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Cortical Auditory Processing of Simple Stimuli is Altered in Autism: A Meta-analysis of Auditory Evoked Responses

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Abstract

Background: Auditory perceptual abnormalities are common in persons on the autism spectrum. The neurophysiologic underpinnings of these differences have frequently been studied using auditory event-related potentials (ERPs) and event-related magnetic fields (ERFs). However, no study to date has quantitatively synthesized this literature to determine whether early auditory ERP/ERF latencies or amplitudes in autistic persons differ from those of typically developing (TD) controls.

Methods: We searched PubMed and ProQuest for studies comparing (a) latencies/amplitudes of P1/M50, N1b, N1c, M100, P2/M200, and/or N2 ERP/ERF components evoked by pure tones and (b) paired-click sensory gating (P1/N1b amplitude suppression) in autistic individuals and TD controls. Effects were synthesized using Bayesian three-level meta-analysis.

Results: In response to pure tones, autistic individuals exhibited prolonged P1/M50 latencies (g=0.341, 95% CrI [0.166,0.546]), prolonged M100 latencies (g=0.319 [0.093,0.550]), reduced N1c amplitudes (g=−0.812 [−1.278,−0.187]), and reduced N2 amplitudes (g=−0.374 [−0.633,−0.179]). There were no practically significant group differences in P2/M200 latencies, N2 latencies, P1/M50 amplitudes, N1b amplitudes, M100 amplitudes, or P2/M200 amplitudes.

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[1]The terms ‘autistic person’ and ‘person on the autism spectrum’ are the preferred language of the majority of people diagnosed with autism (2,4). Out of respect for these preferences, we use these terms to refer to individuals on the spectrum rather than exclusively using person-first language.

Paired-click sensory gating was also reduced in autistic individuals (g=−0.389 [−0.619,−0.112]), although this effect was primarily driven by smaller responses to the first click stimulus.

Conclusions: Relative to typical controls, autistic individuals demonstrate multiple alterations in early cortical auditory processing of simple stimuli. However, most group differences were modest in size and based on small numbers of heterogeneous studies with variable quality. Future work is necessary to understand whether these neurophysiologic measures can predict clinically meaningful outcomes or serve as stratification biomarkers for the autistic population.

Keywords

Autism Spectrum Disorder; Auditory; Event-related Potential; Electroencephalography (EEG); Magnetoencephalography (MEG); Meta-analysis

Introduction

Autism spectrum disorder (hereafter “autism”) is a lifelong neurodevelopmental condition affecting 1 in 54 children in the United States (1). In addition to the cardinal features of social communicative impairment and repetitive behaviors, many autistic[1] individuals exhibit atypical reactions to sensory stimuli, now considered a core feature of the condition (3). Decreased sound tolerance is particularly common, with a lifetime prevalence of 50–70% (5). Autistic individuals also demonstrate other auditory perceptual abnormalities, including excessive loudness perception (6,7), degraded speech-in-noise perception (7,8), impaired auditory-visual integration (9), and temporal processing deficits (10-15). These widespread differences in auditory perception are hypothesized to contribute to the core symptoms of autism by altering the ways in which autistic children interact with and learn from their environment (16,17).

Many studies investigating the underlying integrity of the central auditory system in autism have used auditory event-related potentials (ERPs) and event-related fields (ERFs), measured by electroencephalography (EEG) and magnetoencephalography (MEG) respectively. In particular, studies have focused on the P1–N1b–P2 ERP complex recorded at frontocentral electrodes (and the analogous M50-M100-M200 ERF), reflecting early stimulus feature extraction and integration in primary/secondary auditory cortex (18-24). In young children, the N1b component has not fully matured, and instead a developmentally-specific N2 component is present with a similar topography and generators (25,26), ostensibly representing some of the same processes (27-31). An additional developmentally-sensitive component, the temporal N1c, is generated in the superior temporal gyrus, reflecting the activation of neural generators underlying stimulus encoding and discrimination (22-24). Although N1c is present in adulthood, it is most prominent in young children, decreasing in amplitude with age (28,32).

Disclosures

ZJW serves as a consultant for Roche. He is also a member of the Family Advisory Committee of the Autism Speaks Autism Treatment Network site at Vanderbilt University. The remaining authors report no biomedical financial interests or potential conflicts of interest.

To date, comparisons of auditory ERP/ERF responses between autistic individuals and typically developing (TD) controls have yielded varied results (15,22,33-37). Multiple studies report delayed P1/M50 and N1/M100 latencies in autistic children and adults, ostensibly reflecting a delay central auditory information transfer (38-45). However, others report a lack of consistent group differences (46-54) or even reduced latencies in autistic participants (55,56,58). Similarly, initial findings of decreased N1b amplitudes in autism (47,55-57,59) failed to replicate on several occasions (43,45,54,60-62). Although less frequently studied, reduced N1c (47,63-65) and N2 (42,51,52,66-69) amplitudes have also been found in autism. These results suggest that autism may be characterized by reduced neural synchrony while processing low-level sound features, although this difference may be limited to specific developmental stages/components.

Another line of research on basic auditory processing in autism has examined the brain's ability to filter out or inhibit the processing of redundant sensory information. Known as sensory gating, this process is typically studied using paired broadband click stimuli (70). P1 and/or N1b amplitudes are smaller to the second click than the first, and the degree of amplitude suppression is thought to quantify how effectively one can "gate out" the second stimulus. Decreased sensory gating has been robustly demonstrated in individuals with schizophrenia and other psychotic disorders (71-74), with sensory gating deficits significantly predicting subjective perceptual abnormalities in this population (75,76). However, findings in autism have been inconsistent (37). Some studies have reported large sensory gating deficits in autism (77-79), whereas others have found minimal group differences (45,80-84) or impaired sensory gating only in a subgroup of participants (85,86).

Given the often-contradictory findings regarding early auditory processing in autism, synthesis of this literature is necessary to reach strong conclusions about the presence and directionality of group differences. Thus, the current study sought to meta-analytically compare auditory cortical activity between autistic individuals and TD controls. We focused only on simple, non-linguistic stimuli in order to better answer the question of whether autism is associated with disruptions in basic auditory stimulus processing, which could serve as the neural substrate of altered auditory perception in this population. Although evoked responses to linguistic stimuli may relate more strongly to social communication abilities (87-90), diagnostic group differences in these responses could be confounded by the higher-order deficits in language processing that frequently accompany autism (91). Within the autism ERP/ERF literature, the most frequently utilized non-linguistic auditory stimuli are pure tones and broadband clicks, with the latter primarily being used to assess sensory gating. Accordingly, in the current meta-analysis, we evaluated differences between individuals with and without autism in (a) the amplitudes and/or latencies of tone-evoked early auditory ERP/ERF components and (b) the strength of paired-click sensory gating.

Methods and Materials

Identification and Selection of Studies

The procedures adhered to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines (92). We searched PubMed and ProQuest for publications on autism and auditory ERPs/ERFs, as defined using a combination of keywords and

filters (see Supplemental Materials). Eligible studies included peer-reviewed journal articles, dissertations, and theses published in English between 1/1/1980 and 1/10/2020.

Included studies satisfied the following criteria upon full-text review: (a) included ≥10 autistic participants, (b) included TD control participants, (c) recorded EEG/MEG while presenting pure tone or paired-click stimuli, (d) examined latencies/amplitudes of obligatory ERPs/ERFs in response to tones (P1/M50, N1b, N1c, M100, P2/M200, N2) or P1/N1b amplitude suppression in a paired-click paradigm, and (e) reported statistics necessary for calculation of Hedges' *g* for outcomes of interest (see Supplemental Materials for more details).

Data Extraction

For each study, we extracted group comparison statistics for all outcomes of interest. Many studies reported multiple effect sizes per outcome (e.g., a given ERP amplitude was recorded at multiple electrodes or in multiple task conditions), all of which were extracted and included in our meta-analytic models. In addition, we extracted a number of putative moderator variables, including recording modality (EEG or MEG), laterality (left, right, or midline/bilateral), stimulus/task characteristics (probability, duration, intensity [in dB HL], frequency, inter-stimulus interval, number of presentations, whether attention was directed to task stimuli), bandpass filter settings, and sample characteristics (*N*/age/sex ratio/IQ) (see Supplemental Materials for details). For sensory gating studies, we additionally recorded whether P1 or N1b amplitude suppression was measured and whether the amplitude suppression was measured as a ratio or difference score. Lastly, we graded all studies on a 28-item measure of study quality derived from EEG/MEG study reporting guidelines (93,94). Quality scores (Supplemental Tables S1-S2) were calculated as the mean of all items applicable to a given study, ranging from 0 to 1 with higher scores reflecting relatively higher study quality.

Statistical Analysis

All analyses were performed in R (95). Descriptive statistics, *t*-values, or *F*-values were used to calculate Hedges' *g* effect sizes (96) using the R package *compute.es* (97). The sign of *g* was standardized such that a negative effect size indicated smaller values of a variable in the autism group (e.g., less positive P1 amplitude, less negative N1b amplitude, faster latency, or less effective P1/N1b amplitude suppression), compared to TD controls.

Meta-analytic models were fit for each outcome with data from three or more eligible studies. We utilized three-level random-effects meta-analysis models to accommodate dependent effects (98-100), treating effect size (level 3) as a random effect nested within study (level 2). Parameter estimation was performed in a Bayesian framework using the R package *brms* (101,102) and weakly informative priors (see Supplemental Materials). We utilized the posterior median and the 95% equal-tailed credible interval (CrI) to summarize all model parameters. Summary estimates were tested against the null hypothesis of $g=0$, as well as the interval null hypothesis that the population difference lies within the interval [−0.1,0.1], which represents differences that we deemed "practically insignificant" (i.e., not worthy of interpretation as meaningful effects (103,104)). Table 1 describes the Bayesian

indices used to determine whether the meta-analytic effects were deemed statistically or practically significant (105).

Publication bias in each meta-analytic model was assessed using contour-enhanced funnel plots (106), as well as the Bayesian selection model approach proposed in (107) and implemented in the *RoBMA* R package (see Supplemental Material for details). This method uses Bayesian model averaging (108) to calculate a publication bias Bayes factor (BF_{PB}; see Table 1 for more details) that quantifies evidence for or against the presence of publication bias (107). Notably, this and other quantitative methods for the assessment of publication bias have not been formally extended to the case of three-level meta-analysis, and thus the *RoBMA* implementation of this model ignores the dependencies among effects from the same study in our sample. Nevertheless, as the Bayesian selection model approach shows both high power and low false-positive rates in simulation studies (107), we believe this to be the most accurate quantitative method for ascertaining publication bias in our data.

To assess study heterogeneity, we calculated the multilevel $\hat{I}^2$ statistic (109) as well as the *ICC*(2) statistic (98), which reflects the proportion of heterogeneity attributable to between-study (level 2) variance. We also calculated a model-based 95% predictive interval (110). Additional measures of heterogeneity are presented in Supplemental Table S3.

Moderation analyses were conducted for outcomes with at least 20 included effect sizes (111) using Bayesian meta-regression. Each meta-regression model was compared to its respective baseline (intercept-only) model using a Bayes factor (BF_{10}; Table 1). As developmental effects on the studied ERP/ERF components were of particular interest, we separately reported the moderating effect of age on each outcome. In addition, we conducted subgroup analyses to test (a) whether summary effects differed for EEG and MEG studies considered separately, (b) whether M50/M100 latency effects and N1c amplitude effects varied between hemispheres (38,46,63), and (c) whether sensory gating effects varied between the P1 and N1b ERP components.

Missing data were handled via 10-fold multiple imputation using the *mice* R package(112). Bayes factors derived from multiply imputed data were defined as the arithmetic mean of the Bayes factors computed using each imputed dataset (113).

Results

The initial literature search identified 851 results. After removing duplicates (n=50), authors ZJW/PGA independently screened remaining abstracts to identify studies eligible for full-text review. Agreement between raters was good (90%, κ=0.631), and all articles flagged by either rater were subjected to full-text review (n=159). The same two authors independently reviewed the full texts of these articles, with good agreement between inclusion/exclusion decisions (85%, κ=0.630). In cases of disagreement, the two authors met and discussed the article until consensus was reached. This process resulted in 31 articles meeting the study inclusion criteria. Forward and backward citation tracing of the included articles uncovered an additional 14 eligible references, for a total of 45 articles included in the meta-analysis

(Table 2). A PRISMA flow diagram is presented in Supplemental Figure S1, and the specific studies included in each meta-analysis are described in Supplemental Tables S4-S11.

P1/M50 Latency

P1/M50 latencies were reported for 14 studies (36 effects; N_{AUT}= 498, N_{TD}=359, mean quality=0.741), with effect sizes ranging from −0.717 to 1.139. The meta-analytic model indicated that autistic individuals have prolonged P1/M50 latencies relative to TD controls (g=0.341, 95% CrI [0.184,0.524], BF_{ROPE}=29.26; Figure 1A). Bayes factors provided strong evidence for a prolongation of M50 latency (BF_{ROPE}=22.94) but weak and inconclusive evidence against a prolongation of P1 latency (BF_{ROPE}=0.53; Table 3). Despite these differences, model comparison provided evidence against a moderating effect of recording modality (BF_{ROPE}=0.20), suggesting a negligible difference in effect size between EEG and MEG studies. Group differences in M50 latency were similar across hemispheres ($\beta_{R\text{-}L}$=0.087 [−0.159, 0.331]; Supplemental Figure S2). There was no moderating effect of age on P1/M50 latency effects, although evidence to suggest the absence of an effect was inconclusive (BF_{10}=0.45). Similarly, no other putative moderator explained significant heterogeneity in P1/M50 latency effects, and Bayes factors provided substantial evidence *against* the majority of tested variables (Table 4).

N1b Latency

N1b latencies were reported in eight studies (25 effects; N_{AUT}=146, N_{TD}=139, mean quality=0.554), with effect sizes ranging from −1.442 to 2.208. There was a small and nonsignificant increase in N1b latency in autism (g=0.172 [−0.594,0.915]), although evidence for practical equivalence between groups was inconclusive (BF_{ROPE}=0.36). Moderator analyses indicated the absence of moderation by sample age (BF_{10}=0.06), and no other tested moderator explained significant heterogeneity in N1b latencies (Table 4).

N1c Latency

N1c latencies were reported in two studies (10 effects; N_{AUT}=56, N_{TD}=31, mean quality = 0.426), with effect sizes ranging from 0.274 to 5.566. As fewer than three unique studies reported N1c data, no meta-analysis was conducted. However, it is notable that all effect sizes were positive and relatively large on average (Mdn=0.738, IQR=[0.503,1.092]), indicating prolonged N1c latencies in participants with autism.

M100 Latency

M100 latencies were reported in 12 studies (37 effects; N_{AUT}=516, N_{TD}=305, mean quality=0.759), with effect sizes ranging from −0.893 to 1.050. The meta-analytic model indicated that autistic individuals have significantly prolonged M100 latencies relative to TD controls (g=0.344 [0.135,0.561], BF_{ROPE}=6.60; Figure 1B). Moderator analyses indicated the absence of moderation by sample age (BF_{10}=0.05), and no other tested moderator explained significant heterogeneity in M100 latency effects (Table 4). However, when analyzing laterality effects, the model predicted a 97.3% chance of right-hemisphere M100 latencies being more prolonged in autism ($\beta_{R\text{-}L}$=0.231 [−0.004,0.464]; Supplemental Figure

S5). Nevertheless, there was inconclusive evidence to suggest that the degree of additional prolongation was larger than 0.1 standard deviations (BF_{ROPE}=0.58).

P2/M200 Latency

P2/M200 latencies were reported in four studies (12 effects; N_{AUT}=83, N_{TD}=79, mean quality=0.658), with effect sizes ranging from −0.982 to 0.687. The meta-analytic model demonstrated small and practically insignificant differences in P2/M200 latency between groups (g=0.057 [−0.608,0.611], BF_{ROPE}=0.21). These conclusions did not change when examining only EEG studies (Table 3).

N2 Latency

N2 latencies were reported in seven studies (12 effects; N_{AUT}= 140, N_{TD}= 145, mean quality=0.736), with effect sizes ranging from −0.390 to 0.872. The meta-analytic model demonstrated small and practically insignificant differences in N2 latency between groups (g=0.047 [−0.280,0.223], BF_{ROPE}=0.07).

P1/M50 Amplitude

P1/M50 amplitudes were reported in eight studies (30 effects; N_{AUT}= 182, N_{TD} =154, mean quality=0.695), with effect sizes ranging from −0.863 to 0.652. The meta-analytic model demonstrated small and practically insignificant differences in P1/M50 amplitudes between autism and TD groups (g=0.042 [−0.198,0.324], BF_{ROPE}=0.07; Figure 1C). Results were similar when examining EEG and MEG studies separately (Table 3).

Model comparisons suggested a significant moderating role of stimulus probability (BF_{10}=5.53; Supplemental Figure S8), with larger group differences in P1/M50 amplitudes for lower-probability stimuli. Notably, despite the significant moderation, the 95% CrI for g continued to include zero at all possible stimulus probabilities. The remaining moderators, including sample age (BF_{10}=0.18), did not explain significant heterogeneity in P1/M50 amplitude effects (Table 4).

N1b Amplitude

N1b amplitudes were reported in seven studies (24 effects; N_{AUT}=205, N_{TD}=131, mean quality=0.619), with effect sizes ranging from −1.108 to 0.539. The meta-analytic model demonstrated small and practically insignificant differences in N1b amplitudes between autism and TD groups (g=−0.162 [−0.497,0.157], BF_{ROPE}=0.21).

Model comparisons revealed a significant moderator effect of sample IQ on the magnitude of N1b amplitude differences (BF_{10}=6.03; Supplemental Figure S9). Studies in which the majority of the autism group had an IQ<70 (k=3) demonstrated practically significant group differences (BF_{ROPE}=7.70), with moderately smaller N1b amplitudes in the autism group (g=−0.533 [−0.842,−0.166]). In contrast, studies where the majority of the autism group was of average or higher intelligence (k=4) reported small and practically insignificant amplitude differences (g=0.123 [−0.202,0.349], BF_{ROPE}=0.16). The remaining moderators, including sample age (BF_{10}=0.22), did not explain significant heterogeneity in N1b amplitude effects (Table 4).

N1c Amplitude

N1c amplitudes were reported in three studies (11 effects; N_{AUT}=101, N_{TD}=102, mean quality=0.540), with effect sizes ranging from −2.048 to −0.418. The meta-analytic model indicated that autistic individuals had substantially smaller N1c amplitudes than TD controls (g=−0.812 [−1.278,−0.187], BF_{ROPE}=9.85). Group differences across hemispheres were minimal (β_{R-L}=−0.106 [−0.698,0.455]; Supplemental Figure S10).

M100 Amplitude

M100 amplitudes were reported in five studies (10 effects; N_{AUT}=145, N_{TD}=87, mean quality=0.740), with effect sizes ranging from −0.323 to 0.307. The meta-analytic model demonstrated small and practically insignificant differences in M100 amplitude between groups (g=0.124, [−0.152,0.398], BF_{ROPE}=0.14; Figure 1D)

P2/M200 Amplitude

P2/M200 amplitudes were reported in five studies (13 effects; N_{AUT}=135, N_{TD}=142, mean quality=0.718), with effect sizes ranging from −0.377 to 0.282. The meta-analytic model demonstrated small and practically insignificant differences in P2/M200 amplitude between groups (g=−0.065 [−0.339,0.176], BF_{ROPE}=0.07). These results were similar when examining only EEG studies (Table 3).

N2 Amplitude

N2 amplitudes were reported in nine studies (27 effects; N_{AUT}=191, N_{TD}=197, mean quality=0.735), with effect sizes ranging from −0.820 to 0.051. The meta-analytic model indicated that autistic individuals had significantly reduced N2 amplitudes compared to TD controls (g=−0.374 [−0.633,−0.179], BF_{ROPE}=14.63). There was significant evidence against the moderating role of sample age (BF_{10}=0.09), and no other tested moderator explained significant heterogeneity in N2 amplitude effects (Table 4).

Sensory Gating (P1/N1b Amplitude Suppression)

Sensory gating amplitude differences or ratios were reported in eight studies (21 effects; N_{AUT}=207, N_{TD}=188), with effect sizes ranging from −1.13 to 0.42. The meta-analytic model indicated that sensory gating (i.e., amplitude suppression of P1 or N1b) was significantly reduced in autism compared to TD controls (g=−0.394 [−0.639,−0.099], BF_{ROPE}=3.63; Figure 2A). Analyzing P1 and N1b gating separately, both point estimates were similar in magnitude, but the 95% CrI of the N1b gating estimate included zero (Table 3). Model comparisons provided substantial evidence that neither the ERP component used to measure sensory gating (BF_{10}=0.17) nor the measure of amplitude suppression (ratio vs. difference score; BF_{10}=0.16) significantly moderated between-group effect sizes. Similarly, we found substantial evidence against the moderating role of sample age (BF_{10}=0.02). No other tested moderator explained significant heterogeneity in sensory gating effects (Table 4).

In order to better understand the drivers of altered sensory gating in autism, P1 amplitudes in response to the two click stimuli of paired-click paradigms were analyzed separately

(Table 3; Figure 2B). Meta-analytic models indicated that responses to click 1 were smaller in amplitude in the autism group (g=−0.286 [−0.505,−0.048], BF_{ROPE}=1.51), while responses to click 2 were of approximately equal amplitudes in the two groups (g=0.121, [−0.237,0.445], BF_{ROPE}=0.16).

Publication Bias

Publication bias was examined using contour-enhanced funnel plots (106), with quantitative estimates of the evidence for or against publication bias derived from selection models (107,114). Contour-enhanced funnel plots (Supplemental Figures S11-S18) were generally symmetrical and did not reflect a significance-chasing bias for the majority of outcomes. These judgments were generally supported by publication bias Bayes factor values (Table 2), which demonstrated substantial evidence *against* the presence of publication bias for sensory gating outcomes (BF_{PB}=0.24) and inconclusive evidence for or against the presence of publication bias in all other cases (all other BF_{PB} between 0.34 and 2.80). Notably, the funnel plot for N1c amplitudes (Supplemental Figure S13) showed some evidence for significancechasing, with the publication bias Bayes factor nearly reaching the threshold for indicating significant publication bias (BF_{PB}=2.80).

Discussion

This is the first meta-analysis to quantitatively synthesize studies of (a) obligatory auditory cortical ERP/ERF responses to tone stimuli and (b) sensory gating performance in paired-click paradigms in autistic individuals and TD controls. We found small but practically significant latency delays for P1/M50 and M100, reduced N2 amplitude, and reduced P1/N1b sensory gating in autistic individuals. A large reduction in N1c amplitude was also observed in persons on the autism spectrum, although we consider this finding preliminary due to the small number of low-quality studies analyzed and borderline evidence for publication bias. In addition, Bayes factors provided moderate to strong evidence that group differences in P2/M200 latency, N2 latency, P1/M50 amplitude, N1b amplitude, M100 amplitude, and P2/M200 amplitude were all too small to be of practical significance (i.e., likely falling within the null region [−0.1,0.1]). Evidence for N1b latency differences was inconclusive, with results trending toward a lack of meaningful group differences. Notably, while the N1b amplitude was not significantly different between groups overall, we found significantly smaller responses in studies predominantly comparing autistic individuals with intellectual disability to neurotypical controls. Our results cannot determine whether this reduction in N1b amplitudes is specific to the co-occurrence of autism and intellectual disability; however, two small studies have reported similar group differences when controls also had intellectual disability (47,59). Selection model analyses indicated a lack of publication bias for sensory gating outcomes, but evidence was inconclusive with regard to the presence or absence of publication bias for all other outcomes.

Moderator and subgroup analyses largely indicated that group differences in ERP/ERF components were independent of stimulus characteristics, basic demographics, and methodological choices such as filter settings. In addition, moderation by age was ruled out in all but one case, extending prior studies that reported no diagnosis by age interactions

for M50/M100 latencies (39,53,115). Thus, while the presence of unmeasured confounds cannot be conclusively ruled out, these results suggest that the observed group differences likely reflect changes in underlying brain activity rather than methodological or statistical artifacts.

On average, autistic individuals exhibited delayed stimulus processing at the level of the primary and secondary auditory cortex, as reflected in prolonged P1/M50 and M100 latencies. These delayed responses are hypothesized to reflect alterations in neural conduction velocity or synaptic transmission within the auditory cortex during low-level stimulus processing (39). It is notable that prolonged ERP latencies in autism have also been found in auditory brainstem responses (116,117), the face-sensitive visual N170 potential (118), and some variants of the auditory mismatch negativity (119), raising the possibility of a more generalized deficit in neural processing speed in autism. However, this interpretation is complicated by a lack of diagnostic group differences in a number of other early and late ERP components, including the visual P1 (118), cognitive P3 (120), early somatosensory responses (121), and several other mismatch negativity variants (119,122), as well as poor correlations between brainstem/cortical ERP latencies (123). Additionally, we found equivalent latencies in later cortical potentials such as P2/M200 and N2, suggesting that differences in autistic auditory information processing may be specific to certain neural circuits or perceptual processes. Nevertheless, additional studies are warranted to better understand the relationships between ERP/ERF latencies across multiple sensory modalities and determine whether multimodal information processing delays meaningfully differentiate autistic individuals from TD controls.

In addition to latency delays, autistic individuals exhibited reduced N1c and N2 amplitudes. The N1c is primarily generated in secondary auditory areas of the superior temporal gyrus and is thought to reflect early stages of stimulus feature encoding and discrimination (23,24,28,47,124). Although the role of this component in auditory processing is not fully understood, tone-evoked N1c component amplitudes and latencies have been associated with language ability in children (63,125,126). The developmentally-specific auditory N2 is a precursor of the adult N1b generated in primary/secondary auditory cortical areas, potentially reflecting either fine-grained acoustic analysis or higher-order encoding of sound content features (31). Interestingly, although we found very clear evidence of reduced N2 amplitudes in autistic individuals, there was little evidence for reduced N1b amplitudes (except in the subset with intellectual disability). This finding raises the possibility that certain auditory processing differences are present in autism during the specific developmental periods when the N2 component is prominent, although this difference may simply be masked in adulthood by the activity of multiple other generators of the N1 waveform (19,23,24). While the functional significance of reduced N1c and N2 amplitudes in autism remains unclear, these changes, presumed to reflect decreased neural synchrony in secondary auditory areas, may underlie some of the documented differences in auditory processing and language development seen in autistic persons (12,127).

An additional focus of our analysis was paired-click sensory gating, as measured by P1 and N1b amplitude suppression. Sensory gating ability was slightly reduced in autistic individuals relative to TD controls, irrespective of the method used to quantify amplitude

suppression. This effect seemed to result from lower-amplitude responses to the first click, rather than higher-amplitude responses to the second click (as would be expected if that information were filtered less efficiently). A similar phenomenon is present in schizophrenia, where reduced click 1 amplitudes contribute to group differences in sensory gating (71,72,128). However, in contrast to individuals on the autism spectrum, individuals with schizophrenia also have substantially elevated P1 amplitudes in response to the second click, likely reflecting a true failure to "gate" sensory information (72). Thus, while our results may appear to suggest a superficial similarity between auditory processing abnormalities in autism and schizophrenia, the reduced "sensory gating" seen in autism likely arises from another mechanism. Interestingly, a reduced P1 amplitude in autism was not noted in tone-evoked responses, potentially indicating an effect unique to the paired-click paradigm. One potential mechanism could involve the brief duration of clicks used in sensory gating paradigms. P1 amplitudes grow substantially with increasing stimulus duration (129,130), and it is possible that this growth is reduced in autistic individuals due to less efficient auditory information transfer. However, there are insufficient data to indicate whether the P1 amplitude group effects change when stimulus duration is varied systematically. Additional work is necessary to understand the mechanism and significance of reduced P1/N1b amplitude suppression in autism, as it is currently unclear whether alterations in this process can provide insight into autistic auditory perception.

The current study has a number of limitations. First, the studies included in our analyses were primarily conducted on school-aged children and adolescents with IQs in the average range; relatively few samples contained toddlers, adults, or individuals with cognitive impairments. Additional research is thus necessary to replicate and extend these findings in the broader autism population. Moreover, although we found no moderating effects of age on any outcome, the truncated chronological age range of published studies and scarcity of longitudinal research on this topic limited our ability to draw conclusions about developmental trends in ERP/ERF measures. An additional limitation is the small number of unique studies included in each meta-analysis, which decrease the replicability of results and provide low power to detect potentially important study-level moderators of effect size (111). A further limitation is the fact that only TD control groups were examined, and therefore we were unable to determine whether the group differences reported in our study are specific to autism (though see 47,52,59,131). Lastly, this study did not examine correlations between auditory cortical responses and behavioral outcomes of clinical relevance, such as language ability or auditory sensory reactivity. Further research characterizing the relationships between behavioral and neurophysiologic measures may provide valuable information on the underlying neural substrates of autism symptomatology.

In conclusion, this meta-analysis suggests that autistic individuals as a group differ from typical controls in multiple aspects of early cortical auditory processing. The majority of these differences are small to moderate in magnitude and in some cases primarily driven by a subset of the autism population (40,85,132). Nonetheless, this synthesis highlights the ERP/ERF metrics that have garnered the greatest support for differentiating autistic persons from their typical peers in processing of simple auditory stimuli. Additional research is necessary to ascertain the degree to which ERP/ERF indices of interest to the present synthesis may be useful for explaining heterogeneity in the autism phenotype, stratifying

autism into meaningful subgroups, predicting differential responses to potential treatments, or elucidating the neural mechanisms by which interventions work in autistic persons (e.g., 133-135).

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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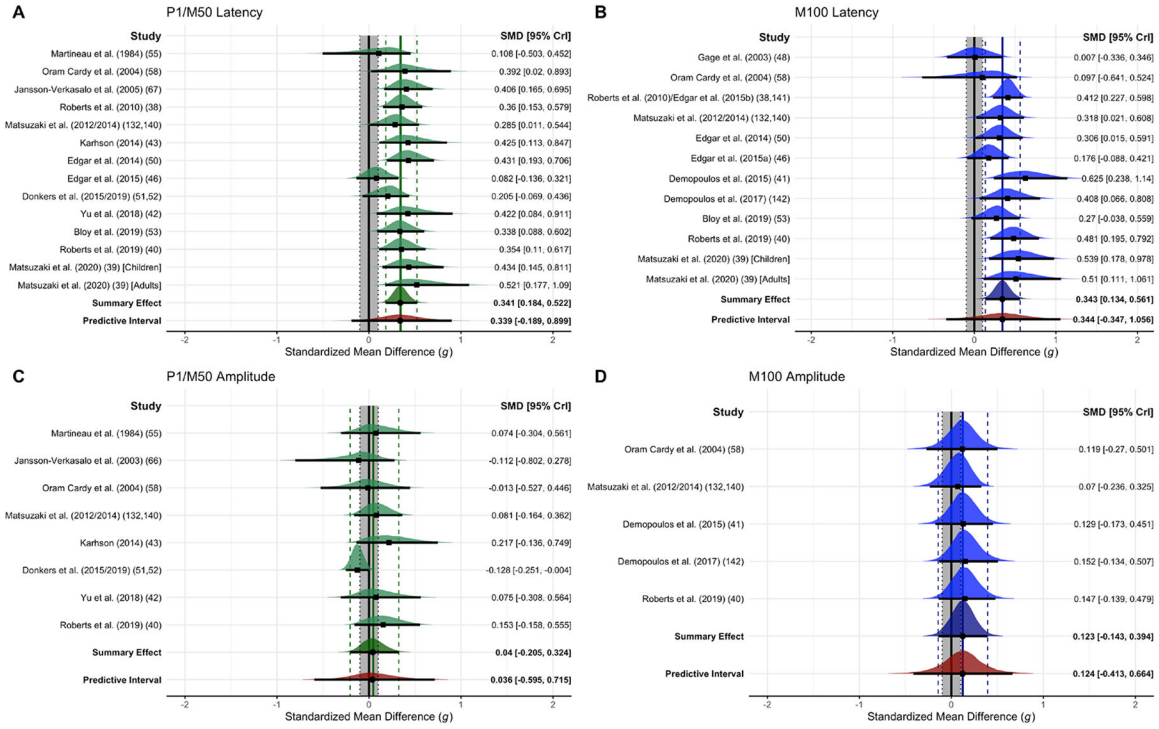

Figure 1.

Posterior density forest plots of: **(A)** P1/M50 latency effects, **(B)** M100 latency effects, **(C)** P1/M50 amplitude effects, and **(D)** M100 amplitude effects. The standardized mean difference (SMD) and 95% credible interval (CrI) for each study represent the posterior distribution of that study's mean effect size, conditional on prior beliefs and the observed data. Negative values of *g* indicate smaller values of a variable in the autism group (i.e., less negative amplitude, faster component latencies), compared to TD controls. The gray shaded areas indicate the region of practical equivalence (ROPE) for each comparison. Raw effect sizes from each study and forest plots for the remaining outcomes can be found in Supplemental Materials.

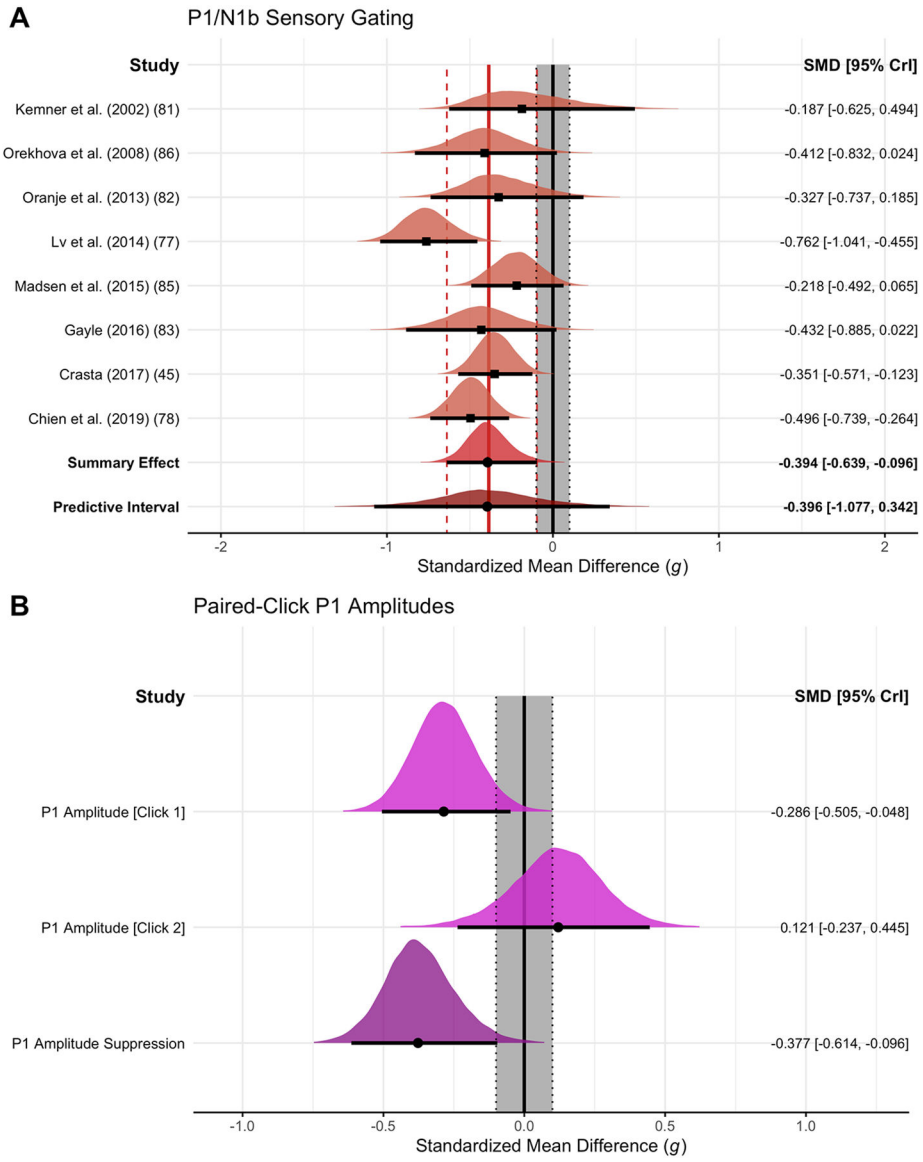

Figure 2.

(A) Posterior density forest plots of P1/N1b amplitude suppression effects. The standardized mean difference (SMD) and 95% credible interval (CrI) for each study represent the posterior distribution of that study's mean effect size, conditional on prior beliefs and the observed data. Negative values of *g* indicate reduced sensory gating ability (i.e., less effective amplitude suppression) in the autism group compared to TD controls. The gray shaded area indicates the region of practical equivalence (ROPE). Raw effect sizes from each study can be found in Supplemental Table S10. **(B)** Summary posterior densities of P1 amplitude differences to the first and second clicks of the paired-click paradigm, as compared to the posterior distribution of P1 amplitude suppression effects. Autistic individuals demonstrate smaller P1 amplitudes in response to the initial click, driving a group difference in amplitude suppression metrics.

Table 1.

Bayesian indices used to quantify evidence for an effect and statistical significance

Index	Description	Interpretation
95% (equal-tailed) Credible Interval (CrI)	The interval between the 2.5th and 97.5th percentiles of a posterior distribution. Conditional on prior information and observed data, there is a 95% chance that the parameter of interest falls between the interval bounds.	As with a frequentist confidence interval, if the 95% CrI of a parameter excludes 0, that parameter can be viewed as being "significantly" greater than or less than 0 at the $\alpha = 0.05$ level.
Posterior predictive distribution and 95% (equal-tailed) posterior predictive interval (PI)	The posterior predictive distribution is generated by the meta-analytic model. This distribution is the predicted distribution of effect sizes expected to be found in future studies of the sort included in the model, accounting for study heterogeneity. Conditional on the data and prior information, there is a 95% chance that a future effect size from this population will lie within the PI.	The posterior predictive distribution is a model-based estimate of the full population of possible study effect sizes, accounting for the observed between- and within-study heterogeneity. The width of the PI can be interpreted as a measure of effect heterogeneity, as wider predictive intervals are characteristic of more heterogeneous effects. The posterior predictive distribution can also be used to calculate the probability that a future effect will be opposite in sign from the meta-analytic estimate.
Probability of direction (P_d (105))	The proportion of the posterior distribution on the same side of 0 as the median (i.e., the probability that a parameter is greater than or less than zero, whichever is more probable).	Bayesian equivalent of a frequentist one-tailed p-value, with values ranging from 0.5 to 1. Values greater than 0.975 indicate that the 95% CrI does not include 0, and thus that the effect can be viewed as "statistically significant."
Bayes factor vs. a region of practical equivalence (BF_{ROPE} (105))	An interval null hypothesis is defined (in this case [−0.1, 0.1]), with all points within this "region of practical equivalence to zero" (ROPE) deemed too small for practical significance (103,104). BF_{ROPE} is defined as the odds of the prior distribution of a parameter falling within vs. outside of the ROPE divided by the odds of the posterior distribution of that parameter falling within vs. outside of the ROPE.	Quantifies degree of evidence *for or against* the interval null hypothesis. Higher values provide more evidence that the true parameter value does not lie within the ROPE, whereas lower values provide more evidence that the true parameter value lies within the ROPE (and thus is practically equivalent to 0). Qualitative descriptions for the degree of evidence are listed below. *BF* values between 1/3 and 3 are typically deemed to provide inconclusive evidence for either hypothesis.
Bayes factor for publication bias (BF_{PB} (107))	Quantifies evidence *for or against* the possibility of publication bias using Bayesian model averaging. BF_{PB} is an "inclusion Bayes factor" (108) for the publication bias parameters.	Qualitative descriptions for the degree of evidence are listed below. BF_{PB} values > 3 suggest publication bias, whereas BF_{PB} values < 1/3 suggest a lack of publication bias. *BF* values between 1/3 and 3 provide inconclusive evidence for or against the possibility of publication bias.
Bayes factor comparing moderated model to baseline model (BF_{10} (136))	Quantifies the evidence *for or against* the inclusion of the tested moderator in the meta-regression model. BF_{10} is defined as the ratio of the marginal likelihood of the moderated model to the marginal likelihood of the baseline (intercept-only) model, calculated via bridge sampling.	

BF_{10}, BF_{ROPE}, or BF_{PB} value	Interpretation (137)
>100	Extreme evidence for H_1
30–100	Very strong evidence for H_1
10–30	Strong evidence for H_1
3–10	Substantial evidence for H_1
1–3	Anecdotal evidence for H_1
1	No evidence
1/3–1	Anecdotal evidence for H_0
1/10–1/3	Substantial evidence for H_0

Index	Description	Interpretation
	1/30–1/10	Strong evidence for H_0
	1/100–1/30	Very strong evidence for H_0
	<1/100	Extreme evidence for H_0

Biol Psychiatry Cogn Neurosci Neuroimaging. Author manuscript; available in PMC 2022 August 01.

Table 2.

Characteristics of included studies

Reference	Components	Technique	Experimental Task	Attention	Sample Size		Sex Ratio (% Female)		Mean Age (Years)		Mean IQ		Quality Score (0–1)
					AUT	TD	AUT	TD	AUT	TD	AUT	TD	
Martineau et al. (1984) (55)	P1, N1b, P2	EEG	Passive listening	−	15	18	46.7	50.0	8.50	8.50	45.0	*n.r.*	0.462
Bruneau et al. (1999) (47)	N1b	EEG	Passive listening	−	16	16	25.0	25.0	6.00	6.00	41.0	*n.r.*	0.667
Gomot et al. (2002) (138)	N2	EEG	Passive listening (silent movie)	−	15	15	20.0	20.0	6.83	6.75	57.0	*n.r.*	0.661
Kemner et al. (2002) (81)	P1 gating	EEG	Paired-click (count clicks)	+	12	11	16.7	0.0	10.40	10.30	96.2	98.5	0.731
Bruneau et al. (2003) (63)	N1c	EEG	Passive listening	−	26	16	15.4	25.0	5.92	5.75	48.0	*n.r.*	0.667
Ferri et al. (2003) (56)	N1b	EEG	Passive listening (silent movie)	−	10	10	0.0	0.0	12.30	12.20	*n.r.* [a]	*n.r.*	0.426
Gage et al. (2003) (48)	M100	MEG	Passive listening	−	13	17	0.0	29.4	11.40	13.50	*n.r.*	*n.r.*	0.540
Jansson-Verkasalo et al. (2003) (66)	P1, N2	EEG	Passive listening (silent movie)	−	10	11	40.0	27.3	9.10	9.60	*n.r.*	*n.r.*	0.643
Oram Cardy et al. (2004) (58)	M50, M100	MEG	Passive listening (silent movie)	−	10	8	0.0	62.5	11.80	12.90	*n.r.*	*n.r.*	0.580
Jansson-Verkasalo et al. (2005) (67)	P1, N2	EEG	Passive listening (silent movie)	−	19	18	26.3	50.0	10.60	10.40	107.0	*n.r.*	0.607
Salmond et al. (2007) (49)	N1b, P2, N2	EEG	Oddball (respond to target)	+	26	19	15.4	63.2	12.30	12.70	87.5	104.0	0.796
Orekhova et al. (2008) (86)	P1 gating	EEG	Paired-click (silent movie)	−	21	21	19.0	14.3	5.92	5.92	77.4	*n.r.*	0.778
Lepistö et al. (2009) (68)	N2	EEG	Passive listening (silent movie)	−	16	14	18.8	14.3	8.10	8.10	106.0	*n.r.*	0.815
Roberts et al. (2010) (38)	M50, M100	MEG	Passive listening (silent movie)	−	22	17	*n.r.*	*n.r.*	10.77	10.20	100.3	110.7	0.780
Gomot et al. (2011) (139)	N2	EEG	Passive listening (silent movie)	−	27	27	22.2	22.2	8.33	8.33	51.0	*n.r.*	0.685
Matsuzaki et al. (2012) (132)	M50, M100	MEG	Passive listening	−	18	12	0.0	0.0	9.52	10.08	99.2	*n.r.*	0.680
Orekhova et al. (2012) (84)	M50 gating	MEG	Passive listening (silent movie)	−	14	15	7.1	13.3	10.58	10.67	92.0	120.0	0.700
Samy et al. (2012) (123)	N1b	EEG	Oddball (count targets)	+	25	25	*n.r.*	*n.r.*	6.50	6.50	*n.r.* [a]	*n.r.*	0.315
Brandwein et al. (2013) (64)	N1c	EEG	Simple reaction time task	+	45	71	17.8	53.5	11.18	11.60	105.9	111.9	0.768
Oranje et al. (2013) (82)	P1 gating	EEG	Paired-click (count clicks)	+	27	12	14.8	8.3	11.69	11.40	96.8	105.8	0.741
Azouz et al. (2014) (65)	N1c	EEG	n.r.	−	30	15	23.3	*n.r.*	5.45	*n.r.*	*n.r.* [a]	*n.r.*	0.185
Edgar et al. (2014) (50)	M50, M100	MEG	Paired-click (silent movie)	−	96	33	*n.r.*	*n.r.*	9.90	10.87	103.9	108.9	0.780
Karhson (2014) (43)	P1, N1b	EEG	Oddball (respond to target)	+	12	13	33.3	38.5	22.50	22.83	105.1	101.3	0.732
Lv et al., 2014 (77)	P1 gating	EEG	Paired-click	−	39	31	5.1	25.8	5.79	6.06	*n.r.*	*n.r.*	0.333
Matsuzaki et al. (2014) (140)	M50, M100	MEG	Passive listening	−	21	15	0.0	0.0	9.45	9.80	98.5	*n.r.*	0.720
Demopoulos et al. (2015) (41)	M100	MEG	Passive listening (silent movie)	−	25	12	28.0	41.7	11.47	13.78	84.2	111.0	0.820

Reference	Components	Technique	Experimental Task	Attention	Sample Size		Sex Ratio (% Female)		Mean Age (Years)		Mean IQ		Quality Score (0–1)
					AUT	TD	AUT	TD	AUT	TD	AUT	TD	
Donkers et al. (2015/2019) (51,52)	P1, N2	EEG	Passive listening (quiet movie)	−	28	39	21.4	20.5	7.62	7.03	82.6	108.5	0.815
Edgar et al. (2015a) (46)	M50, M100, M200	MEG	Paired-click (silent movie)	−	48	60	12.5	8.3	10.10	9.80	107.0	112.6	0.960
Edgar et al. (2015b) (141)	M100	MEG	Paired-click (silent movie)	−	105	36	10.5	52.8	10.07	10.90	103.6	108.8	0.760
Madsen et al. (2015) (85)	P1/N1b gating	EEG	Paired-click	−	31	39	22.6	30.8	11.10	10.80	98.1	107.6	0.768
Gayle (2016) (83)	P1 gating	EEG	Paired-click	−	19	16	*n.r.*	*n.r.*	15.00	*n.r.*	*n.r.*	*n.r.*	0.536
Port et al. (2016) (115)	M100	MEG	Paired-click (silent movie)	−	22	9	0.0	66.7	10.25	10.15	103.6	115.1	0.840
Sokhadze et al. (2016) (44)	N1b	EEG	Passive listening	−	18	14	16.7	28.6	11.06	12.60	*n.r.* [a]	*n.r.*	0.481
Crasta (2017) (45)	P1/N1b gating, N1b, P2	EEG	Sensory gating and tone tasks; passive and active conditions	+/−	24	24	29.2	50.0	23.31	23.70	*n.r.*	*n.r.*	0.554
Demopoulos et al. (2017) (142)	M100, M200	MEG	Passive listening	−	18	18	0.0	0.0	9.82	9.79	101.6	114.0	0.820
Vlaskamp et al. (2017) (69)	N2	EEG	Passive listening (silent movie)	−	35	38	20.0	28.9	11.10	11.10	98.5	107.6	0.741
Hudac et al. (2018) (54)	N1b	EEG	Passive listening (silent movie)	−	102	31	19.6	32.3	12.29	13.27	82.3	115.7	0.696
Yu et al. (2018) (42)	P1, N2	EEG	Passive listening (silent movie)	−	15	16	6.7	18.8	9.60	9.80	88.0	106.0	0.852
Bloy et al. (2019) (53)	M50, M100	MEG	Passive listening (silent movie)	−	62	33	0.0	0.0	11.80	11.80	99.5	115.1	0.840
Chien et al. (2019) (78)	P1/N1b gating	EEG	Passive listening	−	34	34	5.9	5.9	20.60	20.40	100.8	110.5	0.796
Roberts et al. (2019) (40)	M50, M100	MEG	Passive listening (silent movie)	−	71	34	18.3	14.7	10.46	10.18	88.7	112.8	0.800
Matsuzaki et al. (2020) [Children] (39)	M50, M100	MEG	Passive listening (silent movie)	−	58	36	12.1	22.2	10.07	9.21	103.7	113.0	0.740
Matsuzaki et al. (2020) [Adults] (39)	M50, M100	MEG	Passive listening (silent movie)	−	19	19	0.0	0.0	23.80	26.97	108.4	113.8	0.760

Note. n.r. = not reported; Attention: indicates whether the experimental task required the participants to attend to the presented auditory stimuli; ADOS/ADI-R: indicates whether autism diagnoses were confirmed with the Autism Diagnostic Observation Schedule or Autism Diagnostic Interview-Revised (i.e., "gold-standard" measures); +/− indicates that article included studies that both did and did not direct the participants' attention to the stimuli. AUT = autism group; TD = typically developing control group.

[a]While IQ for the AUT group was not reported, study did indicate the proportion of the AUT sample with intellectual disability.

Table 3.

Meta-analytic summary effects for each outcome and a priori defined subgroups with three or more included studies

Outcome	$N_{Effects}$	$N_{Studies}$	N_{AUT}	N_{TD}	g (95% CrI)[a]	P_d	BF_{ROPE}	BF_{PB}	I^2 (95% CrI)	$ICC(2)$	95% PI
Latencies											
P1/M50 Latency	36	14	498	359	**0.341 [0.184, 0.524]**	>0.999	**29.26**	0.937	41.5% [11.4, 73.2]	0.801	[−0.189, 0.913]
P1 Latency	11	5	89	104	0.273 [−0.189, 0.700]	0.910	0.53	—	62.9% [15.7, 90.9]	0.582	[−0.854, 1.344]
M50 Latency	25	9	409	255	**0.365 [0.185, 0.592]**	>0.999	**22.94**	—	39.5% [8.5, 75.4]	0.829	[−0.153, 0.936]
N1b Latency	25	8	146	139	0.172 [−0.594, 0.915]	0.690	0.36	2.316	90.5% [77.3, 97.0]	0.995	[−2.250, 2.563]
M100 Latency	37	12	516	305	**0.344 [0.135, 0.561]**	0.998	**6.60**	1.769	52.4% [19.2, 79.9]	0.749	[−0.339, 1.056]
P2/M200 Latency	12	4	83	79	0.057 [−0.608, 0.611]	0.591	*0.21*	1.372	71.2% [15.0, 95.5]	0.967	[−1.416, 1.400]
P2 Latency	10	3	65	61	−0.108 [−0.864, 0.480]	0.656	*0.23*	—	66.8% [9.7, 96.0]	0.956	[−1.592, 1.206]
N2 Latency	12	7	140	145	−0.047 [−0.280, 0.223]	0.656	*0.07*	0.402	28.0% [1.4, 74.8]	0.571	[−0.585, 0.537]
Amplitudes											
P1/M50 Amplitude	30	8	182	154	0.042 [−0.198, 0.324]	0.640	*0.07*	0.589	42.8% [6.8, 81.8]	0.912	[−0.580, 0.713]
P1 Amplitude	23	5	80	97	−0.018 [−0.392, 0.445]	0.539	*0.18*	—	56.2% [6.0, 92.1]	0.932	[−0.926, 0.965]
M50 Amplitude	7	3	102	57	0.140 [−0.319, 0.546]	0.780	*0.21*	—	31.8% [1.4, 86.5]	0.679	[−0.688, 0.923]
N1b Amplitude	24	7	205	131	−0.162 [−0.497, 0.157]	0.861	*0.21*	0.807	55.1% [26.0, 83.8]	0.964	[−1.021, 0.676]
N1c Amplitude	11	3	101	102	**−0.812 [−1.278, −0.187]**	0.988	**9.85**	2.800	61.2% [12.5, 93.4]	0.578	[−1.922, 0.451]
M100 Amplitude	10	5	145	87	0.124 [−0.152, 0.398]	0.831	*0.14*	0.391	21.4% [0.9, 70.2]	0.638	[−0.423, 0.667]
P2/M200 Amplitude	13	5	135	142	−0.065 [−0.339, 0.176]	0.720	*0.07*	0.350	26.2% [1.4, 75.1]	0.749	[−0.622, 0.444]
P2 Amplitude	10	3	65	61	0.046 [−0.328, 0.397]	0.628	*0.09*	—	26.4% [1.1, 85.0]	0.747	[−0.634, 0.695]
N2 Amplitude	27	9	191	197	**−0.374 [−0.633, −0.179]**	0.999	**14.63**	0.731	32.5% [2.4, 76.3]	0.897	[−0.933, 0.116]
Sensory Gating											
P1/N1 Suppression	21	8	207	188	**−0.394 [−0.639, −0.099]**	0.992	**3.63**	0.237	52.3% [17.8, 83.0]	0.832	[−1.077, 0.348]
P1 Suppression	16	8	207	188	**−0.382 [−0.633, −0.082]**	0.992	**3.07**	—	51.9% [16.0, 82.7]	0.771	[−1.068, 0.365]
N1 Suppression	5	3	86	97	−0.389 [−0.853, 0.151]	0.942	1.16	—	58.2% [6.2, 93.8]	0.677	[−1.371, 0.671]
P1 Amplitude (Click 1)	10	7	171	148	**−0.286 [−0.505, −0.048]**	0.989	1.51	0.344	20.1% [0.8, 66.6]	0.538	[−0.731, 0.197]
P1 Amplitude (Click 2)	8	6	157	133	0.121 [−0.237, 0.445]	0.776	*0.16*	1.604	51.2% [7.0, 85.8]	0.740	[−0.693, 0.900]

Note. There was insufficient data to meta-analyze N1c latency, and thus this outcome is not reported. Publication bias Bayes factors are not reported for subgroup analyses. 95% credible intervals that do not overlap 0 are bolded. BF_{ROPE} values greater than 3 (providing significant evidence that the true effect lies outside [−0.1,0.1]) are bolded, whereas BF_{ROPE} values less than 1/3 (providing significant evidence that the true effect lies within [−0.1,0.1]) are italicized. AUT = autism group; TD = typically developing control group; CrI = Equal-tailed Credible Interval; P_d = Probability of Direction (the probability that the effect is of the same sign as the point estimate); BF_{ROPE} = Bayes factor vs. the interval null hypothesis [−0.1,0.1], i.e., the region of practical equivalence to 0 (ROPE); BF_{PB} =

Bayes factor testing the hypothesis of publication bias (107); I^2 = standardized heterogeneity estimate across levels 2 (study) and 3 (effect size) of the meta-analytic model; *ICC*(2) = the proportion of heterogeneity attributed to level 2 (i.e., between-study heterogeneity); PI = posterior predictive interval.

[a]Negative *g* values indicate smaller values of a variable in the autism group (e.g., less positive P1/P2 amplitude, less negative N1/N2 amplitude, faster component latency, or less effective P1/N1b amplitude suppression), compared to TD controls.

Biol Psychiatry Cogn Neurosci Neuroimaging. Author manuscript; available in PMC 2022 August 01.

Table 4.

Bayes factor values for each tested moderator variable

Moderator	P1/M50 Latency	N1b Latency	M100 Latency	P1/M50 Amplitude	N1b Amplitude	N2 Amplitude	Sensory Gating
Stimulus/Paradigm Factors							
Stimulus Probability	*0.301*	0.537	0.688	**4.356**	0.398	1.537	—
Stimulus Duration	*0.002*	*0.023*	*0.001*	*0.002*	*0.002*	*0.001*	*0.165*
Stimulus Intensity	*0.006*	*0.037*	*0.023*	*0.009*	*0.040*	*0.034*	*0.038*
Stimulus Frequency (log scale)	*0.303*	*0.197*	*0.244*	*0.303*	*0.213*	0.930	—
Inter-stimulus Interval	0.347	0.369	*0.163*	0.347	*0.301*	*0.001*	*<0.001*
Number of Trials	*<0.001*	*0.214*	*0.001*	*0.009*	*< 0.001*	1.187	*0.004*
Active Task	—	*0.272*	—	1.170	*0.194*	—	*0.214*
Analysis Factors							
EEG or MEG	*0.204*	—	—		—	—	—
P1 or N1b Gating	—	—	—	—	—	—	*0.170*
Gating Ratio or Difference	—	—	—	—	—	—	*0.158*
Lowpass Filter	*0.003*	*0.018*	*0.002*	*0.002*	*0.003*	*0.015*	*0.015*
Highpass Filter	*0.053*	0.983	*0.061*	*0.046*	0.975	0.355	*0.023*
Laterality of Recording	*0.024*	—	*0.182*	*0.044*	—	*0.021*	—
Sample Factors							
Total *N*	*0.002*	*0.205*	*0.002*	*0.005*	*0.008*	*0.008*	*0.020*
Mean Age (AUT Group)	0.450	*0.064*	*0.051*	*0.183*	*0.221*	*0.094*	*0.022*
Proportion Female (AUT Group)	1.053	0.957	1.310	0.660	0.782	0.868	*0.023*
Mean IQ (AUT Group)[a]	*0.120*	0.750	*0.052*	*0.012*	**6.034**	*0.036*	*0.017*
Study Quality (logit transformed)	*0.219*	0.557	*0.173*	*0.240*	*0.268*	*0.227*	0.700
Publication Year	*0.090*	0.550	*0.049*	*0.015*	*0.291*	*0.022*	*0.059*

Note. Moderator analyses were conducted for studies in which 20 or more effect sizes were included in the meta-analysis. Omitted values indicate that (a) a given moderator was not applicable to the tested component or (b) there was insufficient variance in the moderator across studies to test it in a meta-regression. Bayes factors in bold provide significant evidence for the inclusion of the moderator. Bayes factors in italics provide significant evidence *against* the inclusion of the moderator. All other values are inconclusive. AUT = autism spectrum disorder.

[a]For the sample of studies reporting N1b amplitudes and latencies, mean IQ in the AUT group was treated as a binary variable (i.e., indexing whether or not the majority of the sample had IQ < 70), as quantitative IQ scores were not available in many of these studies.