

1 Event History Analyses for psychological time-to-event data: A tutorial in R with examples  
2 in Bayesian and frequentist workflows

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## Abstract

15 Time-to-event data such as response times, saccade latencies, and fixation durations form a  
16 cornerstone of experimental psychology, and have had a widespread impact on our  
17 understanding of human cognition. However, the orthodox method for analysing such data  
18 – comparing means between conditions – is known to conceal valuable information about  
19 the timeline of psychological effects, such as their onset time and duration. The ability to  
20 reveal finer-grained, “temporal states” of cognitive processes can have important  
21 consequences for theory development by qualitatively changing the key inferences that are  
22 drawn from psychological data. Moreover, well-established analytical approaches, such as  
23 event history analysis, are able to evaluate the detailed shape of time-to-event distributions,  
24 and thus characterise the timeline of psychological states. One barrier to wider use of event  
25 history analysis, however, is that the analytical workflow is typically more time-consuming  
26 and complex than orthodox approaches. To help achieve broader uptake, in this paper we  
27 outline a set of tutorials that detail how to implement one distributional method known as  
28 discrete-time event history analysis. We illustrate how to wrangle raw data files and  
29 calculate descriptive statistics, as well as how to calculate inferential statistics via Bayesian  
30 and frequentist multi-level regression modelling. We touch upon several key aspects of the  
31 workflow, such as how to specify regression models, the implications for experimental  
32 design, as well as how to manage inter-individual differences. We finish the article by  
33 considering the benefits of the approach for understanding psychological states, as well as  
34 the limitations and future directions of this work. Finally, the project is written in R and  
35 freely available, which means the general approach can easily be adapted to other data  
36 sets, and all of the tutorials are available as .html files to widen access beyond R-users.

37       *Keywords:* response times, event history analysis, Bayesian multi-level regression  
38 models, experimental psychology, cognitive psychology

39 Word count: X

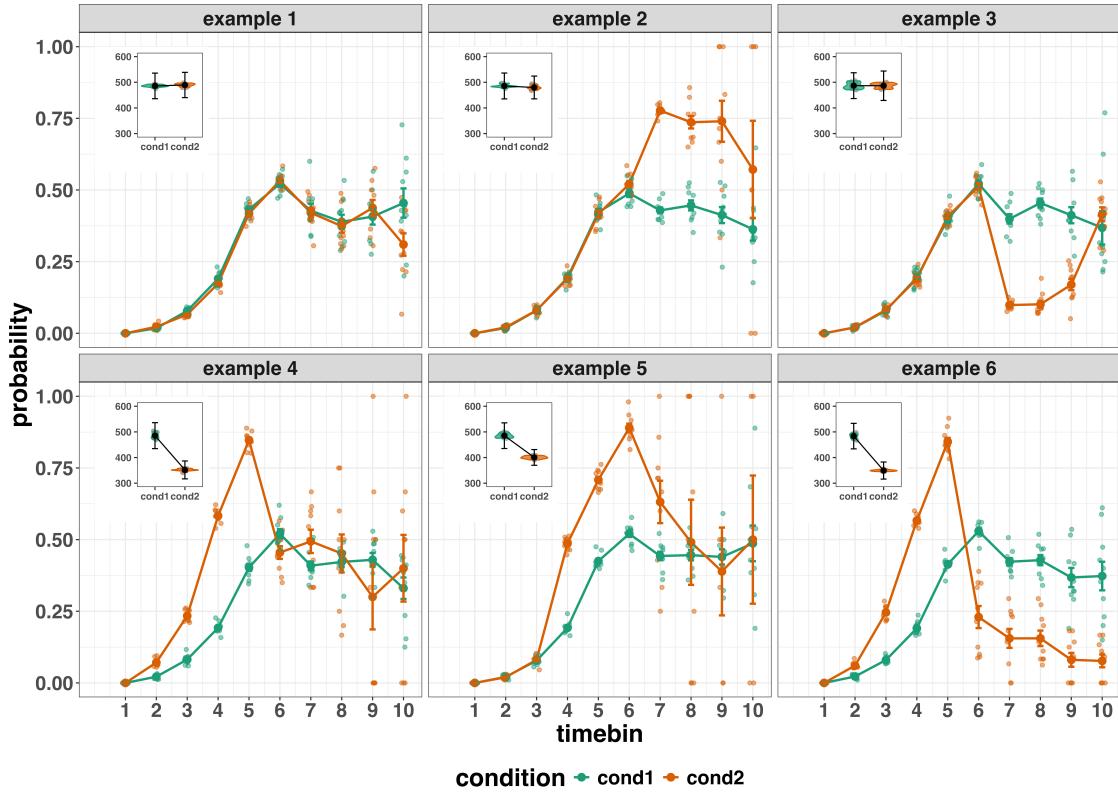
40 Event History Analyses for psychological time-to-event data: A tutorial in R with examples  
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42 **1. Introduction**

43 **1.1 Motivation and background context: Comparing means versus  
44 distributional shapes**

45 In experimental psychology, it is standard practice to analyse response times (RTs),  
46 saccade latencies, and fixation durations by calculating average performance across a series  
47 of trials. Such mean-average comparisons have been the workhorse of experimental  
48 psychology over the last century, and have had a substantial impact of theory development  
49 and our understanding of the structure of cognition and brain function. However,  
50 differences in mean RT conceal when an experimental effect starts, how long it lasts, how it  
51 evolves with increasing waiting time, and whether its onset is time-locked to other events  
52 (Panis, 2020; Panis, Moran, Wolkersdorfer, & Schmidt, 2020; Panis & Schmidt, 2016, 2022;  
53 Panis, Torfs, Gillebert, Wagemans, & Humphreys, 2017; Panis & Wagemans, 2009). Such  
54 information is useful not only for interpretation of the effects, but also for cognitive  
55 psychophysiology and computational model selection (Panis, Schmidt, Wolkersdorfer, &  
56 Schmidt, 2020).

57 As a simple illustration, Figure 1 shows the results of several simulated RT datasets,  
58 which show how mean-average comparisons between two conditions can conceal the shape  
59 of the underlying RT distributions. For instance, in examples 1-3, mean RT is always  
60 comparable between two conditions, while the distribution differs (Figure 1, top row). In  
61 contrast, in examples 4-6, mean RT is lower in condition 2 compared to condition 1, but  
62 the RT distribution differs in each case (Figure 1, bottom row). Therefore, a comparison of  
63 means would lead to a similar conclusion in examples 1-3, as well as examples 4-6, whereas  
64 a comparison of the distribution would lead to a different conclusion in every case.



*Figure 1.* Means versus distributional shapes for six different simulated dataset examples. For our purposes here, it is enough to know that the distributions plotted represent the probability of an event occurring in that timebin, given that it has not yet occurred. Insets show mean reaction time per condition.

65 Why does this matter for research in psychology? Compared to the aggregation of  
 66 data across trials, a distributional approach offers the possibility to reveal the timecourse of  
 67 psychological states. As such, the approach permits different kinds of questions to be  
 68 asked, different inferences to be made, and it holds the potential to discriminate between  
 69 different theoretical accounts of psychological and/or brain-based processes. For example,  
 70 the distributions in Example 4 show that the effect starts around 200 ms and is gone by  
 71 600 ms. In contrast, in Example 5, the effect starts around 400 ms and is gone by 800 ms.  
 72 And in the Example 6, the effect reverses around between 500 and 600 ms. What kind of  
 73 theory or set of theories could account for such effects? Are there new auxiliary

74 assumptions that theories need to adopt? And are there new experiments that need to be  
75 run to test the novel predictions that follow from these analyses? As we show later using  
76 concrete examples from past experimental data, for many psychological questions this  
77 “temporal states” information can be theoretically meaningful by leading to more  
78 fine-grained understanding of psychological processes as well as adding a relatively  
79 under-used dimension – the passage of waiting time – to our theory building toolkit.

80 From a historical perspective, it is worth noting that the development of analytical  
81 tools that can estimate or predict whether and when events will occur is not a new  
82 endeavour. Indeed, hundreds of years ago, analytical methods were developed to predict  
83 time to death (REFs). The same logic has been applied to psychological time-to-event  
84 data, as previously demonstrated (Panis et al., 2020). Here, in the paper, we hope to show  
85 the value of EHA for knowledge and theory building in cognitive psychology and related  
86 areas of research, such as cognitive neuroscience, as well as provide practical tutorials that  
87 provide step-by-step code and instructions in the hope that we can enable others to use  
88 EHA in a more routine, efficient and effective manner.

## 89 1.2 Aims and structure of the paper

90 In this paper, we focus on a distributional method for time-to-event data known as  
91 discrete-time event history analysis, a.k.a. hazard analysis, duration analysis, failure-time  
92 analysis, survival analysis, and transition analysis. We first provide a brief overview of  
93 hazard analysis to orient the reader to the basic concepts that we will use throughout the  
94 paper. However, this will remain relatively short, as this has been covered in detail before  
95 (Allison, 1982, 2010; Singer & Willett, 2003), and our primary aim here is to introduce a  
96 set of tutorials, which explain **how** to do such analyses, rather than repeat in any detail  
97 **why** you should do them.

98 We then provide six different tutorials, each of which is written in the R

99 programming language and publicly available on our Github and the Open Science  
100 Framework (OSF) pages, along with all of the other code and material associated with the  
101 project. The tutorials provide hands-on, concrete examples of key parts of the analytical  
102 process, so that others can apply the analyses to their own time-to-event data sets. Each  
103 tutorial is provided as an RMarkdown file, so that others can download and adapt the code  
104 to fit their own purposes. Additionally, each tutorial is made available as .html file, so that  
105 it can be viewed by any web browser, and thus available to those that do not use R.

106 In Tutorial 1a, we illustrate how to process or “wrangle” a previously published RT +  
107 accuracy data set to calculate descriptive statistics when there is one independent variable.  
108 The descriptive statistics are plotted, and we comment on their interpretation. In Tutorial  
109 1b we provide a generalisation of this approach to illustrate how one can calculate the  
110 descriptive statistics when using a more complex design, such as when there are two  
111 independent variables. In Tutorial 2a, we illustrate how one can fit Bayesian multi-level  
112 regression models to RT data using the R package brms. We discuss possible link  
113 functions, and plot the model-based effects of our predictors of interest. In Tutorial 2b we  
114 fit Bayesian multi-level regression models to *timed* accuracy data to perform a micro-level  
115 speed-accuracy tradeoff (SAT) analysis, which complements the hazard analysis. In  
116 Tutorial 3a, we illustrate how to fit the same type of hazard regression models in a  
117 frequentist framework using the R package lme4. We then briefly compare and contrast  
118 these inferential frameworks when applied to EHA. In Tutorial 3b, we illustrate how to  
119 perform the SAT analysis in a frequentist framework.

120 In summary, even though event history analyses is a widely used statistical tool and  
121 there already exist many excellent reviews (REFs) and tutorials (Allison, 2010) on its  
122 general use-cases, we are not aware of any tutorials that are aimed at psychological  
123 time-to-event data, and which provide worked examples of the key data processing and  
124 multi-level regression modelling steps. Therefore, our ultimate goal is twofold: first, we  
125 want to convince readers of the many benefits of using hazard analysis when dealing with

126 time-to-event data with a focus on psychological time-to-event data, and second, we want  
127 to provide a set of practical tutorials, which provide step-by-step instructions on how you  
128 actually perform a discrete-time hazard analysis on time-to-event data, as well as a  
129 complementary discrete-time SAT analysis on timed accuracy data.

130 **2. A brief introduction to hazard analysis**

131 For a comprehensive background context to hazard analysis, we recommend several  
132 excellent textbooks (Singer & Willett, 2003). Likewise, for general introduction to  
133 understanding regression equations, we recommend several introductory level textbooks  
134 (REFs). Our focus here is not on providing a detailed account of the underlying regression  
135 equations, since this topics has been comprehensively covered many times before. Instead,  
136 we want to provide an intuition to how EHA works in general as well as in the context of  
137 experimental psychology. As such, we only supply regression equations in the  
138 supplementary material (part D) and then refer to them in the text whenever relevant.

139 **2.1 Basic features of hazard analysis**

140 To apply event history analysis (EHA), one must be able to:

- 141 1. define an event of interest that represents a qualitative change that can be situated in  
142 time (e.g., a button press, a saccade onset, a fixation offset, etc.)
- 143 2. define time point zero (e.g., target stimulus onset, fixation onset)
- 144 3. measure the passage of time between time point zero and event occurrence in discrete  
145 or continuous time units.

146 The definition of hazard and the type of models employed depend on whether one is  
147 using continuous or discrete time units. Since our focus here is on hazard models that use

148 discrete time units, we describe that approach. After dividing time in discrete, contiguous  
149 time bins indexed by  $t$  (e.g.,  $t = 1:10$  timebins), let  $RT$  be a discrete random variable  
150 denoting the rank of the time bin in which a particular person's response occurs in a  
151 particular experimental condition. For example, the first response might occur at 546 ms  
152 and it would be in timebin 6 (any RTs from 501 ms to 600). Continuous RT data is treated  
153 here as interval-censored data.

154 Discrete-time EHA focuses on the discrete-time hazard function and the discrete-time  
155 survivor function (Figure 2). The equations that define both of these functions are reported  
156 in the supplementary material (equations 1 and 2 in part A). The discrete-time hazard  
157 probability gives you the probability that the event occurs (sometime) in bin  $t$ , given that  
158 the event does not occur in previous bins. In other words, it reflects the instantaneous  
159 likelihood that the event occurs in the current bin, given that it has not yet occurred in the  
160 past, i.e., in one of the prior bins. This conditionality in the definition of hazard is what  
161 makes the hazard function so diagnostic for studying event occurrence, as an event can  
162 physically not occur when it has already occurred before. In contrast, the discrete-time  
163 survivor function cumulates the bin-by-bin risks of event *nonoccurrence* to obtain the  
164 probability that the event occurs after bin  $t$ . In other words, the survivor function reflects  
165 the likelihood that the event occurs in the future, i.e., in one of the subsequent timebins.

166 The survivor function can help to qualify or provide context to the interpretation of  
167 the hazard function. For example, it can give a sense of how many trials contribute to each  
168 part of the hazard distribution. If a participant completes 100 trials in an experiment, and  
169 the survivor function reaches a probability of 0.03 at the end of timebin (400,500], then  
170 only 3% of trials remain beyond this point, which in this case would amount to 3 trials.  
171 Therefore, the error bars in later parts of the hazard function would be wider and less  
172 precise compared to earlier parts.

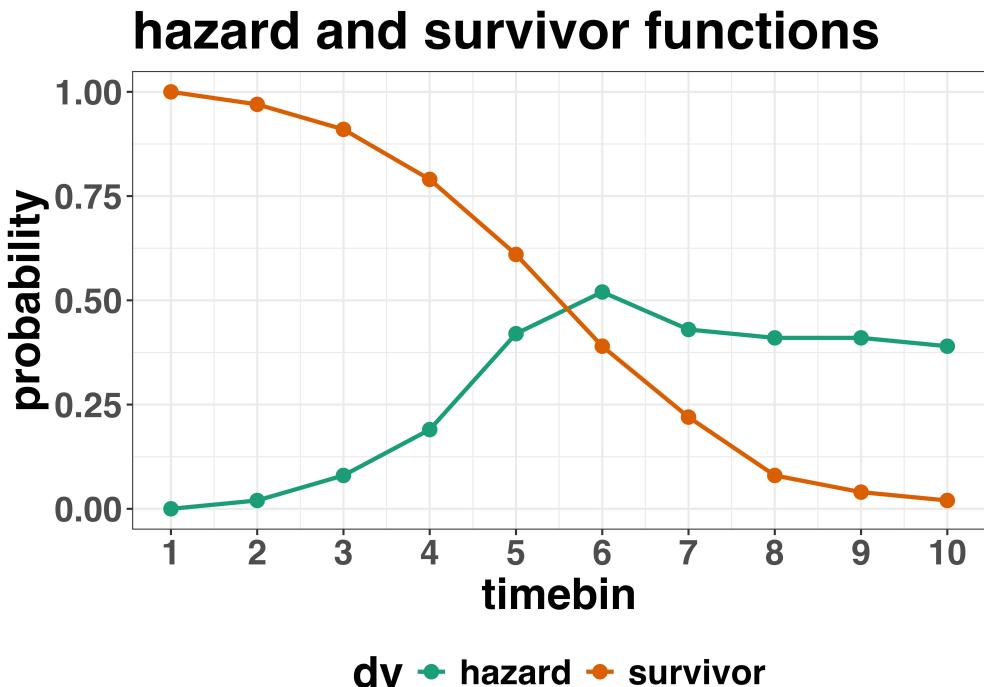


Figure 2. Hazard and survivor functions

<sup>173</sup> **2.2 Hazard analysis in the context of experimental psychology**

<sup>174</sup> **2.2.1 A worked example.** In the context of experimental psychology, it is  
<sup>175</sup> common for participants to be presented with a task that has a right and a wrong answer.  
<sup>176</sup> For example, a task may involve choosing between two response options with only one of  
<sup>177</sup> them being correct. For such two-choice RT data, the discrete-time hazard analysis can be  
<sup>178</sup> extended with a discrete-time SAT analysis. Specifically, the hazard function of event  
<sup>179</sup> occurrence can be extended with the discrete-time conditional accuracy function (see  
<sup>180</sup> equation 5 in part A of the supplementary material), which gives you the probability that a  
<sup>181</sup> response is correct given that it is emitted in time bin t (Allison, 2010; Kantowitz &  
<sup>182</sup> Pachella, 2021; Wickelgren, 1977).

<sup>183</sup> Integrating results between hazard and conditional accuracy functions can be  
<sup>184</sup> informative for understanding psychological processes. To illustrate, we consider a  
<sup>185</sup> hypothetical example that is inspired by real data (Panis et al., 2016), but simplified to

186 make the main point clearer (Figure 3). In a standard response priming paradigm, there is  
187 a prime stimulus (e.g., an arrow pointing left or right) followed by a target stimulus  
188 (another arrow pointing left or right). The prime can then be congruent or incongruent  
189 with the target. Figure 3 shows that the early upswing in hazard is equal for both prime  
190 conditions, and that early responses are always correct in the congruent condition and  
191 always incorrect in the incongruent condition. These results show that for early responses  
192 (< bin 6), responses always follow the prime (and not the target, as instructed). And then  
193 for later bins, response hazard is lower in incongruent compared to congruent trials, and all  
194 responses emitted in these later bins are correct. This is interesting because mean-average  
195 RT would only represent the overall ability of cognition to overcome interference, on  
196 average, across trials. And such a conclusion is not supported when the effects are explored  
197 over a timeline. Instead, the psychological conclusion is much more nuanced and suggests  
198 that multiple states start, stop and possibly interact over a particular temporal window.

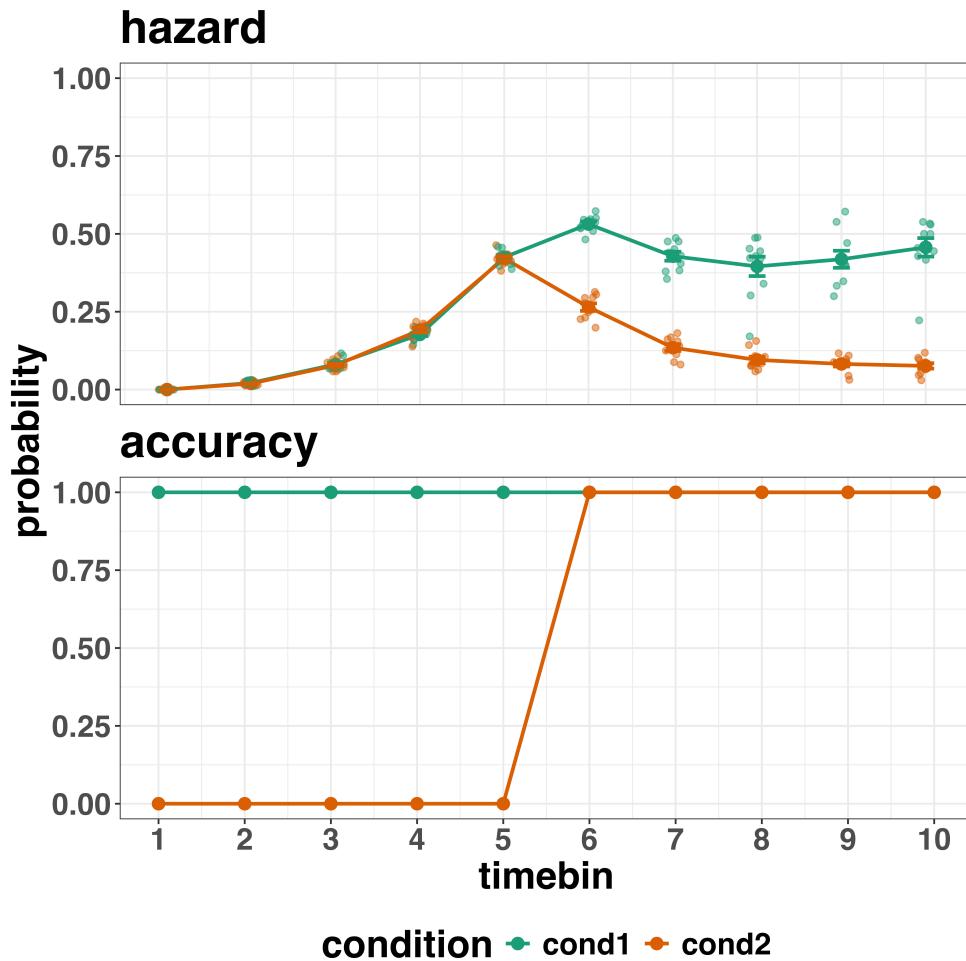


Figure 3. Hazard and conditional accuracy

199       Unlocking the temporal states of cognitive processes can be revealing in and of itself  
 200      for theory development and the understanding of basic psychological processes. Possibly  
 201      more importantly, however, is that it simultaneously opens the door to address many new  
 202      and previously unanswered questions. Do all participants show similar temporal states or  
 203      are there individual differences? Do such individual differences extend to those individuals  
 204      that have been diagnosed with some form of psychopathology? How do temporal states  
 205      relate to brain-based mechanisms that might be studied using other methods from cognitive  
 206      neuroscience? And how much of theory in cognitive psychology would be in need of  
 207      revision if mean-average comparisons were supplemented with a temporal states approach?

**2.2.2 Implications for designing experiments.**

Performing hazard analyses in experimental psychology has implications for how experiments are designed. Indeed, if trials are categorised as a function of when responses occur, then each timebin will only include a subset of the total number of trials. For example, let's consider an experiment where each participant performs 2 conditions and there are 100 trial repetitions per condition. Those 100 trials must be distributed in some manner across the chosen number of bins.

In such experimental designs, since the number of trials per condition are spread

across bins, it is important to have a relatively large number of trial repetitions per participant and per condition. Accordingly, experimental designs using this approach typically focus on factorial, within-subject designs, in which a large number of observations are made on a relatively small number of participants (so-called small- $N$  designs). This approach emphasizes the precision and reproducibility of data patterns at the individual participant level to increase the inferential validity of the design (Baker et al., 2021; Smith & Little, 2018).

In contrast to the large- $N$  design that typically average across many participants

without being able to scrutinize individual data patterns, small- $N$  designs retain crucial information about the data patterns of individual observers. This can be advantageous whenever participants differ systematically in their strategies or in the time-courses of their effects, so that averaging them would lead to misleading data patterns. Note that because statistical power derives both from the number of participants and from the number of repeated measures per participant and condition, small- $N$  designs can still achieve what are generally considered acceptable levels of statistical power, if they have have a sufficient amount of data overall (Baker et al., 2021; Smith & Little, 2018).

We used R (Version 4.4.0; R Core Team, 2024)<sup>1</sup> for all reported analyses. The

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<sup>1</sup> We, furthermore, used the R-packages *bayesplot* (Version 1.11.1; Gabry, Simpson, Vehtari, Betancourt, &

<sup>233</sup> content of the tutorials is mainly based on Allison (2010), Singer and Willett (2003),  
<sup>234</sup> McElreath (2018), Kurz (2023a), and Kurz (2023b).

<sup>235</sup> **3. An overview of the general analytical workflow**

<sup>236</sup> Although the focus is on EHA, we also want to briefly comment on broader aspects of  
<sup>237</sup> our general analytical workflow, which relate more to data science and data analysis  
<sup>238</sup> workflows.

<sup>239</sup> **3.1 Data science workflow and descriptive statistics**

<sup>240</sup> Descriptive, data science workflow. Data wrangling via tidyverse principles and a  
<sup>241</sup> functional programming approach (cite R4DS textbook here). Functional programming  
<sup>242</sup> basically means you don't write your own loops but instead use functions that have been  
<sup>243</sup> built and tested by others. [[more here, as necessary]].

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Gelman, 2019), *brms* (Version 2.21.0; Bürkner, 2017, 2018, 2021), *citr* (Version 0.3.2; Aust, 2019), *cmdstanr* (Version 0.8.1.9000; Gabry, Češnovar, Johnson, & Brander, 2024), *dplyr* (Version 1.1.4; Wickham, François, Henry, Müller, & Vaughan, 2023), *forcats* (Version 1.0.0; Wickham, 2023a), *ggplot2* (Version 3.5.1; Wickham, 2016), *lme4* (Version 1.1.35.5; Bates, Mächler, Bolker, & Walker, 2015), *lubridate* (Version 1.9.3; Grolemund & Wickham, 2011), *Matrix* (Version 1.7.0; Bates, Maechler, & Jagan, 2024), *nlme* (Version 3.1.166; Pinheiro & Bates, 2000), *papaja* (Version 0.1.2.9000; Aust & Barth, 2023), *patchwork* (Version 1.2.0; Pedersen, 2024), *purrr* (Version 1.0.2; Wickham & Henry, 2023), *RColorBrewer* (Version 1.1.3; Neuwirth, 2022), *Rcpp* (Eddelbuettel & Balamuta, 2018; Version 1.0.12; Eddelbuettel & François, 2011), *readr* (Version 2.1.5; Wickham, Hester, & Bryan, 2024), *RJ-2021-048* (Bengtsson, 2021), *standist* (Version 0.0.0.9000; Girard, 2024), *stringr* (Version 1.5.1; Wickham, 2023b), *tibble* (Version 3.2.1; Müller & Wickham, 2023), *tidybayes* (Version 3.0.6; Kay, 2023), *tidyrr* (Version 1.3.1; Wickham, Vaughan, & Girlich, 2024), *tidyverse* (Version 2.0.0; Wickham et al., 2019), and *tinylabels* (Version 0.2.4; Barth, 2023).

### 244 3.2 Inferential statistical approach

245 Our lab adopts a estimation approach to multi-level regression (Kruschke & Liddel,  
246 2018; Winter, 2019), which is heavily influenced by Bayesian approach as suggested by  
247 Richard McElreath (McElreath, 2020; Kurz, 202?). We also use a “keep it maximal”  
248 approach to specifying varying (or random) effects (Barr et al., 2013). This means that  
249 wherever possible we include varying intercepts and slopes per participant To make  
250 inferences, we use two main approaches. We compare models of different complexity, using  
251 information criteria, such as WAIC or LOO, to evaluate out-of-sample predictive accuracy.  
252 We also take the most complex model and evaluate key parameters of interest using point  
253 and interval estimates.

## 254 4. Tutorials

255 Tutorials 1a and 1b show how to calculate and plot the descriptive statistics when  
256 there are one and two independent variables, respectively. Tutorials 2a and 2b illustrate  
257 how to use Bayesian multilevel modeling to fit hazard and conditional accuracy models,  
258 respectively. Tutorials 3a and 3b show how to implement, respectively, multilevel models  
259 for hazard and conditional accuracy in the frequentist framework. Additionally, to further  
260 simplify the process for other users, the tutorials rely on a set of our own custom functions  
261 that make sub-processes easier to automate, such as data wrangling and plotting functions  
262 (see part B in the supplemental material for a list of the custom functions).

263 Our list of tutorials is as follows:

- 264 • 1a. Wrangle raw data and descriptive stats for one independent variable.
- 265 • 1b. Wrangle raw data and descriptive stats for two independent variables.
- 266 • 2a. Bayesian multilevel modeling for  $h(t)$
- 267 • 2b. Bayesian multilevel modeling for  $ca(t)$

- 268     • 3a. Frequentist multilevel modeling for  $h(t)$   
269     • 3b. Frequentist multilevel modeling for  $ca(t)$

270       Planning (T4) - if we get a simulation and power analysis script working, which we

271       are happy with then we could include it here.

272   **4.1 Tutorial 1a: Calculating descriptive statistics using a life table**

273       **4.1.1 Data wrangling aims.** Our data wrangling procedures serve two related

274       purposes. First, we want to summarise and visualise descriptive statistics that relate to our  
275       main research questions about the timecourse of psychological processes. Second, we want  
276       to produce two different data sets that can each be submitted to different types of  
277       inferential modelling approaches. The two types of data structure we label as ‘person-trial’  
278       data and ‘person-trial-bin’ data. The ‘person-trial’ data (Table 1) will be familiar to most  
279       researchers who record behavioural responses from participants, as it represents the  
280       measured RT and accuracy per trial within an experiment. This data set is used when  
281       fitting conditional accuracy models.

Table 1  
*Data structure for ‘person-trial’ data*

pid	trial	condition	rt	accuracy
1	1	congruent	373.49	1
1	2	incongruent	431.31	1
1	3	congruent	455.43	0
1	4	incongruent	622.41	1
1	5	incongruent	535.98	1
1	6	incongruent	540.08	1
1	7	congruent	511.07	1
1	8	incongruent	444.42	1
1	9	congruent	678.69	1
1	10	congruent	549.79	1

*Note.* The first 10 trials for participant 1 are shown. These data are simulated and for illustrative purposes only.

282 In contrast, the ‘person-trial-bin’ data (Table 2) has a different, more extended  
 283 structure, which indicates in which bin a response occurred, if at all, in each trial.  
 284 Therefore, the ‘person-trial-bin’ dataset generates a 0 in each bin until an event occurs and  
 285 then it generates a 1 to signal an event has occurred in that bin. This data set is used  
 286 when fitting hazard models. It is worth pointing out that there is no requirement for an  
 287 event to occur at all (in any bin), as maybe there was no response on that trial or the event  
 288 occurred after the time window of interest. Likewise, when the event occurs in bin 1 there  
 289 would only be one row of data for that trial in the person-trial-bin data set.

Table 2  
*Data structure for ‘person-trial-bin’ data*

pid	trial	condition	timebin	event
1	1	congruent	1	0
1	1	congruent	2	0
1	1	congruent	3	0
1	1	congruent	4	1
1	2	incongruent	1	0
1	2	incongruent	2	0
1	2	incongruent	3	0
1	2	incongruent	4	0
1	2	incongruent	5	1

*Note.* The first 2 trials for participant 1 from Table 1 are shown. The width of the time bins is 100 ms. These data are simulated and for illustrative purposes only.

290       **4.1.2 A real data wrangling example.** To illustrate how to quickly set up life  
 291 tables for calculating the descriptive statistics (functions of discrete time), we use a  
 292 published data set on masked response priming from Panis and Schmidt (2016). In their  
 293 first experiment, Panis and Schmidt (2016) presented a double arrow for 94 ms that  
 294 pointed left or right as the target stimulus with an onset at time point zero in each trial.  
 295 Participants had to indicate the direction in which the double arrow pointed using their  
 296 corresponding index finger, within 800 ms after target onset. Response time and accuracy  
 297 were recorded on each trial. Prime type (blank, congruent, incongruent) and mask type  
 298 were manipulated. Here we focus on the subset of trials in which no mask was presented.

299 The 13-ms prime stimulus was a double arrow with onset at -187 ms for the congruent  
 300 (same direction as target) and incongruent (opposite direction as target) prime conditions.

301 There are several data wrangling steps to be taken. First, we need to load the data  
 302 before we (a) supply required column names, and (b) specify the factor condition with the  
 303 correct levels and labels.

304 The required column names are as follows:

- 305 • “pid”, indicating unique participant IDs;
- 306 • “trial”, indicating each unique trial per participant;
- 307 • “condition”, a factor indicating the levels of the independent variable (1, 2, ...) and  
     the corresponding labels;
- 309 • “rt”, indicating the response times in ms;
- 310 • “acc”, indicating the accuracies (1/0).

311 In the code of Tutorial 1a, this is accomplished as follows.

```
data_wr <- read_csv("../Tutorial_1_descriptive_stats/data/DataExp1_6subjects_wrangled.csv")
colnames(data_wr) <- c("pid", "bl", "tr", "condition", "resp", "acc", "rt", "trial")
data_wr <- data_wr %>%
  mutate(condition = condition + 1, # original levels were 0, 1, 2.
        condition = factor(condition, levels=c(1,2,3), labels=c("blank", "congruent", "incongruent")))
```

312 Next, we can set up the life tables and plots of the discrete-time functions  $h(t)$ ,  $S(t)$ ,  
 313  $ca(t)$ , and  $P(t)$  – see part A of the supplementary material. To do so using a functional  
 314 programming approach, one has to nest the data within participants using the  
 315 `group_nest()` function, and supply a user-defined censoring time and bin width to our  
 316 custom function “`censor()`”, as follows.

```
data_nested <- data_wr %>% group_nest(pid)
data_final <- data_nested %>%
  mutate(censored = map(data, censor, 600, 40)) %>% # ! user input: censoring time, and bin width
```

```

mutate(ptb_data = map(censored, ptb)) %>%      # create person-trial-bin dataset
mutate(lifetable = map(ptb_data, setup_lt)) %>%    # create life tables without ca(t)
mutate(condacc = map(censored, calc_ca)) %>%      # calculate ca(t)
mutate(lifetable_ca = map2(lifetable, condacc, join_lt_ca)) %>%    # create life tables with ca(t)
mutate(plot = map2(.x = lifetable_ca, .y = pid, plot_eha,1)) # create plots

```

317        Note that the censoring time should be a multiple of the bin width (both in ms). The  
 318        censoring time should be a time point after which no informative responses are expected  
 319        anymore. In experiments that implement a response deadline in each trial the censoring  
 320        time can equal that deadline time point. Trials with a RT larger than the censoring time,  
 321        or trials in which no response is emitted during the data collection period, are treated as  
 322        right-censored observations in EHA. In other words, these trials are not discarded, because  
 323        they contain the information that the event did not occur before the censoring time.

324        Removing such trials before calculating the mean event time can introduce a sampling bias  
 325        (REFs). The person-trial-bin oriented dataset has one row for each time bin of each trial  
 326        that is at risk for event occurrence. The variable “event” in the person-trial-bin oriented  
 327        data set indicates whether a response occurs (1) or not (0) for each bin.

328        The next step is to plot the data using our custom function `plot_eha()`. When  
 329        creating the plots, some warning messages will likely be generated, like these:

- 330        • Removed 2 rows containing missing values or values outside the scale range  
 331        (`geom_line()`).
- 332        • Removed 2 rows containing missing values or values outside the scale range  
 333        (`geom_point()`).
- 334        • Removed 2 rows containing missing values or values outside the scale range  
 335        (`geom_segment()`).

336        The warning messages are generated because some bins have no hazard and  $ca(t)$   
 337        estimates, and no error bars. They can thus safely be ignored. One can now inspect

338 different aspects, including the life table for a particular condition of a particular subject,  
339 and a plot of the different functions for a particular participant. It is important to visually  
340 inspect the functions first for each participant, in order to identify possible cheaters (e.g., a  
341 flat conditional accuracy function at .5 indicates (s)he was only guessing), outlying  
342 individuals, and/or different groups with qualitatively different behavior.

343 Table 3 shows the life table for condition “blank” (no prime stimulus presented) for  
344 participant 6 – compare to Figure 1. A life table includes for each time bin, the risk set  
345 (number of trials that are event-free at the start of the bin), the number of observed  
346 events, and the estimates of  $h(t)$ ,  $S(t)$ , possibly  $ca(t)$ , and their estimated standard errors  
347 ( $se$ ). At time point zero, no events can occur and therefore  $h(t)$  and  $ca(t)$  are undefined.

348 Figure 4 displays the discrete-time hazard, survivor, conditional accuracy, and  
349 probability mass functions for each prime condition for participant 6. By using  
350 discrete-time  $h(t)$  functions of event occurrence – in combination with  $ca(t)$  functions for  
351 two-choice tasks – one can provide an unbiased, time-varying, and probabilistic description  
352 of the latency and accuracy of responses based on all trials of any data set.

353 For example, for participant 6, the estimated hazard values in bin (240,280] are 0.03,  
354 0.17, and 0.20 for the blank, congruent, and incongruent prime conditions, respectively. In  
355 other words, when the waiting time has increased until *240 ms* after target onset, then the  
356 conditional probability of response occurrence in the next 40 ms is more than five times  
357 larger for both prime-present conditions, compared to the blank prime condition.

358 Furthermore, the estimated conditional accuracy values in bin (240,280] are 0.29, 1,  
359 and 0 for the blank, congruent, and incongruent prime conditions, respectively. In other  
360 words, if a response is emitted in bin (240,280], then the probability that it is correct is  
361 estimated to be 0.29, 1, and 0 for the blank, congruent, and incongruent prime conditions,  
362 respectively.

Table 3

*The life table for the blank prime condition of participant 6.*

bin	risk_set	events	hazard	se_haz	survival	se_surv	ca	se_ca
0.00	220.00	NA	NA	NA	1.00	0.00	NA	NA
40.00	220.00	0.00	0.00	0.00	1.00	0.00	NA	NA
80.00	220.00	0.00	0.00	0.00	1.00	0.00	NA	NA
120.00	220.00	0.00	0.00	0.00	1.00	0.00	NA	NA
160.00	220.00	0.00	0.00	0.00	1.00	0.00	NA	NA
200.00	220.00	0.00	0.00	0.00	1.00	0.00	NA	NA
240.00	220.00	0.00	0.00	0.00	1.00	0.00	NA	NA
280.00	220.00	7.00	0.03	0.01	0.97	0.01	0.29	0.17
320.00	213.00	13.00	0.06	0.02	0.91	0.02	0.77	0.12
360.00	200.00	26.00	0.13	0.02	0.79	0.03	0.92	0.05
400.00	174.00	40.00	0.23	0.03	0.61	0.03	1.00	0.00
440.00	134.00	48.00	0.36	0.04	0.39	0.03	0.98	0.02
480.00	86.00	37.00	0.43	0.05	0.22	0.03	1.00	0.00
520.00	49.00	32.00	0.65	0.07	0.08	0.02	1.00	0.00
560.00	17.00	9.00	0.53	0.12	0.04	0.01	1.00	0.00
600.00	8.00	4.00	0.50	0.18	0.02	0.01	1.00	0.00

*Note.* The column named “bin” indicates the endpoint of each time bin (in ms), and includes time point zero. For example the first bin is (0,40] with the starting point excluded and the endpoint included. se = standard error. ca = conditional accuracy.

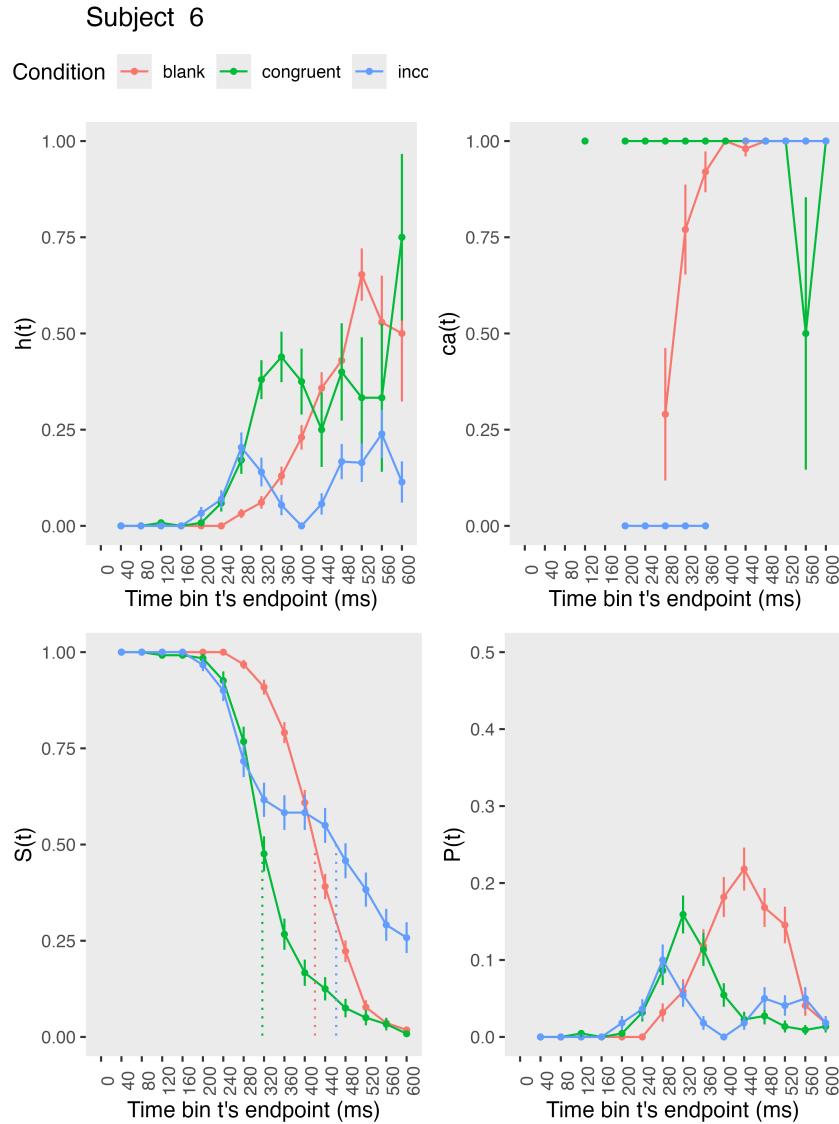


Figure 4. Estimated discrete-time hazard, survivor, and conditional accuracy functions for participant 6, as a function of the passage of discrete waiting time.

363 However, when the waiting time has increased until 400 ms after target onset, then  
 364 the conditional probability of response occurrence in the next 40 ms is estimated to be  
 365 0.36, 0.25, and 0.06 for the blank, congruent, and incongruent prime conditions,  
 366 respectively. And when a response does occur in bin (400,440], then the probability that it  
 367 is correct is estimated to be 0.98, 1, and 1 for the blank, congruent, and incongruent prime

368 conditions, respectively.

369 When participants show qualitatively the same distributional patterns, one might  
370 consider to aggregate their data and make one plot (see Tutorial\_1a.Rmd).

371 These results suggest that the participant is initially responding to the prime even  
372 though (s)he was instructed to only respond to the target, that response competition  
373 emerges in the incongruent prime condition around 300 ms, and that only later responses  
374 are fully controlled by the target stimulus. Qualitatively similar results were obtained for  
375 the other five participants.

376 These results go against the (often implicit) assumption that all observed responses  
377 are primed responses to the target stimulus. Instead, the distributional data show that  
378 early responses are triggered exclusively by the prime stimulus, while only later responses  
379 reflect primed responses to the target stimulus.

380 At this point, we have calculated, summarised and plotted descriptive statistics for  
381 the key variables in EHA. As we will show in Tutorials 2 and 3, statistical models for  $h(t)$   
382 and  $ca(t)$  can be implemented as generalized linear mixed regression models predicting  
383 event occurrence (1/0) and accuracy (1/0) in each bin of a selected time window for  
384 analysis. As such, multi-level regression is what we turn to in those tutorials. But first we  
385 consider calculating the descriptive statistics for two independent variables.

## 386 **4.2 Tutorial 1b: Generalising to a more complex design**

387 So far in this paper, we have used a simple experimental design, which involved one  
388 condition with three levels. But psychological experiments are often more complex, with  
389 crossed factorial designs with more conditions and more than three levels. The purpose of  
390 Tutorial 1b, therefore, is to provide a generalisation of the basic approach, which extends  
391 to a more complicated design. We felt that this might be useful for researchers in  
392 experimental psychology that typically use crossed factorial designs.

393 To this end, Tutorial 1b illustrates how to calculate and plot the descriptive statistics

394 for the full data set of Experiment 1 of Panis and Schmidt (2016), which includes two

395 independent variables: mask type and prime type. As we use the same functional

396 programming approach as in Tutorial 1a, we simply present the sample-based functions for

397 participant 6 in Figure 5.

398 In the no-mask condition (column 1 in Figure 5), we observe a positive compatibility

399 effect in the hazard and  $ca(t)$  functions, as congruent primes temporarily generate higher

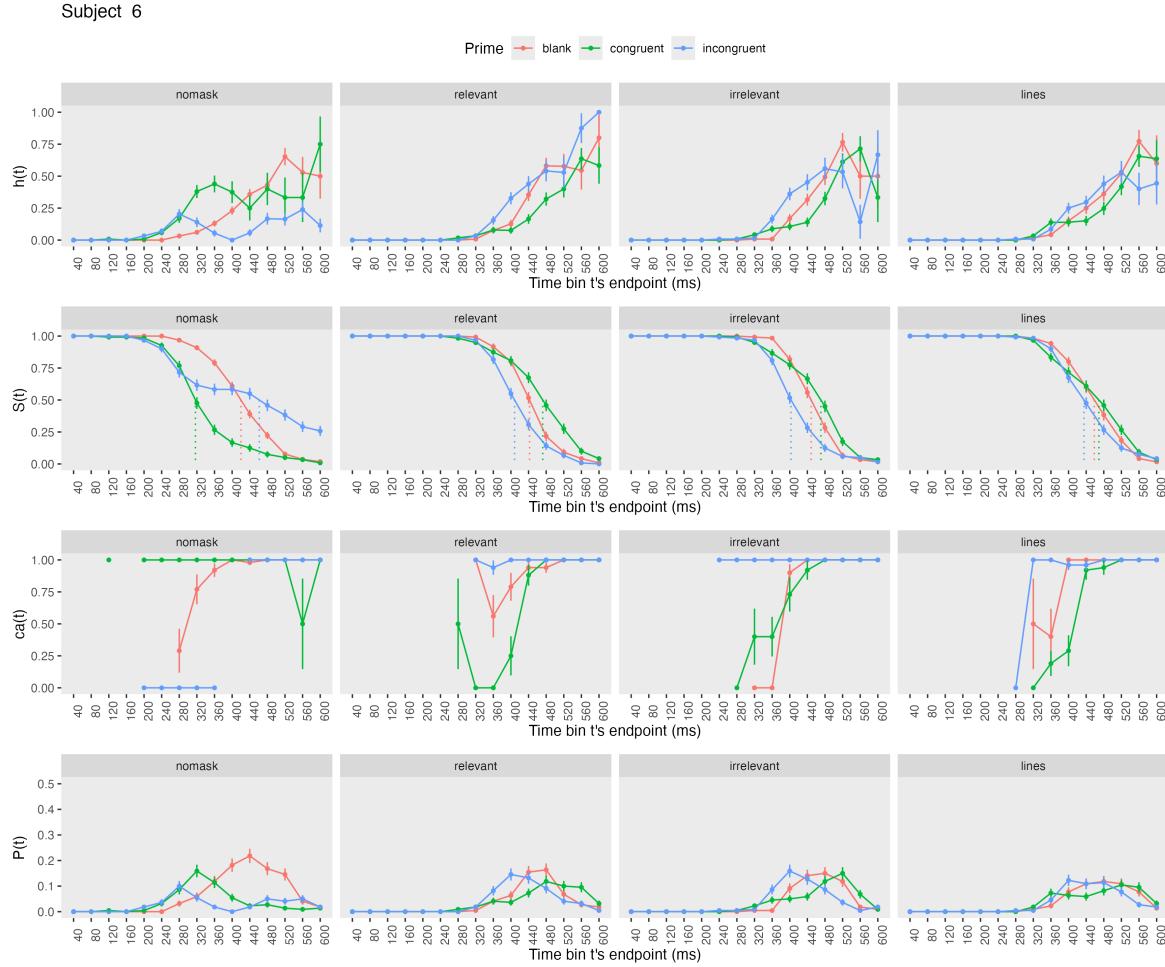
400 values for hazard and conditional accuracy compared to incongruent primes. However,

401 when a (relevant, irrelevant, or lines) mask is present (columns 2-4), there is a negative

402 compatibility effect in the hazard and conditional accuracy functions, as congruent primes

403 temporarily generate *lower* values for hazard and conditional accuracy compared to

404 incongruent primes.



*Figure 5.* Sample-based discrete-time hazard, survivor, and conditional accuracy functions for participant 6, as a function of prime type (blank, congruent, incongruent) and mask type (no mask, relevant, irrelevant, lines).

#### 405 4.3 Tutorial 2a: Fitting Bayesian hazard models to time-to-event data

406 In this third tutorial, we illustrate how to fit Bayesian hazard regression models to  
 407 the masked response priming data set used in Tutorial 1a. Fitting (Bayesian or  
 408 non-Bayesian) regression models to the data is important when you want to study how the  
 409 shape of the hazard function depends on various predictors (Singer & Willett, 2003).

**4.3.1 Hazard model considerations.** There are several analytic decisions one

410 has to make when fitting a hazard model. First, one has to select an analysis time window,  
411 i.e., a contiguous set of bins for which there is enough data for each participant. Second,  
412 given that the dependent variable is binary, one has to select a link function (see part C in  
413 the supplementary material). The cloglog link is preferred over the logit link when events  
414 can occur in principle at any time point within a bin, which is the case for RT data (Singer  
415 & Willett, 2003). Third, one has to choose a specification of the effect of discrete TIME  
416 (i.e., the time bin index  $t$ ). One can choose a general specification (one intercept per bin)  
417 or a functional specification, such as a polynomial one (compare model 1 with models 2, 3,  
418 and 4 below). We provide relevant example regression formulas in part D of the  
419 supplementary material.

420 In the case of a large- $N$  design without repeated measurements, the parameters of a

421 discrete-time hazard model can be estimated using standard logistic regression software  
422 after expanding the typical person-trial-oriented data set into a person-trial-bin-oriented  
423 data set (Allison, 2010). When there is clustering in the data, as in the case of a small- $N$   
424 design with repeated measurements, the parameters of a discrete-time hazard model can be  
425 estimated using population-averaged methods (e.g., Generalized Estimating Equations),  
426 and Bayesian or frequentist generalized linear mixed models (Allison, 2010).

427 In general, there are three assumptions one can make or relax when adding

428 experimental predictor variables and other covariates: The linearity assumption for  
429 continuous predictors (the effect of a 1 unit change is the same anywhere on the scale), the  
430 additivity assumption (predictors do not interact), and the proportionality assumption  
431 (predictors do not interact with TIME).

432 In this tutorial we will fit four Bayesian multilevel models (i.e., generalized linear

433 mixed models) that differ in complexity to the person-trial-bin oriented data set that we  
434 created in Tutorial 1a. We select the analysis range (200,600] and the cloglog link. The

436 data is prepared as follows.

```
# load person-trial-bin oriented data set
ptb_data <- read_csv("../Tutorial_1_descriptive_stats/data/inputfile_hazard_modeling.csv")

# select analysis time range: (200,600] with 10 bins (time bin ranks 6 to 15)
ptb_data <- ptb_data %>% filter(period > 5)

# create factor condition, with "blank" as the reference level
ptb_data <- ptb_data %>% mutate(condition = factor(condition, labels = c("blank", "congruent", "incongruent")))

# center TIME (period) on bin 9, and trial on trial 1000 and rescale; Add dummy variables for each bin.
ptb_data <- ptb_data %>%
  mutate(period_9 = period - 9,
         trial_c = (trial - 1000)/1000,
         d6 = if_else(period == 6, 1, 0),
         d7 = if_else(period == 7, 1, 0),
         d8 = if_else(period == 8, 1, 0),
         d9 = if_else(period == 9, 1, 0),
         d10 = if_else(period == 10, 1, 0),
         d11 = if_else(period == 11, 1, 0),
         d12 = if_else(period == 12, 1, 0),
         d13 = if_else(period == 13, 1, 0),
         d14 = if_else(period == 14, 1, 0),
         d15 = if_else(period == 15, 1, 0))
```

437 **4.3.2 Prior distributions.** To get the posterior distribution of each model

438 parameter given the data, we need to specify a prior distribution for each parameter. The  
 439 middle column of Figure 12 in part E of the supplementary material shows seven examples  
 440 of prior distributions on the logit and/or cloglog scales.

441 While a normal distribution with relatively large variance is often used as a weakly  
 442 informative prior for continuous dependent variables, rows A and B in Figure 12 show that  
 443 specifying such distributions on the logit and cloglog scales leads to rather informative  
 444 distributions on the original probability scale, as most mass is pushed to probabilities of 0  
 445 and 1. The other rows in Figure 9 show prior distributions on the logit and cloglog scale  
 446 that we use instead.

447       **4.3.3 Model 1: A general specification of TIME, and main effects of**

448       **congruency and trial number.** When you do not want to make assumptions about the  
 449       shape of the hazard function, or its shape is not smooth but irregular, you can use a  
 450       general specification of TIME, i.e., one intercept per time bin. In this first model, we use a  
 451       general specification of TIME for the selected baseline condition (blank prime), and assume  
 452       that the effects of prime-target congruency and trial number are proportional and additive,  
 453       and that the effect of trial number is linear. Before we fit model 1, we remove unnecessary  
 454       columns from the data, and specify our priors. In the code of Tutorial 2a, model M1 is  
 455       specified as follows.

```
plan(multicore)

model_M1 <-
  brm(data = M1_data,
       family = binomial(link="cloglog"),
       event | trials(1) ~ 0 + d6 + d7 + d8 + Intercept + d10 + d11 + d12 + d13 + d14 + d15 +
              condition + trial_c +
              (d6 + d7 + d8 + 1 + d10 + d11 + d12 + d13 + d14 + d15 + condition + trial_c | pid),
       prior = priors_M1,
       chains = 4, cores = 4, iter = 3000, warmup = 1000,
       control = list(adapt_delta = 0.999, step_size = 0.04, max_treedepth = 12),
       seed = 12, init = "0",
       file = "Tutorial_2_Bayesian/models/model_M1")
```

456       After selecting the binomial family and the cloglog link, the model formula is

457       specified. The fixed effects include 9 dummy variables, the explicit Intercept variable  
 458       (which represents bin 9 in this example), and the main effects of priming condition and  
 459       centered trial number. Each of these effects is allowed to vary across individuals.

460       Estimating model M1 took about 70 minutes on a MacBook Pro (Sonoma 14.6.1 OS, 18GB  
 461       Memory, M3 Pro Chip).

462       **4.3.4 Model 2: A polynomial specification of TIME, and main effects of**

463       **congruency and trial number.** When the shape of the hazard function is rather

smooth, as it is here for RT data, one can fit a more parsimonious model by using a polynomial specification of TIME. For our second example model, we thus use a third-order polynomial specification of TIME for the baseline condition (blank prime), and again assume that the effects of prime-target congruency and centered trial number are proportional and additive, and that the effect of trial number is linear. The model formula for model M2 looks as follows.

```
event | trials(1) ~ 0 + Intercept + period_9 + I(period_9^2) + I(period_9^3) +
       condition + trial_c +
       (1 + period_9 + I(period_9^2) + I(period_9^3) +
       condition + trial_c | pid),
```

Because TIME is centered on bin 9, and trial number on trial 1000, the Intercept represents the cloglog-hazard in bin 9 for the blank prime condition in trial 1000. Estimating model M2 took about 144 minutes.

**4.3.5 Model 3: A polynomial specification of TIME, and relaxing the proportionality assumption.** So far, we assumed that the effect of our predictors condition and centered trial number are the same in each time bin. However, the descriptive plots suggest that the effect of condition can vary across time bins. Previous research has indeed shown that psychological effects typically change over time (Panis, 2020; Panis, Moran, et al., 2020; Panis & Schmidt, 2022; Panis et al., 2017; Panis & Wagemans, 2009). For the third model, we thus use a third-order polynomial specification of TIME for the baseline condition (blank prime), and relax the proportionality assumption for the predictor variables prime-target congruency (variable “condition”) and centered trial number (variable “trial\_c”).

```
event | trials(1) ~ 0 + Intercept +
       condition*period_9 +
       condition*I(period_9^2) +
       condition*I(period_9^3) +
       trial_c*period_9 +
       trial_c*I(period_9^2) +
```

```

trial_c*I(period_9^3) +
(1 + condition*period_9 +
condition*I(period_9^2) +
condition*I(period_9^3) +
trial_c*period_9 +
trial_c*I(period_9^2) +
trial_c*I(period_9^3) | pid),

```

483 Note that duplicate terms in the model formula are ignored. Estimating model M3

484 took about 268 minutes.

#### 485 4.3.6 Model 4: A polynomial specification of TIME, and relaxing all three

486 assumptions. Based on previous work (Panis, 2020; Panis, Moran, et al., 2020; Panis &

487 Schmidt, 2022; Panis et al., 2017; Panis & Wagemans, 2009), we relax all three

488 assumptions in model 4. We thus add a squared term for the continuous predictor centered

489 trial number – I(trial\_c^2) – and include interaction terms.

```

event | trials(1) ~ 0 + Intercept +
      condition*period_9*trial_c +
      condition*period_9*I(trial_c^2) +
      condition*I(period_9^2)*trial_c +
      condition*I(period_9^2)*I(trial_c^2) +
      condition*I(period_9^3) +
      trial_c*I(period_9^3) +
      (1 + condition*period_9*trial_c +
      condition*period_9*I(trial_c^2) +
      condition*I(period_9^2)*trial_c +
      condition*I(period_9^2)*I(trial_c^2) +
      condition*I(period_9^3) +
      trial_c*I(period_9^3) | pid)

```

490 Again, duplicate terms in the model formula are ignored. Estimating model M4 took

491 about 8 hours.

#### 492 4.3.7 Compare the models. We can compare the four models using the Widely

493 Applicable Information Criterion (WAIC) and Leave-One-Out (LOO) cross-validation, and

494 look at model weights for both criteria (Kurz, 2023a; McElreath, 2018).

```
model_weights(model_M1, model_M2, model_M3, model_M4, weights = "loo") %>% round(digits = 3)
```

```
495 ## model_M1 model_M2 model_M3 model_M4
496 ##      0      0      0      1
```

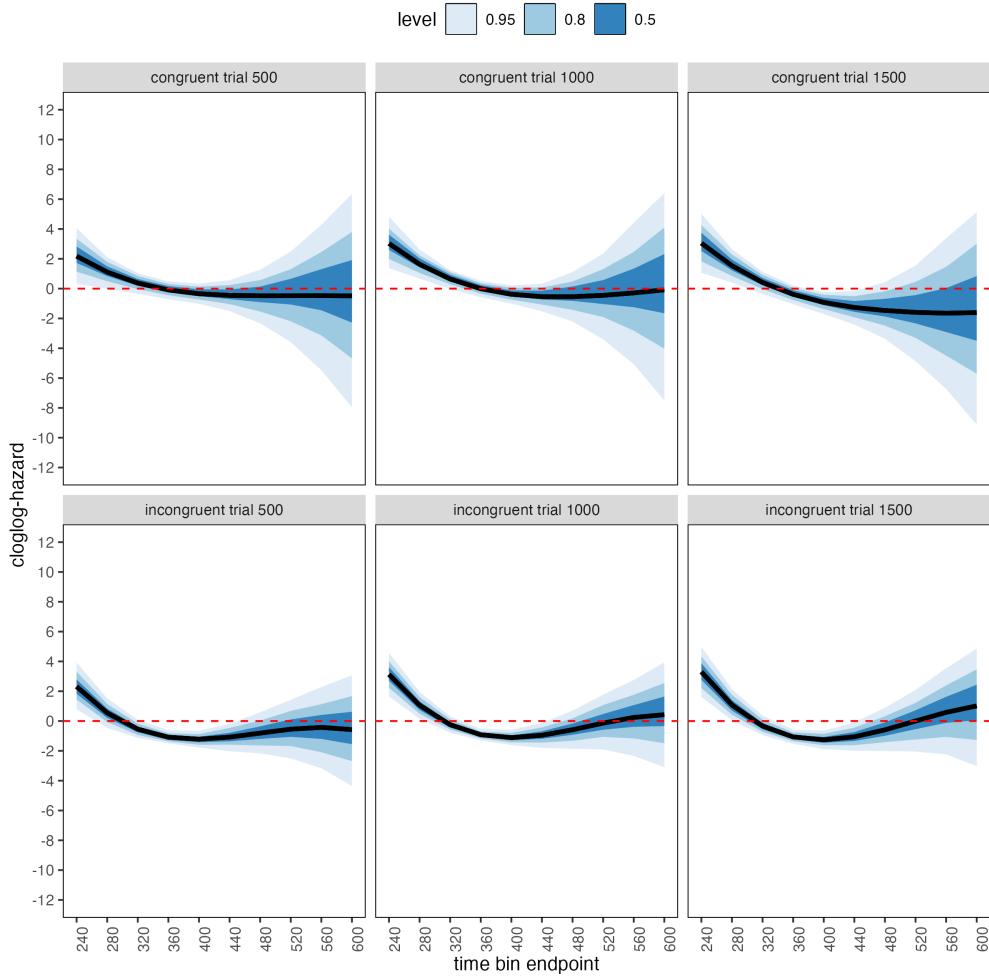
```
model_weights(model_M1, model_M2, model_M3, model_M4, weights = "waic") %>% round(digits = 3)
```

```
497 ## model_M1 model_M2 model_M3 model_M4
498 ##      0      0      0      1
```

499        Clearly, both the loo and waic weighting schemes assign a weight of 1 to model M4,  
 500 and a weight of 0 to the other three simpler models.

501        **4.3.8 Evaluate parameter estimates.** To make inferences from the parameter  
 502 estimates in model M4, we summarize the draws from the posterior distributions of the  
 503 effect of congruent and incongruent primes relative to the blank prime condition, in each  
 504 time bin for trial numbers 500, 1000, and 1500, in terms of point and interval estimates.

505        Figure 6 shows one point (mean) and three highest posterior density interval  
 506 (50/80/95%) estimates for the effects of congruent and incongruent primes relative to  
 507 neutral primes, for each time bin in trial numbers 500, 1000, and 1500.



*Figure 6.* Means and 50/80/95% highest posterior density intervals of the draws from the posterior distributions representing the effect of congruent and incongruent primes relative to neutral primes in each bin for trial numbers 500, 1000, and 1500.

508 Table 4 shows the summaries of the draws from the posterior distributions of the  
 509 effects of congruent and incongruent primes relative to the blank prime condition in trials  
 510 500, 1000, and 1500, in terms of a point estimate (the mean) and the upper and lower  
 511 bounds of the 95% highest posterior density interval. Also, by exponentiating the mean we  
 512 obtain an effect size in terms of a hazard ratio.

Table 4

*Point and 95% highest posterior density interval estimates,  
and hazard ratios.*

bin_endpoint	condition	mean	.lower	.upper	.width	hazard ratio
240.00	c500	2.18	0.33	4.05	0.95	8.82
280.00	c500	1.11	-0.02	2.11	0.95	3.03
320.00	c500	0.37	-0.34	1.04	0.95	1.45
360.00	c500	-0.09	-0.70	0.48	0.95	0.91
400.00	c500	-0.35	-1.02	0.34	0.95	0.71
440.00	c500	-0.45	-1.50	0.56	0.95	0.64
480.00	c500	-0.48	-2.32	1.27	0.95	0.62
520.00	c500	-0.48	-3.57	2.52	0.95	0.62
560.00	c500	-0.52	-5.69	4.27	0.95	0.60
600.00	c500	-0.66	-8.56	6.99	0.95	0.52
240.00	c1000	3.03	1.37	4.82	0.95	20.63
280.00	c1000	1.63	0.68	2.63	0.95	5.13
320.00	c1000	0.64	-0.02	1.24	0.95	1.90
360.00	c1000	-0.01	-0.57	0.52	0.95	0.99
400.00	c1000	-0.38	-1.01	0.22	0.95	0.68
440.00	c1000	-0.54	-1.52	0.32	0.95	0.58
480.00	c1000	-0.54	-2.20	1.11	0.95	0.58
520.00	c1000	-0.45	-3.40	2.35	0.95	0.64
560.00	c1000	-0.34	-5.78	3.90	0.95	0.71
600.00	c1000	-0.25	-8.34	6.73	0.95	0.78
240.00	c1500	3.05	1.07	5.02	0.95	21.02
280.00	c1500	1.54	0.40	2.65	0.95	4.66
320.00	c1500	0.42	-0.36	1.13	0.95	1.52
360.00	c1500	-0.38	-1.05	0.21	0.95	0.68
400.00	c1500	-0.92	-1.70	-0.24	0.95	0.40
440.00	c1500	-1.26	-2.41	-0.18	0.95	0.28
480.00	c1500	-1.47	-3.36	0.43	0.95	0.23
520.00	c1500	-1.60	-4.86	1.58	0.95	0.20
560.00	c1500	-1.71	-7.01	3.37	0.95	0.18
600.00	c1500	-1.88	-10.07	5.98	0.95	0.15
240.00	i500	2.31	0.79	3.93	0.95	10.10
280.00	i500	0.55	-0.46	1.52	0.95	1.72
320.00	i500	-0.54	-1.13	0.08	0.95	0.58
360.00	i500	-1.08	-1.50	-0.61	0.95	0.34
400.00	i500	-1.22	-1.78	-0.65	0.95	0.30

Table 4 continued

bin_endpoint	condition	mean	.lower	.upper	.width	hazard ratio
440.00	i500	-1.08	-2.03	-0.19	0.95	0.34
480.00	i500	-0.81	-2.16	0.59	0.95	0.44
520.00	i500	-0.55	-2.50	1.42	0.95	0.58
560.00	i500	-0.42	-3.16	2.28	0.95	0.65
600.00	i500	-0.58	-4.35	3.10	0.95	0.56
240.00	i1000	3.12	1.66	4.58	0.95	22.68
280.00	i1000	1.06	0.15	1.95	0.95	2.88
320.00	i1000	-0.24	-0.78	0.31	0.95	0.78
360.00	i1000	-0.92	-1.30	-0.52	0.95	0.40
400.00	i1000	-1.11	-1.61	-0.59	0.95	0.33
440.00	i1000	-0.95	-1.80	-0.12	0.95	0.39
480.00	i1000	-0.58	-1.86	0.70	0.95	0.56
520.00	i1000	-0.14	-1.90	1.77	0.95	0.87
560.00	i1000	0.24	-2.33	2.75	0.95	1.27
600.00	i1000	0.42	-3.17	3.85	0.95	1.52
240.00	i1500	3.30	1.63	4.98	0.95	27.07
280.00	i1500	1.08	0.05	2.14	0.95	2.94
320.00	i1500	-0.33	-0.94	0.36	0.95	0.72
360.00	i1500	-1.06	-1.52	-0.57	0.95	0.35
400.00	i1500	-1.26	-1.88	-0.65	0.95	0.28
440.00	i1500	-1.06	-1.99	-0.09	0.95	0.35
480.00	i1500	-0.59	-2.01	0.88	0.95	0.55
520.00	i1500	0.00	-2.05	2.09	0.95	1.00
560.00	i1500	0.58	-2.23	3.54	0.95	1.79
600.00	i1500	1.01	-3.02	4.86	0.95	2.75

Note. c500 = effect of congruent primes in trial 500; c1000 = effect of congruent primes in trial 1000; c1500 = effect of congruent primes in trial 1500; i500 = effect of incongruent primes in trial 500; i1000 = effect of incongruent primes in trial 1000; i1500 = effect of incongruent primes in trial 1500.

514 Interpretation...

515 To conclude this Tutorial 2a, Figure 7 shows the model-based hazard functions for  
 516 each prime type for participant 6, in trial 500, 1000, and 1500.

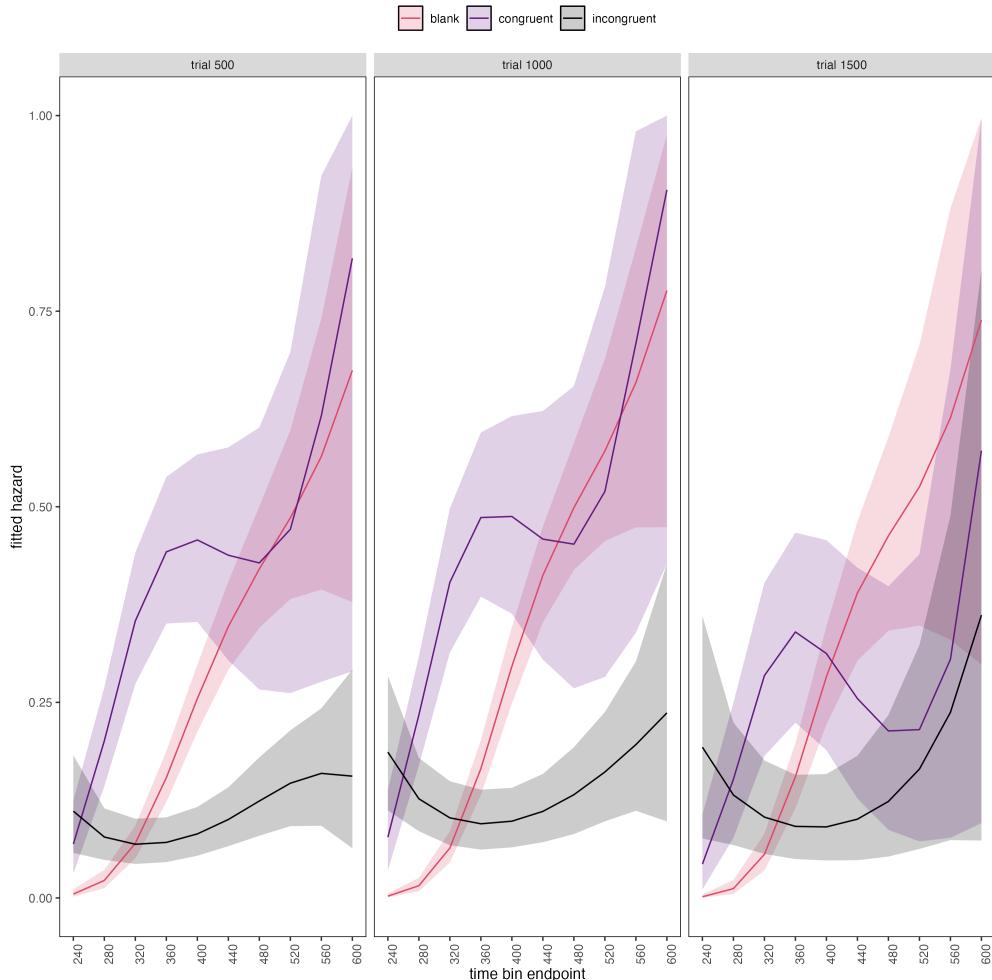


Figure 7. Model-based hazard functions for participant 6 in trial 500, 1000, and 1500.

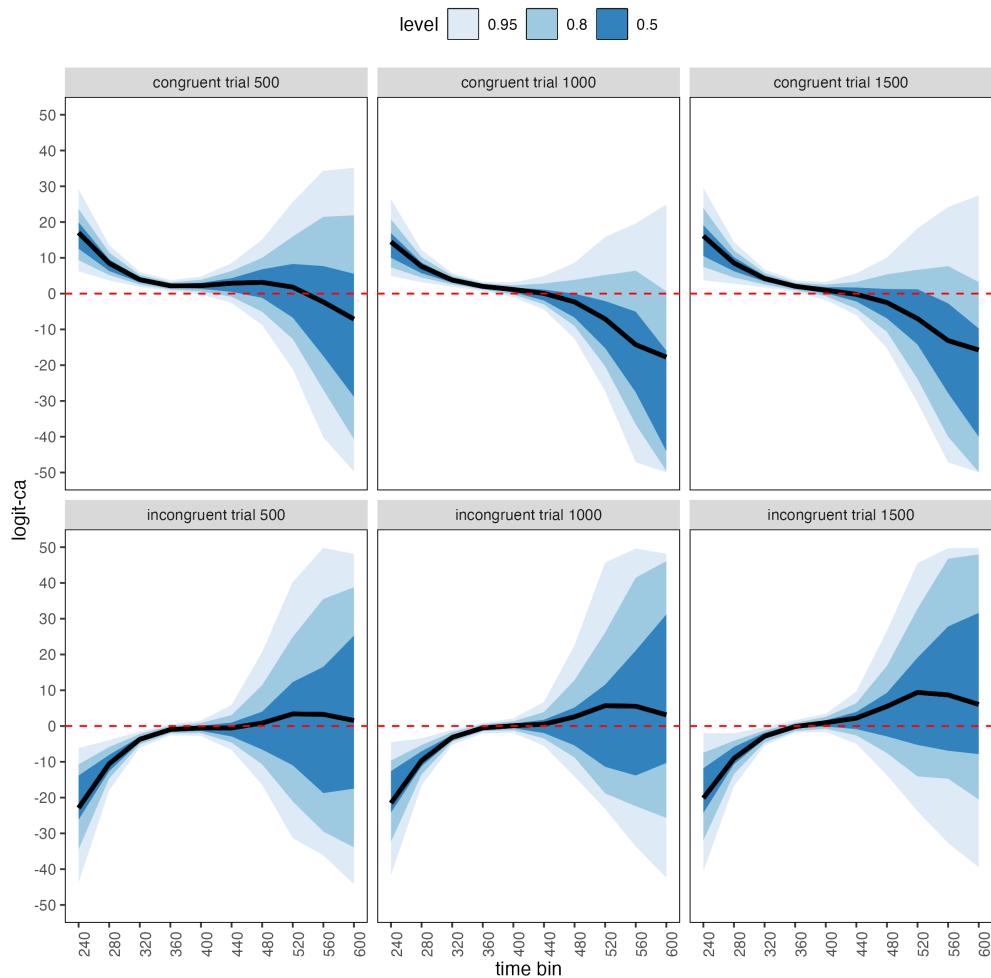
#### 517 4.4 Tutorial 2b: Fitting Bayesian conditional accuracy models

518 In this fourth tutorial, we illustrate how to fit a Bayesian regression model to the  
 519 timed accuracy data from the masked response priming data set used in Tutorial 1a. For  
 520 illustration purposes, we only fitted the effects of model M4 (see Tutorial 2a).

521 To make inferences from the parameter estimates in model M4\_ca, we summarize the

522 draws from the posterior distributions of the effect of congruent and incongruent primes  
 523 relative to the blank prime condition, in each time bin for trial numbers 500, 1000, and  
 524 1500, in terms of point and interval estimates.

525 Figure 8 shows one point (mean) and three highest posterior density interval  
 526 (50/80/95%) estimates for the effects of congruent and incongruent primes relative to  
 527 neutral primes on logit-ca, for each time bin in trial numbers 500, 1000, and 1500.



*Figure 8.* Means and 50/80/95% highest posterior density intervals of the draws from the posterior distributions representing the effect of congruent and incongruent primes relative to neutral primes in each bin for trial numbers 500, 1000, and 1500.

528 Table 5 shows the summaries of the draws from the posterior distributions of the

529 effects of congruent and incongruent primes relative to the blank prime condition in trials  
 530 500, 1000, and 1500, in terms of a point estimate (the mean) and the upper and lower  
 531 bounds of the 95% highest posterior density interval. Also, by exponentiating the mean we  
 532 obtain an effect size in terms of an odds ratio.

Table 5

*Point and 95% highest posterior density interval estimates, and odds ratios.*

bin_endpoint	condition	mean	.lower	.upper	.width	odds ratio
240.00	c500	17.02	6.26	29.22	0.95	24,618,458.61
280.00	c500	8.49	3.71	13.54	0.95	4,846.09
320.00	c500	3.91	1.88	6.05	0.95	49.79
360.00	c500	2.19	0.69	3.75	0.95	8.89
400.00	c500	2.22	-0.25	4.75	0.95	9.19
440.00	c500	2.91	-2.56	8.54	0.95	18.31
480.00	c500	3.15	-8.77	15.10	0.95	23.41
520.00	c500	1.86	-20.13	26.73	0.95	6.40
560.00	c500	-2.08	-39.94	42.41	0.95	0.12
600.00	c500	-9.77	-73.17	61.54	0.95	0.00
240.00	c1000	14.46	4.94	26.35	0.95	1,899,836.02
280.00	c1000	7.58	3.21	12.18	0.95	1,961.83
320.00	c1000	3.80	1.90	5.71	0.95	44.87
360.00	c1000	2.02	0.72	3.35	0.95	7.57
400.00	c1000	1.14	-0.99	3.11	0.95	3.14
440.00	c1000	0.06	-4.41	4.87	0.95	1.06
480.00	c1000	-2.32	-12.62	8.61	0.95	0.10
520.00	c1000	-7.10	-27.24	15.97	0.95	0.00
560.00	c1000	-15.39	-54.71	23.54	0.95	0.00
600.00	c1000	-28.27	-92.96	35.54	0.95	0.00
240.00	c1500	16.12	3.74	29.48	0.95	10,001,085.39
280.00	c1500	8.54	2.78	14.43	0.95	5,124.44
320.00	c1500	4.22	1.75	6.70	0.95	68.12
360.00	c1500	2.06	0.48	3.71	0.95	7.82
400.00	c1500	0.95	-1.75	3.26	0.95	2.58
440.00	c1500	-0.20	-6.03	5.65	0.95	0.82
480.00	c1500	-2.49	-15.23	10.07	0.95	0.08
520.00	c1500	-7.03	-30.41	18.55	0.95	0.00

Table 5 continued

bin_endpoint	condition	mean	.lower	.upper	.width	odds ratio
560.00	c1500	-14.91	-58.81	27.21	0.95	0.00
600.00	c1500	-27.22	-95.59	43.50	0.95	0.00
240.00	i500	-23.34	-44.42	-4.87	0.95	0.00
280.00	i500	-10.55	-17.93	-3.94	0.95	0.00
320.00	i500	-3.71	-6.06	-1.51	0.95	0.02
360.00	i500	-0.97	-2.57	0.56	0.95	0.38
400.00	i500	-0.52	-2.75	1.55	0.95	0.59
440.00	i500	-0.53	-6.67	5.86	0.95	0.59
480.00	i500	0.83	-16.41	20.71	0.95	2.30
520.00	i500	5.40	-32.44	52.48	0.95	222.04
560.00	i500	15.00	-58.75	104.35	0.95	3,282,435.93
600.00	i500	31.47	-90.20	190.08	0.95	46,319,712,352,328.76
240.00	i1000	-21.85	-43.05	-4.10	0.95	0.00
280.00	i1000	-9.67	-16.56	-3.46	0.95	0.00
320.00	i1000	-3.17	-5.23	-0.99	0.95	0.04
360.00	i1000	-0.53	-2.03	0.89	0.95	0.59
400.00	i1000	0.09	-1.88	2.11	0.95	1.10
440.00	i1000	0.52	-5.54	6.73	0.95	1.68
480.00	i1000	2.58	-14.16	22.53	0.95	13.20
520.00	i1000	8.10	-28.51	55.88	0.95	3,307.44
560.00	i1000	18.92	-51.75	111.96	0.95	164,758,701.84
600.00	i1000	36.86	-89.39	191.12	0.95	10,165,856,639,901,592.00
240.00	i1500	-20.51	-42.95	-2.49	0.95	0.00
280.00	i1500	-9.04	-16.80	-2.03	0.95	0.00
320.00	i1500	-2.86	-5.47	-0.25	0.95	0.06
360.00	i1500	-0.14	-1.81	1.67	0.95	0.87
400.00	i1500	0.94	-1.61	3.40	0.95	2.57
440.00	i1500	2.22	-4.97	9.63	0.95	9.21
480.00	i1500	5.52	-13.87	26.57	0.95	249.51
520.00	i1500	12.67	-31.46	58.41	0.95	318,500.40
560.00	i1500	25.50	-53.08	115.21	0.95	119,299,568,240.94
600.00	i1500	45.85	-86.60	200.06	0.95	81,670,189,671,651,033,088.00

Table 5 continued

bin_endpoint	condition	mean	.lower	.upper	.width	odds ratio
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*Note.* c500 = effect of congruent primes in trial 500; c1000 = effect of congruent primes in trial 1000; c1500 = effect of congruent primes in trial 1500; i500 = effect of incongruent primes in trial 500; i1000 = effect of incongruent primes in trial 1000; i1500 = effect of incongruent primes in trial 1500.

533

534 Interpretation...

535 plot 9 ca(t)

536 **4.5 Tutorial 3a: Fitting Frequentist hazard models**

537 In this fifth tutorial we illustrate how to fit a multilevel hazard regression model in  
 538 the frequentist framework, for the data set used in Tutorial 1a. The general process is  
 539 similar to that in Tutorial 2a, except that there are no priors to set. For illustration  
 540 purposes, we only fitted the effects from model M3 (see Tutorial 2a) using the function  
 541 `glmer()` from the R package `lme4`. Alternatively, one could also use the function  
 542 `glmmPQL()` from the R package `MASS`. The resulting hazard model is called `M3_f`.

543 In Figure 10 we compare the parameter estimates of model M3 from `brm()` with those  
 544 of model `M3_f` from `glmer()`.

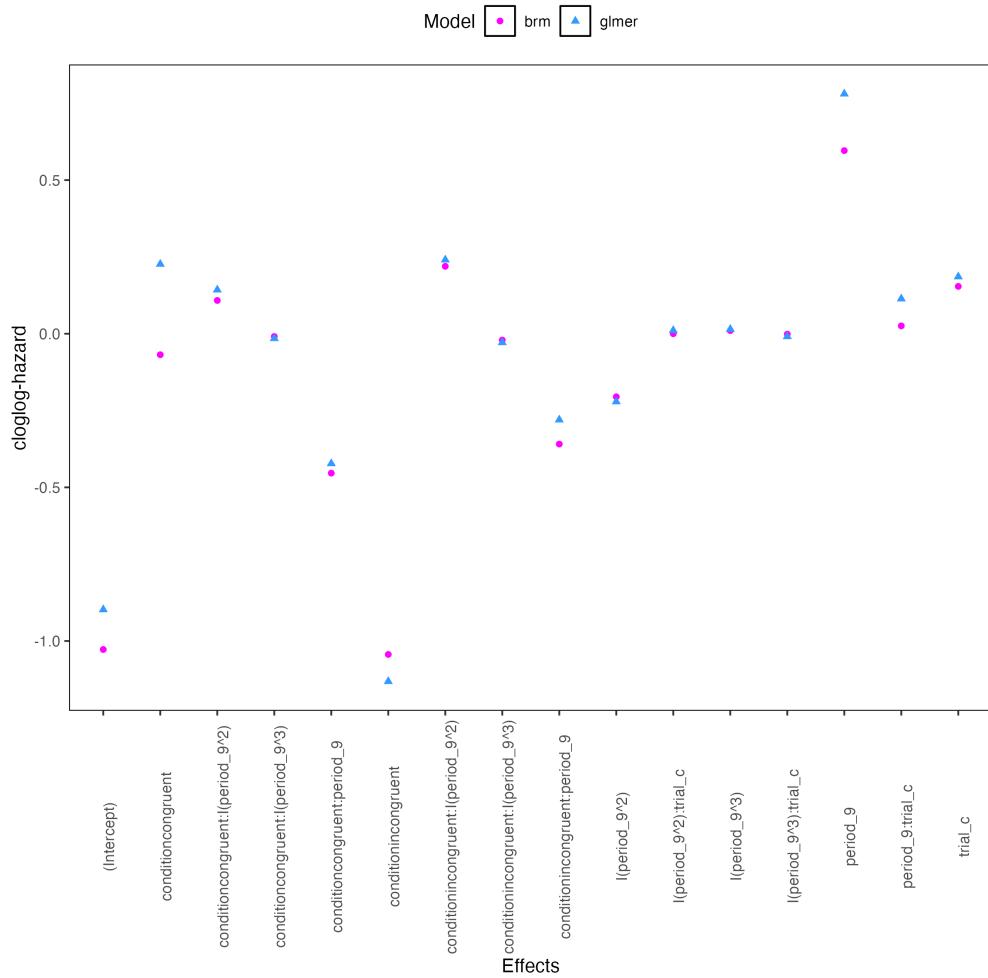


Figure 9. Parameter estimates for model M3 from `brm()` and model M3\_f from `glmer()`.

545       Figure 10 confirms that the parameter estimates from both Bayesian and frequentist

546       models are pretty similar. However, the random effects structure of model M3\_f was

547       already too complex for the frequentist model as it did not converge and resulted in a

548       singular fit. This is of course one of the reasons why Bayesian modeling has become so

549       popular in recent years. But the price you pay for being able to fit more complex models in

550       a Bayesian framework is computation time. In other words, as we have noted throughout,

551       some of the Bayesian models in Tutorials 2a and 2b took several hours to build.

552 **4.6 Tutorial 3b: Fitting Frequentist conditional accuracy models**

553 In this sixth tutorial we illustrate how to fit a multilevel regression model to the  
554 timed accuracy data in the frequentist framework, for the data set used in Tutorial 1a. For  
555 illustration purposes, we only fitted the effects from model M3 (see Tutorial 2a) using the  
556 function glmer() from the R package lme4. Alternatively, one could also use the function  
557 glmmPQL() from the R package MASS. Again, the resulting conditional accuracy model  
558 M3\_ca\_f did not converge and resulted in a singular fit.

559 **5. Discussion**

560 This main motivation for writing this paper is the observation that event history  
561 analysis remains under-used in psychological research, which means the field of research is  
562 not taking full advantage of the many benefits EHA provides compared to more  
563 conventional analyses. By providing a freely available set of tutorials, which provide  
564 step-by-step guidelines and ready-to-use R code, we hope that researchers will feel more  
565 comfortable using EHA in the future. Indeed, we hope that our tutorials may help to  
566 overcome a barrier to entry with EHA, which is the increase in analytical complexity  
567 compared to mean-average comparisons. While we have focused here on within-subject,  
568 factorial, small- $N$  designs, it is important to realize that event history analysis can be  
569 applied to other designs as well (large- $N$  designs with only one measurement per subject,  
570 between-subject designs, etc.). As such, the general workflow and associated code can be  
571 modified and applied more broadly to other contexts and research questions. In the  
572 following, we discuss issues relating to model complexity versus interpretability, individual  
573 differences, limitations of the approach, and future extensions.

574 **5.1 Advantages of hazard analysis**

575 Statisticians and mathematical psychologists recommend focusing on the hazard  
576 function when analyzing time-to-event data for various reasons. First, as discussed by  
577 Holden, Van Orden, and Turvey (2009), “probability density functions can appear nearly  
578 identical, both statistically and to the naked eye, and yet are clearly different on the basis  
579 of their hazard functions (but not vice versa). Hazard functions are thus more diagnostic  
580 than density functions” (p. 331) when one is interested in studying the detailed shape of a  
581 RT distribution (see also Figure 1 in Panis, Schmidt, et al., 2020). Therefore, when the  
582 goal is to study how psychological effects change over time, hazard functions are preferred.

583 Second, because RT distributions may differ from one another in multiple ways,  
584 Townsend (1990) developed a dominance hierarchy of statistical differences between two  
585 arbitrary distributions A and B. For example, if  $h_A(t) > h_B(t)$  for all t, then both hazard  
586 functions are said to show a complete ordering. Townsend (1990) concluded that stronger  
587 conclusions can be drawn from data when comparing the hazard functions using EHA. For  
588 example, when mean A < mean B, the hazard functions might show a complete ordering  
589 (i.e., for all t), a partial ordering (e.g., only for  $t > 300$  ms, or only for  $t < 500$  ms), or they  
590 may cross each other one or more times.

591 Third, EHA does not discard right-censored observations when estimating hazard  
592 functions, that is, trials for which we do not observe a response during the data collection  
593 period in a trial so that we only know that the RT must be larger than some value (i.e., the  
594 response deadline). This is important because although a few right-censored observations  
595 are inevitable in most RT tasks, a lot of right-censored observations are expected in  
596 experiments on masking, the attentional blink, and so forth. In other words, by using EHA  
597 you can analyze RT data from experiments that typically do not measure response times.  
598 As a result, EHA can also deal with long RTs in experiments without a response deadline,  
599 which are typically treated as outliers and are discarded before calculating a mean. This

600 orthodox procedure can lead to a sampling bias, however, which results in underestimation  
601 of the mean. By introducing a fixed censoring time for all trials at the end of the analysis  
602 time window, trials with long RTs are not discarded but contribute to the risk set of each  
603 bin.

604 Fourth, hazard modeling allows incorporating time-varying explanatory covariates  
605 such as heart rate, electroencephalogram (EEG) signal amplitude, gaze location, etc.  
606 (Allison, 2010). This is useful for linking physiological effects with behavioral effects when  
607 performing cognitive psychophysiology (Meyer, Osman, Irwin, & Yantis, 1988).

608 Finally, as explained by Kelso, Dumas, and Tognoli (2013), it is crucial to first have a  
609 precise description of the macroscopic behavior of a system (here:  $h(t)$  and  $ca(t)$  functions)  
610 in order to know what to derive on the microscopic level. EHA can thus solve the problem  
611 of model mimicry, i.e., the fact that different computational models can often predict the  
612 same mean RTs as observed in the empirical data, but not necessarily the detailed shapes  
613 of the empirical RT hazard distributions. Also, fitting parametric functions or  
614 computational models to data without studying the shape of the empirical discrete-time  
615  $h(t)$  and  $ca(t)$  functions can miss important features in the data (Panis, Moran, et al.,  
616 2020; Panis & Schmidt, 2016).

## 617 **5.2 Model complexity versus interpretability**

618 Models for discrete-time  $h(t)$  and  $ca(t)$  can quickly become very complex when  
619 adding more than 1 time scale, due to the many possible higher-order interactions. For  
620 example, model M4 contains two time scales as covariates: the passage of time on the  
621 across-bin or within-trial time scale (variable period\_9), and the passage of time on the  
622 across-trial or within-experiment time scale (variable trial\_c). However, when trials are  
623 presented in blocks, and blocks of trials within sessions, and when the experiment  
624 comprises three sessions, then four time scales can be defined (across-bin or within-trial,

625 across-trial or within-block, across-block or within-session, and across-session or  
626 within-experiment). From a theoretical perspective, adding more than 1 time scale is  
627 important to capture plasticity (e.g., proactive control) and other learning effects that play  
628 out on such longer time scales (across-trials, across-blocks, across-sessions), and that are  
629 probably present in each experiment in general. From a practical perspective, therefore, it  
630 might be interesting for interpretational purposes to limit the number of experimental  
631 variables, because adding time scales quickly increases model complexity.

### 632 5.3 Individual differences

633 One important issue is that of possible individual differences in the overall location of  
634 the distribution, and the time course of psychological effects. For example, when you wait  
635 for a response of the participant on each trial, you allow the participant to have control  
636 over the trial duration, and some participants might respond only when they are confident  
637 that their emitted response will be correct. These issues can be avoided by introducing a  
638 (relatively short) response deadline in each trial, e.g., 600 ms for simple detection tasks,  
639 1000 ms for more difficult discrimination tasks, or 2 s for tasks requiring extended  
640 high-level processing. Because EHA can deal in a straightforward fashion with  
641 right-censored observations (i.e., trials without an observed response), introducing a  
642 response deadline is recommended when designing RT experiments. Furthermore,  
643 introducing a response deadline and asking participants to respond before the deadline as  
644 much as possible, will also lead to individual distributions that overlap in time, which is  
645 important when selecting a common analysis time window when fitting hazard models.

646 But even when using a response deadline, participants can differ qualitatively in the  
647 effects they display (see Panis, 2020). One way to deal with this is to describe and  
648 interpret the different patterns. Another way is to run a clustering algorithm on the  
649 individual hazard estimates across all conditions. The obtained dendrogram can then be  
650 used to identify a (hopefully big) cluster of participants that behave similarly, and to

651 identify a (hopefully small) cluster of participants with outlying behavioral patterns. One  
652 might then exclude the outlying participants before fitting a hazard model.

653 Another approach: fit models to individual subjects and describe prevalence... REF

654 **5.4 Limitation(s)**

655 Compared to the orthodox method – comparing mean-averages between conditions –  
656 the most important limitation of multilevel hazard modeling is that it might take a long  
657 time to estimate the parameters using Bayesian methods or the model might have to be  
658 simplified significantly to use frequentist methods.

659 Another issue is that you need a relatively large number of trials per condition to  
660 estimate the hazard function with high temporal resolution. Indeed, in general, there is a  
661 trade-off between the number of trials per condition and the temporal resolution (i.e., bin  
662 width) of the hazard function. Therefore, we recommend researchers to collect as many  
663 trials as possible per experimental condition, given the available resources and considering  
664 the participant experience (e.g., fatigue and boredom). For instance, if the maximum  
665 session length deemed reasonable is between 1 and 2 hours, what is the maximum number  
666 of trials per condition that you could reasonably collect? After consideration, it might be  
667 worth conducting multiple testing sessions per participant and/or reducing the number of  
668 experimental conditions. Finally, there is a user-friendly online tool for calculating  
669 statistical power as a function of the number of trials as well as the number of participants,  
670 and this might be worth consulting to guide the research design process (Baker et al., 2021).

671 We did not discuss continuous-time hazard analysis. As indicated by Allison (2010),  
672 learning discrete-time methods first will help in learning continuous-time methods. Given  
673 that RT is typically treated as a continuous variable, it is possible that continuous-time  
674 methods will ultimately prevail. However, they require much more data to estimate the  
675 continuous-time hazard (rate) function well. Thus, by trading a bit of temporal resolution

676 for a lower number of trials, discrete-time methods seem ideal for dealing with typical  
677 psychological time-to-event data sets for which there are less than ~200 trials per condition  
678 per experiment.

679 **5.5 Extensions**

680 The hazard models in this tutorial assume that there is one event of interest. For RT  
681 data, this event constitutes a single transition between an “idle” state and a “responded”  
682 state. However, in certain situations, more than one event of interest might exist. For  
683 example, in a medical or health-related context, an individual might transition back and  
684 forth between a “healthy” state and a “depressed” state, before a final “death” state.  
685 When you have data on the timing of these transitions, one can apply multi-state models,  
686 which generalize survival analysis to transitions between three or more states (Steele,  
687 Goldstein, & Browne, 2004). Also, the predictor variables in this tutorial are  
688 time-invariant, i.e., their value did not change over the course of a trial. Thus, another  
689 extension is to include time-varying predictors, i.e., predictors whose value can change  
690 across the time bins within a trial (@ Allison, 2010). For example, when gaze position is  
691 tracked during a visual search trial, the gaze-target distance will vary during a trial when  
692 the eyes move around before a manual response is given; shorter gaze-target distances  
693 should be associated with a higher hazard of response occurrence. Note that the effect of a  
694 time-varying predictor (e.g., an occipital EEG signal) can itself vary over time.

695 **6. Conclusions**

696 RT and accuracy distributions are a rich source of information on the time course of  
697 cognitive processing, which have been largely undervalued in the history of experimental  
698 psychology and cognitive neuroscience. Statistically controlling for the passage of time  
699 during data analysis is thus equally important as experimental control during the design of  
700 an experiment, to better understand human behavior in experimental paradigms. We hope

701 that by providing a set of hands-on, step-by-step tutorials, which come with custom-built  
702 and freely available code, researchers will feel more comfortable embracing event history  
703 analysis and investigating the temporal profile of cognitive states. On a broader level, we  
704 think that wider adoption of such approaches will have a meaningful impact on the  
705 inferences drawn from data, as well as the development of theories regarding the structure  
706 of cognition.

707

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840

**Supplementary material**

841   **A. Definitions of discrete-time hazard, survivor, and conditional accuracy**  
 842   **functions**

843       The shape of a distribution of waiting times can be described in multiple ways (Luce,  
 844       1991). After dividing time in discrete, contiguous time bins indexed by  $t$ , let  $RT$  be a  
 845       discrete random variable denoting the rank of the time bin in which a particular person's  
 846       response occurs in a particular experimental condition. Discrete-time EHA focuses on the  
 847       discrete-time hazard function

848       
$$h(t) = P(RT = t | RT \geq t) \quad (1)$$

849       and the discrete-time survivor function

850       
$$S(t) = P(RT > t) = [1-h(t)].[1-h(t-1)].[1-h(t-2)] \dots [1-h(1)] \quad (2)$$

851       and not on the probability mass function

852       
$$P(t) = P(RT = t) = h(t).S(t-1) \quad (3)$$

853       nor the cumulative distribution function

854       
$$F(t) = P(RT \leq t) = 1-S(t) \quad (4)$$

855       The discrete-time hazard function of event occurrence gives you the probability that  
 856       the event occurs (sometime) in bin  $t$ , given that the event has not occurred yet in previous  
 857       bins. While the discrete-time hazard function assesses the unique risk of event occurrence  
 858       associated with each time bin, the discrete-time survivor function cumulates the bin-by-bin  
 859       risks of event *nonoccurrence* to obtain the probability that the event occurs after bin  $t$ . The  
 860       probability mass function cumulates the risk of event occurrence in bin  $t$  with the risks of  
 861       event nonoccurrence in bins 1 to  $t-1$ . From equation 3 we find that hazard in bin  $t$  is equal  
 862       to  $P(t)/S(t-1)$ .

863 For two-choice RT data, the discrete-time hazard function can be extended with the

864 discrete-time conditional accuracy function

865  $ca(t) = P(\text{correct} \mid RT = t)$  (5)

866 which gives you the probability that a response is correct given that it is emitted in time

867 bin  $t$  (Allison, 2010; Kantowitz & Pachella, 2021; Wickelgren, 1977). This latter function is

868 also known as the micro-level speed-accuracy tradeoff (SAT) function.

869 The survivor function provides a context for the hazard function, as  $S(t-1) = P(RT >$

870  $t-1) = P(RT \geq t)$  tells you on how many percent of the trials the estimate  $h(t) = P(RT =$

871  $t \mid RT \geq t$ ) is based. The probability mass function provides a context for the conditional

872 accuracy function, as  $P(t) = P(RT = t)$  tells you on how many percent of the trials the

873 estimate  $ca(t) = P(\text{correct} \mid RT = t)$  is based.

874 When time is treated as a continuous variable, let  $RT$  be a continuous random variable

875 denoting a particular person's response time in a particular experimental condition.

876 Because waiting times can only increase, continuous-time EHA does not focus on the

877 cumulative distribution function  $F(t) = P(RT \leq t)$  and its derivative, the probability

878 density function  $f(t) = F(t)'$ , but on the survivor function  $S(t) = P(RT > t)$  and the

879 hazard rate function  $\lambda(t) = f(t)/S(t)$ . The hazard rate function gives you the instantaneous

880 *rate* of event occurrence at time point  $t$ , given that the event has not occurred yet.

## 881 B. Custom functions for descriptive discrete-time hazard analysis

882 We defined 13 custom functions that we list here.

- 883 • `censor(df,timeout,bin_width)` : divide the time segment  $(0, \text{timeout}]$  in bins, identify

884 any right-censored observations, and determine the discrete RT (time bin rank)

- 885 • `ptb(df)` : transform the person-trial data set to the person-trial-bin data set

- 886 • `setup_lt(ptb)` : set up a life table for each level of 1 independent variable

- setup\_lt\_2IV(ptb) : set up a life table for each combination of levels of 2 independent variables
- calc\_ca(df) : estimate the conditinal accuracies when there is 1 independent variable
- calc\_ca\_2IV(df) : estimate the conditional accuraiies when there are 2 independent variables
- join\_lt\_ca(df1,df2) : add the ca(t) estimates to the life tables (1 independent variable)
- join\_lt\_ca\_2IV(df1,df2) : add the ca(t) estimates to the life tables (2 independent variables)
- extract\_median(df) : estimate quantiles S(t).50 (1 independent variable)
- extract\_median\_2IV(df) : estimate quantiles S(t).50 (2 independent variables)
- plot\_oha(df,subj,haz\_yaxis) : create plots of the discrete-time functions (1 independent variable)
- plot\_oha\_2IV(df,subj,haz\_yaxis) : create plots of the discrete-time functions (2 independent variables)
- plot\_oha\_agg(df,subj,haz\_yaxis) : create 1 plot for aggregated data (1 independent variable)

When you want to analyse simple RT data from a detection experiment with one independent variable, the functions calc\_ca() and join\_lt\_ca() should not be used, and the code to plot the conditional accuracy functions should be removed from the function plot\_oha(). When you want to analyse simple RT data from a detection experiment with two independent variables, the functions calc\_ca\_2IV() and join\_lt\_ca\_2IV() should not be used, and the code to plot the conditional accuracy functions should be removed from the function plot\_oha\_2IV().

911 **C. Link functions**

912 Popular link functions include the logit link and the complementary log-log link, as

913 shown in Figure 11.

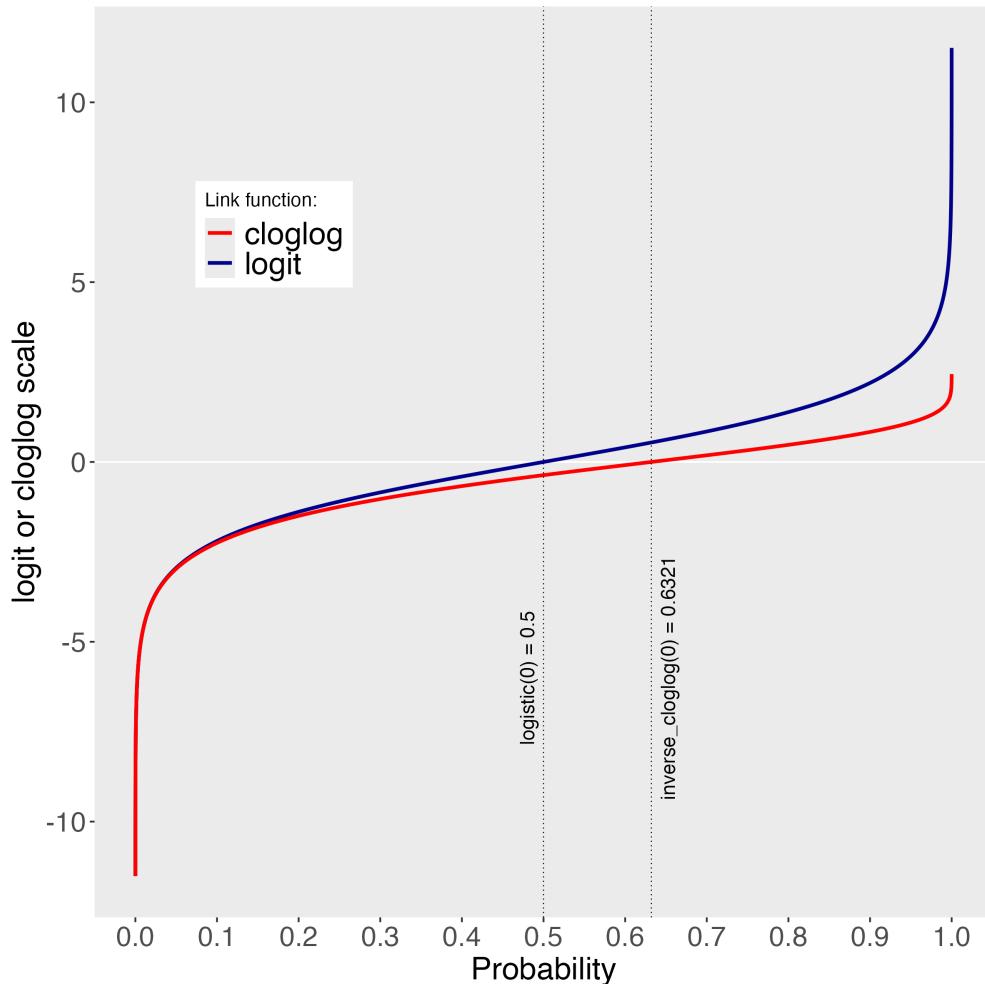


Figure 10. The logit and cloglog link functions.

914 **D. Regression equations**

915 An example (single-level) discrete-time hazard model with three predictors (TIME,

916  $X_1$ ,  $X_2$ ), the cloglog link function, and a third-order polynomial specification for TIME can

917 be written as follows:

$$918 \quad \text{cloglog}[h(t)] = \ln(-\ln[1-h(t)]) = [\alpha_1 \text{ONE} + \alpha_2(\text{TIME}-9) + \alpha_3(\text{TIME}-9)^2] + [\beta_1 X_1 +$$

$$919 \quad \beta_2 X_2 + \beta_3 X_2(\text{TIME}-9)]$$

920        The main predictor variable TIME is the time bin index t that is centered on value 9  
 921      in this example. The first set of terms within brackets, the alpha parameters multiplied by  
 922      their polynomial specifications of (centered) time, represents the shape of the baseline  
 923      cloglog-hazard function (i.e., when all predictors  $X_i$  take on a value of zero). The second set  
 924      of terms (the beta parameters) represents the vertical shift in the baseline cloglog-hazard  
 925      for a 1 unit increase in the respective predictor variable. Predictors can be discrete,  
 926      continuous, and time-varying or time-invariant. For example, the effect of a 1 unit increase  
 927      in  $X_1$  is to vertically shift the whole baseline cloglog-hazard function by  $\beta_1$  cloglog-hazard  
 928      units. However, if the predictor interacts linearly with TIME (see  $X_2$  in the example), then  
 929      the effect of a 1 unit increase in  $X_2$  is to vertically shift the predicted cloglog-hazard in bin  
 930      9 by  $\beta_2$  cloglog-hazard units (when  $\text{TIME}-9 = 0$ ), in bin 10 by  $\beta_2 + \beta_3$  cloglog-hazard  
 931      units (when  $\text{TIME}-9 = 1$ ), and so forth. To interpret the effects of a predictor, its  $\beta$   
 932      parameter is exponentiated, resulting in a hazard ratio (due to the use of the cloglog link).  
 933      When using the logit link, exponentiating a  $\beta$  parameter results in an odds ratio.

934        An example (single-level) discrete-time hazard model with a general specification for  
 935      TIME (separate intercepts for each of six bins, where D1 to D6 are binary variables  
 936      identifying each bin) and a single predictor ( $X_1$ ) can be written as follows:

$$937 \quad \text{cloglog}[h(t)] = \ln(-\ln[1-h(t)]) = [\alpha_1 D1 + \alpha_2 D2 + \alpha_3 D3 + \alpha_4 D4 + \alpha_5 D5 + \alpha_6 D6] +$$

$$938 \quad [\beta_1 X_1]$$

## 939 E. Prior distributions

940        To gain a sense of what prior *logit* values would approximate a uniform distribution

941      on the probability scale, Kurz (2023a) simulated a large number of draws from the

942      Uniform(0,1) distribution, converted those draws to the log-odds metric, and fitted a

943 Student's t distribution. Row C in Figure 4 shows that using a t-distribution with 7.61  
 944 degrees of freedom and a scale parameter of 1.57 as a prior on the logit scale, approximates  
 945 a uniform distribution on the probability scale. According to Kurz (2023a), such a prior  
 946 might be a good prior for the intercept(s) in a logit-hazard model, while the  $N(0,1)$  prior in  
 947 row D might be a good prior for the non-intercept parameters in a logit-hazard model, as it  
 948 gently regularizes  $p$  towards .5 (i.e., a zero effect on the logit scale).

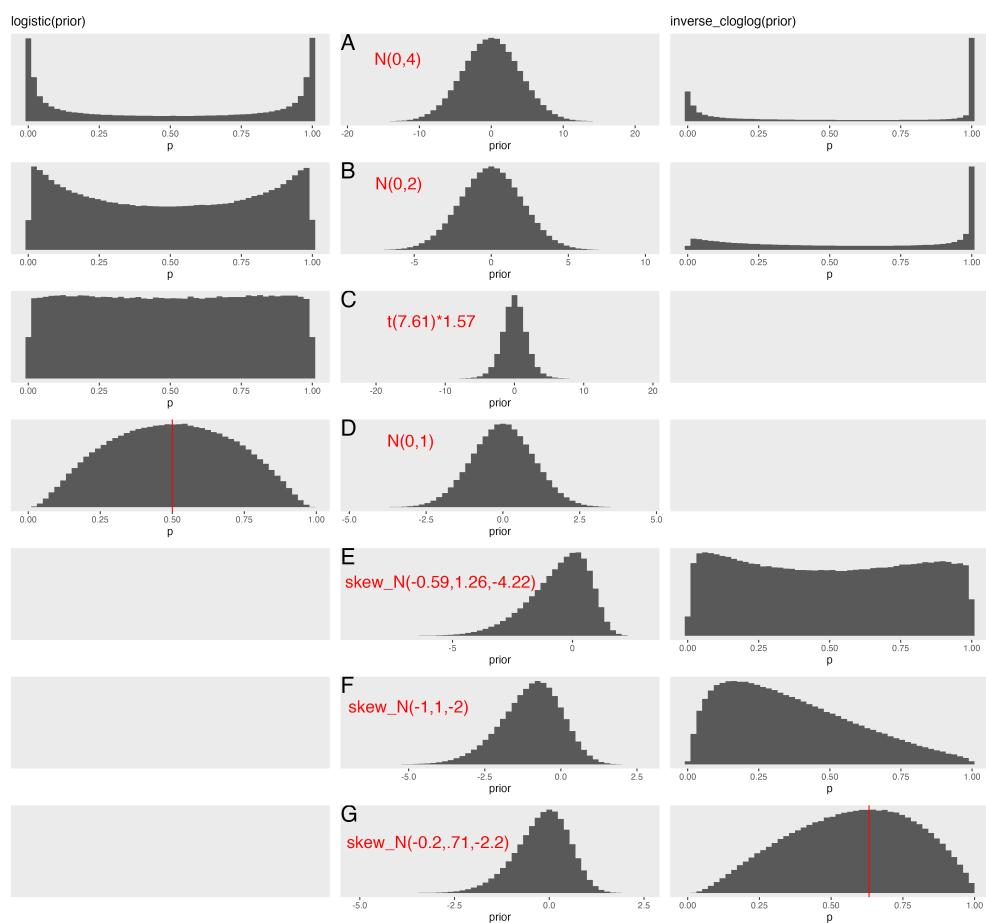


Figure 11. Prior distributions on the logit and/or cloglog scales (middle column), and their implications on the probability scale after applying the inverse-logit (or logistic) transformation (left column), and the inverse-cloglog transformation (right column).

949 To gain a sense of what prior *cloglog* values would approximate a uniform distribution  
 950 on the hazard probability scale, we followed Kurz's approach and simulated a large number

of draws from the Uniform(0,1) distribution, converted them to the cloglog metric, and fitted a skew-normal model (due to the asymmetry of the cloglog link function). Row E shows that using a skew-normal distribution with a mean of -0.59, a standard deviation of 1.26, and a skewness of -4.22 as a prior on the cloglog scale, approximates a uniform distribution on the probability scale. However, because hazard values below .5 are more likely in RT studies, using a skew-normal distribution with a mean of -1, a standard deviation of 1, and a skewness of -2 as a prior on the cloglog scale (row F), might be a good weakly informative prior for the intercept(s) in a cloglog-hazard model. A skew-normal distribution with a mean of -0.2, a standard deviation of 0.71, and a skewness of -2.2 might be a good weakly informative prior for the non-intercept parameters in a cloglog-hazard model as it gently regularizes p towards .6321 (i.e., a zero effect on the cloglog scale).