# 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, 73, 70, 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 [14].

The literature suggest two perspectives from which speech intelligibility can be assessed: the message and listener's perspective [5, 6]. 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) [64]. Whereas the second, also known as perceptual studies, is centered on making holistic assessments of the speech stimuli, e.g. measure their perceived quality [5, 6]. On both instances, the stimuli (children's utterances) can be generated from reading at loud, contextualized utterances, or spontaneous speech tasks<sup>1</sup>.

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 words contained in an utterance [6]. In contrast, under *subjective rating* methods, listeners directly infer the intelligibility score by assessing the speech sample's quality through specific procedures, e.g. absolute holistic, analytic, or comparative judgments, among others.

It is easy to deduce that *objective rating* methods produce more valid<sup>2</sup> and reliable<sup>3</sup> scores than its *subjective* counterpart, and as a result, are usually used as an objective measure of intelligibility [6, 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, 7, 37, 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 investigates 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 in the Qualtrics environment [76]. Finally, the transcriptions were transformed into entropy measures 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 measures 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, i.e. a reliability score.

Second, we use Directed Acyclic Graph (DAG) [57, 18] 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, 78, 63].

 $<sup>^1\</sup>mathrm{ordered}$  on increasing level of ecological validity [30, 24]

<sup>&</sup>lt;sup>2</sup>the extent to which scores are appropriate for their intended interpretation and use [48, 68].

<sup>&</sup>lt;sup>3</sup>the extend to which a measure would give us the same result over and over again [68], 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 method.

### 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<sup>4</sup>. 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 automated auditory brainstem response hearing tests for newborns, and receive 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 2, 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 [6]. The transcriptions were only used 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 [76]. 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.

Entropy is a measure bounded in the continuum [0, 1], and it was used as a quantification of (dis)agreement between listeners' transcriptions, where utterances yielding a high degree of agreement between

<sup>&</sup>lt;sup>4</sup>similar to one outlined in Faes et al. [26]

transcribers were considered highly intelligible, and therefore registered a lower entropy  $(H \to 0)$ . In contrast, utterances yielding a low degree of agreement were considered as exhibiting low intelligibility, and therefore registered a higher entropy  $(H \to 1)$  [6, 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, 18] 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*.

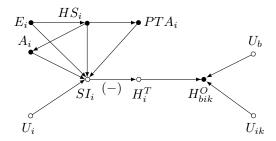


Figure 1: DAG: causal diagram describing the relationships among the analyzed variables

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, ..., 10 utterances, i = 1, ..., 32 children, and b = 1, ..., 5 blocks. Moreover,  $H_i^T$  and  $SI_i$  denotes the child's true entropy and speech intelligibility scores, respectively. In addition,  $A_i$  denotes the children's hearing age,  $E_i$  the etiology of the disease that led to the hearing impairment,  $HS_i$  the hearing status group, and  $PTA_i$  the post-implant pure tone average.

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 that the observed entropy replicates  $H_{bik}^O$  represent multiple realizations of a child's true entropy  $H_i^T$ . Finally, it shows 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 hierarchy 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. Therefore, we expected a hierarchy with children, replicates and block levels ( $U_i$ ,  $U_{ik}$  and  $U_b$ , respectively).

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 [79, 58, 52, 12, 77, 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, 7, 37, 27], and how these influence the children's intelligibility of speech. Furthermore, it also reveals that we assume the variables are independent, beyond the described relationships. Here follows a description of our causal hypothesis related to the relevant variables.

About *hearing age* and *hearing status*. For the former, we expect it to be one of the main responsible for the increase in speech intelligibility in children. Several studies provide evidence that for NH children, intelligibility increases with chronological age [16, 17, 30, 31, 3, 9, 43], and similar evidence can be found for the HI/CI children. Moreover, recent literature seem to suggest the effects are independent of the

children's hearing status [6]. For the latter, we do not have a clear expectation about the intelligibility levels among the groups. Previous literature suggest that some HI/CI children catch up with their NH counterparts [74, 39, 8, 35, 10, 20, 75]. However, other studies also seem to indicate the HI/CI children never reach similar levels than their NH counterparts [54, 12, 15, 36, 34, 23, 38].

Additionally, we expect *pure tone average* to have a small or null effect on speech intelligibility, as the evidence seem to suggest [6]. *Pure tone average* is the child's subjective hearing sensitivity, aided or unaided, by their hearing apparatus.

Furthermore, we expect the *Etiology* of the disease, that led to the hearing impairment, 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.

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, 78, 63].

Finally, we advise the reader to follow the supplementary section A.4 for a extended view of our assumptions and the reasons why other variables, deemed relevant by the literature, were not considered in our hypothesis.

### 2.5. Statistical analysis

Using the DAG described in previous section we developed multiple bayesian probabilistic models [44], each reflecting our different hypothesis.

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:

$$H_{bik}^{O} \sim \text{BetaProp}\left(\bar{H}_{bi}, M_{i}\right)$$
 (1)

$$\bar{H}_{bi} = \log i t^{-1} (a_b - SI_i) \tag{2}$$

$$H_i^T = \log i t^{-1} (-SI_i) \tag{3}$$

where BetaProp( $\mu$ ,  $\theta$ ) defined the beta-proportion distribution with parameters  $\mu$  and  $\theta$  [29, 46]; where  $\mu \in [0,1]$  and  $\theta > 0$ . Furthermore,  $\bar{H}_{bi}$  represented the "average" entropy measure nested within blocks ( $b = 1, \ldots, 5$ ) and children ( $i = 1, \ldots, 32$ ), while  $M_i$  denoted the "sample size", possibly nested within children. Furthermore,  $H_{bik}^O$ ,  $H_i^T$  and  $SI_i$  were defined as in the previous section, and  $a_b$  represented the block random effects. Finally, logit  $H_i^T$  are  $H_i^T$  and  $H_i^T$  are  $H_i^T$  are  $H_i^T$  and  $H_i^T$  are  $H_i^T$  are  $H_i^T$  and  $H_i^T$  are  $H_i^T$  and  $H_i^T$  are  $H_i^T$  and  $H_i^T$  are  $H_i^T$  are  $H_i^T$  and  $H_i^T$  are  $H_i^T$  are  $H_i^T$  and  $H_i^T$  are  $H_i^T$  are  $H_i^T$  are  $H_i^T$  and  $H_i^T$  are  $H_i^T$  and  $H_i^T$  are  $H_i^T$  are  $H_i^T$  and  $H_i^T$  are  $H_i^T$  are  $H_i^T$  are  $H_i^T$  and  $H_i^T$  are  $H_i^T$  are  $H_i^T$  are  $H_i^T$  and  $H_i^T$  are  $H_i^T$ 

From the previous algebraic structure we can notice. First, we modeled the "average" entropy of the replicates with  $\mu = \bar{H}_{bi}$ , which was a non-linear transformation of the block random effects and the children's speech intelligibility. Second, the children 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 around the "average" using  $\theta = M_i$ . See supplementary section A.6.1 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:

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

$$\tag{4}$$

where  $E_i$  and  $HS_i$  are defined as in the previous section,  $A_i$  is defined 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  $\alpha$  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.

One important thing need to notice from the previous algebraic structure is that 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.

From the previous descriptions, thirteen statistical models were derived. Each model expressed one specific way our outlined research hypothesis could be investigated. The models were characterized by two aspects: (i) the use of interactions and which variables interacted, and (ii) the "sample size" that captures the replicates variability. For the former, the options were: intercept only model (model 1), and multivariate regression with no interactions (models 2 to 4), with age and hearing status interaction (models 5, 8, and 11), with etiology and hearing status interaction (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, 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.6.2, where we expand about the presence of robust models.

Table 1 provides an overview of their parameterizations. Notice, all models contemplate the use of block and children's random effects ( $a_b$  and  $a_i$ , respectively), as well as a fixed effect intercept ( $\alpha$ ). Moreover, all models contemplate the interaction of the standardized pure tone average and the hearing status. The latter was imposed because the collection of the data did not contemplated the measurement of the pure tone average for the NH group, therefore, we needed to only estimate the parameter for the HI/CI children, and this one was the easier way?.

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

Table 1: Proposed statistical models.

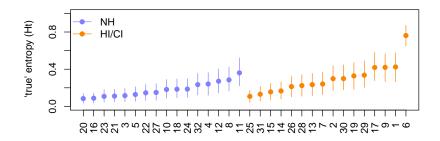
Finally, for the model implementation we proposed some priors for our parameters, decide on estimation procedure, performed a pre-processing phase for the data, and conducted a set of simulation studies. All of these are detailed in their corresponding supplementary section.

### 3. Results

- 3.1. Model selection and results
- 3.2. Speech intelligibility scale
- 3.3. Posterior predictive
- 3.4. Outlying observations

### 4. Discussion

talk about decision statements or thinking-at-loud tasks.. the listener provide a decision statement on why the selected stimulus sounded more intelligible



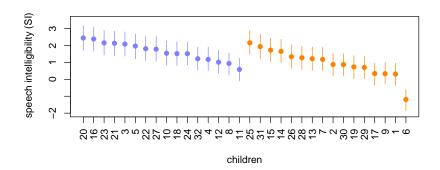


Figure 2: Posterior predictive: "true" entropy and speech intelligibility scales

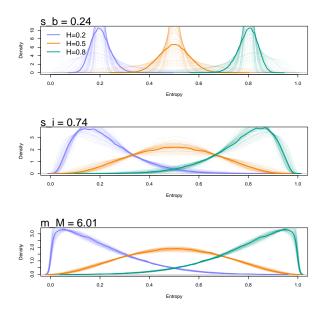


Figure 3: Posterior predictive: levels of variability

We relied on the DAG presented in section 2.4 only to define the analyses described 2.5. A better planning could be done by designing the experimental design according to our hypothesis described by the causal framework. but ....

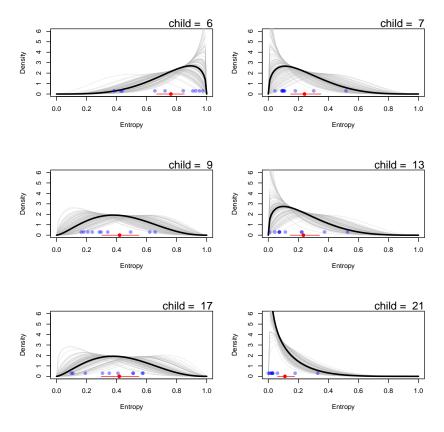


Figure 4: Posterior predictive: entropy replicates

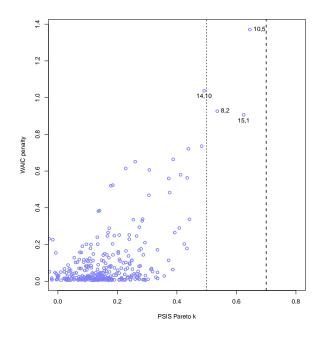


Figure 5: Outlying observations. Pairs (child, utterance) are reported for specific observations.

### 5. 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.

# 6. Financial support

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

## 7. Conflicts of interest

The authors declare they have no conflict of interest.

# 8. 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.

# A. Supplementary

### A.1. Children characteristics

Table 2 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.

Child         age (y;m)         of use (y;m)         age (y;m)         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;03         06;03         CMV         115         25           6         male         07;00         06;08         06;08         Genetic         93         32           7         female         07;00         06;08         06;08         Genetic         93         32           7         female         07;00         05;05         05;05         Unknown         112         42           9         male         07;00         05;05         05;05         Unknown         112         42           9         male         07;01         05;05         05;05         Unknown         112         42           10         female		Gender	Chronological	Device length	Hearing	Etiology	PTA (dB.)	
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;08         06;08         Genetic         93         32           6         male         07;00         05;08         05;08         Genetic         117         17           8         female         07;00         05;05         05;05         Unknown         112         42           9         male         07;00         05;05         05;05         Unknown         112         42           9         male         07;00         05;05         05;05         Unknown         112         42           10         female         07;00         05;05         05;05         CMV         120         35           11         male         07;01         05;07<	Child		age $(y;m)$	of use $(y;m)$	age $(y;m)$		unaided	aided
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;08         06;08         Genetic         93         32           6         male         07;00         05;08         05;08         Genetic         117         17           8         female         07;00         05;05         05;05         Unknown         112         42           9         male         07;00         05;05         05;05         Unknown         112         42           9         male         07;00         05;05         05;05         Unknown         112         42           10         female         07;00         05;05         05;05         CMV         120         35           11         male         07;01         05;07<		HI/CI						
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         Unknown         112         42           9         male         07;00         05;05         05;05         UNknown         112         42           9         male         07;00         05;05         05;05         UNknown         112         42           10         female         07;01         05;07         05;05         CMV         120         35           11         male         07;01         05;07<	1			05;00	05;00	Genetic	120	19
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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;01         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         120         37           13         male         07;02         06;05         06;05         Genetic         120         37           13         male         07;08         06;10         06;10         CMV         114         27           14         male         07;08         06;02         06;02         CMV         120         35           15         male         08;07         07;10	3	male	06;07	05;10	05;10	Genetic	114	35
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         120         37           13         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;07         07;10	4	female	06;10	06;00	06;00	Unknown	120	20
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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         120         35           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         35           15         male         08;08         09;09         09;09         Genetic         95         27           NH children           17         female         06;05         n.a.         n.a.         n.a.         n.a.           18         female	6	male	07;00	05;08	05;08	Genetic	93	32
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	7	female	07;00	06;08	06;08	Genetic	117	17
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	8	female	07;00	05;05	05;05	Unknown	112	42
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	9	male	07;00	05;05	05;05	CMV	120	15
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	10	female	07;01	05;11	05;11	Genetic	120	35
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	11	male	07;01	05;07	05;07	Genetic	113	42
$\begin{array}{c ccccccccccccccccccccccccccccccccccc$	12	male	07;02	06;05	06;05	Genetic	120	37
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.         n.a.           18         female         06;06         n.a.         06;06         n.a.         n	13	male	07;08	06;10	06;10	CMV	114	27
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. </td <td>14</td> <td>male</td> <td>07;09</td> <td>06;02</td> <td>06;02</td> <td>CMV</td> <td>120</td> <td>35</td>	14	male	07;09	06;02	06;02	CMV	120	35
NH children           17         female         06;05         n.a.         06;05         n.a.         n.a.         n.a.         n.a.           18         female         06;06         n.a.         06;06         n.a.	15	male	08;07	07;10	07;10	CMV	120	33
17       female       06;05       n.a.       06;05       n.a.	16	male	08;08	09;09	09;09	Genetic	95	27
18       female       06;06       n.a.       06;06       n.a.		NH chi	ldren					
19       female       06;07       n.a.       06;07       n.a.	17	female	06;05	n.a.	06;05	n.a.	n.a.	n.a.
20       female       06;09       n.a.       06;09       n.a.       n.a.       n.a.       n.a.         21       female       06;09       n.a.       06;09       n.a.       n.a.       n.a.       n.a.         22       male       06;09       n.a.       06;09       n.a.	18	female	06;06	n.a.	06;06	n.a.	n.a.	n.a.
21       female       06;09       n.a.       06;09       n.a.       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.	19	female	06;07	n.a.	06;07	n.a.	n.a.	n.a.
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$\begin{array}{cccccccccccccccccccccccccccccccccccc$	21	female	06;09	n.a.	06;09	n.a.	n.a.	n.a.
$\begin{array}{cccccccccccccccccccccccccccccccccccc$	1	male	06;09	n.a.	06;09	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.		male	06;09	n.a.	06;09	n.a.	n.a.	n.a.
26       male       07;01       n.a.       07;01       n.a.	1	male	06;10	n.a.	06;10	n.a.	n.a.	n.a.
27       male       07;04       n.a.       07;04       n.a.		female	07;01	n.a.	07;01	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.	1		,	n.a.	07;01	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.			· · · · · · · · · · · · · · · · · · ·	n.a.		n.a.	n.a.	n.a.
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31 female 08;00 n.a. 08;00 n.a. n.a. n.a.			07;08	n.a.	07;08	n.a.	n.a.	n.a.
, ,	1	female	07;09	n.a.	07;09	n.a.	n.a.	n.a.
$\begin{array}{ c c c c c c c c c c c c c c c c c c c$			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 2: Characteristics of selected children.

### A.2. Experiment details

### A.2.1. Transcription task

The setting for the transcription task comprised the following steps [5, 6]:

- 1. the listener took a seat in front of a computer screen, located at the campus' computer laboratory.
- 2. the listener opened Qualtrics [76] 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, e.g. the listeners were instructed to write one X to replace an unintelligible word, part of an utterance, or a complete utterance,
  - b) the aspects not to consider 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 environment.

### A.2.2. Entropy calculation

The outcome from the transcription task was obtained following a two step procedure [6]. 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 3. This step was repeated for every one of the 6400 transcriptions. Lastly, we computed the entropy measure of the aligned transcriptions as in Shannon [65]:

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

$$(5)$$

where H is bounded in the continuum [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	Utterance					
number	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]	$\operatorname{cook}$	
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 3: Alignment and entropy calculation. Extracted from Boonen et al. [6], 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  $(H \to 0)$ . In contrast, utterances yielding a low degree of agreement were considered as exhibiting low intelligibility, and therefore registered a higher entropy  $(H \to 1)$  [6, 26].

To exemplify relevant scenarios for the procedure, we generate the entropy for utterances 2, 4 and 5 present in Table 3. 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:

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

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:

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

Finally, for the fifth utterance, we observe that all of the transcriptions identify it with different words. Notice we consider the unidentifiable word [X] in the second transcription, as being different from the one in the last. This is done to avoid the artificial reduction of the entropy measure, as [X] values already indicate the lack of intelligibility of the word. Therefore, 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:

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

### 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, 73, 70, 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 words contained in an utterance [6].

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. Causal framework details

In this section we make explicit some of the assumptions that guided our causal framework, and later, our statistical analysis.

For *hearing age*, and its relevance in our research hypothesis, we describe how the variable is constructed and its inherent assumptions.

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 2). The variable tries to approximate the amount of time a child has been actively hearing and developing his(her) language. However, no short of evidence has been presented in favor of using others surrogate measures, like chronological age [32, 39, 38] or age at implantation [55, 7, 10, 20]. We argue that the feasibility of using any 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 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). Moreover, the variable serve two additional purposes: (i) control for sampling bias (see section A.5), and (ii) de-confound the parameter estimates of hearing status [18].

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 might cause problems in the modeling procedure, as including variables that provide "similar information" might lead to a problem known as multicollinearity, in which our estimates would be biased and less precise [28], leading us to wrong conclusions.

Regarding *hearing status*, 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.

In the case of *pure tone average*, the variable was included for two reasons. First, given that previous modeling efforts did not capture the full hierarchy of variability, it is possible that the effects of PTA on speech intelligibility has been largely overlooked. As one can infer, it might be 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 the signal in normal hearing scenarios [21]. Finally, as with *hearing age*, the variable might be useful to de-confound the parameter estimates of *hearing status* [18].

For *etiology*, it is possible that its effects on speech intelligibility has also been largely overlooked, due to the lack of control on the full hierarchy of variability, similar to the *pure tone average* case. Moreover, as with its predecessors, the variable might also be useful to de-confound the parameter estimates of *hearing status* [18], assuming our DAG is appropriate.

Finally, it is important to highlight the reason for the absence of other variables 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 it is measured. This in turn means that its inclusion/exclusion would not confound our estimates. Additionally, given that most of the children underwent though sequential bilateral implantation (eleven in total), we anticipated the effect of variable already permeates the sample, therefore, if we wanted to investigate its effect, a larger sample size would be required.

For the case of *additional disabilities*, e.g. mental retardation or speech motor problems, there was no need to consider it, as no additional comorbidities were reported.

In the case of *environmental factors*, such as communication modality, 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).

Last but not least, **gender** was not included in our hypothesis as no theoretical nor empirical evidence have been found on its effects [6].

### A.5. 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 [18, 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.6. Model details

### A.6.1. 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 an "average" entropy  $\mu = 0.5$ , 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.

In this sense, we expect that if the posterior estimates for  $M_i$  reach lower values, it would imply the entropy replicates  $H_{bik}^O$  have high dispersion; and therefore, we could say the replicates have low reliability to reflect the *true* entropy. In contrast, the higher the values for  $M_i$ , the lower the dispersion, and higher the reliability of the replicates to reflect the *true* entropy.

Then, as one can notice, the approach uses the "sample size" parameter to model the replicates' heterogeneity, effectively estimating an entropy's measurement error model [11].

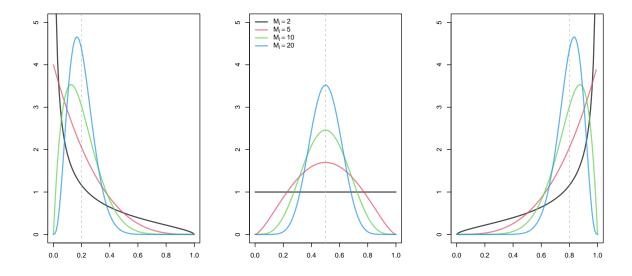


Figure 6: Variability in a beta-proportional distribution. Discontinuous lines describe the "average" value for the distribution ( $\mu$ ), solid lines describe the distribution assuming different  $\theta = M_{ik}$ .

#### A.6.2. 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 [5, 69, 6], and the reason for it, is that the identification of influential observations cannot be done outside the context of a full model [51], i.e. what can behave as an *outlier* based on a univariate analysis, might behave as expected under the appropriate model. Therefore, we believe the use of preliminary or univariate procedures might lead to erroneous identification of *outliers* and biased conclusions.

Considering the previous, we proposed a set of models that were *robust* against influential observations, rather than to eliminate them based on other analysis (see Table 1). Playing on the strengths of the Bayesian framework, the proposal of such model was a trivial task.

#### A.6.3. Priors

We determined priors for all models through prior predictive simulation. The resulting priors are weakly informative, specifying low probability for impossible outcomes and inducing regularized estimates, but allowing a very wide range of results. The priors used for the model are

Third, we use mildly informative priors and hyper-priors to state our uncertainty regarding the direction and magnitude of the effects $^5$ .

 $\sigma_a \sim \text{Exp}(1)$ 

$$M_{ik} \sim \text{LN}(\mu_M, \sigma_M)$$
 (6)  
 $a_i \sim \text{N}(\mu_a, \sigma_a)$  (7)  
 $\alpha \sim \text{N}(0, 0.3)$  (8)  
 $\alpha_{E[i], HS[i]} \sim \text{N}(0, 0.3)$  (9)  
 $\beta_{A, HS[i]} \sim \text{N}(0, 0.3)$  (10)  
 $\beta_P \sim \text{N}(0, 0.3)$  (11)  
 $\mu_M \sim \text{N}(0, 5)$  (12)  
 $\sigma_M \sim \text{Exp}(1)$  (13)  
 $\mu_a \sim \text{N}(0, 0.5)$  (14)

(15)

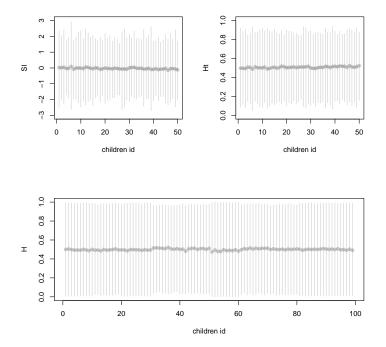


Figure 7: Prior distribution implications: speech intelligibility, true entropy and observed entropy scales.

#### A.6.4. Estimation

The proposed models, described in section 2.5, were estimated under the Bayesian framework<sup>6</sup>. More specifically, we used the No-U-Turn Hamiltonian Monte Carlo algorithm (No-U-Turn HMC) [4, 22, 41, 53] implemented in Stan [67]. Additionally, we used R [61] and its integration packages [66] to analyze its outputs.

<sup>&</sup>lt;sup>5</sup>see Rivera [62] (p. 18-19) for an intuition on prior elicitation.

<sup>&</sup>lt;sup>6</sup>see Rivera [62] (p. 11-13, 15-27) for a detailed description of its benefits and shortcomings.

#### A.6.5. Simulation

Preliminary to the data collection, we simulated data in silico to test the models and inform data collection procedure. The simulation code is available in the GitHub repository. [33]

#### A.6.6. Model selection

Following the successful and comprehensive analysis in van Daal [69] and Lesterhuis [48], the current research will also use the Information-Theoretic Approach (ITA) [2, 13] for the selection of competing models. The approach considers three steps: (1) state our hypothesis into statistical models, (2) select among competing models, and (3) make inferences based on one or multiple models.

First, for the translation of our working hypotheses into statistical models, we will use Directed Acyclic Graphs (DAG) and probabilistic programming [44]. A DAG is the simplest representation of a Graphical Causal Model (GCM), a heuristic model that contains information not purely statistical, but unlike a detailed statistical model, it allow us to deduce which variable relationships can provide valid causal inferences [40, 51]. In summary, a DAG is a reasonable way to state our hypothesis, and make our assumption more transparent. However, abide by the *no-free lunch* rule, the causal inferences produced under the DAG will only be valid if the assumed DAG is correct. In contrast, the probabilistic programming will serve as the algebraic formalist to define our statistical models.

Second, to select among competing models, we will use the Widely Applicable Information Criterion (WAIC) [72], and the Pareto-smoothed importance sampling cross-validation (PSIS) [71]<sup>7</sup>. Two reasons justify our decision. First, both criteria allow us to embrace the full flexibility and information of our bayesian implementation (outlined in Section ??). 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. McElreath [51] points out that is a rather benign characteristic of the model's selection procedure that we do not need the KL divergence's absolute value, as the *true* distribution of our data is not available (otherwise, we would not need a statistical model). But rather, using the difference in deviance between competing models, we can measure which model is the farthest from *perfect* (*predictive*) accuracy for our data<sup>8</sup>.

Finally, considering the evidence provided by the previous step, we proceed to make inferences based on one or multiple models.

<sup>&</sup>lt;sup>7</sup>van Daal [69] used the Akaikes Information Criterion (AIC) [1] with similar purposes.

<sup>8</sup> see McElreath [51] (p. 202-211) for the intuition and detailed derivation of the argument.

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