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Reinterpreting the Human ABR Binaural Interaction Component: Isolating Attention from Stimulus Effects

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Highlights

- Attention to tone-pips or clicks increases the effect size of the presence of DN1.
- Such selective attention to tone-pips, but not clicks, affected ABR wave Vs.
- There were isolable attention-independent stimulus effects upon DN1 amplitudes.
- The mediation of that stimulus effect on DN1 differs from that on binaural wave V.

Abstract

Subtracting the sum of left and right monaural auditory brainstem responses (ABRs) from the corresponding binaural ABR isolates the binaural interaction component (ABR-BIC). In a previous investigation (Ikeda, 2015), during auditory yet not visual tasks, tone-pips elicited a significant difference in amplitude between summed monaural and binaural ABRs. With click stimulation, this amplitude difference was task-independent. This self-critical reanalysis's purpose was to establish that a difference waveform (i.e., ABR-BIC DN1) reflected an auditory selective attention effect that was isolable from stimulus factors. Regardless of whether stimuli were tone-pips or clicks, effect sizes of the DN1 peak amplitudes relative to zero improved during auditory tasks over visual tasks. Auditory selective attention effects on the monaural and binaural ABR wave-V amplitudes were tone-pip specific. Those wave-V effects thus could not explain the stimulus-universal effect of auditory selective attention on DN1 detectability, which was thus entirely binaural. In a manner isolated from auditory selective attention, multiple mediation analyses indicated that the higher right monaural wave-V amplitudes mediated individual differences in how clicks, relative to tone-pips, augmented DN1 amplitudes. There are implications of these findings for advancing ABR-BIC measurement.

Keywords: auditory brainstem response binaural interaction component (ABR-BIC), monaural, selective attention, tone, click.

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1. Introduction

The binaurally evoked neural response to sound differs from the artificial sum of the corresponding monaural responses (Benichoux *et al.*, 2018; Dobie and Berlin, 1979; Gardi and Berlin, 1981; Jewett, 1970; Melcher, 1996; Tolnai and Klump, 2020; Ungan and Yağcioğlu, 2002; Wada and Starr, 1989). This difference between such responses is a sign of binaural interaction in the auditory system. An oft-measured deflection of the binaural interaction component (BIC) is the DN1 component of the human auditory brainstem response (ABR). This negative deflection reflects the reduced amplitude of the binaural response relative to the sum of each monaural response (Dobie and Norton, 1980; Ito *et al.*, 1988; Jiang, 1996; Levine, 1981; Polyakov and Pratt, 1999; Riedel and Kollmeier, 2006). Subtracting summed monaural waveforms from binaural ABRs results in the first negative difference potential at the active vertex electrode at the latency of ABR wave V or later. Dobie and Berlin (1979) thus termed this deflection of the BIC as the DN1. Dobie and Berlin (1979) similarly named the preceding first positive difference wave as the DP1.

Historically, influences of vigilance and attention upon the human ABR have been denied (Amadeo and Shagass, 1973; Picton and Hillyard, 1974; Woldorff *et al.*, 1987). On the other hand, human studies employing fixed ear stimulation have found a task-dependent ABR modulation (Galbraith *et al.*, 2003; Lukas, 1980; Sörqvist, Stenfelt, and Rönnberg, 2012; for an alternative perspective, see the literature review of Varghese *et al.*, 2015). Lukas (1980) and Sörqvist *et al.* (2012) also observed that visual distraction reduced wave V amplitudes. Indeed, binaural stimulation with 1000-Hz tone bursts elicited ABRs that revealed such attentional influences (Lukas, 1980; Sörqvist *et al.*, 2012). These findings thus raise the research question as to whether the attended modality modulates the ABR-BIC. Ikeda (2015) found evidence consistent with an attentional modulation of the ABR-BIC: At the DN1 latency, the amplitude difference between the binaural and summed monaural

responses to 1000-Hz tone-pips was not statistically significant when elicited during the visual task. By contrast, this amplitude difference attained weak significance for the same stimuli during the auditory task. However, using clicks as stimuli, a significant difference in amplitude between the binaural and summed monaural responses was robustly present during both visual and auditory tasks. Ikeda (2015) thus interpreted that attending to the auditory modality is necessary for the tone-pip DN1 but not for the click DN1.

In a recent advance, Ikeda (2019) found outcomes of the DN1 that were inconsistent with the above interpretation (Ikeda, 2015). A methodological shortcoming of the previous work (Ikeda, 2015) was the waveform analysis, which did not deal with the DN1 difference wave itself. To address this analytical concern, Ikeda (2019) measured the DN1 amplitude from the difference wave. Doing so determined that an auditory task improved the detectability of DN1 amplitudes, the effects of which seemed universal across stimuli. Participants in the study received low-passed (< 1000 Hz) and high-passed (> 2000 Hz) clicks at 30-dB SL intensity during auditory and visual tasks. For both kinds of clicks, the detection of DN1 amplitudes as compared to zero was better in the auditory task than in the visual task. If an improvement in DN1 detectability with auditory selective attention is stimulus-universal, as seen in Ikeda (2019), a prediction is that clicks as well as tone-pips would reveal the task-related effects on DN1 presence as compared to zero. This inference motivated a reanalysis of the previous dataset (Ikeda, 2015), now using difference waveform amplitudes, to explore effects of auditory selective attention on DN1 presence.

With binaural cochlear implant users, Hu and Dietz (2015) revealed an intriguing correlation when stimulating different electrodes in the inner ears. Parenthetically, the primary purpose of recording the electrically evoked DN1 for Hu and Dietz (2015) was to identify the best inter-aural electrode pairing according to the identical best frequency between bilateral auditory nerves (Brown *et al.*, 2019). This approach addresses how the depth of surgical placement of intendedly corresponding cochlear implant electrodes, with respect to the tonotopic organization of the cochleas, typically varies between ears. However, a separate finding of Hu and Dietz's (2015) investigation is germane here, a

correlation of intra-individual variables: as the monaural eV became more positive, the electrically evoked DN1 became more negative. That correlation held out for most participants. Hu and Dietz (2015) revealed that, when controlling for this correlation, there was still an influence of the choice of the inter-aural electrode pair. This pairing, as an intra-individual variable, influenced the electrically evoked DN1. These findings thus raise a question here with acoustical rather than electric hearing. The question is whether the intra-individual variability in stimulus type or attended modality directly influences the DN1 or the binaural ABR. Another question is whether there are such indirect influences via the monaural left or right ABRs. Indeed, Hu and Dietz's correlation may suggest such indirect effects. This correlation that Hu and Dietz control for by normalizing (electrically evoked) BIC amplitudes according to monaural wave V amplitudes is thus of interest here.

Drawing these connected themes together, the purpose of the present new analyses was thus threefold. First, the present investigation further explored the hypothesis that the presence of the DN1 itself exhibited an attentional modulation. To achieve this purpose, the difference of DN1 amplitudes from zero level was evaluated. The effect of selective attention on the DN1 to clicks or tone-pips was then assessed by comparing auditory and visual tasks. Secondly, to assay if the DN1 itself exhibited an attentional modulation that reflected purely binaural processing, there was an assessment of the contribution of binaural and monaural ABRs to attentional effects on the DN1. The reason for that assay is that either binaural or monaural ABRs could affect their difference waveform component. That is, theoretically, an increment of the DN1 could be brought either from reduced binaural wave V amplitudes or an augmentation of monaural wave V amplitudes. A third purpose of this investigation was concerned with inter-individual and intra-individual variability. While inter-individual variability concerned the left and right monaural ABRs, intra-individual variability was related to the stimulus type, electrode reference, and attended modality. This investigation thus determined which individual variability was associated with the DN1 and the binaural ABR, as well as modelling *how* those correlations were mediated.

2. Materials and Methods

The task, EEG recordings, and the derivation of behavioral and ABR data analyzed here are described in detail in Ikeda (2015). The new analytical approach (section 2.5) self-critically reassessed attentional modulations that seemed apparent in the DN1. Attentional effects upon monoaural ABRs may hypothetically contribute to such modulations. Further, these analyses also investigate if, and how, monaural ABRs and intra-individually variable factors predict DN1 and binaural ABRs.

2.1. Participants

Twelve persons had healthy hearing, healthy or corrected-to-healthy visual acuity, and right-hand dominance (8 females; mean age 20.71 years, 95% Confidence Interval (CI) [19.65, 21.77]). In accordance with the Declaration of Helsinki, volunteers gave their informed written consent prior to participating in the experiment in exchange for a small honorarium. The research was also approved by the ethics committee of Tokyo Gakugei University.

2.2. Stimulation

Auditory stimuli were either 1000-Hz tone-pips, 10-msec sinusoids with 5-msec rise and fall times without plateau, or clicks, 0.1-msec rectangular waveforms. Stimuli were delivered via headphones at an 80 dB peak equivalent sound pressure level (pe SPL). To clarify, the stimulus level at 80 dB pe SPL was less than 67 dB SPL, below the minimal threshold for ipsilateral and contralateral middle-ear acoustic reflexes (Ikeda, 2015). With an otherwise constant SOA of 180 msec, occasionally stimulus omissions extended the SOA to 360 msec at the probability of 0.01. The leading phase of the stimulus alternated on each successive trial. During binaural blocks, both ears received the stimuli simultaneously without noise masking. In monaural blocks, either the left or right ear was exposed to the stimuli. In those monaural blocks, the contralateral ear received continuous white noise at an intensity of 38 dB(A). For visual stimuli, participants saw a silent movie on a liquid crystal

display. Visual targets were changes from previous movie scenes that were defined as almost the same actors existing at same location. The definition of a movie scene was based upon the participant's own standard. The occurrence of such visual targets was more infrequent than auditory omissions (Table 1) since the frequency of auditory omissions was at least 20 within one block. These auditory and visual stimuli were delivered to participants in parallel during each block.

2.3. Procedure

All participants were instructed to reduce body tension during the tasks. Each participant underwent three blocks according to the ear of Presentation, i.e., binaural, monaural left, and monaural right. In each such block, participants conducted two separate experimental tasks – auditory and visual – thus manipulating the Attended modality. In the auditory task, participants pressed a button upon detecting a sound omission. For the visual task, participants identified each new scene of the movie. In this visual task, whenever another new movie scene appeared, participants were thus required to press a button. During all tasks, participants fixated the display's central cross. A Latin-square design distributed the order of three conditions by two tasks to participants. The responding hand (left or right) and the order of auditory stimulus types were balanced across participants.

2.4. Electrophysiological Recordings

Ag/AgCl electrodes for recording electroencephalogram (EEG) were located, bilaterally, both at the earlobes (A1 and A2) and at the mastoids (M1 and M2). These electrodes were referenced to the vertex (Cz) with a forehead ground. A separate bipolar montage measured electrooculogram between the supraorbital rim and lateral canthus of the right eye. Signals were amplified within a 0.16–2110 Hz (-3 dB point) bandpass and were digitally sampled at 10 kHz. Measurement during each task in a block continued until recordings contained 2000 artifact-free 22-msec epochs, inclusive of a 2-mec pre-stimulus baseline, during which the vertex-earlobe potentials were within ± 30 μV in any channel.

Recordings were band-pass filtered offline with a zero-phase shift at 20–2000 Hz (-12 dB/octave). The filtered signal was epoched and the artifact-free epochs were then averaged for each response. Re-referencing of the active electrode, Cz, to the average of each pair of inverting electrodes took place after this offline signal averaging.

2.5. Data Analysis

A correct response during auditory tasks was defined as a button press that was 200–800 msec after the onset of an omission according to the preceding constant SOA. Such responses during the auditory tasks gave rise to two behavioral measures: auditory reaction time (ART) and auditory correct response rate (ACR). For visual tasks, the number of button presses was the visual response count (VRC). These behavioral measures were each examined with a 2(Stimulus: tone-pips, click) × 3(Presentation: binaural, left monaural, right monaural) repeated-measures Analysis of Variance (ANOVA).

For the subsequent data analyses, the waveforms obtained from the two lateral derivations were averaged (Cz - [A1 + A2] / 2; Cz - [M1 + M2] / 2). In addition, subtracting the artificial sum of monaural waveforms from the corresponding binaural response obtained the difference waveform. Then, separately for each derivation, separately for each stimulus, difference waveforms, monaural ABRs, summed monaural ABRs, and binaural ABRs were grand-averaged.

Then for each derivation separately, for each stimulus separately, there were three plots: Firstly, the grand-averaged BIC difference wave for the two tasks was overplotted (Fig. 1 A-D). Secondly, in a plot for the visual task, and, thirdly, in another plot for the auditory task, the grand-averaged summed monaural ABRs and grand-averaged binaural ABRs were overplotted (Fig. 1 E-L). Additionally, for each derivation separately, for each stimulus separately, there were two plots, one for left monaural presentation, and another for right monaural presentation, in which the grand-averaged ABRs for the two tasks were overplotted (Fig. 3 A-H).

The analysis method for ABRs consisted of four stages reliant upon ANOVAs, which

were repeated-measures throughout. The first stage detected time intervals of mean amplitudes exhibiting the binaural interaction in the averaged potentials: An ANOVA compared binaural with summed monaural amplitudes within successive 0.5-msec bins from 2 msec post-stimulus onset onwards. Thus, the test made no assumption about the direction of effects (e.g., binaural < summed monaural). This inferential statistical analysis (Ikeda, 2015) identified the DN1 time window. The time window was fixed to a duration of 1 msec starting at the onset of a significant interval covering the ABR wave V in the auditory task. The resulting DN1 windows were 6.5–7.5 msec for clicks and 9.5–10.5 msec for tone-pips, which were analysis windows unemployed in Ikeda (2015) and being the same for all participants. Inspection of grand averages in Fig. 1 reveals that the DN1 analysis window focuses on the wave V peak rather than a slope following the wave V peak. The second stage for each stimulus, derivation, and attended modality, quantified the DN1 peak amplitudes. That is, measurements of the difference waveform were taken within these time windows. The DN1 peak was defined as the minimum voltage within the time window. To begin to assay whether the presence of DN1 itself exhibited an attentional modulation that affected purely binaural processing, eight ANOVAs then evaluated the difference of the DN1 peak amplitude from zero. Each such ANOVA with "actual data or zero" as the independent variable had two levels: i) actual DN1 peak negative amplitude and ii) zero. *p*-values were Bonferroni-corrected. To clarify, as shall be revealed, the difference between peak DN1 and zero did not equate with the difference between peak binaural and summed monaural responses: The source generators that dominate the peak DN1 are thus arguably distinct from those determining the peak binaural and summed monaural response – typically the wave V peak. After Näätänen *et al.* (2004), this methodology, comparing a difference to zero, detects whether a component's presence is significant. There was then the examination of these peak DN1 amplitudes with, for each derivation, a 2 (Stimulus: tone-pips, clicks) × 2(Attended modality: auditory, visual) ANOVA.

Third and fourth stages assessed each contribution of the binaural and monaural ABRs to the DN1, characterizing how attention affects monaural ABRs. In the third stage,

the peak amplitude measures of binaural and summed monaural potentials in the appropriate DN1 time window were quantified. The binaural and summed monaural peaks were defined as the maximum voltage within the time window. There was then the examination of these peak amplitudes with, for each derivation, a 2(Stimulus: tone-pips, clicks) × 2(Attended modality: auditory, visual) × 2(Response: binaural responses, summed monaural responses) ANOVA. In the fourth stage, the amplitude measures of the left and right monaural potentials were quantified by using the DN1 time window. There was then the examination of these peak amplitudes with, for each derivation, a 2(Stimulus: clicks, tone-pips) × 2(Attended modality: auditory, visual) × 2(Laterality: left monaural response, right monaural response) ANOVA. The reported degrees of freedom were uncorrected since Mauchly tests of sphericity did not reveal correction as necessary. Planned comparisons were conducted only for the amplitude difference between the auditory and visual tasks. Further comparisons were Bonferroni-corrected.

Turning to intra-individual and inter-individual variability, a fifth phase of the analysis characterized the influence of Stimulus, Attended modality, and Reference upon individual differences in peak amplitudes of the monaural ABRs, binaural ABRs, and DN1s. This phase also characterized the influence of those differences in monaural amplitude on individual differences in the DN1 (Hu and Dietz, 2015) and upon binaural ABRs. In this phase, binaural ABRs from different conditions of Stimulus and Attended modality were pooled across references and overplotted against the corresponding left ABR, right ABR, and summed monaural ABRs. DN1 data from different conditions of Stimulus and Attended modality were pooled across references and overplotted as a function of the corresponding binaural ABR, left ABR, right ABR, and summed monaural ABRs. For each such scatterplot, the approach of Pernet *et al.* (2013) excluded univariate and bivariate outliers. Whether according to the boxplot rule, the MAD-median rule, or an S-estimator, all outliers were excluded. Pearson Product-Moment Correlations then evaluated the association between monaural ABR measures and binaural ABRs, as well as assessing the association of monaural and binaural ABR measures with DN1. Introducing coefficients for Stimulus

(Tone-pips: -0.5, Clicks: 0.5), Attended modality (Visual: -0.5, Auditory: 0.5) and Reference (Mastoid derivation: -0.5, Earlobe derivation: 0.5), permitted point-biserial correlations. Those correlations assessed the association between these intra-individually variable factors and individual differences not only in the binaural ABR but also in the DN1. To determine what factors could make independent predictive contributions to individual differences, first, in DN1 and, second, in binaural ABRs, there were two multiple stepwise linear regressions. The first considered the contributions of Stimulus, Attended modality, Reference, left ABR, right ABR, and binaural ABR to predicting DN1. The second considered the contributions of Stimulus, Attention, Reference, left ABR, and right ABR to predicting binaural ABR. Parallel Multiple Mediator models in PROCESS (Hayes, 2017) then established how promising intra-individually variable factors, either directly or indirectly, predict binaural ABRs and DN1s. 5000 samples assessed 95% bootstrap confidence intervals for model coefficients. The approach centered only continuous variables that define products. HC2 heteroscedasticity-consistent inference was employed to estimate the model's regression coefficients. For DN1, an exploratory Serial Multiple Mediator model also resolved any interpretational quandary about the inter-relations of multiple mediators. Critical α was set to 0.05 throughout.

3. Results

The main tendencies in the data were as follows. The detection of auditory omissions tended to ceiling, driving effects into the speed domain (Table 1; Section 3.1): Overall, participants detected the omission of binaural sounds faster than sounds emanating from the right, particularly for clicks. Participants detected tone-pip omissions faster than click omissions, albeit only for right-ear stimulation. Turning to the electrophysiological findings, DN1s were apparent, whether stimuli were clicks (Fig. 1A and C) or tone-pips (Fig. 1B and D). Concerning whether the DN1 itself reflected the attentional modulation, auditory selective attention increased the presence of the DN1 (difference from zero level), in terms of effect sizes, for both tone-pips and clicks (Table 2; Section 3.2). There appeared to be an

augmented DN1 negativity during auditory yet not visual tasks (Fig. 1B, C, and D). This observation warranted an investigation of the influence of auditory selective attention on DN1 peak amplitudes (Section 3.2). However, the ANOVA for assessing the effects of auditory selective attention on DN1 amplitudes revealed no significance. Only DN1 presence itself exhibited a significant attentional modulation reflecting purely binaural processing: Contrary to both the presence and amplitude of the DN1, tone-pips during the auditory task elicited an increase in monoaural ABR amplitudes in the DN1 time range. Independently of ear, this task-related increase of monaural amplitudes was not found for clicks (Fig. 2E and F; Section 3.4). Attending to the auditory task rather than the visual task seemed to increase the summed monaural and binaural tone-pip ABRs comparably (Fig. 1F vs J, H vs L; 2C-D; Section 3.3). In contrast, there was no such increase of summed monaural and binaural click ABRs by auditory selective attention (Fig. 1G vs K, 2D; Section 3.3). Attending to the auditory task rather than the visual task seemed to increase both left and right monaural ABRs for tone-pips (Fig. 2E-F, 3E-H; Section 3.4). There was also a right-ear advantage, with higher amplitudes for clicks presented to the right ear than to the left ear (Fig. 2E-F; Fig. 3A vs B; Fig. 3C vs D; Section 3.4).

Throughout correlational analyses (Section 3.5, Fig. 4), increases in the left, right, and summed monaural ABRs, each showed an association with increases in the binaural ABRs, and augmented DN1 negativities. There was also a stronger association of DN1 amplitude with the right ABR than the association with the left ABR (Section 3.5, Fig. 4). Of intra-individually variable factors, Stimulus, yet neither Reference nor Attended modality, predicted DN1 and binaural ABR peak amplitudes. Facets of both the predictions of DN1 and ABR peak amplitudes from Stimulus proved direct (Fig. 5). Stimulus also predicted DN1 amplitudes in a manner indirectly and distinctly mediated via the right monaural ABR wave V (Fig. 5A). As well, an indirect effect revealed that Stimulus predicted binaural ABRs via bilateral monaural wave Vs (Fig. 5B).

3.1. Behavioral responses

PLEASE INSERT TABLE 1 ABOUT HERE

Table 1 summarizes the behavioral descriptive statistics. These values were either derived from correct responses during the auditory task (ARTs, ACRs) or from response numbers during the visual task (VRCs). Presentation seemed to affect ARTs, which were overall faster for binaural presentation. Stimulus also appeared to affect ARTs, which were significantly faster for tone-pips than clicks only with right monaural presentation. These influences of Presentation and Stimulus in the speed domain (ARTs) lacked any obvious homologues in the accuracy domain (ACRs). The accuracy indices were at ceiling, whether Attended modality was auditory (ACRs) or visual (VRCs).

Inferential statistical analyses corroborated this pattern of behavioral findings: A 2 (Stimulus: clicks, tone-pips) × 3 (Presentation: binaural, left monaural, right monaural) ANOVA for ARTs revealed that the Presentation main effect was significant, $F(2, 22) = 8.205$, $p = 0.002$, $\eta_p^2 = 0.427$ (binaural: 450.226 msec, 95% CI [420.669, 479.783], left monaural: 470.701 msec, 95% CI [444.770, 496.632], right monaural: 484.342 msec, 95% CI [454.512, 514.173]). Bonferroni-corrected multiple comparisons revealed that participants detected the omission of binaural sounds faster than of right monaural sounds (binaural < right monaural), $F(1, 11) = 21.036$, $p < 0.003$, $\eta_p^2 = 0.657$, 95% CI [13.140, 55.092]. Bonferroni-corrected comparisons did not reveal significant differences in ARTs between left monoaural and either of the other two conditions of Presentation, Fs < 5.399.

The ANOVA interaction for ARTs was marginal, $F(2, 22) = 3.308$, $p = 0.055$, $\eta_p^2 = 0.231$. Significant differences underpinned this marginal interaction: Bonferroni-corrected linear contrasts revealed Stimulus differences in ARTs to be significant, $F(1,11) = 7.896$, $p = 0.017$, $\eta_p^2 = 0.418$, 95% CI [6.594, 54.258], only for right monaural presentation (tone pip, right monaural: 469.129 msec, 95% CI [439.680, 498.579] < click, right monaural: 499.555 msec, 95% CI [464.966, 534.144]). For binaural or left monaural presentation, ARTs were

roughly equivalent across Stimulus, *F*s < 1. There was a significant ART advantage for binaural presentation, $F(1,11) = 17.908$, $p = 0.004$, $\eta_p^2 = 0.619$, 95% CI [16.702, 83.430], for the click level of Stimulus (binaural, clicks: 449.489 msec, 95% CI [420.996, 477.983] < right monoaural, clicks: 499.555 msec, 95% CI [464.966, 534.144]). The ART left ear advantage was marginal, $F(1,11) = 7.398$, $p = 0.060$, $\eta_p^2 = 0.402$, 95% CI [-1.293, 71.490], for the click level of Stimulus (left monaural, clicks: 464.457 msec, 95% CI [438.296, 490.617] < right monoaural, clicks: 499.555 msec, 95% CI [464.966, 534.144]). Other effects on ARTs in Bonferroni-corrected linear contrasts were not significant, *F*s < 4.033. Further ANOVAs neither revealed significant effects nor interactions for ACRs and VRCs, *F*s < 2.378.

PLEASE INSERT FIG. 1 ABOUT HERE

3.2. The DN1

Fig. 1A-D illustrates the grand-average difference (binaural minus summed monaural) waveforms for auditory and visual tasks. Montages contained Cz and bilateral electrodes either at the earlobes (Fig. 1A, B) or at the mastoids (Fig. 1C, D). Visible in each plot (Fig. 1A–D) is some indication of a DN1 deflection of the ABR-BIC component. DN1 was apparent for each Attended modality, whether auditory or visual, whether the Stimulus was a click (Fig. 1A and C) or a tone-pip (Fig. 1B and D). However, when the Attended modality was auditory, whether the Stimulus was a click (Fig. 1C) or a tone-pip (Fig. 1D), the presence of DN1s seemed more prominent, at least at the mastoids.

PLEASE INSERT TABLE 2 ABOUT HERE

It was first necessary to establish whether or not the DN1 of the ABR-BIC was significantly present (Näätänen *et al.*, 2004). Table 2 thus reports the comparisons of the peak

DN1 amplitude measure with zero. DN1 effect sizes proved that, although large throughout (η_p^2 > 0.14, Cohen, 1988), they were smaller for tone-pips (Fig. 1B and D) than for the corresponding click DN1s (Fig. 1A and C). Effect sizes also proved larger for auditory tasks (Fig. 1, dash-dot lines) than for visual ones (Fig. 1, dotted lines) in the Attended modality. Consideration now turns from the effect sizes to the significance levels in Table 2. With tone-pips (Fig. 1B and D), the significant presence of DN1s in auditory tasks declined to marginal tendencies in visual tasks. These marginal tendencies were nonsignificant following Bonferroni correction. Accordingly, DN1s for tone-pips seemed to be present only when attending to the auditory rather than the visual modality. Withstanding Bonferroni correction, all DN1s with clicks (Fig. 1A and C) were significantly present regardless of the Attended modality, albeit with larger effect sizes when attending to the auditory modality. In short, auditory selective attention's influence on the DN1 presence would appear, at first, to be more evident for tone-pips than for clicks.

PLEASE INSERT FIG. 2 ABOUT HERE

Inasmuch that the Attended modality appeared to influence the presence of the DN1 in Table 2, an open question remained: Did auditory selective attention affect DN1 amplitudes? Initially, DN1 amplitudes seemed to be more prominent when attending to the auditory modality, particularly with the mastoid derivation (Fig. 1B, C, and D). Figs. 2A and B summarize DN1 peak amplitude descriptive statistics. The slopes of the functions in Figs. 2A and B were inconsistent for the influence of Attended modality. At a glance, this attentional influence on DN1 amplitudes seemed to vary with Stimulus and derivation. At the same time, auditory tasks reduced confidence intervals of the mean, except for click-evoked responses with the mastoid derivation (Figs. 2A and B). The following inferential statistical analyses were basically negative for the influence of Attended modality on DN1 amplitudes. Those analyses were simultaneously sensitive to a significant DN1 augment for clicks relative to tones. This Stimulus effect was in accord with Table 2's larger effect sizes concerning DN1

presence for clicks than tone-pips. That effect of Stimulus was contingent on reference, being more apparent with the mastoid (Fig. 2B) rather than the earlobe derivation (Fig. 2A).

Inferential statistics turn from the first to the second stage of data analysis. Whether the derivation was earlobes (Fig. 2A) or mastoids (Fig. 2B), a 2 (Stimulus: clicks, tone-pips) × 2 (Attended modality: auditory, visual) ANOVA revealed neither a main effect of Attended modality nor a Stimulus × Attended modality interaction, Fs ≤ 2.301. Selective attention planned linear contrasts, for each derivation and each stimulus, did not demonstrate a positive result. As depicted in black in Fig. 2B, there was a marginal increase of click-evoked DN1 amplitude due to auditory attention with the mastoid derivation (visual, clicks: -0.155 μV, 95% CI [-0.237, -0.072] > auditory, clicks: -0.250 μV, 95% CI [-0.348, -0.153], $F(1, 11) = 3.443$, $p = 0.091$, $\eta_p^2 = 0.237$, 95% CI [-0.018, 0.210]). All other such selective attention planned linear contrasts were not significant, Fs < 1. Consideration now shifts from influences of Attended modality to those of Stimulus on DN1 amplitude. The earlobe derivation (Fig. 2A) did not reveal significance of the Stimulus main effect, $F < 1$, $\eta_p^2 = 0.063$. For the mastoid derivation (Fig. 2B), there was a large Stimulus main effect, $F(1, 11) = 8.007$, $p = 0.016$, $\eta_p^2 = 0.421$, 95% CI [-0.165, -0.021] depicting an augmented DN1 for clicks over tone-pips (tone-pips: -0.110 μV, 95% CI [-0.158, -0.061] > clicks: -0.202 μV, 95% CI [-0.273, 0.132]).

3.3. The binaural and summed monaural responses

The grand-averaged Wave Vs seen in Figs. 1E-L, Figs. 2C and D summarize the peak amplitudes for binaural and summed monaural responses within the DN1 time range: Evident were higher amplitudes for clicks (black symbols) than for tone-pips (other symbols). Also evident were lower amplitudes for the binaural responses (bold large symbols) than the summed monaural responses (ordinary size symbols). Crucially, the slopes of the lines suggested that auditory selective attention generally produced an amplitude augment. This attentional tendency was held for tone-pips whether considering binaural (bold large symbols) or summed monaural ABR responses (other symbols). However, this attentional tendency did not hold out for clicks (black symbols).

Inferential statistical analysis corroborated these tendencies. Planned comparisons of the amplitudes between the auditory and visual tasks were considered in the 2 (Stimulus: clicks, tone-pips) × 2 (Attended modality: auditory, visual) × 2 (Response: binaural responses, summed monaural responses) ANOVA. Both binaural and summed monaural responses exhibited significant differences in the comparisons only for tone-pips. Thus, considering each slope in Figs. 2C and D, whether responses to tone-pips were binaural or summed monaural, there was an amplitude augment when attending to the auditory rather than the visual modality. This pattern held for tone-pips with both derivations (ps ≤ 0.045), yet not for clicks with either derivation (Fs ≤ 1.997, ps > 0.1).

The Stimulus main effect, whereby peak amplitudes were lower for tone-pips than clicks, was highly significant not only with the earlobe derivation (tone-pips: 0.567 μV, 95% CI [0.488, 0.647] < clicks: 1.067 μV, 95% CI [0.924, 1.209], $F(1, 11) = 119.983$, $p < 0.001$, $\eta_p^2 = 0.916$, 95% CI [0.399, 0.600]), but also with the mastoid derivation (tone-pips: 0.581 μV, 95% CI [0.501, 0.660] < clicks: 1.144 μV, 95% CI [0.975 1.313], $F(1, 11) = 111.351$, $p < 0.001$, $\eta_p^2 = 0.910$ μV, 95% CI [0.446, 0.681]). The Response main effect revealed a reduced amplitude for binaural responses. This Response effect was significant not only with the earlobe derivation (binaural: 0.773 μV, 95% CI [0.683, 0.864] < summed monaural: 0.861 μV, 95% CI [0.740, 0.982], $F(1, 11) = 13.617$, $p = 0.004$, $\eta_p^2 = 0.553$, 95% CI [-0.140, -0.035]), but also with the mastoid derivation (binaural: 0.882 μV, 95% CI [0.717, 0.927] < summed monaural: 0.903 μV, 95% CI [0.766, 1.040], $F(1, 11) = 8.873$, $p = 0.013$, $\eta_p^2 = 0.446$, 95% CI [-0.141, -0.021]). The main effect of Attended modality constituted a large amplitude augment of responses by auditory selective attention. This Attended modality effect was not significant with the earlobe derivation (auditory: 0.839 μV, 95% CI [0.737, 0.941] ≈ visual: 0.795 μV, 95% CI [0.682, 0.909], $F(1, 11) = 2.673$, $p > 0.1$, $\eta_p^2 = 0.196$, 95% CI [-0.015, 0.102]), but significant with the mastoid derivation (auditory: 0.899 μV, 95% CI [0.782, 1.015] > visual: 0.826 μV, 95% CI [0.702, 0.950], $F(1, 11) = 13.153$, $p = 0.004$, $\eta_p^2 = 0.545$, 95% CI [-0.117, -0.029].

Turning to interactions, the effect of Attended modality was significantly stronger for

tones. That is, the Stimulus × Attended modality interaction was significant with the earlobe derivation, $F(1, 11) = 8.419$, $p = 0.014$, $\eta_p^2 = 0.434$, albeit marginal with the mastoid derivation, $F(1, 11) = 4.028$, $p = 0.070$, $\eta_p^2 = 0.268$. This interaction revealed a large amplitude augment of responses to tone-pips by auditory selective attention. This attentional augment for tone-pips was not only significant with the earlobe derivation (auditory: 0.622 μV, 95% CI [0.544, 0.699] > visual: 0.513 μV, 95% CI [0.421, 0.604], F(1, 11) = 16.601, η_p^2 = 0.601, $p = 0.002$, 95% CI [0.050, 0.168]), but also with the mastoid derivation (auditory: 0.646 μV, 95% CI [0.565, 0.727] > visual: 0.516 μV, 95% CI [0.425, 0.607], F(1, 11) = 18.439, $p = 0.001$, $\eta_p^2 = 0.626$, 95% CI [0.063, 0.197]). In contrast, for clicks, the Stimulus × Attended modality interaction failed to exhibit the attentional augment with both derivations, Fs < 1.

Concerning Fig. 2C, the Attended modality × Response interaction was not significant with the earlobe derivation, $F < 1$. Concerning Fig. 2D, this interaction was marginal with the mastoid derivation, $F(1, 11) = 3.244$, $p = 0.099$, $\eta_p^2 = 0.228$. Significant differences underpinned this marginal interaction (Fig. 2D): Firstly, there was a significant amplitude advantage of summed monoaural responses over binaural responses under auditory selective attention (binaural, auditory: 0.841 μV, 95% CI [0.742, 0.939] < summed monaural, auditory: 0.957 μV, 95% CI [0.815, 1.098], $F(1, 11) = 12.624$, $p = 0.005$, η_p^2 = 0.534, 95% CI [-0.187, -0.044]. Attending to the visual modality attenuated this summed monaural response advantage to nonsignificance (binaural, visual: 0.803 μV, 95% CI [0.686, 0.920] ≈ summed monaural, visual: 0.850 μV, 95% CI [0.709, 0.990], $F(1, 11) = 1.894$, $p >$ 0.1). Secondly, another facet of the marginal Attended modality × Response interaction concerned how attentional effects varied with Response. There was a significant increase in the summed monaural response when attending to the auditory rather than the visual modality (summed monaural, auditory: 0.957 μV, 95% CI [0.815, 1.098] > summed monaural, visual: 0.850 μV, 95% CI [0.709, 0.990], $F(1, 11) = 11.403$, $p = 0.006$, η_p^2 = 0.509, 95% CI [0.037, 0.177]). This significance was not found for the binaural response (binaural, auditory: 0.841 μV, 95% CI [0.742, 0.939] ≈ binaural, visual: 0.803 μV, 95% CI

[0.686, 0.920], $F(1, 11) = 2.776$, $p > 0.1$). All other interactions were not significant, Fs ≤ 1.332. In summary, focusing on tone-pips, binaural and summed monaural responses were comparably augmented by auditory selective attention (Figs. 2C and D). As shown in Figs. 2C and D, the origins behind the marginal Attended modality × Response interaction at the mastoids seemed to be responses to clicks. However, planned comparisons have already confirmed that attentional modality did not significantly influence any responses to clicks.

3.4. The left and right monaural responses

PLEASE INSERT FIG. 3 ABOUT HERE

Fig. 3 presents the grand-averaged ABRs to monaural stimuli. Figs. 2E and F demonstrate averages of the peak amplitudes for the left and right monaural responses. The slopes of Figs. 2E and F indicated an influence of Attended modality on monaural responses to tone-pips, yet not for clicks. These slopes were consistent with the influence of attention on wave V amplitudes in Figs. 3E-H, as unapparent in Figs. 3A-D. Monaural responses were higher in amplitude for clicks than tones (Figs. 2E and F) as shown in wave V amplitudes (Figs. 3A-D vs. 3E-H). Also apparent were higher amplitudes with right than left clicks, i.e., a right ear advantage, which did not affect responses to tone-pips (Figs. 2E and F). This right ear advantage can be seen in the wave V by comparing Figs. 3A and C with B and D for clicks, whereas it is not seen in Figs. 3E-H for tone-pips.

Inferential statistical analyses confirmed the general tendencies visible in Fig. 2. Selective attention planned comparisons were considered for the amplitudes between the auditory and visual tasks in the 2 (Stimulus: tone-pips, click) × 2 (Attended modality: auditory, visual) × 2 (Laterality: left monaural, right monaural) ANOVA. These comparisons tested monoaural effects of auditory selective attention. Thus, each slope in Figs. 2E-F received consideration in turn. The large influence of Attended auditory modality augmented the monaural response to a tone-pip in the left ear, that was significant both with the earlobe

derivation (auditory: 0.346, [0.263, 0.429] > visual: 0.282 μV, 95% CI [0.219, 0.344], F(1, 11) = 4.851, p = 0.0499, η_p^2 =0.306, 95% CI [0.000044, 0.128]) and with the mastoid derivation (auditory: 0.350 μV, 95% CI [0.221, 0.318] > visual: 0.270 μV, 95% CI [0.221, 0.318], F(1, 11) = 8.154, p = 0.016, η_p^2 = 0.426, 95% CI [0.018, 0.142]). A similar influence for the right ear tone-pip was marginal with the earlobe derivation (auditory: 0.341 μV, 95% CI [0.300, 0.382] > visual: 0.274 μV, 95% CI [0.192, 0.357], F(1, 11) = 4.247, η_p^2 =0.279, p = 0.064, 95% CI [-0.005, 0.139]), although it was nonsignificant for the mastoid derivation, F < 1. Considering the slopes for clicks in Figs. 2E and F, the planned linear contrasts revealed no monaural significant influences of auditory selective attention, Fs ≤ 1.668, ps ≥ 0.223.

Both Stimulus and Laterality main effects were effective for both derivations, whereas the Attentional modality effect was significant only with the mastoid derivation. The three main effects for the amplitude measures were as follows. The Stimulus main effect was highly significant not only with the earlobe derivation (tone-pips: 0.311 μV, 95% CI = [0.258, 0.364] < clicks: 0.562 μV, 95% CI = [0.479, 0.644], F(1, 11) = 79.754, p < 0.001, η_p^2 = 0.879, 95% CI = [-0.313, -0.189]), but also with the mastoid derivation (tone-pips: 0.312 μV, 95% CI = [0.267, 0.357] < clicks: 0.605 μV, 95% CI = [0.507, 0.703], F(1, 11) = 82.826, p < 0.001, η_p^2 = 0.883, 95% CI = [-0.364, -0.222]). The Laterality main effect was marginal both with the earlobe derivation (left: 0.408 μV, 95% CI [0.333, 0.483] < right: 0.464 μV, 95% CI [0.403, 0.526], F(1, 11) = 4.702, p = 0.053, η_p^2 = 0.299, 95% CI [-0.001, 0.114]) and with the mastoid derivation (left: 0.429 μV, 95% CI [0.350, 0.509] < right: 0.487 μV, 95% CI [0.418, 0.557], F(1, 11) = 3.997, p = 0.071, η_p^2 = 0.267, 95% CI [-0.006, 0.122]). Although the Attended modality main effect was nonsignificant for the earlobe derivation (auditory: 0.450 μV, 95% CI [0.391, 0.509] ≈ visual: 0.422 μV, 95% CI [0.350, 0.494], F(1, 11) = 2.135, p > 0.1, η_p^2 = 0.163, 95% CI = [-0.071, 0.014]), this effect was significant with the mastoid derivation (auditory: 0.482 μV, 95% CI [0.412, 0.553] > visual: 0.434 μV, 95% CI [0.365, 0.504], F(1, 11) = 8.121, p = 0.016, η_p^2 = 0.425, 95% CI = [-0.085, -0.011]). As shall be seen in planned comparisons, this main effect seemed to be due to an augment of

monaural responses by auditory selective attention that confined to tone-pips.

Turning to the interactions, the Stimulus × Laterality interaction was significant with the earlobe derivation, $F(1, 11) = 9.046$, $p = 0.012$, $\eta_p^2 = 0.451$, and also marginal with the mastoid derivation, $F(1, 11) = 4.151$, $p = 0.066$, $\eta_p^2 = 0.274$. The large amplitude advantage of a click over a tone-pip was robust for both left and right ear stimulation. This robust pattern held for both derivations, demonstrating larger effect sizes with the right ear: For the earlobe derivation, the left ear stimulation (tone-pips: 0.314 μV, 95% CI [0.247, 0.380] < clicks: 0.502 μV, 95% CI [0.403, 0.602], $F(1, 11) = 27.920$, $p < 0.001$, $\eta_p^2 = 0.717$, 95% CI [-0.267, -0.110]), the right ear stimulation (tone-pips: 0.308 μV, 95% CI [0.254, 0.362] < clicks: 0.621 μV, 95% CI = [0.535, 0.707], $F(1, 11) = 84.766$, $p < 0.001$, $\eta_p^2 = 0.885$, 95% CI [-0.388, -0.238]), and for the mastoid derivation, the left ear stimulation (tone-pips: 0.310 μV, 95% CI [0.256, 0.363] < clicks: 0.549 μV, 95% CI [0.434, 0.664], $F(1, 11) = 41.341$, $p < 0.001$, $\eta_p^2 = 0.790$, 95% CI [-0.321, -0.157]), as well as the right ear stimulation (tone-pips: 0.314 μV, 95% CI = [0.263, 0.365] < clicks: 0.661 μV, 95% CI [0.551, 0.770], $F(1, 11) = 57.946$, $p < 0.001$, $\eta_p^2 = 0.840$, 95% CI [-0.447, -0.246]). The Stimulus × Laterality interaction also revealed that the laterality advantage of the right over the left ear stimulation for clicks (Figs. 2E and F) was lost for tone-pips with either derivation, Fs < 1. This large right-ear advantage for clicks was significant with both the earlobe derivation (left, clicks: 0.502 μV, 95% CI [0.403, 0.602] < right, clicks: 0.621 μV, 95% CI [0.535, 0.707], $F(1, 11) = 9.275$, $p = 0.011$, $\eta_p^2 = 0.457$, 95% CI [-0.204, -0.033]) and the mastoid derivation (left, clicks: 0.549 μV, 95% CI [0.434, 0.664] < right, clicks: 0.661 μV, 95% CI [0.551, 0.770], $F(1, 11) = 5.064$, $p = 0.046$, $\eta_p^2 = 0.315$, 95% CI [-0.221, -0.002]).

The Stimulus × Attended modality interaction was marginal with the earlobe derivation, $F(1, 11) = 4.293$, $p = 0.063$, $\eta_p^2 = 0.281$, and nonsignificant with the mastoid derivation, $F < 1$, $p > 0.1$. The very large amplitude advantage of a click over a tone-pip was robust with both derivations, although effect sizes were greater when attending to the visual modality. With the earlobe derivation, this click advantage was highly significant for the auditory task (tone-pips: 0.344 μV, 95% CI [0.296, 0.391] < clicks: 0.557 μV, 95% CI [0.469,

0.645], $F(1, 11) = 35.665$, $p < 0.001$, $\eta_p^2 = 0.764$, 95% CI [0.135, 0.292]) and for the visual task (tone-pips: 0.278 μV, 95% CI [0.210, 0.345] < clicks: 0.566 μV, 95% CI = [0.477, 0.656], $F(1, 11) = 87.288$, $p < 0.001$, $\eta_p^2 = 0.888$, 95% CI [0.220, 0.356]). With the mastoid derivation, the amplitude advantage of clicks was kept robust for the auditory task (tone-pips: 0.344 μV, 95% CI [0.302, 0.386] < clicks: 0.620 μV, 95% CI [0.506, 0.734], $F(1, 11) = 37.577$, $p < 0.001$, $\eta_p^2 = 0.774$, 95% CI [0.177, 0.375]) and for the visual task (tone-pips: 0.279 μV, 95% CI = [0.218, 0.341] < clicks: 0.589 μV, 95% CI [0.498, 0.680], $F(1, 11) = 95.658$, $p < 0.001$, $\eta_p^2 = 0.897$, 95% CI [0.240, 0.380]). In the Stimulus × Attended modality interaction, the large monaural auditory selective attention advantage for tone-pips was significant not only with the earlobe derivation (auditory, tone-pips: 0.344 μV, 95% CI [0.296, 0.391] > visual, tone-pips: 0.278 μV, 95% CI [0.210, 0.345], $F(1, 11) = 8.969$, $p = 0.012$, $\eta_p^2 = 0.449$, 95% CI [0.017, 0.114]) but also with the mastoid derivation (auditory, tone-pips: 0.344 μV, 95% CI [0.302, 0.386] > visual, tone-pips: 0.279 μV, 95% CI [0.218, 0.341], $F(1, 11) = 6.557$, $p = 0.026$, $\eta_p^2 = 0.373$, 95% CI [0.009, 0.121]). The same effect of auditory selective attention with clicks revealed none of significance, as apparent in either derivation, Fs ≤ 1.085. All other interactions were nonsignificant, Fs ≤ 1.103, ps > 0.1.

3.5. Relating individual differences between DN1, binaural, and monaural ABR amplitudes

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PLEASE INSERT FIG. 4 ABOUT HERE

Strong and highly significant point-biserial correlations corroborated the main effect of Stimulus. These correlations with Stimulus revealed that peak amplitudes were higher with clicks than tone-pips for the left ABR, $r(79) = 0.621$, $p < 0.001$, the right ABR, $r(82) = 0.794$, $p < 0.001$, the summed ABR, $r(81) = 0.781$, $p < 0.001$, and the binaural ABR, $r(71) = 0.781$, $p < 0.001$. There was a marginal negative correlation of the DN1 with Stimulus, $r(74) = -0.207$, $p = 0.073$. This null result accorded with the main effect of Stimulus on the DN1 – the significance of which depended upon the reference. Thus

including only data with the mastoid derivation, this point-biserial correlation of the DN1 with Stimulus was significant, $r(34) = -0.339$, $p = 0.043$. This result confirmed the existence of an association between the click stimulation and an augmented DN1. However, there were no significant point-biserial correlations involving either Attention or Reference, ps > 0.162.

In scrutiny of Fig. 4, it was necessary to determine what factors could make independent predictive contributions to individual differences in the DN1 and Binaural ABRs. Stepwise multiple linear regressions thus considered first those independent contributions to predicting the DN1 that Stimulus, Attended modality, Reference, the left ABR, the right ABR, and the binaural ABR made. The second stepwise multiple linear regression then considered the predictive contributions that Stimulus, Attended modality, Reference, the left ABR, and the right ABR made to the binaural ABR.

In the first stepwise procedure to predict DN1 amplitudes (Fig. 4D–F), a model containing three predictor variables – the binaural ABR, the left ABR, and the right ABR – explained the DN1s, adjusted $R^2 = 0.918$, $F(3, 71) = 126.098$, $p < 0.001$. The binaural ABR, $B = 0.733$, $p < 0.001$, the left ABR, $B = -0.707$, $p < 0.001$, and the right ABR, $B = -0.929$, $p < 0.001$, all significantly predicted DN1s. When the influence of the binaural, left, and right ABRs was controlled, the excluded variable, Stimulus, did not predict DN1s, $p > 0.1$. The effect was marginal for the excluded predictive contribution of Attended modality, $B = 0.90$, $p > 0.065$, and of Reference, $B = 0.80$, $p > 0.091$. Although a point-biserial correlation had revealed a significant association of DN1 augments with using click stimuli only for the mastoid derivation, Stimulus made no predictive contribution to the DN1 independently from the binaural, left, and right ABRs. As shall be seen, some of these ABR variables thus mediated the association between Stimulus and the DN1.

In the second stepwise procedure, a model containing three predictor variables – the left ABR, the right ABR, and Stimulus – explained the binaural ABR (Fig. 4A and B), adjusted $R^2 = 0.938$, $F(3, 69) = 169.902$, $p < 0.001$. The left ABR, $B = 0.997$, $p < 0.001$, the right ABR, $B = 0.407$, $p = 0.001$, and Stimulus, $B = 0.147$, $p < 0.001$, all significantly predicted the binaural ABR, whereas the excluded variables, Attention and Reference, did not, ps > 0.774.

PLEASE INSERT FIG. 5 ABOUT HERE

Having established with a point-biserial correlation that Stimulus can predict the DN1, at least with the mastoid derivation, and with stepwise regression that stimulus predicts binaural ABR, it was necessary to determine how Stimulus relates to the DN1 and the binaural ABR. A mediation analysis within PROCESS (Hayes, 2017) tested parallel multiple mediator models with left and right ABRs as candidate mediators (Fig. 5). To predict the consequent variable *Y* from the antecedent variable, *X*, each such model requires an equation of each of *k* mediator variables *M*,

$$M_i = i_{M_i} + a_i X + e_{M_i} \quad \text{for } i = 1 \text{ to k} \tag{1}$$

and one for the consequent, *Y*,

$$Y = i_Y + c' + \sum_{i=1}^{k} b_i M_i + e_Y \tag{2}$$

As shown in Fig. 5A, the first model found that the total effect of Stimulus on the DN1 was marginal, $R^2 = 0.037$, $F(1, 73) = 6.591$, $p = 0.097$, effect size = -0.040, which augmented the negativity of DN1 amplitudes for clicks relative to tone-pips. However, the direct effect of Stimulus on the DN1 that attenuated this negativity for clicks relative to tone-pips was highly significant, $t(71) = 4.365$, $p < 0.001$, effect size = 0.130. This marginality of the total effect was in line with how the significance of the point-biserial correlation depended upon using only observations from the mastoid reference. As shall be revealed, the marginality of the total effect was due to a significant indirect effect of opposing polarity. Consider the indirect pathways in Fig. 5A that subtly augmented the negativity of the DN1 for clicks relative to tones. The indirect effect via the left ABR had a bootstrapped mean effect size of -0.014, 95% CI [-0.035, 0.015], which was nonsignificant with CI spanning zero. By contrast, the indirect pathway via the right ABR significantly mediated the augmented negativity of the DN1 for clicks relative to tones, having a bootstrapped mean effect size of -0.156, 95% CI [-0.235, -0.100]. A significant contrast of indirect effects

confirmed stronger mediation via the right ABR than the left ABR, bootstrapped mean effect size: 0.142, 95% CI [0.077, 0.238].

Parenthetically, the left and right ABRs were positively correlated, as warranted scrutiny. Even so, exploratory follow-up with serial multiple mediator models (Hayes, 2017) confirmed that the left ABR only significantly predicted the DN1 when mediated by the right ABR. In that model, mediation of the influence of Stimulus on the DN1 by the right ABR alone was significantly stronger. However, probing for interactions in the first parallel multiple mediator model, a significant left ABR × Stimulus interaction confirmed that the left ABR moderated, rather than mediated, the direct effect of Stimulus on the DN1, $F(1, 70) = 9.601$, $p = 0.003$: The level of the left ABR thus affected the direct influence of Stimulus on the DN1 when the left ABR was more negative. That is, the stimulus being a click rather than a tone more strongly augmented the negative amplitude of the DN1. When the left ABR was more positive, the stimulus being a click rather than a tone attenuated the negative amplitude of the DN1.

Accordingly, although clicks enhanced the left ABR amplitude relative to tone-pips, the left ABR amplitude did not significantly mediate the influence on the DN1 in the way that the right ABR did (Fig. 5A). Laterality of monaural stimulation thus affects how the influence of Stimulus on the DN1 is mediated by individual differences in Stimulus effects upon monoaural ABRs.

As shown in Fig. 5B, the second parallel multiple mediator model confirmed that the total effect of Stimulus on the binaural ABR was highly significant, $R^2 = 0.624$, $F(1, 71) = 112.949$, $p < 0.001$, effect size = 0.418, which augmented the positive binaural ABR peak amplitudes for clicks relative to tone-pips. The direct effect of Stimulus on the binaural ABR was significant that also augmented the positivity for clicks relative to tone-pips, $t(69) = 3.700$, $p < 0.001$, effect size = 0.147. The two indirect pathways in Fig. 5B were both significant, augmenting the positive peak of binaural ABRs for clicks relative to tones. The indirect effect with the left ABR had a bootstrapped mean effect size of 0.160, 95% CI [0.101, 0.228]. The indirect effect with the right ABR had a bootstrapped mean effect size of

0.111, 95% CI [0.038, 0.178]. The indirect contrast confirmed these indirect effects as comparable, bootstrapped effect size, 0.050, 95% CI [-0.035, 0162]. Individual differences in Stimulus effects on the monaural ABRs thus comparably mediated the influence of Stimulus on the binaural ABR in the DN1 time range, regardless of laterality. This pattern of findings concerning laterality and the mediation of click-related augments in the binaural ABR (Fig. 5B) thus corroborates the click-related DN1 attenuation with the direct Stimulus effect (Fig. 5A).

4. Discussion

The results show that auditory selective attention improved DN1 detectability relative to zero level – an effect that is universal across stimuli. That is, the increased effect size for detecting the DN1 occurs not only for tone-pips but also for clicks (Table 2). These outcomes accord with the recent results of Ikeda (2019), revealing that the increased DN1 detectability during the auditory task is stimulus-universal. Concomitantly, effects of auditory selective attention on DN1 amplitudes were either absent for tone-pips (Fig. 1B and D, 2A and B) or marginal for clicks (Fig. 1C and 2B). The above results are at odds with the previous interpretation of Ikeda (2015) that auditory attention influences the tone-pip DN1 yet not the click DN1. The following now considers the results in more depth with respect to – as mentioned in the Introduction – each of the three purposes of the research in turn, which then leads into a more integrative discussion.

Concerning the first purpose of the research, DN1 presence itself proved to exhibit an attentional modulation (cf. Ikeda, 2015). Tenable assumptions include that this attentional modulation of DN1 detectability is isolable from the effect of auditory selective attention on the binaural and monaural ABRs. A further plausible assumption is that the effect of auditory selective attention on DN1 detectability is distinct from what determines DN1 peak amplitudes, which contrastingly goes unaffected by the task. Problematic for the conclusion that attention modulated the DN1 is a methodological issue that the left, right, and binaural ABRs were recorded at completely different times (i.e., blocked stimulus design) inasmuch

that block-to-block fluctuations in attentional state may occur in a manner independent of stimulus and/or attentional instruction. Ikeda (2019) has overcome this methodological shortcoming of the present investigation by presenting binaural, monaural left, and monaural right sounds pseudo-randomly within blocks. By this improved procedure, Ikeda (2019) has also observed the attention-related modulation of DN1 detection. An alternative source of the apparent auditory selective attention effects on DN1 detectability, which shall be cautiously excluded, is myogenic artifact. Sammeth *et al.* (2021) observed challenges in recording the DN1 when participants exhibited muscle activity. Sammeth *et al.* (2021) identified myogenic activity as a disrupting factor in DN1 measurement. Thus, at first, it might have seemed that some increase in muscle tension during the visual relative to the auditory tasks might have given rise to the apparent effect-size improvement in DN1 peak amplitudes. However, the present participants were carefully instructed to reduce body tension during the tasks. As a consequence, the grand mean of the number of rejected epochs was less than 4% of that of the averaging epochs (Ikeda, 2015). Further, that number of rejected epochs revealed no significant difference between the auditory and visual tasks in the present data (Ikeda, 2015). This epoch rejection rate thus excludes the possibility that the cause of the apparent stimulus-universal auditory selective attention effects on the DN1, shown here, is contamination by myogenic artifact. The current findings rather indicate that, when participant's muscle activity is carefully reduced, auditory selective attention is genuinely an advantageous state for identifying the DN1. Previous animal studies have evinced the corticofugal influence on the midbrain auditory localization system (Nakamoto *et al.*, 2008; Zhang *et al.*, 2000; Zhou and Jen, 2005). Behavioral evidence demonstrated that the corticofugal pathway's function is related to the environment-dependent plasticity of the midbrain localization system rather than primary localization (Bajo *et al.*, 2010). This indirect nature of a corticofugal pathway over the midbrain localization system accords well with the present investigation's auditory selective attention effect that improved DN1 presence without increasing DN1 peak amplitudes. The corticofugal influence on the midbrain auditory localization system is primarily shifting cell responses to localization cues

(Nakamoto *et al.*, 2008; Zhang *et al.*, 2000; Zhou and Jen, 2005) possibly resulting in behavioral adaptation to shifted localization cues (Bajo *et al.*, 2010). This efferent influence is functioning even under anesthesia because cooling auditory cortices alters azimuthal tuning of the auditory midbrain cells in anesthetized animals (Nakamoto *et al.*, 2008). At the same time, the present task-related effects are similar to the corticofugal effects established by cortical stimulation for anesthetized animals (Zhang *et al.*, 2000; Zhou and Jen, 2005). Examining mean DN1 peaks in Fig. 2A and B has revealed reduced error bars in the auditory task relative to the visual task. Therefore, the optimization of binaural processing achieved by the cortical efferent pathway might balance excitatory and inhibitory inputs into binaural neurons. DN1 negative amplitudes are improved if the deviation to excitatory inputs is corrected, whereas excessive DN1 amplitudes are reduced if the deviation to inhibitory inputs is adjusted. As a consequence, the effect size of DN1 detection can be improved without affecting mean DN1 peaks across task conditions.

Turning to the second purpose of the research, to assay if DN1 itself exhibits an attentional modulation that affects purely binaural processing, the results do not demonstrate a contribution of binaural and monaural ABRs to attentional effects on the DN1. Consistent with Ikeda's (2015) observation, the auditory task during tone-pip stimulation augments both the monaural and binaural ABRs, whereas the task-related difference during click stimulation is not found for the monaural and binaural ABR amplitudes (Fig. 2C-F). The effects of auditory selective attention on the monaural and binaural ABR amplitudes are not universal across stimuli. The stimulus-universal effect of auditory selective attention on DN1 detectability cannot be some by-product of the tone-pip specific effect of auditory selective attention on the monaural and binaural ABR amplitudes. Theoretically, the increased amplitude of the binaural ABR does not contribute to DN1 augmentation (i.e., BIC = binaural ABR - summed monaural ABRs): The attentional modulation of the binaural ABR is arguably independent of the effect of auditory selective attention on DN1 presence. Accordingly, the DN1 itself exhibits an attentional modulation that affects purely binaural processing.

PLEASE INSERT TABLE 3 ABOUT HERE

To clarify the foregoing discussion of attentional effects upon an isolable DN1, Table 3 denotes the number of participants revealing a DN1 peak below zero, [DN1 < 0], as well as those who exhibited a binaural peak lower in amplitude than the summed monaural peak, [B < L + R]. The discrepancy between these two numbers, i.e., [DN1 < 0] and [B < L + R], suggests that monaural components contaminate only [B < L + R]. That is, monaural componentry affects [B < L + R], whereas that monaural componentry has already been subtracted out when considering [DN1 < 0]. Table 3 typically reveals a consistency between [DN1 < 0] and [B < L + R] for the auditory task, as is unapparent for the visual task conditions. For the auditory task conditions, auditory selective attention thus strengthens the purely binaural process for [B < L + R]. One exception is the outcome for tone-pip stimulation and the mastoid derivation during the auditory task. A possible interpretation for this exception is that the influence of monaural components on [B < L + R] remains in this condition. The improvement of the DN1 detection with the auditory task (Table 2) accords with this increase in the number of participants showing the effect [DN1 < 0] with the auditory task (Table 3). Those outcomes can contribute to advancing the ABR-BIC measurements. Since the ABR-BIC amplitude is small, detection of this component becomes difficult at relatively weak stimulus intensities (Cone-Wesson *et al.*, 1997; Ito *et al.*, 1988), as contamination by myogenic activity can substantially obscure (Sammeth *et al.*, 2021). Reducing participant's myogenic activity to a large extent, whilst paying attention to evoking stimuli, would thus improve ABR-BIC detection. Broadly speaking, selective attention to the auditory modality – via the auditory corticofugal system – can promote ABR-BIC detection, whereas vigilance, through the global activation of brain areas, does not (Sammeth *et al.*, 2021; Suzuki *et al.*, 1991).

The third purpose of the research further concerns how inter- and intra-individual variability of the monaural ABRs, Stimulus, Reference, and Attended modality associated

with differences in the DN1 and in the binaural ABRs. There are no such significant associations involving Attention or Reference. Attention does not make an independent contribution to predicting the DN1 and the binaural ABRs. Collapsing across reference and attentional condition, a multiple parallel mediator model (Hayes, 2017) establishes that the association of Stimulus with the DN1 and binaural ABR amplitudes is mediated by monaural ABR amplitudes (Fig. 5). In particular, the right ABRs, yet not the left ABRs, mediate the influence of Stimulus on the DN1 (Fig. 5A). This result accords with Hu and Dietz's (2015) observation that many participants revealed amplitude correlations of the right monaural ABR with the DN1. There is also a direct influence of Stimulus on the DN1 (Fig. 5A), which attenuates DN1 amplitudes for clicks relative to tone-pips. The monaural origin of the click relative to the tone-pip DN1 augment (Fig. 1A-D, Fig. 2A and B) is thus independent of the stimulus-universal effect of auditory selective attention on DN1 presence (Table 2).

Inasmuch that the effect-size increase of the DN1 with auditory selective attention (Table 2) is independent of an attentional modulation of the binaural and monaural ABRs, the auditory selective attention effect on DN1 presence is considered purely binaural in origin.

The neuronal population critically supporting binaural interaction in the auditory brainstem is classified into two types, i.e., excitatory-inhibitory versus excitatory-excitatory (EI vs. EE) neurons. Traditionally, EI cells were bound to the lateral superior olivary (LSO) functions for identifying spatial cues with higher sound frequencies and EE cells to the medial superior olivary (MSO) functions for catching the cues with lower sound frequencies (e.g., Goksoy *et al.*, 2005; Melcher, 1996; Riedel and Kollmeier, 2002, 2006; Ungan and Yağcioğlu, 2002; Ungan *et al.*, 1997). Do EI or EE cells contribute more to the auditory selective attention effect on DN1 presence? The interpretation offered is that the EI cell population predominantly determines this effect in the auditory brainstem.

This notion of EI predominance in the purely binaural process is in line with the following evidence: First, the explicit presence of the ABR binaural interaction for click stimuli is explained by a binaural model based on EI cells (Laumen *et al.*, 2016). In previous

ABR studies (Goksoy *et al.*, 2005; Riedel and Kollmeier, 2002, 2006; Ungan *et al.*, 1997), a model of EI interaction (van Bergeijk, 1962) acceptably predicted DN1 latency as a function of interaural time differences (ITDs) for a broadband stimulus, whereas models of EE interaction (Jeffress, 1948; Young and Rubel, 1983) did not predict the ABR results. Second, recent findings elucidate that EI interactions in the LSO have coverage for broadband frequencies (Grothe *et al.*, 2010; Laumen *et al.*, 2016). Through phase-locked EI interactions, low-frequency LSO neurons exhibited the trough-type ITD sensitivity (Tollin and Yin, 2005). Third, recent neurophysiological evidence has not only directly (Tolnai and Klump, 2020) but also indirectly (Benichoux *et al.*, 2018) proved LSO neuronal activity to be a major source of the DN1. Tolnai and Klump (2020) demonstrated that, through simultaneous recordings of gerbils' scalp DN1 and single-cell firings in the MSO or LSO, the best correspondence of the DN1 with binaurally evoked neuronal firings occurred for the LSO rather than the MSO. Benichoux *et al.* (2018) revealed that DN1 modulations due to ITDs were identical in a wide range of different mammals, yet some species do not have a binaurally functional MSO. These previous findings support the assumption that there is an EI mechanism in the brainstem that greatly contributes to the ABR-BIC modulation.

Candidates for anatomical substrates of the DN1 attentional modulation might be corticofugal projections (Coomes Peterson and Schofield, 2007) to the inferior colliculus (IC) or to the superior olivary complex (SOC). The amount of corticofugal projections to the IC is much greater than those to the SOC (Winer, 2006). As previously mentioned, the corticofugal influences on the auditory localization system are found in the IC (Bajo *et al.*, 2010; Nakamoto *et al.*, 2008; Zhang *et al.*, 2000; Zhou and Jen, 2005). Since the majority of ITD-sensitive neurons in the IC have trough-type tuning possibly originating from EI interactions (Ono *et al.*, 2020), cortical efferent effects on the auditory localization system in the IC can be a basis of the DN1 attentional modulation. Supporting this, an origin of the human magnetic ABR wave V has been estimated at the lateral lemniscus to IC level (Parkkonen *et al.*, 2009). Also, a case with unilateral IC lesions exhibited both a deficit in psychophysical sound localization and a marked decrease in wave V amplitude (Litovsky *et*

al. 2002). On the other hand, the corticofugal projections to the SOC in guinea pigs are mainly terminated at the ventral nucleus of the trapezoid body (NTB) and the superior paraolivary nucleus, added to fractional contacts at the LSO and medial NTB (Coomes and Schofield, 2004; Coomes Peterson and Schofield, 2007). Interestingly, no contacts are found at the MSO. Even though cortical efferent pathways toward the SOC are fractional in comparison with those to the IC (Winer, 2006), those SOC-bound pathways may contribute to the DN1 attentional modulation if periolivary cell projections to main olivary nuclei (Coomes Peterson and Schofield, 2007) facilitate EI interactions in binaural processing. This inference may be consistent with the evidence that the trapezoid body lesions in humans slightly influence the DN1 orientation (Pratt *et al.*, 1998).

The discussion now proceeds to monaural influences on the DN1 that are independent of auditory selective attention. The multiple parallel mediator model revealed that, without assuming any influence of auditory selective attention, click stimulation enhanced DN1 peaks via the right monaural ABR (Fig. 5A). The right-ear advantage for enhancing the click-evoked wave V amplitudes has been found in previous human neonate studies (Sininger and Cone-Wesson, 2006; Sininger *et al.*, 1998). As the electrically evoked DN1 increased, so did the right monaural electrically evoked wave V amplitude for the majority of participants of Hu and Dietz (2015). Those findings are consistent with the present laterality outcomes (Figs. 2E and F) for clicks, yet not tone-pips, and suggest that the right monaural pathway in the brainstem dominates during task-independent corticopetal processing (Sininger and Cone-Wesson, 2006; Sininger *et al.*, 1998). Thus, one interpretation for the multiple parallel mediator model analysis is that the dominance of the right monaural pathway in click stimulation determines DN1 peaks, the effect of which is independent of the binaural interaction pathway. However, an alternative interpretation, which considers some involvement of the right monaural inputs in the binaural interaction pathway, is possible. In a perceptual context of horizontal sound localization, the right ear stimulation activated both the left and right brain structures above the cochlear nucleus level. In juxtaposition, the left ear stimulation principally activated right hemispheric structures

(Schönwiesner *et al.*, 2007). Without the spatial context, this asymmetry of ear stimulation for hemispheric activation was abolished. Schönwiesner *et al.* (2007) inferred, considering the lateral asymmetry, that a local corticofugal pathway modulated the subcortical auditory activities during a visual task (i.e., without auditory selective attention). A corticofugal pathway of auditory spatial processing could prime the brainstem localization mechanism, e.g., EI cells, giving priority to the right monaural inputs. In future assessment of this question, case studies of the unilateral brainstem lesions examining psychophysical sound lateralization (Furst *et al.*, 2000) may prove revealing.

5. Conclusion

This investigation critically reinterpreted the attentional modulation of the ABR-BIC (Ikeda, 2015). This reanalysis establishes that the effect of auditory selective attention on ABR-BIC DN1 presence reflects a purely binaural form of processing isolable from stimulus factors. This is a stimulus-universaleffect of auditory selective attention on DN1 presence: Whether stimuli are tone-pips or clicks, effect sizes of DN1 presence are larger with auditory than visual tasks. Effects of auditory selective attention on binaural and monaural ABRs confines to tone-pips, thus indicating that the stimulus-universal influence of auditory selective attention on DN1 presence relies on a purely binaural process. Those monaural attentional effects are isolated from a much stronger effect of stimulus type, such that clicks more vigorously elicit both left and right monaural ABRs, as well as binaural and summed monaural ABRs. Regardless of any null effect of attention, wave V of the right monaural ABR mediates the influence of stimulus type on the DN1. Both left and right monaural ABRs mediate the influence of stimulus type on the binaural ABRs. There are also direct effects of stimulus type on the DN1 and binaural ABR amplitudes.

Contributions

Kazunari Ikeda: Conceptualization; Data curation; Formal analysis; Investigation; Methodology; Project administration; Resources; Validation; Writing - original draft.

Tom Campbell: Data curation; Formal analysis; Funding acquisition; Resources; Software; Visualization; Writing - review & editing.

Declarations of interest

None.

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Table 1

Behavioral outcomes for the Attended modality as a function of Stimulus and Presentation

Measure	Stimulus	Binaural		Left monaural		Right monaural	
		Mean	(*SD*)	Mean	(*SD*)	Mean	(*SD*)
ART (msec)	Tone-pip	450.96	(57.31)	476.95	(58.32)	469.13	(46.35)
	Click	449.49	(44.85)	464.46	(41.17)	499.56	(54.44)
ACR (%)	Tone-pip	93.22	(6.89)	91.16	(5.40)	88.89	(6.60)
	Click	90.65	(7.81)	88.96	(5.76)	87.25	(6.99)
VRC (count)	Tone-pip	3.50	(1.45)	3.58	(2.15)	5.25	(2.80)
	Click	4.42	(1.83)	4.50	(2.75)	4.92	(2.81)

ART, auditory reaction time; ACR, auditory correct rate; VRC, visual response count; SD, standard deviation.

Table 2

Comparison of the DN1 peak amplitude measures with zero voltages by ANOVA

Stimulus	Derivation	Attended Modality	$F(1, 11)$	p	$p_{\alpha/8}$	η_p^2	95% CI
Tone-pip	(A1+A2)/2	Auditory	17.774‡	0.001	0.012	0.618	[-0.214, -0.067]
		Visual	4.769 *	0.052	0.412	0.302	[-0.225, -0.001]
	(M1+M2)/2	Auditory	10.926 †	0.007	0.056	0.498	[-0.188, -0.038]
		Visual	3.866 *	0.075	0.600	0.260	[-0.225, -0.013]
Click	(A1+A2)/2	Auditory	26.045 ‡	< 0.001	0.003	0.703	[-0.225, -0.090]
		Visual	16.301 ‡	0.002	0.016	0.597	[-0.257, -0.076]
	(M1+M2)/2	Auditory	31.998 ‡	< 0.001	0.001	0.744	[-0.348, -0.153]
		Visual	16.910 ‡	0.002	0.014	0.606	[-0.237, -0.072]

‡ Significant with a critical α of 0.05, as withstands the Bonferroni correction at $\alpha/8$.

† Significant with a critical α of 0.05, marginal after the Bonferroni correction at $\alpha/8$.

* Marginal with a critical α of 0.05, nonsignificant after the Bonferroni correction at$\alpha/8$.

Table 3

Number of participants with a DN1 peak below zero and with a binaural peak below the summed monaural peak

Stimulus	Derivation	Attended Modality	DN1 < 0	%	B < L+R	%
Tone-pip	(A1+A2)/2	Auditory	10/12	83.33	10/12	83.33
		Visual	8/12	66.67	7/12	58.33
	(M1+M2)/2	Auditory	11/12	91.67	8/12	66.67
		Visual	8/12	66.67	7/12	58.33
Click	(A1+A2)/2	Auditory	11/12	91.67	11/12	91.67
		Visual	10/12	83.33	9/12	75.00
	(M1+M2)/2	Auditory	11/12	91.67	11/12	91.67
		Visual	10/12	83.33	7/12	58.33

DN1 < 0, participants with a DN1 peak below zero; B < L+R, participants with a binaural peak below the summed monaural peak.

Figure Legends

Fig. 1. The grand-average BIC difference waveforms (binaural minus summed monaural; **A-D**) with visual and auditory selective attention, alongside the corresponding ABRs (**E-L**). In each column, there are the corresponding binaural and summed monaural ABR grand-averages that are subtracted for the visual task (**E-H**) and for the auditory task (**I-L**). The first two columns plot the vertex-earlobe derivation; the second two, the vertex-mastoid derivation. The BIC peak nomenclature follows that of Dobie and Berlin (1979), whereas the ABRs followed the Jewett and Williston (1971) convention. An analysis window of the DN1 peaks for tones (9.5–10.5 msec) is later than that for clicks (6.5–7.5 msec), for which there are fewer discernible ABR peaks.

Fig. 2. Peak amplitude averages of the DN1 and the ABR wave V as a function of Attended modality and Stimulus: **A** and **B**, the DN1; **C** and **D**, the binaural and summed monaural ABR potentials; **E** and **F**, the monaural left and right ABR potentials. The left column concerns the vertex-earlobe derivation [Cz - (A1 + A2) / 2]; the right, the vertex-mastoid derivation [Cz - (M1 + M2) / 2]. Error bars denote 95% confidence intervals (CIs).

Fig. 3. With visual and auditory selective attention, the grand-average monaural ABRs. The upper row plots the ABRs for clicks; the lower row, tone-pips. The first two columns plot the vertex-earlobe derivation; the second two, the vertex-mastoid derivation. The nomenclature followed that of Jewett and Williston (1971).

Fig. 4. Scatterplots of peak amplitudes, in the DN1 time range, of binaural ABR amplitudes (**A–C**) and DN1s (**E–G**) as a function of the left (**A** and **E**), right (**B** and **F**), and summed (**C** and **G**) monaural responses, as well as DN1 amplitudes as a function of binaural responses (**D**).

Fig. 5. Two parallel multiple mediator models reveal that Stimulus directly predicts the DN1

and the binaural ABR. The models also reveal indirect pathways mediated by the right ABRs for the DN1; mediated by left and right ABRs for the binaural ABR. Eqs. 1 and 2 predict the consequent variable, *Y*, which is either the peak amplitude of the DN1 (**A**) or the binaural ABR (**B**). This prediction is from the antecedent variable Stimulus, *X*, with two, *k*, mediators, M_i, the peak amplitude of the ABR when the stimulus is presented to the left ear, M_1, or the right ear, M_2. In indirect paths, a model parameter coefficient a_i denotes the relation between the antecedent, Stimulus, *X*, and a mediator, M_i. A coefficient b_i, denotes the relation of those mediators to the consequent. Coefficient c' denotes the direct relation between antecedent and consequent. 95% bootstrapped CIs concern effect sizes from 5000 samples. Inferential statistical testing reveals the significance of each parameter, such that *, significant; ‡, highly significant.

Figure 1

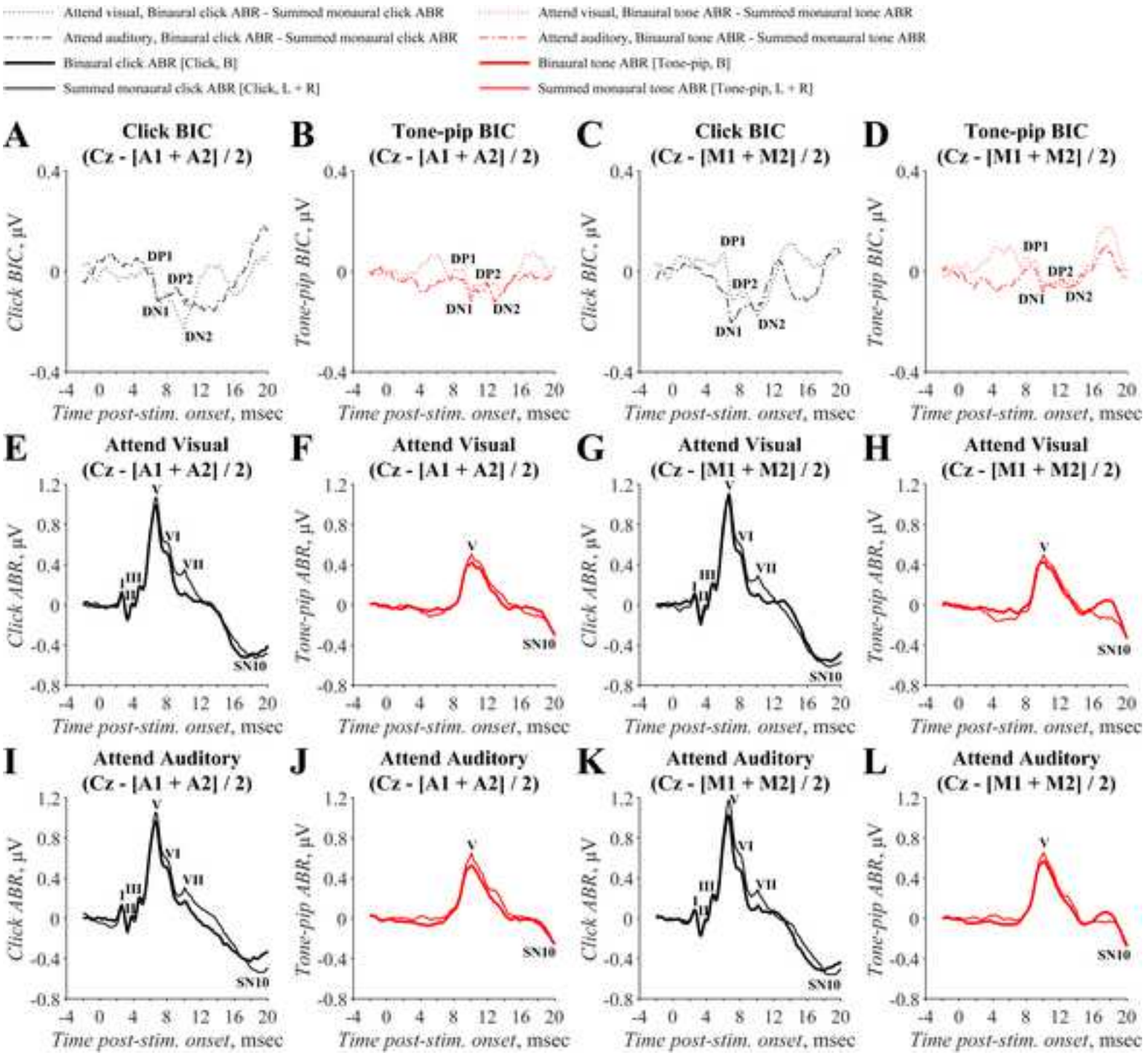


Figure 2

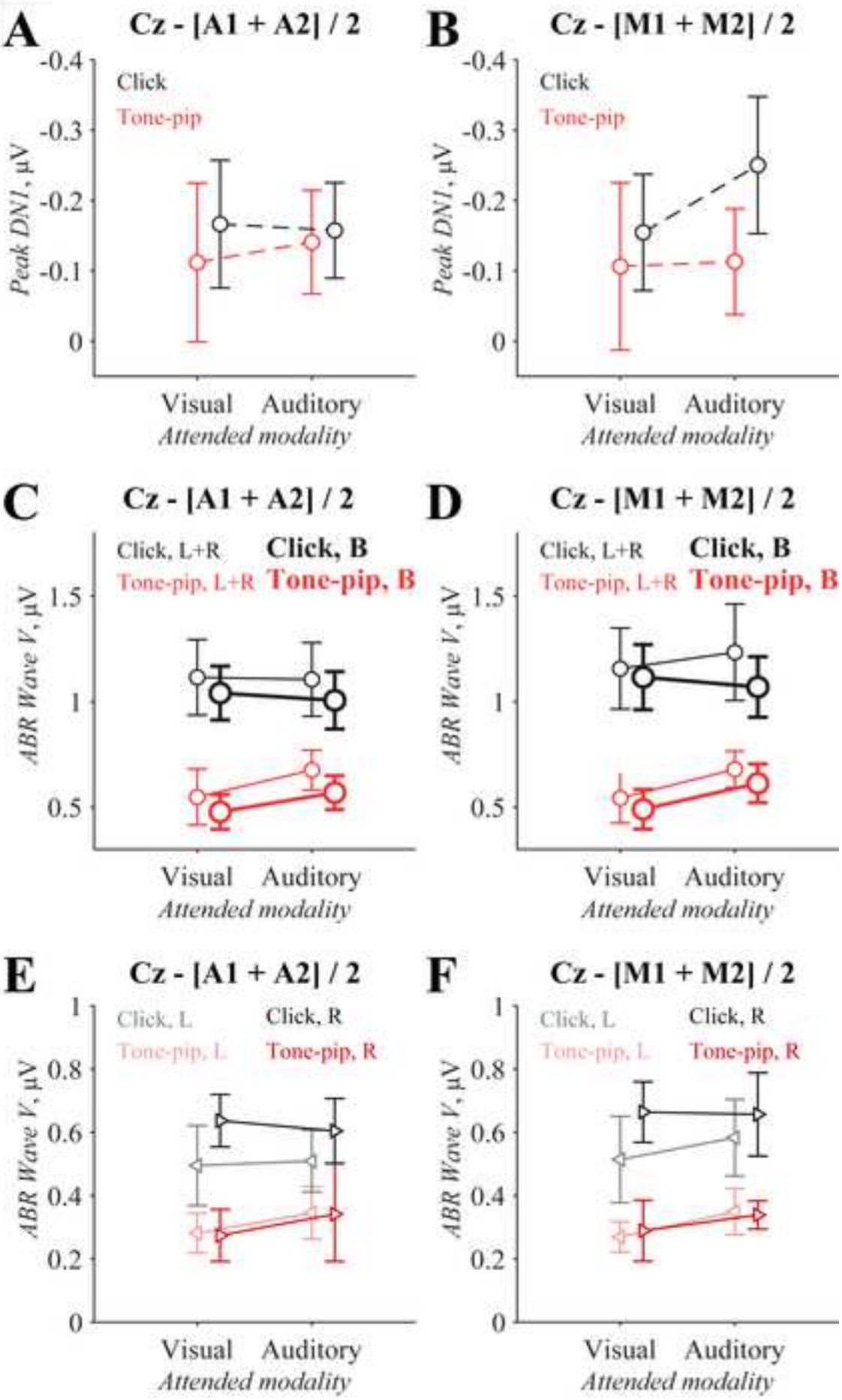


Figure 3

Click here to access/download;Figure;IkeCam_Fig3R.tif

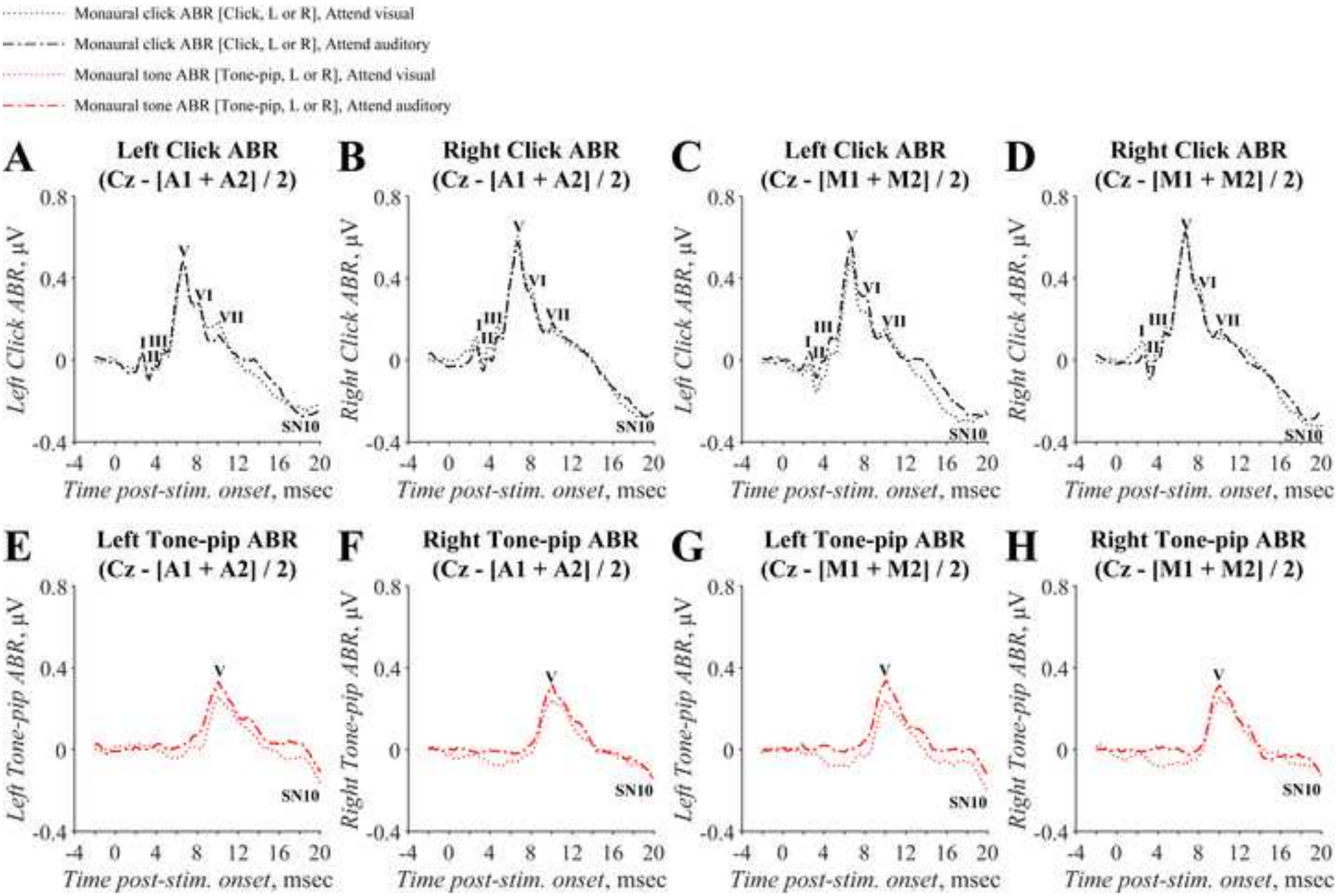


Figure 4

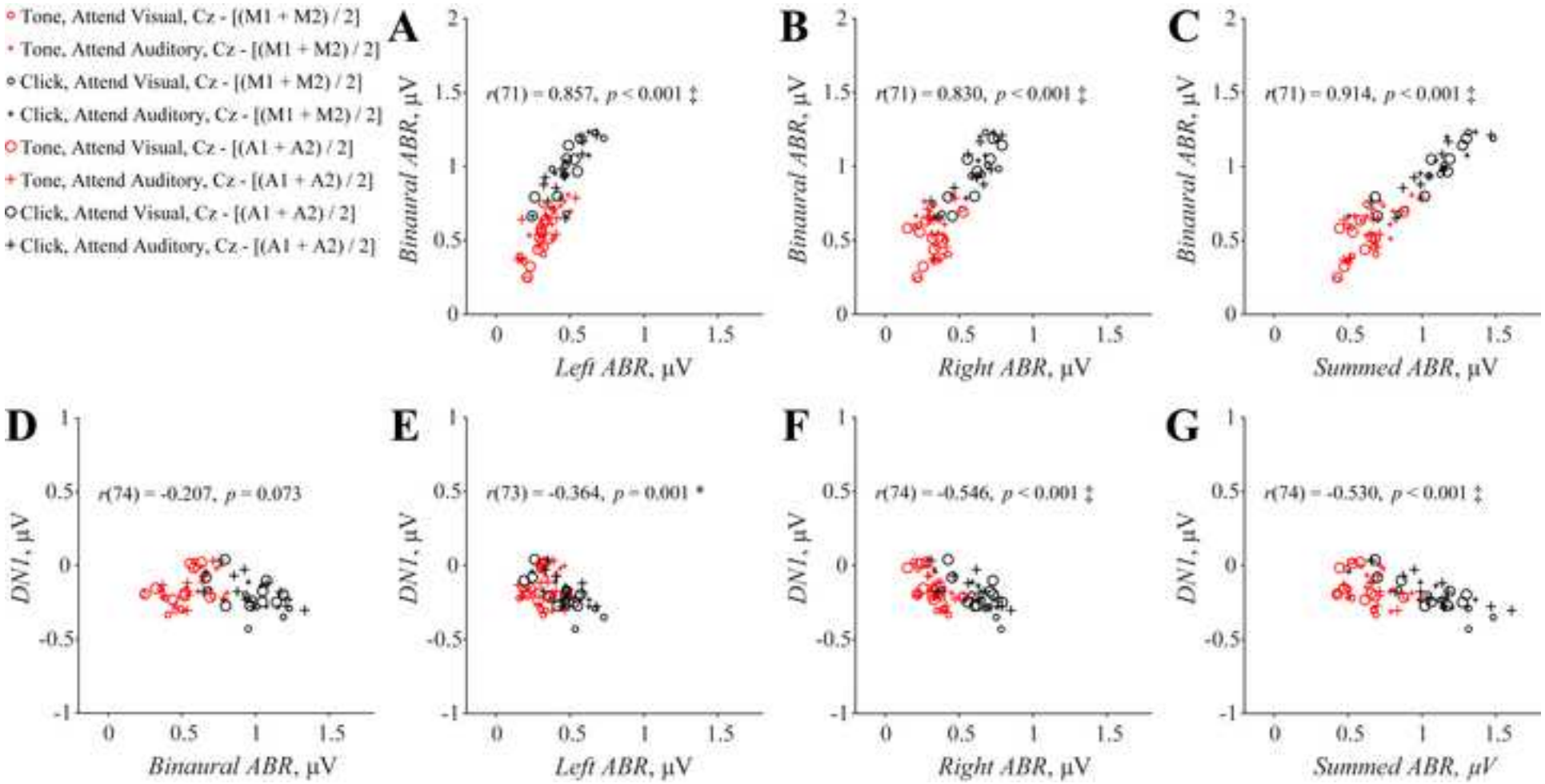
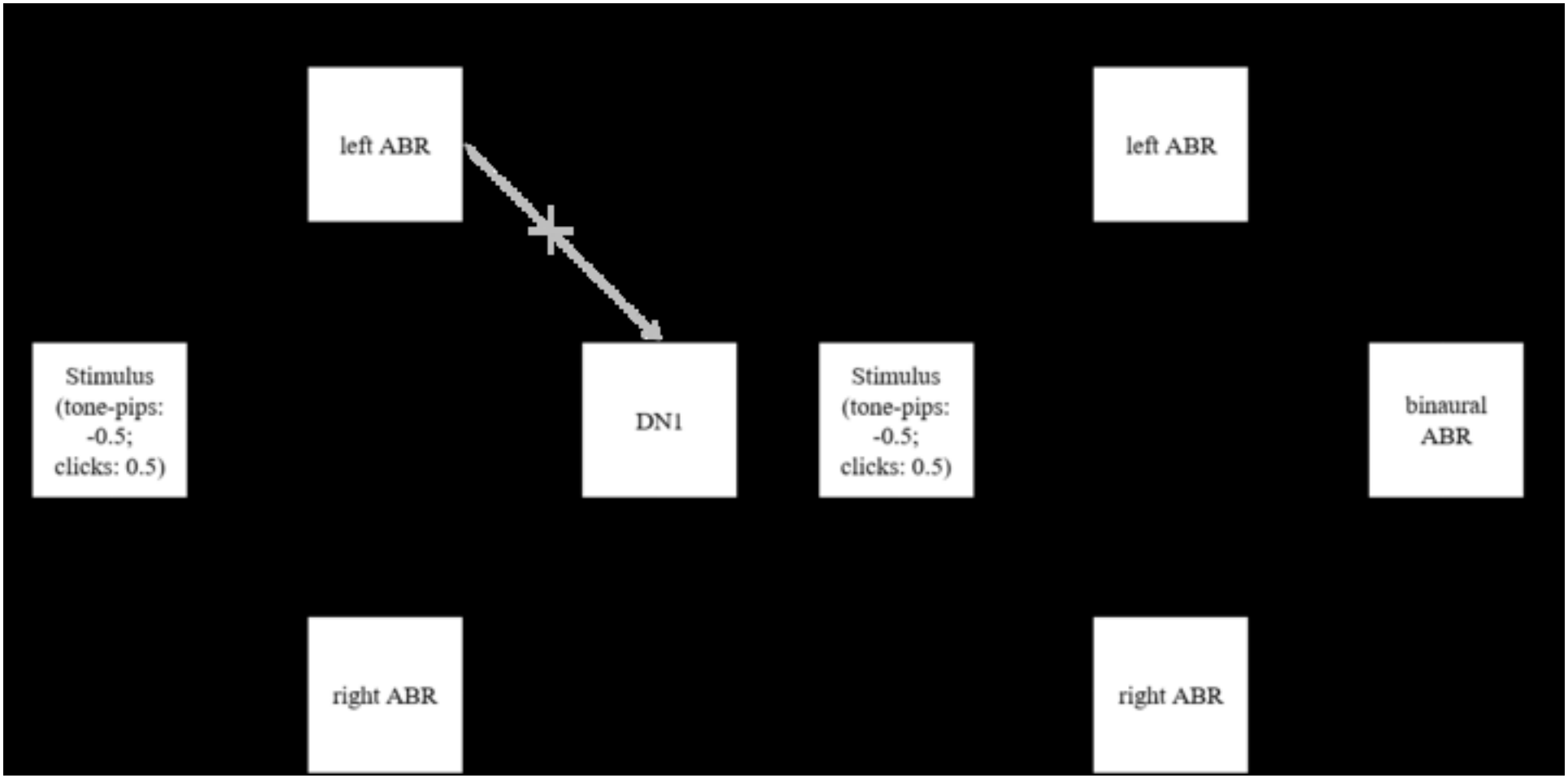


Figure 5



Author Contributions

Kazunari Ikeda: Conceptualization; Data curation; Formal analysis; Funding acquisition; Investigation; Methodology; Project administration; Resources; Validation; Writing - original draft.

Tom Campbell: Data curation; Formal analysis; Funding acquisition; Resources; Software; Visualization; Writing - review & editing.