation Study of the Number of Events per Variable in Logistic Regression Analysis. *J. Clin. Epidemiol.* **1996**, *49*, 1373–1379. [CrossRef] [PubMed]
26. Benjamini, Y.; Hochberg, Y. Controlling the False Discovery Rate: A Practical and Powerful Approach to Multiple Testing. *J. R. Stat. Soc. Ser. B Stat. Methodol.* **1995**, *57*, 289–300. [CrossRef]
27. Hosmer, D.W.; Lemeshow, S.; Sturdivant, R.X. *Applied Logistic Regression*, 1st ed.; Wiley Series in Probability and Statistics; Wiley: Hoboken, NJ, USA, 2013; ISBN 978-0-470-58247-3.
28. Steyerberg, E.W.; Harrell, F.E.; Borsboom, G.J.J.M.; Eijkemans, M.J.C.; Vergouwe, Y.; Habbema, J.D.F. Internal Validation of Predictive Models. *J. Clin. Epidemiol.* **2001**, *54*, 774–781. [CrossRef] [PubMed]
29. Slobodin, O.; Heffler, K.F.; Davidovitch, M. Screen Media and Autism Spectrum Disorder: A Systematic Literature Review. *J. Dev. Behav. Pediatr. JDBP* **2019**, *40*, 303–311. [CrossRef] [PubMed]
30. Chonchaiya, W.; Nuntnarumit, P.; Pruksananonda, C. Comparison of Television Viewing between Children with Autism Spectrum Disorder and Controls. *Acta Paediatr.* **2011**, *100*, 1033–1037. [CrossRef] [PubMed]
31. Heffler, K.F.; Sienko, D.M.; Subedi, K.; McCann, K.A.; Bennett, D.S. Association of Early-Life Social and Digital Media Experiences with Development of Autism Spectrum Disorder-Like Symptoms. *JAMA Pediatr.* **2020**, *174*, 690–696. [CrossRef] [PubMed]
32. Mazurek, M.O.; Shattuck, P.T.; Wagner, M.; Cooper, B.P. Prevalence and Correlates of Screen-Based Media Use Among Youths with Autism Spectrum Disorders. *J. Autism Dev. Disord.* **2012**, *42*, 1757–1767. [CrossRef] [PubMed]
33. Munzer, T.; Parga-Belinkie, J.; Milkovich, L.M.; Tomopoulos, S.; Ajumobi, T.; Cross, C.; Gerwin, R.; Madigan, S.; Psych, R. Council on Communications and Media. Digital Ecosystems, Children, and Adolescents: Policy Statement. *Pediatrics* **2026**, *157*, e2025075320. [CrossRef] [PubMed]
34. Ozonoff, S.; Young, G.S.; Carter, A.; Messinger, D.; Yirmiya, N.; Zwaigenbaum, L.; Bryson, S.; Carver, L.J.; Constantino, J.N.; Dobkins, K.; et al. Recurrence Risk for Autism Spectrum Disorders: A Baby Siblings Research Consortium Study. *Pediatrics* **2011**, *128*, e488–e495. [CrossRef] [PubMed]
35. Ozonoff, S.; Young, G.S.; Bradshaw, J.; Charman, T.; Chawarska, K.; Iverson, J.M.; Klaiman, C.; Landa, R.J.; McDonald, N.; Messinger, D.; et al. Familial Recurrence of Autism: Updates from the Baby Siblings Research Consortium. *Pediatrics* **2024**, *154*, e2023065297. [CrossRef] [PubMed]
36. Gardener, H.; Spiegelman, D.; Buka, S.L. Prenatal Risk Factors for Autism: Comprehensive Meta-Analysis. *Br. J. Psychiatry J. Ment. Sci.* **2009**, *195*, 7–14. [CrossRef] [PubMed]
37. Loomes, R.; Hull, L.; Mandy, W.P.L. What Is the Male-to-Female Ratio in Autism Spectrum Disorder? A Systematic Review and Meta-Analysis. *J. Am. Acad. Child Adolesc. Psychiatry* **2017**, *56*, 466–474. [CrossRef] [PubMed]
38. Zeidan, J.; Fombonne, E.; Scorah, J.; Ibrahim, A.; Durkin, M.S.; Saxena, S.; Yusuf, A.; Shih, A.; Elsabbagh, M. Global Prevalence of Autism: A Systematic Review Update. *Autism Res. Off. J. Int. Soc. Autism Res.* **2022**, *15*, 778–790. [CrossRef] [PubMed]
39. Jurek, A.M.; Greenland, S.; Maldonado, G.; Church, T.R. Proper Interpretation of Non-Differential Misclassification Effects: Expectations vs Observations. *Int. J. Epidemiol.* **2005**, *34*, 680–687. [CrossRef] [PubMed]
40. Hviid, A.; Hansen, J.V.; Frisch, M.; Melbye, M. Measles, Mumps, Rubella Vaccination and Autism: A Nationwide Cohort Study. *Ann. Intern. Med.* **2019**, *170*, 513–520. [CrossRef] [PubMed]
41. Lipkin, P.H.; Macias, M.M.; Council on Children with Disabilities, Section on Developmental and Behavioral Pediatrics; Norwood, K.W.; Brei, T.J.; Davidson, L.F.; Davis, B.E.; Ellerbeck, K.A.; Houtrow, A.J.; Hyman, S.L.; et al. Promoting Optimal Development: Identifying Infants and Young Children with Developmental Disorders Through Developmental Surveillance and Screening. *Pediatrics* **2020**, *145*, e20193449. [CrossRef] [PubMed]
42. Grynszpan, O.; Weiss, P.L.T.; Perez-Diaz, F.; Gal, E. Innovative Technology-Based Interventions for Autism Spectrum Disorders: A Meta-Analysis. *Autism* **2014**, *18*, 346–361. [CrossRef] [PubMed]
43. Budisteanu, M.; Rad, F.; Tudosie, V.; Zgura, R.; Budisteanu, B.; Papuc, S.M.; Tutulan-Cunita, A.; Arghir, A.; Dobrescu, I. Prevalence Study of Autism Spectrum Disorder in Romania. *Eur. J. Paediatr. Neurol.* **2017**, *21*, e143. [CrossRef]

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