

Speech intelligibility measurement

A latent variable approach on utterances' transcriptions

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Abstract

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1. Introduction

Intelligible speech can be defined as the extent to which the elements in an speaker’s acoustic signal, e.g. phonemes or words, can be correctly recovered by a listener [45, 70, 67, 34]. Because intelligible spoken language requires all core components of speech perception, cognitive processing, linguistic knowledge, and articulation to be mastered [34], its attainment carries an important societal value, as it is a milestone in children’s language development, the ultimate checkpoint for the success of speech therapy, and has been qualified as the “gold standard” for assessing the benefit of cochlear implantation [13].

The literature suggest two perspectives from which *speech intelligibility* can be assessed: the message and listener’s perspective [4, 5]. The first, also known as acoustic studies, is focused on assessing separately particular characteristics of the speech samples, e.g. their pitch, duration or stress (supra segmental characteristics), or the articulation of vowels and consonants (segmental characteristics) [61]. Whereas the second, also known as perceptual studies, is centered on making holistic assessments of the speech stimuli, e.g. measure their perceived quality [4, 5]. On both instances, the stimuli (children’s utterances) can be generated from reading at loud, contextualized utterances, or spontaneous speech tasks¹.

Furthermore, perceptual studies can use multiple approaches to measure intelligibility, but they can be largely grouped into two: objective and subjective ratings [43]. In *objective rating* methods, listeners transcribe the children’s utterances orthographically or phonetically, and use such information to construct a score. In that sense, in the transcription task, intelligibility can be inferred from the extent a set of transcribers can identify the word contained in an utterance [5]. In contrast, under *subjective rating* methods, listeners directly infer the utterance’s intelligibility score through specific procedures, e.g. absolute holistic, analytic, or comparative judgments, among others.

It is easy to deduce that *objective rating* methods produce more valid² and reliable³ scores than its *subjective* counterpart, and as a result, are usually used as an objective measure of intelligibility [5, 26].

Accompanying the intelligibility assessment methods, the literature supply a myriad of factors that are thought also contribute to the (under)development of intelligible spoken language [55, 6, 38, 27]. Among these are audiology related factors, such chronological age, age at implantation, the duration of device use, *hearing age*, bilateral or contralateral cochlear implantation, and the children’s preoperative and postoperative hearing levels. On the other hand, there are also child related factors, such as the cause of the hearing impairment (genetic, infections), additional disabilities (mental retardation, speech motor problems), and gender. Finally, there are also environmental factors, such as communication modality.

Considering all of the above, this paper seeks to investigate the speech intelligibility levels of normal hearing (NH) versus hearing-impaired children with cochlear implants (HI/CI). For that purpose, ten utterances recordings, from thirty two NH and HI/CI children, were selected from a large corpus of *spontaneously spoken speech* collected by the CLiPS research center. Additionally, we set up an experiment, where one hundred language students transcribed each stimuli to the Qualtrics environment [74]. Finally, the transcriptions were transformed into an entropy measure per utterance, which served as our outcome variable.

We believe this paper make three specific contributions to the understanding of the factors that drive the intelligibility of spoken language. First, we develop a novel analysis using a latent variable approach [25]. More specifically, we model *speech intelligibility* as a latent variable that can be inferred from the entropy measure replicates. This method offers three specific benefits. On the one hand, the method “constructs” an intelligibility score, which in turn, allow us to test different hypothesis and even make individual comparisons at the appropriate level. On the other hand, it allow us to control for different sources of variation. This is particularly important as, by failing to account for the appropriate hierarchies in the data, we could be ‘manufacturing’ false confidence in the parameter’s estimates, leading us to incorrect inferences [51]. Finally, the method also provides a ‘criterion’ on how reliable are the entropy replicates to measure speech intelligibility.

Second, we use Directed Acyclic Graph (DAG) [57, 17] to depict all the relevant variables though to influence speech intelligibility. We describe in detail our causal and non-causal hypothesis, and supplement our description with a causal diagram. The benefit of the method lies, not only, in that it makes the assumptions of our hypothesis more transparent, but also allow us to derive statistical procedures from the aforementioned causal assumptions [51, 76, 60].

¹ordered on increasing level of ecological validity [30, 24]

²the extent to which scores are appropriate for their intended interpretation and use [48, 65].

³the extend to which a measure would give us the same result over and over again [65], i.e. measure something, free from error, in a consistent way.

Third and final, we wrap the analysis procedure under the Bayesian framework, providing the assumptions, and the steps required to reproduce the computational implementation of the models.

2. Materials and Methods

We set up an experiment where speech samples were transcribed by a group of listeners. The current section succinctly describes the participating children, the stimuli used, and the experimental setup, while also delve into the causal and statistical framework of analysis.

2.1. Children

Thirty two children were selected using a large corpus of *spontaneously spoken speech*, collected by the Computational Linguistics, Psycholinguistics and Sociolinguistics research center (CLiPS). The selection followed a two step procedure [26]. First, a sample of sixteen hearing-impaired children (ten boys, six girls) were selected based on the quality of their registered stimuli. Second, an additional matched sample of sixteen normal hearing children was also selected (six boys, ten girls), and served as a comparison group.

For the first group, all the hearing-impaired children with cochlear implants (HI/CI) were native speakers of Belgian Dutch, living in Flanders, the Dutch speaking area of Belgium. They were all raised orally using monolingual Dutch, with a limited support of signs. All of the children were screened by the Universal Neonatal Hearing Screening (UNHS), using an automated auditory brainstem response hearing tests for newborns, and all received the cochlear implantation before the age of two. Their medical and audiological records did not ascertain any additional health or developmental issues. Hence, no known additional comorbidities were though present. Finally, at the date of the measurement, they were all enrolled in the mainstream educational system.

For the second group, the sixteen normal hearing children (NH) were closely matched to the HI/CI group based on chronological age. All children were also native speakers of Belgian Dutch, and enrolled in the mainstream educational system. None reported hearing loss or additional disabilities, judged from the UNHS screening procedure and their respective parental report.

The characteristics of the selected children is detailed in Table 3, at the supplementary section A.1.

2.2. Stimuli

The stimuli consisted of children’s utterances, i.e. sentences of similar length, recovered from previously mentioned CLiPS corpus. More specifically, we use a portion of the corpus that consisted of ten utterances recordings for each of the thirty two selected children, adding to a total of 320 stimuli.

The stimuli were documented when the child was telling a story cued by the picture book ‘Frog, where are you’ [50] to a caregiver ‘who does not know the story’.

The recordings were orthographically transcribed with the CLAN editor in CHAT format [49]. The quality of the stimuli was ensured by selecting utterances with no syntactically ill-formed or incomplete sentences, any background noise, cross-talk, long hesitations, revisions or non-words [5]. The aforementioned transcriptions were used only in the selection process of the stimuli for the experiment.

2.3. Experimental setup

The experiment was setup to perform a transcription task in the Qualtrics environment [74]. One hundred language students from the University of Antwerp participated. The participants were native speakers of Belgian Dutch, without any particular experience with the speech of hearing-impaired children.

The participants and stimuli were divided into five groups, where each group of 20 students transcribed 64 stimuli on their series. The stimuli were presented to the listeners in a random order. As a result, the setup produced 20 transcriptions per utterance, adding to a total of 6400 transcriptions. The steps that comprised the task are detailed in the supplementary section A.2.1.

The data resulting from the transcription task was then processed and converted into one entropy measure per utterance (H), which served as our outcome variable.

The entropy measure was bounded in the unit interval $[0, 1]$, and used as a quantification of (dis)agreement between listeners’ transcriptions, where utterances yielding a high degree of agreement between transcribers were considered highly intelligible, and therefore registered a lower entropy ($H \rightarrow 0$). In

contrast, utterances yielding a low degree of agreement were considered as exhibiting low intelligibility, and therefore registered a higher entropy ($H \rightarrow 1$) [5, 26]. The procedure followed to calculate the entropies is detailed in the supplementary section A.2.2.

2.4. Causal framework

The analysis was informed by a preliminary work aimed at describing the causal and non-causal factors influencing speech intelligibility. More specifically, the current research uses a Directed Acyclic Graph (DAG) [57, 17] to describe all the relevant variables though to influence intelligibility. A DAG is a type of *structural causal model* that can be represented, among other ways, by a *causal diagram*.

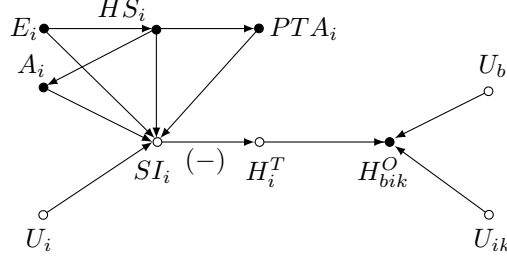


Figure 1: DAG: causal diagram describing the relationships among the analyzed variables. Open circles describe latent/unobserved variables, full circles describe observed variables.

Figure 1 shows the *causal diagram* for our research hypothesis. In the figure, H_{bik}^O denote the *observed* entropy replicates nested within children and experimental blocks, where $k = 1, \dots, 10$ utterances, $i = 1, \dots, 32$ children, and $b = 1, \dots, 5$ blocks. Moreover, H_i^T and SI_i denotes the child’s *true* entropy and speech intelligibility scores, respectively. In addition, HS_i denotes the children’s hearing status group, A_i their *hearing age*, PTA_i their post-implant pure tone average, and E_i the etiology of the disease that led to the hearing impairment.

Three main features can be emphasized from the figure. First, the children’s *speech intelligibility* and *true* entropy scores are thought to be latent/unobservable variables [25] (drawn with open circles, see supplementary section A.3 about the appropriate interpretations of the scores). The figure also shows the scores are thought to be inferable from the *observed* entropy replicates. More specifically, we are asserting the *observed* entropy replicates H_{bik}^O represent multiple realizations of a child’s *true* entropy H_i^T . Finally, we assume the *intelligibility* score SI_i is inversely/negatively related to the *true* entropy H_i^T , i.e. the lower the intelligibility the higher the entropy and vice versa, as expected from our the theory.

Second, the figure reflects the expected multilevel structure of variability in our data. This is particularly important as, by failing to account for the appropriate dependencies in the data, we could be ‘manufacturing’ false confidence in the parameter’s estimates, leading us to incorrect inferences [51].

Based on the experimental setup described in section 2.3, we anticipated the ten utterances, originated from each of the thirty two children, were also observed within a group of transcribers (series) assigned to the observation, i.e. our data has a hierarchy with children, replicates and block levels (U_i , U_{ik} and U_b , respectively). In this regard, we expect that if the experiment was ‘set up right’, the block random effects would explain a small amount of variability in the data, and its inclusion/exclusion in the model would not change the parameter estimates. Moreover, we expect a larger variability between children’s *speech intelligibility*, at least larger than the block random effects. Several evidence suggest this is particularly true among HI/CI children [77, 58, 52, 11, 75, 56, 34]. Finally, we did not had any comparable expectation for the variability in the replicates, as this feature has not been investigated before.

Third, the figure shows the assumed relationship among the relevant variables [55, 6, 38, 27], and how these influence the children’s intelligibility of speech. Furthermore, it also reveals we assume the variables are independent, beyond the described relationships. Here follows a description of our causal hypothesis related to the relevant variables.

For *hearing status* (HS_i), it is clear that its inclusion directly corresponds with the main purpose of the current research endeavor. i.e. compare the levels of *speech intelligibility* among NH and HI/CI children. Yet, we do not have a clear expectation about the levels among the groups. In terms of intelligibility, previous literature suggest that some HI/CI children manage to ‘catch up’ with their NH

counterparts [71, 40, 7, 35, 9, 20, 72]. However, other studies also indicate HI/CI children never reach similar levels than their NH analogues [54, 11, 14, 36, 34, 23, 39].

About **hearing age** (A_i), we expect it to be one of the main responsible for the increase in children’s *speech intelligibility*, no matter the children’s hearing status [5]. *Hearing age* is a composite variable constructed by combining the *chronological age* for the NH group, and the *device length of use* for the HI/CI group [26] (see table 3). The variable tries to approximate the amount of time a child has been actively hearing and developing his(her) language.

Several studies provide evidence that intelligibility increases with age [15, 16, 30, 31, 2, 8, 43], and no short of evidence has been presented in favor of using others surrogate measures, like *chronological age* [32, 40, 39] or *age at implantation* [55, 6, 9, 20]. We argue that the feasibility of using any other proxy measure, largely depends on the assumed reliability of the surrogate to approximate the variable of interest. In that sense, although we recognize *hearing age* is not a ‘perfect’ proxy [26], we argue it is the most appropriate to test our hypothesis, based on the relevant literature review and its assumed reliability to capture children’s language development (although the latter has not been tested). Furthermore, we believe the variable serve two additional purposes: (i) control for sampling bias (expanded in supplementary section A.4), and (ii) de-confound the parameter estimates of *hearing status* [17].

Finally, it is important to highlight that for modeling purposes, using more than one of the aforementioned proxies in tandem is not recommended. It is apparent from the previous description, the three surrogate measures share high similarities in their data construction. This in turn, could cause problems in the modeling procedure, as including variables that provide ‘similar information’ might lead to a statistical problem known as multicollinearity, in which our estimates get biased and less precise [28], leading us to incorrect inferences and conclusions.

For **pure tone average** (PTA_i), we expect it to have a small or null effect on *speech intelligibility*, as the empirical evidence seem to suggest [5]. *Pure tone average* is the child’s subjective hearing sensitivity, aided or unaided, by their hearing apparatus.

However, beyond the empirical evidence, the variable was included for two additional reasons. First, given that previous modeling efforts did not capture the full data hierarchy, it is possible that the effects of PTA on *speech intelligibility* has been largely overlooked. Although evidence seem to suggest the variable has no effect, it is also sensible to think that HI/CI children with severe hearing loss, as accounted by the variable, might develop their language at a slower rate. This is especially true, if we consider the signal provided by the cochlear implant is still degraded compared to normal hearing scenarios [21]. Lastly, a second reason is that the variable might be useful to de-confound the parameter estimates of *hearing status* [17], a purpose shared with the variable *hearing age*.

In the case of the **Etiology** of the disease that led to the hearing impairment (E_i), we expect it to have a differential effect on *speech intelligibility*, within the HI/CI group. However, since the severity of the etiology cannot be easily ascertain nor ordered, we cannot foresee the direction of such effects, i.e. genetic factors not necessarily lead to worse levels of language development and intelligibility, than factors related to infections.

The previous assumption does not have a correspondence with the empirical evidence, were the variable was deemed unimportant [5]. However, we argue that it is possible its effects have also been largely overlooked, due to the lack of statistical control of the full data hierarchy, similar to the PTA case. Moreover, as with its predecessors, the variable might also be useful to de-confound the parameter estimates of *hearing status* [17], assuming our DAG is appropriate.

Finally, it is important to highlight the reason for the absence of other variables, deemed relevant by the literature, in our causal hypothesis.

For the **type of cochlear implantation**, i.e. bilateral or contralateral, the variable was not included because we did not expect it to be related to other variables in the DAG, i.e. the decision on receiving one or the other is solely based on the intelligibility outcome, no matter how the latter is measured. This in turn means that its inclusion/exclusion would not confound our estimates. Additionally, given that most of the children underwent through sequential bilateral implantation (eleven in total), we anticipated the effect of variable already permeates the sample.

For the case of **additional disabilities**, e.g. mental retardation or speech motor problems, as children did not reported any additional comorbidities, the variable was not necessary.

In the case of **environmental factors**, such as communication modality, as the current sample of HI/CI children were raised orally using monolingual Dutch with a limited support of signs, a scenario similar to the NH group (see section 2.1), the variable was also not necessary.

Last but not least, **gender** was not included in our hypothesis, as no theoretical nor empirical evidence have been found on its effects [5].

As expected, it is possible that other unobserved confounding variables are not accounted by our assumptions, and therefore, our causal diagram. This is true for any type of social, behavioral and educational research. However, we argue that the additional transparency of our approach, and its ability to derive statistical procedures from causal assumptions, is its main strength [51, 76, 60].

2.5. Statistical analysis

Using the DAG, described in the previous section, we describe the algebraic formalism for our multiple Bayesian probabilistic models [44]. Each model targeted a different manner in which our research hypothesis could be investigated.

In general terms, every model was composed of two parts: a latent measurement model [25] and a structural equation model [42]. For the former, we represented the child’s *speech intelligibility* as a latent variable, inversely related to the child’s (latent) *true* entropy. Moreover, we modeled the latter as a variable that can be inferred from the (observed) entropy replicates in the following way:

$$H_{bik}^O \sim \text{BetaProp}(\bar{H}_{bi}, M_i) \quad (1)$$

$$\bar{H}_{bi} = \text{logit}^{-1}(a_b - SI_i) \quad (2)$$

$$H_i^T = \text{logit}^{-1}(-SI_i) \quad (3)$$

where $\text{BetaProp}(\mu, \theta)$ defines the beta-proportion distribution with parameters μ and θ [29, 46]; with $\mu \in [0, 1]$ and $\theta > 0$. Furthermore, \bar{H}_{bi} represented the “average” entropy measure nested within blocks and children ($b = 1, \dots, 5$ and $i = 1, \dots, 32$, respectively), while M_i denotes the ‘sample size’ of the distribution, possibly nested within children. Furthermore, H_{bik}^O , H_i^T and SI_i denotes the children’s entropy replicates, *true* entropy and *speech intelligibility* score, respectively. Finally, a_b represented the block random effects, and $\text{logit}^{-1}(x) = \exp(x)/(1 + \exp(x))$ represented the inverse-logit transformation.

From the previous algebraic structure we can notice. First, we modeled the average entropy of the replicates with $\mu = \bar{H}_{bi}$, which is a non-linear transformation of the block random effects and the children’s *speech intelligibility*. Second, the children’s *true* entropy H_i^T is inversely and non-linearly related to the *speech intelligibility* SI_i . Third and last, we captured the variability of the entropy replicates using $\theta = M_i$. See supplementary section A.5.1 for a detailed overview of the implications of using this approach.

For the second component we used a structural model [42]. In this portion, different iterations of our research hypothesis were proposed. Hereby we present the general representation from which the others can be derived:

$$SI_i = a_i + \alpha + \alpha_{E[i], HS[i]} + \beta_{A, HS[i]}(A_i - \bar{A}) + \beta_{P, HS[i]}sPTA_i \quad (4)$$

where E_i and HS_i are defined as in the previous section, A_i is the *hearing age* in years, while $sPTA_i$ is the standardized version of the post-implant pure tone average PTA_i . Moreover, a_i denoted the children’s random intercepts, and α the fixed intercept. On the other hand, $\alpha_{E[i], HS[i]}$ denoted the fixed effects intercept per etiology and hearing status group, while $\beta_{A, HS[i]}$ denoted the slope of *hearing age* per hearing status group. Finally, $\beta_{P, HS[i]}$ described the slope for the standardized pure tone average per hearing status group.

In the previous algebraic structure it is important to highlight, all parameters are estimated in the logit scale and centered at $sPTA_i = 0$ and $\bar{A} = 5$, the latter denoting the minimum *hearing age* in the sample. This is done to facilitate the interpretation of the parameters.

Thirteen statistical models were derived from the previous general description. Each model expressed one specific way in which our research hypothesis could be investigated. The models were characterized by two aspects: (i) the use of interactions and the selection of interacting variables, and (ii) the ‘sample size’ set to capture the replicates’ variability. For the former, the options were: intercept only (model 1), and multivariate regression with no interactions (models 2 to 4), with age and hearing status interaction only (models 5, 8, and 11), with etiology and hearing status interaction only (models 6, 9, and 12), and a full interaction model comprising the previous two (models 7, 10 and 13). In a similar fashion, for the latter the options were: one fixed ‘sample size’ (models 1, 2, 5, 6, and 7), one estimated ‘sample size’ (models 3, 8, 9, and 10), and finally, one ‘sample size’ per child (models 4, 11, 12, and 13), which we

dubbed *robust* models. See supplementary section A.5.2, where we expand about the need for *robust* models.

Table 1 provides an overview of the parameterization for the full set of proposed models. Notice all used block and children’s random effects (a_b and a_i , respectively), as well as the fixed effect intercept (α). Moreover, the models with one fixed ‘sample size’ used a value of ten, corresponding to number of utterances per child (see supplementary section A.5.1 on its implications for modeling). Finally, all models contemplated the interaction of the standardized pure tone average and the hearing status. The latter was imposed, because the collection of data did not contemplated the measurement of the pure tone average for the NH group. Therefore, the only interpretable parameter is the one estimated for the HI/CI children.

It is important to highlight that for all model implementations, the visual inspection of trace, trace-rank and autocorrelation plots was performed. Additionally, we also evaluated the Gelman-Rubin diagnostic and effective number of samples [37]. In all cases, the plots and statistics indicated the parameters achieved convergence, good mixing and lack of serial autocorrelation, all necessary requirements to be able to interpret the model estimates.

Additional details concerning the Bayesian implementation of the models, such as the representation of variability in a beta-proportion distribution, the parameters’ priors and hyper-priors, the estimation procedure, the data pre-processing phase, and simulation studies are expanded in the supplementary section A.5.

Model	Name	Parameters						
		M_i	a_b	a_i	α	$\alpha_{E[i],HS[i]}$	$\beta_{A,HS[i]}$	$\beta_{P,HS[i]}$
1	Intercept only (fixed ‘size’)	10	a_b	a_i	α	–	–	–
2	No interaction (fixed ‘size’)	10	a_b	a_i	α	$\alpha_{HS[i]}$	β_A	$\beta_{P,HS[i]}$
3	No interaction (one ‘size’)	M	a_b	a_i	α	$\alpha_{HS[i]}$	β_A	$\beta_{P,HS[i]}$
4	No interaction (robust)	M_i	a_b	a_i	α	$\alpha_{HS[i]}$	β_A	$\beta_{P,HS[i]}$
5	Age interaction (fixed ‘size’)	10	a_b	a_i	α	$\alpha_{HS[i]}$	$\beta_{A,HS[i]}$	$\beta_{P,HS[i]}$
6	Etiology interaction (fixed ‘size’)	10	a_b	a_i	α	$\alpha_{E[i],HS[i]}$	β_A	$\beta_{P,HS[i]}$
7	Full interaction (fixed ‘size’)	10	a_b	a_i	α	$\alpha_{E[i],HS[i]}$	$\beta_{A,HS[i]}$	$\beta_{P,HS[i]}$
8	Age interaction (one ‘size’)	M	a_b	a_i	α	$\alpha_{HS[i]}$	$\beta_{A,HS[i]}$	$\beta_{P,HS[i]}$
9	Etiology interaction (one ‘size’)	M	a_b	a_i	α	$\alpha_{E[i],HS[i]}$	β_A	$\beta_{P,HS[i]}$
10	Full interaction (one ‘size’)	M	a_b	a_i	α	$\alpha_{E[i],HS[i]}$	$\beta_{A,HS[i]}$	$\beta_{P,HS[i]}$
11	Age interaction (robust)	M_i	a_b	a_i	α	$\alpha_{HS[i]}$	$\beta_{A,HS[i]}$	$\beta_{P,HS[i]}$
12	Etiology interaction (robust)	M_i	a_b	a_i	α	$\alpha_{E[i],HS[i]}$	β_A	$\beta_{P,HS[i]}$
13	Full interaction (robust)	M_i	a_b	a_i	α	$\alpha_{E[i],HS[i]}$	$\beta_{A,HS[i]}$	$\beta_{P,HS[i]}$

Table 1: Proposed statistical models. Parameterizations.

3. Results

3.1. About our hypothesis

The current research used the Information-Theoretic Approach [1, 12] for model selection and inference. The application of the approach required: (i) the expression of the research hypothesis into statistical models, (ii) the selection of the most plausible models, and (iii) to produce inferences based on one or multiple selected models. The first requirement of the approach is covered in sections 2.4 and 2.5, and expanded in supplementary section A.5. Here we use the results of the second requirement, detailed in the supplementary section A.5.6, to produce the final inferences.

As detailed in the aforementioned supplementary section, the final conclusions of our research will be drawn from the comparisons of two models: (i) a no interaction model, with one estimated ‘sample size’ (model 3), and (ii) a full interaction model, with one estimated ‘sample size’ (model 10). The former is selected as it is the model with highest probabilistic support. The latter is considered because it encompasses the remaining highest supported models. Furthermore, no *robust* model is inspected, as we prefer a more parsimonious depiction of our hypothesis. Table 2 summarizes the parameters posterior estimates and contrasts of interest.

Parameter	Mean	SD	CI		HPDI		n eff.	Rhat
			2.5%	97.5%	2.5%	97.5%		
Model 3: No interaction (one ‘size’)								
parameters:								
a	0.179	0.189	-0.197	0.554	-0.198	0.552	3677.705	1.001
bP[2]	-0.117	0.166	-0.439	0.207	-0.440	0.202	1738.855	1.000
bA	0.432	0.141	0.154	0.715	0.170	0.723	1815.243	1.001
aHS[1]	0.284	0.235	-0.178	0.761	-0.203	0.728	2719.833	1.000
aHS[2]	0.116	0.217	-0.303	0.537	-0.304	0.537	2646.671	1.000
contrast:								
aHS[2]-aHS[1]	-0.168	0.246	-0.661	0.371	-0.650	0.324	n.a.	n.a.
Model 10: Full interaction (one ‘size’)								
parameters:								
a	0.217	0.179	-0.142	0.562	-0.118	0.580	3902.629	0.999
bP[2]	-0.122	0.173	-0.457	0.220	-0.460	0.216	1659.639	1.002
bAHS[1]	0.435	0.157	0.127	0.745	0.123	0.741	1477.460	1.000
bAHS[2]	0.237	0.177	-0.119	0.596	-0.089	0.615	1844.785	1.001
aEHS[1,1]	0.183	0.278	-0.358	0.726	-0.349	0.729	2524.451	1.000
aEHS[2,2]	0.212	0.241	-0.259	0.684	-0.259	0.684	2418.275	0.999
aEHS[3,2]	0.077	0.245	-0.402	0.547	-0.383	0.561	3015.559	0.999
aEHS[4,2]	0.007	0.269	-0.522	0.530	-0.547	0.499	3268.043	1.000
contrast:								
bAHS[2]-bAHS[1]	-0.197	0.208	-0.600	0.213	-0.590	0.223	n.a.	n.a.
aEHS[2,2]-aEHS[1,1]	0.029	0.352	-0.673	0.723	-0.714	0.682	n.a.	n.a.
aEHS[3,2]-aEHS[1,1]	-0.106	0.365	-0.823	0.607	-0.822	0.609	n.a.	n.a.
aEHS[4,2]-aEHS[1,1]	-0.176	0.380	-0.906	0.568	-0.928	0.535	n.a.	n.a.
aEHS[3,2]-aEHS[2,2]	-0.135	0.299	-0.719	0.453	-0.730	0.439	n.a.	n.a.
aEHS[4,2]-aEHS[2,2]	-0.205	0.344	-0.888	0.453	-0.902	0.430	n.a.	n.a.
aEHS[4,2]-aEHS[3,2]	-0.070	0.344	-0.744	0.612	-0.735	0.617	n.a.	n.a.

CI = compatibility interval

HDPI = highest posterior density interval

n eff. = effective number samples

Rhat = Gelman-Rubin diagnostic

n.a. = not available / not applicable

[1] = NH children, [2] = HI/CI children

[1,i] = NH children, [2,i] = genetic, [3,i] = CMV infection, [4,i] = unknown etiology

Table 2: Selected statistical models: results.

Before any interpretation of the parameters, it is important to highlight a problematic that will permeate all of our inferences. Given the large amount of variability registered at the children and replicates levels, the models will not be able to produce unequivocal null hypothesis rejections (compatibility and HPD intervals not including zero). The reasons for this are further expanded in section 3.2. However, from supplementary section A.5.5, we can be certain that our models can affirm the parameter values with at least 60% power, depending on the size of the effects. Therefore we will continue interpreting the parameters, but the reader should cautious into understand the CI and HPDI indicate our data size is not enough to ultimately define the conclusions here arrived.

First, about *hearing status*, model three reveals that HI/CI children have a modest lower level of *speech intelligibility*, compared to their NH counterparts (aHS [2] - aHS [1]). The result then seem to support previous evidence on the matter [54, 11, 14, 36, 34, 23, 39].

However, model ten paints a more nuanced story. The contrasts of the model reveal that HI/CI children with genetic etiology manage to reach similar levels of *intelligibility* as NH children (aEHS [2, 2] - aEHS [1, 1]), but the same cannot be said when the hearing impairment is caused either by a CMV infection (aEHS [3, 2] - aEHS [1, 1]) or other unknown causes (aEHS [4, 2] - aEHS [1, 1]). The former result

seem intuitive, as children with genetic causes have grown developing their language using the hearing apparatus, and therefore, also developing the appropriate neural connections to benefit from the new hearing input from the start, even when that input is slightly degraded [21]. In contrast, the evidence seem to suggest a different scenario for HI/CI children with other etiology status. What is hinted is that a child with other etiology status might need to ‘rewire’ his(her) neural connection in order to fully take advantage of the new hearing input signal, as it is assumed they begin their life hearing normally, and then loosing the ability to process sound. This result is particularly important, as it reveals that the factors that caused the hearing impairment matter, even when the children receive the new hearing input signal at a young age.

work in progress

3.2. About the hierarchy of variability

As expected, three hierarchies of variability were present in our data: the blocks and children’s random effects, as well as, the variability of the entropy replicates.

Evidence from the posterior estimates reveal the block random effects explained a small amount of variability in the data (top panel of figure 2), and its inclusion/exclusion in the model did not change the parameter estimates. The previous implies the experiment was correctly ‘set up’, as the series in which the utterances were transcribed did not explain a significant amount of variation, nor its exclusion biased the parameter estimates.

On the contrary, we observe a significantly larger variability between children’s *speech intelligibility*, more precisely, more than three times the block effects variability (middle panel of figure 2). The previous corroborated preliminary evidence on the matter [77, 58, 52, 11, 75, 56, 34, 5]. Additionally, it implied that given such a large amount of between variability, the statistical models might have a harder time delimiting the *hearing status* groups location on the intelligibility scale.

Finally, for the variability of the entropy replicates, the posterior estimates reveal a reasonable finding: the amount of variability at the replicates level is even larger than the one observed at the children level (bottom panel of figure 2). The latter implies there is significant error in measuring speech intelligibility with the entropy replicates. This further emphasize the difficulty of producing unequivocal inferences, in respect to the intelligibility levels of the *hearing status* groups.

3.3. The speech intelligibility scale

work in progress

3.4. Posterior predictive

work in progress

3.5. Influential observations

work in progress

4. Discussion

work in progress

5. Acknowledgments

A special thanks to Richard McElreath, for timely response and help.

6. Author contributions

All authors contributed to the development of the causal hypothesis. Jose Rivera performed the statistical analysis, Sven de Maeyer supervised the production of the documents and statistical results, and Steven Gillis collected the data.

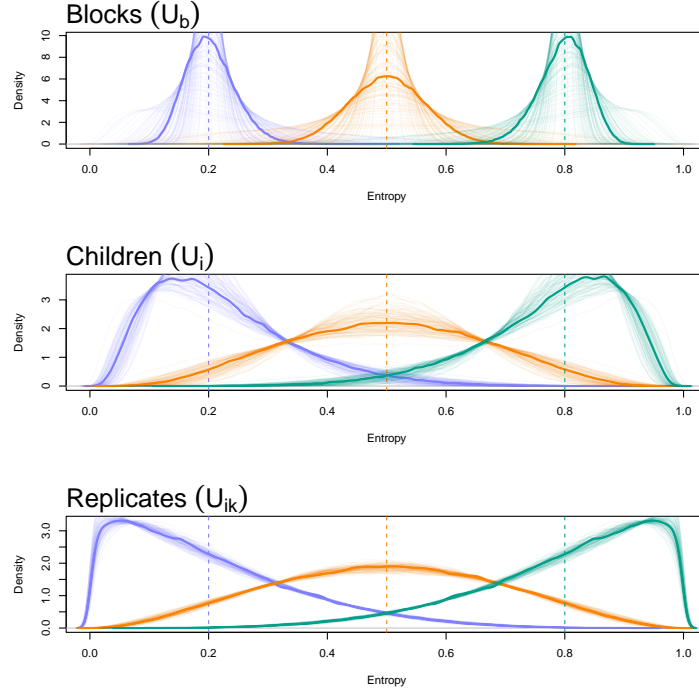


Figure 2: Posterior predictive: hierarchy of variability in the data. Distributions are plotted at the entropy replicates scale, considering three different average entropy values: $\mu = 0.2$, $\mu = 0.5$, and $\mu = 0.8$ (discontinuous lines). Thick solid lines represent the marginal distribution, thin solid lines depict 100 random posterior samples.

7. Financial support

The project was financed by the Flemish Government through the University Research Fund (BOF).

8. Conflicts of interest

The authors declare they have no conflict of interest.

9. Research transparency and reproducibility

The model simulation procedures and testing that support the findings of this study are openly available at https://github.com/jriveraespejo/PhD_UA_paper1.

Due to the privacy and confidentiality of subjects, the data set in which the model was implemented cannot be put online.

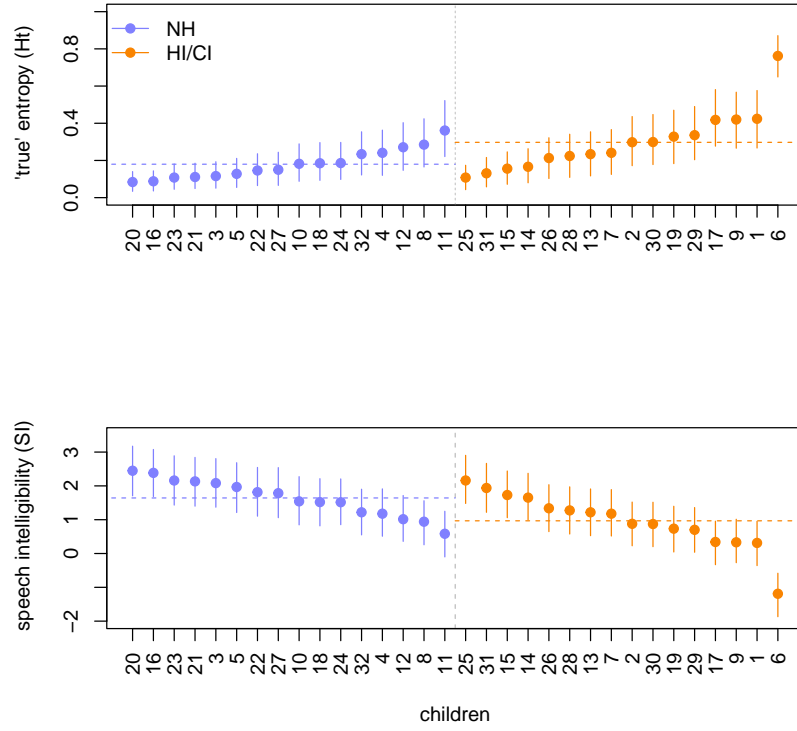


Figure 3: Posterior predictive: *true* entropy and *speech intelligibility* scales. Circles represent mean values, lines depict 95% highest posterior density intervals (HDPI). Horizontal discontinuous lines represent the marginal average for the *hearing status* group.

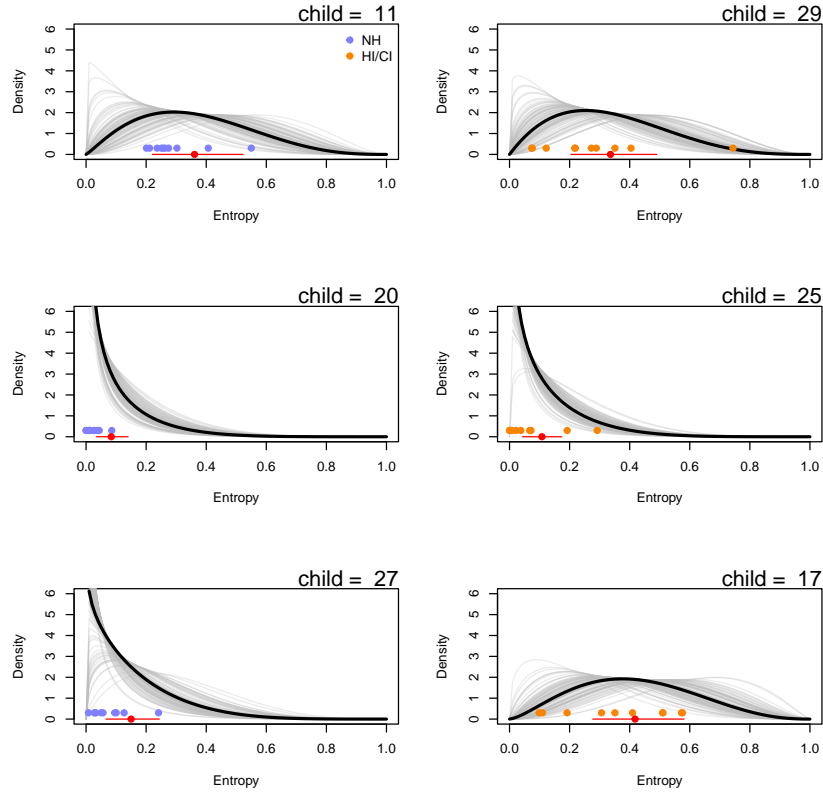


Figure 4: Posterior predictive: entropy replicates. Red point with lines represent the mean *true* entropy with 95% highest posterior density interval (HPDI). Thick solid line represents the marginal distribution, thin solid lines depicts 100 random posterior samples.

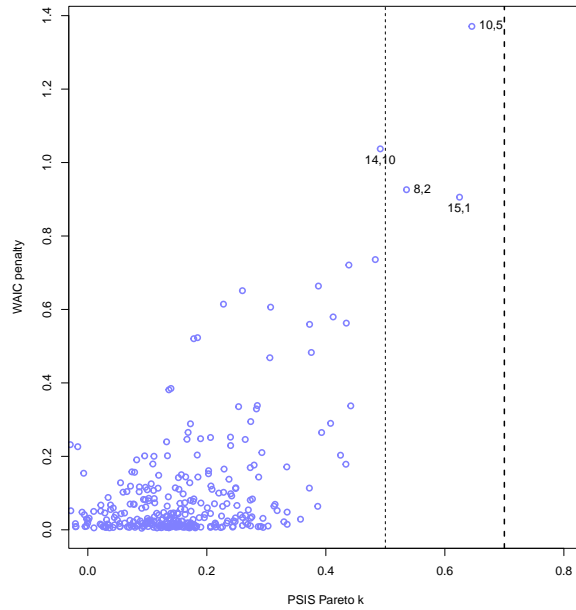


Figure 5: Influential observations. Pairs (child, utterance) are reported for specific observations.

A. Supplementary

A.1. Children characteristics

Table 3 shows the detailed information of the sampled children. The referred table includes the variable used for the matching procedure, i.e. chronological age, while also additional variables thought to be relevant for our hypothesis. No other variables are included as no known additional comorbidities, beside their hearing impairment, are suspected. Additionally, notice the pure tone average (PTA) data was not collected for the NH children.

Child	Gender	Chronological age (y;m)	Device length of use (y;m)	Hearing age (y;m)	Etiology	PTA (dB.)	
						unaided	aided
	HI/CI children						
1	female	05;07	05;00	05;00	Genetic	120	19
2	male	06;04	05;09	05;09	CMV	106	23
3	male	06;07	05;10	05;10	Genetic	114	35
4	female	06;10	06;00	06;00	Unknown	120	20
5	female	07;00	06;03	06;03	CMV	115	25
6	male	07;00	05;08	05;08	Genetic	93	32
7	female	07;00	06;08	06;08	Genetic	117	17
8	female	07;00	05;05	05;05	Unknown	112	42
9	male	07;00	05;05	05;05	CMV	120	15
10	female	07;01	05;11	05;11	Genetic	120	35
11	male	07;01	05;07	05;07	Genetic	113	42
12	male	07;02	06;05	06;05	Genetic	120	37
13	male	07;08	06;10	06;10	CMV	114	27
14	male	07;09	06;02	06;02	CMV	120	35
15	male	08;07	07;10	07;10	CMV	120	33
16	male	08;08	09;09	09;09	Genetic	95	27
	NH children						
17	female	06;05	n.a.	06;05	n.a.	n.a.	n.a.
18	female	06;06	n.a.	06;06	n.a.	n.a.	n.a.
19	female	06;07	n.a.	06;07	n.a.	n.a.	n.a.
20	female	06;09	n.a.	06;09	n.a.	n.a.	n.a.
21	female	06;09	n.a.	06;09	n.a.	n.a.	n.a.
22	male	06;09	n.a.	06;09	n.a.	n.a.	n.a.
23	male	06;09	n.a.	06;09	n.a.	n.a.	n.a.
24	male	06;10	n.a.	06;10	n.a.	n.a.	n.a.
25	female	07;01	n.a.	07;01	n.a.	n.a.	n.a.
26	male	07;01	n.a.	07;01	n.a.	n.a.	n.a.
27	male	07;04	n.a.	07;04	n.a.	n.a.	n.a.
28	female	07;08	n.a.	07;08	n.a.	n.a.	n.a.
29	male	07;08	n.a.	07;08	n.a.	n.a.	n.a.
30	female	07;09	n.a.	07;09	n.a.	n.a.	n.a.
31	female	08;00	n.a.	08;00	n.a.	n.a.	n.a.
32	female	08;01	n.a.	08;01	n.a.	n.a.	n.a.

(y;m) = (years;months)

n.a. = not applicable / not available

Table 3: Characteristics of selected children.

A.2. Experiment details

A.2.1. Transcription task

The setting for the transcription task comprised the following steps [4, 5]:

1. the listener took a seat in front of a computer screen, located at the campus’ computer laboratory.
2. the listener opened Qualtrics [74] and select the transcription task.
3. the listener read two set of instructions presented on the computer screen about:
 - a) *how to perform the task*,
 - b) *the aspects not considered for the task*.
4. the listener hear the stimuli through high quality headphones, set at a comfortable volume.
5. the listener wrote the orthographic transcriptions of the utterances, in a free text field in the software environment.

A.2.2. Entropy calculation

The outcome from the transcription task was obtained following a two step procedure [5]. First, we aligned the participant’s orthographic transcriptions, at the utterance level, in a column-like grid structure similar to the one presented in Table 4. This step was repeated for every one of the 6400 transcriptions. Lastly, we computed the entropy measure of the aligned transcriptions as in Shannon [62]:

$$H = H(\mathbf{p}) = \frac{-\sum_{i=1}^n p_i \cdot \log_2(p_i)}{\log_2(N)} \quad (5)$$

where H is bounded in the unit interval $[0, 1]$, n denotes the number of word occurrences within each utterance, p_i the probability of such word occurrence, and N the total number of aligned transcriptions per utterance.

Transcription number	Utterance				
	1	2	3	4	5
1	de	jongen	ziet	een	kikker
	the	boy	see	a	frog
2	de	jongen	ziet	de	[X]
	the	boy	sees	the	[X]
3	de	jongen	zag	[B]	kokkin
	the	boy	saw	[B]	cook
4	de	jongen	zag	geen	kikkers
	the	boy	saw	no	frogs
5	de	hond	zoekt	een	[X]
	the	dog	searches	a	[X]
Entropy	0	0.3109	0.6555	0.8277	1

[B] = blank space, [X] = unidentifiable word

Table 4: Alignment and entropy calculation. Extracted from Boonen et al. [5], and slightly modified with illustrative purposes.

Entropy was used as a quantification of (dis)agreement between listeners’ transcriptions, i.e. utterances yielding a high degree of agreement between transcribers were considered highly intelligible, and therefore registered a lower entropy ($H \rightarrow 0$). In contrast, utterances yielding a low degree of agreement were considered as exhibiting low intelligibility, and therefore registered a higher entropy ($H \rightarrow 1$) [5, 26].

To exemplify relevant scenarios for the procedure, we generate the entropy for utterances 2, 4 and 5 present in Table 4. To make the example easy to calculate, we assume our data consisted only of five transcriptions in total ($N = 5$).

For the second utterance, we observe that four transcriptions identify it with the word *jongen*, while the last with the word *hond*. Therefore, we registered two word occurrences ($n = 2$), with probabilities $\mathbf{p} = (p_1, p_2) = (4/5, 1/5)$, and entropy measure equal to:

$$\begin{aligned} H &= \frac{-\sum_{i=1}^2 p_i \cdot \log_2(p_i)}{\log_2(5)} \\ &= \frac{-[0.8 \log_2(0.8) + 0.2 \log_2(0.2)]}{\log_2(5)} \\ &\approx 0.3109 \end{aligned}$$

For the fourth utterance, we observe that two transcriptions identify it with the word *een*, one with *de*, one with *geen*, and one with a blank space [B]. Notice the blank space was not expected in such position, therefore, it was considered as a different word occurrence. As a result, the scenario had four word occurrences ($n = 4$), with probabilities $\mathbf{p} = (p_1, p_2, p_3, p_4) = (2/5, 1/5, 1/5, 1/5)$, and entropy measure equal to:

$$\begin{aligned} H &= \frac{-\sum_{i=1}^4 p_i \cdot \log_2(p_i)}{\log_2(5)} \\ &= \frac{-[0.4 \log_2(0.4) + 3 \cdot 0.2 \log_2(0.2)]}{\log_2(5)} \\ &\approx 0.8277 \end{aligned}$$

Finally, for the fifth utterance, we observe that all transcriptions identify it with different words. Notice that when a transcriber did not managed to identify (part of) the complete utterance, (s)he was instructed to write [X] to replace it. However, if more than one transcriber used [X] for an unidentifiable word, each was considered as being different from one another. The latter is done to avoid the artificial reduction of the entropy measure, as [X] values already indicate the lack of intelligibility of the word. Therefore, for the fifth utterance we registered five word occurrences ($n = 5$), with probabilities $\mathbf{p} = (p_1, \dots, p_5) = (1/5, \dots, 1/5)$, and entropy measure equal to:

$$\begin{aligned} H &= \frac{-\sum_{i=1}^5 p_i \cdot \log_2(p_i)}{\log_2(5)} \\ &= \frac{-5 \cdot 0.2 \log_2(0.2)}{\log_2(5)} \\ &= 1 \end{aligned}$$

A.3. About speech intelligibility

As described in the introduction, intelligible speech can be defined as the extent to which the elements in an speaker’s acoustic signal, e.g. phonemes or words, can be correctly recovered by a listener [45, 70, 67, 34]. More specifically, in the context of the transcription task, speech intelligibility can be inferred from the extent a set of transcribers can identify the word contained in an utterance [5].

Therefore in this paper, through the implementation of our proposed model, *speech intelligibility* is interpreted as a latent trait of individuals (hypothetical construct), which underlies the probability of observing a set of entropy replicates; that in turns, describes the ability of transcribers to identify the words in an utterance. Henceforth, statements such ‘*speech intelligibility is influenced by*’ can be read as ‘*the probability of observing a set of entropy replicates for each individual in the sample is influenced by*’. Similar interpretation can be extended to the (latent) *true* entropy measures.

Despite this practical approach, we emphasize we did our best to ensure the construct validity of our study, by ensuring the transcription task was well understood and appropriately performed by the transcribers.

We then expect speech intelligibility, as measured by our model, to reflect the (general) intelligibility of speech possessed by individuals, but do not deal with general epistemological considerations on the connection between the two.

A.4. Sampling bias

As it happens in most observational, and some experimental studies, ours can also be a potential victim of sampling bias. While stratifying on the selection variables can help to balance the samples, and even ‘correct’ the estimates [17, 19], as we do here by controlling for *hearing age*. Given the sample’s selection and matching procedures, we cannot ensure the HI/CI nor the NH groups are representative of their respective populations.

Nevertheless, we argue that by controlling for other relevant confounders, the qualitative results presented in this study holds. However, we cannot discard the presence of unobservable variables that could bias our results, and in that sense, inferences beyond this particular set of children must be taken with care.

A.5. Model details

A.5.1. Variability in the beta-proportion distribution

Figure 6 shows the implications of different ‘sample sizes’ (M_i) on the dispersion of the beta-proportion distribution [46]. The panels show different average entropies: the middle panel assumes $\mu = 0.5$, the left $\mu = 0.2$, and the right $\mu = 0.8$ (as shown by the discontinuous lines).

In all three panels we notice two prevalent pattern: (i) the higher the ‘sample size’, the less dispersed is the distribution, and (ii) as expected from non-linear models, the behavior of the dispersion depends on the location of the distribution, i.e. their average value.

Concerning the first patterns, we expect that if the posterior estimates for M_i reach lower values, it would imply the entropy replicates H_{bik}^O had high dispersion. In contrast, higher M_i estimates would imply a lower dispersion in the replicates. In this particular case, a good comparison point is the number of utterances/replicates. If $M_i < 10$, it would imply that our effective ‘sample size’ (measuring points) is less than the actual number of replicates, and therefore, we would need more replicates to provide an accurate measure of the (latent) *true* entropy. In contrast, $M_i > 10$ would imply the opposite.

As a final point, it is important to highlight that as the approach uses the ‘sample size’ parameter to model the replicates’ heterogeneity, we are effectively estimating a measurement error model [10] on entropy values.

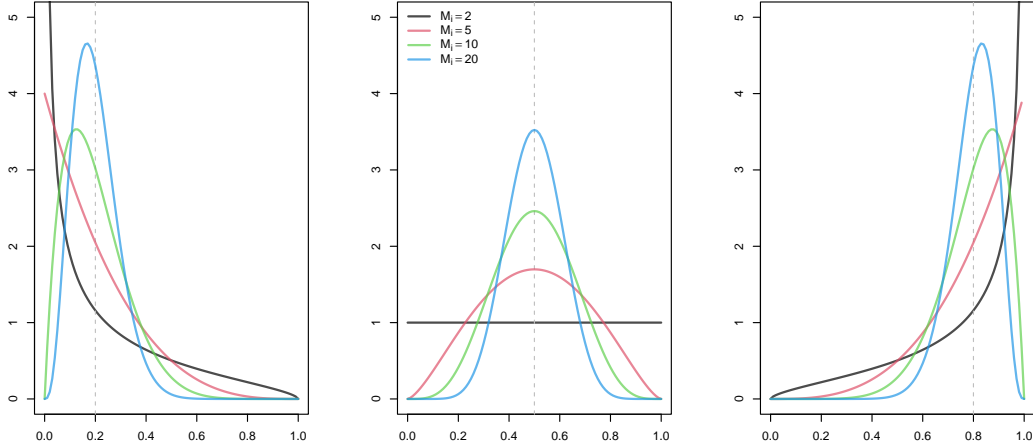


Figure 6: Variability in a beta-proportional distribution. Discontinuous lines describe the average value for the distribution (μ), solid lines describe the distribution assuming different $\theta = M_i$.

A.5.2. Data pre-processing

Besides the exclusion of corrupted observations, e.g. no available transcription, no other information was excluded before the modeling process. This decision departs from what has been done in previous research

[4, 66, 5]. The reason is that we believe the identification of influential observations, through preliminary or univariate procedures, might lead to erroneous exclusion of information, ultimately biasing our results. We believe the identification of *outliers* should not be done outside the context of a full modeling effort [51], as what can behave as an *outlier* based on a univariate analysis, might behave as expected under the appropriate model.

Considering the previous, instead of eliminating information based on preliminary analysis, we proposed a set of models that can be qualified as *robust* against influential observations. Considering the flexibility of the Bayesian framework, the proposal of such models was a trivial task (see Table 1).

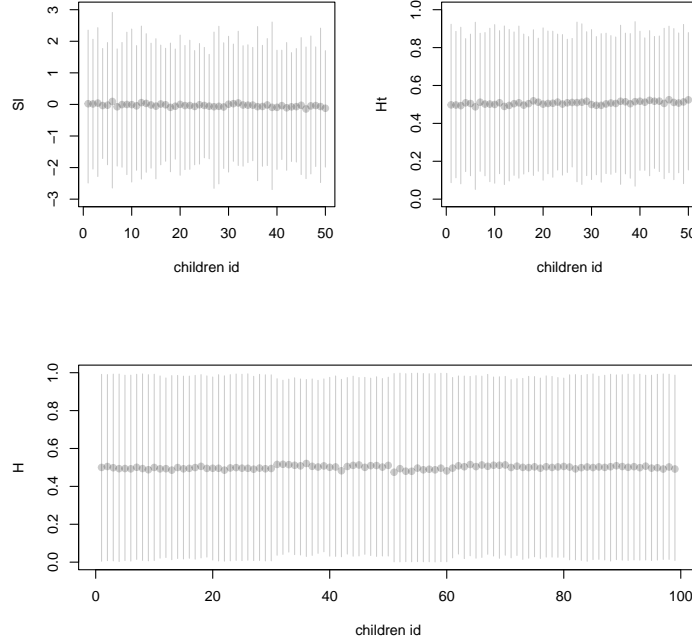


Figure 7: Prior distribution implications: speech intelligibility, *true* entropy and observed entropy scales. Circles represent mean values, lines depict the 95% compatibility intervals.

A.5.3. Priors and hyper-priors

For all models, the selection of priors and hyper-priors was done through prior predictive simulation [51]. The selected priors were considered mildly informative and regularizing.

Figure 7 shows the implication of our assumptions on the three outcome scales of interest: the *speech intelligibility*, the *true* entropy, and the *observed* entropy replicates. Notice, no undesirable assumption has crept in any of the scales, consequently, the estimates are free to visit a wide range of results, while also establishing a low probability of reaching impossible outcomes.

Hereby follows a description of the priors:

$$M_i \sim \text{Log-Normal}(\mu_M, \sigma_M) \quad (6)$$

$$a_b \sim \text{Normal}(\mu_b, \sigma_b) \quad (7)$$

$$a_i \sim \text{Normal}(\mu_i, \sigma_i) \quad (8)$$

$$\alpha \sim \text{Normal}(0, 0.2) \quad (9)$$

$$\alpha_{E[i], HS[i]} \sim \text{Normal}(0, 0.3) \quad (10)$$

$$\beta_{A, HS[i]} \sim \text{Normal}(0, 0.3) \quad (11)$$

$$\beta_{P, HS[i]} \sim \text{Normal}(0, 0.3) \quad (12)$$

while the hyper-priors were defined as follows:

$$\mu_M \sim \text{Normal}(0, 0.5) \quad (13)$$

$$\sigma_M \sim \text{Exponential}(1) \quad (14)$$

$$\mu_b \sim \text{Normal}(0, 0.2) \quad (15)$$

$$\sigma_b \sim \text{Exponential}(1) \quad (16)$$

$$\mu_i \sim \text{Normal}(0, 0.2) \quad (17)$$

$$\sigma_i \sim \text{Exponential}(1) \quad (18)$$

A.5.4. Estimation procedure

The proposed models in Table 1 were estimated under the Bayesian framework. More specifically, we used the No-U-Turn Hamiltonian Monte Carlo algorithm (No-U-Turn HMC) [3, 22, 41, 53] implemented in **Stan** [64]. We used four chains of 2000 iterations each, from which the first 1000 were discarded as a warm-up required by the software, leaving 1000 effective iterations.

Additionally, all procedures were performed in R 4.1.2 [59] and its integration packages [63], in a 64-bit system with a MINGW 32-bit cross-compiler (x86_64-w64-mingw32/x64).

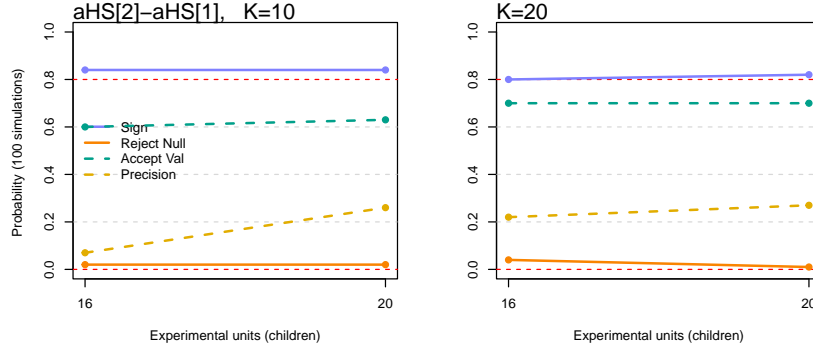


Figure 8: Group contrasts: power on small hearing group differences (≈ 0.15)

A.5.5. Simulation

Prior to fitting real data, all the proposed models were tested on simulated data. The simulation was done with two purposes: (i) to test the models' ability to recover the 'real' parameter values [33], and (ii) to test the power of our data size and assumed hypothesis [46]. On both instances, we used the *equivalent prior sampling* method [73], informed by the appropriate children population [18].

The results of our simulations highlight three key points. First, the probabilistic implementations work as intended, i.e. the statistical models manage to recover the 'real' estimate values, if enough sample size is available.

Second, considering our data size (sixteen children per group), if the assumed hearing status group difference is small (≈ 0.15), we can be certain that at least 60% of the time our models will affirm the predicted value (see green discontinuous line in figure 8). Moreover, the previous power threshold surpasses 80% if we assume medium group differences (≈ 0.4 , not shown). In the case of other parameters of interest, even if we assume small effects, the models always affirm the predicted value with power above 80%, e.g. for $bA = 0.15$ or $bP = 0.1$.

Third, the statistical precision of our estimates do not reach similar power results with our current data size (see yellow discontinuous line in figure 8). The latter implies that with sixteen children per group, the standard error of our estimates will not allow us to produce definite conclusions, e.g. we will not be certain if the two groups are not different because they are truly not statistically different or because the sample size is not enough to produce unequivocal inferences. A great part of this result is due to the assumed variability at the children and replicates levels, which we assumed moderate. However, further model testing indicated that such unequivocal results can be accomplished, if we managed to grab a hold on sample sizes similar to the full population size (70 children per group).

For more details on the assumed effects and the simulation code, refer to the GitHub repository: https://github.com/jriveraespejo/PhD_UA_paper1

A.5.6. Model selection

As stated in previous sections, the current research used the Information-Theoretic Approach [1, 12] for model selection. The application of the approach required: (i) the expression of the research hypothesis into statistical models, (ii) the selection of the most plausible models, and (iii) to produce inferences based on one or multiple selected models. Since the first step is covered in sections 2.4 and 2.5, and expanded in supplementary section A.5, here we proceed with the intermediate step.

We used the widely applicable information criterion (WAIC) [69], and the Pareto-smoothed importance sampling cross-validation (PSIS) [68] as the criteria to select among competing models. Two reasons justify our decision. First, both criteria allow us to use all the information of our Bayesian models, i.e. the posterior distribution of the parameters. Last, and more important, both criteria provide us with the best approximations for the out-of-sample (cross-validated) deviance [51]. The deviance is the best approximation for the Kullback-Liebler (KL) divergence [47], i.e. a measure of how far a model is from describing the *true* distribution of our data. In that sense, by comparing the deviance between competing models, we can measure which model is the farthest from ‘perfect (predictive) accuracy’ for our data [51].

Table 5 and 6 report the criteria for all the proposed models, sorted by the appropriate statistic. Three patterns are visible from the tables. First, we notice that around 63% of the evidence (WE) support two types of models: (a) no interaction and one estimated ‘sample size’, and (b) three (similar) interaction models with one estimated ‘sample size’. Furthermore, 35% of the evidence is condensed in the diverse types of *robust* models, while the remaining 2% is negligible dispersed in the remaining ones.

Second, comparing the differences in WAIC/PSIS from the ‘best’ model (dWAIC and dPSIS, respectively), we arrive at the same conclusion as the previous paragraph, i.e. the model with no interaction and one estimated ‘sample size’ is the best model, followed by the interaction models with one ‘sample size’, and a bit further away, by the *robust* models. One important point to highlight is that, even when there seem to be a hierarchy of fit among the models, effectively, all of these are indistinguishable from each other, as it can be inferred from their precision estimate (dSE). However, we can also observe, a more precise support for models with any type of interaction and one estimated ‘sample size’, than for the *robust* models.

Third, we observe a greater over-fitting penalty (pWAIC and pPSIS, respectively) for models with a fixed ‘sample size’ than any other model. This is quite particular, as one would expect that a model with less parameters would over-fit less the data. However in this case, it implies the complexity of the data requires more parameters, especially considering there are some highly influential observations that might sway the estimates of the less parameterized models (see section 3.5).

Model	Name	WAIC	SE	dWAIC	dSE	pWAIC	WE
3	No interaction (one 'size')	-621.0	42.99	0.0	–	31.1	0.18
10	Full interaction (one 'size')	-621.0	42.98	0.1	0.78	31.2	0.18
9	Etiology interaction (one 'size')	-620.7	43.07	0.4	0.48	31.2	0.15
8	Age interaction (one 'size')	-620.2	42.91	0.8	0.76	32.1	0.12
11	Age interaction (robust)	-620.1	42.70	0.9	2.21	34.2	0.12
12	Etiology interaction (robust)	-619.9	42.77	1.1	2.06	34.4	0.10
4	No interaction (robust)	-619.2	42.81	1.8	2.18	34.8	0.07
13	Full interaction (robust)	-618.8	42.65	2.2	2.17	35.0	0.06
5	Age interaction (fixed 'size')	-578.2	52.51	42.8	16.85	50.7	0.00
6	Etiology interaction (fixed 'size')	-578.2	52.63	42.8	16.95	50.7	0.00
2	No interaction (fixed 'size')	-577.6	52.59	43.5	16.97	51.0	0.00
1	Intercept only (fixed 'size')	-576.8	52.98	44.3	17.40	51.8	0.00
7	Full interaction (fixed 'size')	-575.8	52.58	45.3	16.96	52.1	0.00
SE	WAIC approximate standard error						
dWAIC	difference in WAIC from the best model						
dSE	standard error for the difference in WAIC from the best model						
pWAIC	WAIC over fitting penalty						
WE	weight of evidence						

Table 5: Fit of statistical models: WAIC.

Model	Name	PSIS	SE	dPSIS	dSE	pPSIS	WE
3	No interaction (one 'size')	-619.8	42.98	0.0	–	31.8	0.22
10	Full interaction (one 'size')	-619.7	42.97	0.0	0.82	31.8	0.22
9	Etiology interaction (one 'size')	-619.2	43.05	0.5	0.51	32.0	0.17
8	Age interaction (one 'size')	-619.0	42.89	0.7	0.81	31.8	0.15
11	Age interaction (robust)	-617.8	42.70	2.0	2.19	35.4	0.08
12	Etiology interaction (robust)	-617.7	42.77	2.1	2.07	35.5	0.08
4	No interaction (robust)	-616.5	42.81	3.3	2.19	36.1	0.04
13	Full interaction (robust)	-616.3	42.64	3.4	2.13	36.2	0.04
6	Etiology interaction (fixed 'size')	-576.0	52.63	43.7	17.00	51.7	0.00
5	Age interaction (fixed 'size')	-575.9	52.54	43.9	16.94	51.9	0.00
2	No interaction (fixed 'size')	-575.3	52.59	44.4	17.03	52.1	0.00
1	Intercept only (fixed 'size')	-574.6	52.97	45.1	17.44	52.9	0.00
7	Full interaction (fixed 'size')	-573.8	52.61	46.0	17.01	53.1	0.00
SE	WAIC approximate standard error						
dPSIS	difference in PSIS from the best model						
dSE	standard error for the difference in PSIS from the best model						
pPSIS	PSIS over fitting penalty						
WE	weight of evidence						

Table 6: Fit of statistical models: PSIS.

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