

1 A tutorial on Bayesian and Frequentist Event History Analyses for psychological  
2 time-to-event data

3 Sven Panis<sup>1</sup> & Richard Ramsey<sup>1</sup>

4 <sup>1</sup> ETH Zürich

5 Author Note

6 Neural Control of Movement lab, Department of Health Sciences and Technology

7 (D-HEST).

8 The authors made the following contributions. Sven Panis: Conceptualization,  
9 Writing - Original Draft Preparation, Writing - Review & Editing; Richard Ramsey:  
10 Conceptualization, Writing - Review & Editing, Supervision.

11 Correspondence concerning this article should be addressed to Sven Panis, ETH  
12 GLC, room G16.2, Gloriastrasse 37/39, 8006 Zürich. E-mail: sven.panis@hest.ethz.ch

13

## Abstract

14 Time-to-event data such as response times, saccade latencies, and fixation durations are  
15 ubiquitous in experimental psychology. The orthodox method for analysing such data –  
16 comparing means with analysis-of-variance – is actually hiding a lot of information about  
17 psychological effects, such as their onset time and duration, and whether they are  
18 time-locked to stimuli. Such information can change key conclusions about psychological  
19 processes and can be revealed by using distributional measures.

20 Here we provide a set of tutorials on how to implement one particular distributional  
21 method known as discrete-time event history analysis, a.k.a. hazard analysis, duration  
22 analysis, failure-time analysis, survival analysis, and transition analysis. We illustrate how  
23 one can calculate the descriptive statistics, and how one can implement Bayesian and  
24 frequentist regression models, using the R packages tidyverse, brms, and lme4. The R code  
25 is publicly available on Github and OSF, and can easily be adapted for other data sets. We  
26 further discuss how to manage inter-individual differences, implications for experimental  
27 design, and how to select among various options when analysing time-to-event data using  
28 discrete-time hazard analysis.

29 *Keywords:* response times, event history analysis, Bayesian regression models

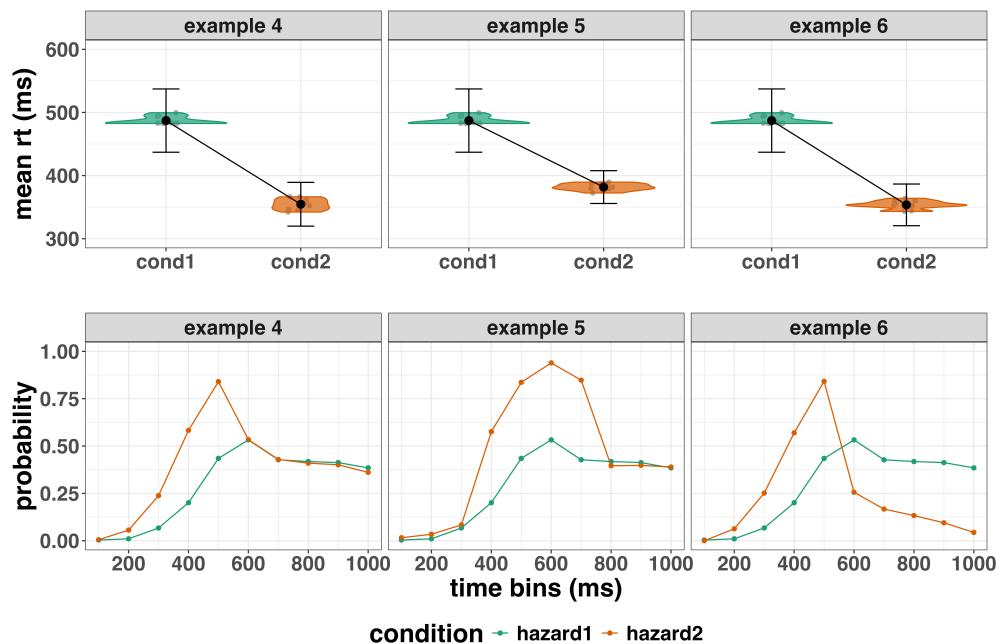
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31 A tutorial on Bayesian and Frequentist Event History Analyses for psychological  
 32 time-to-event data

33 **Introduction**

34 **Means versus distributional shapes**

35 In experimental psychology, it is still standard practice to analyse response times  
 36 (RTs), saccade latencies, and fixation durations by calculating average performance across  
 37 a series of trials. However, differences in means conceal when an experimental effect starts,  
 38 how long it lasts, how it evolves over increasing waiting time, and whether its onset is  
 39 time-locked to other events. Such information is useful not only for interpretation, but also  
 40 for cognitive psychophysiology and computational model selection (Panis, Schmidt,  
 41 Wolkersdorfer, & Schmidt, 2020). As a simple illustration, Figure 1 shows three examples  
 42 of how an observed difference in mean response times (RTs) between two experimental  
 43 conditions conceals differences in the shapes of the underlying RT distributions.



*Figure 1.* Means versus distributional shapes.

<sup>44</sup> In each example, the mean RT is lower in condition 2 compared to condition 1.  
<sup>45</sup> However, the distributions in the first example show that the effect starts around 200 ms  
<sup>46</sup> and is gone by 600 ms. In the second example, the distributional effect starts around 400  
<sup>47</sup> ms and is gone by 800 ms. And in the third example, the distributional effect reverses  
<sup>48</sup> around 550 ms.

<sup>49</sup> **Outline of the paper**

<sup>50</sup> In this paper we focus on a distributional method known as discrete-time event  
<sup>51</sup> history analysis, a.k.a. hazard analysis, duration analysis, failure-time analysis, survival  
<sup>52</sup> analysis, and transition analysis. We first provide a brief overview of hazard analysis to  
<sup>53</sup> orient the reader to the basic concepts and ideas that we will use throughout the paper.  
<sup>54</sup> However, this will remain relatively short, and for detailed treatment, see Singer and  
<sup>55</sup> Willett (2003), Allison (1982), and Allison (2010).

<sup>56</sup> We then provide four different tutorials, each of which is written in R code and  
<sup>57</sup> publicly available on Github and the Open Science Framework (OSF). The tutorials  
<sup>58</sup> provide hands-on, concrete examples of key parts of the analytical process, so that others  
<sup>59</sup> can apply the analyses to their own time-to-event data sets. In Tutorial 1 we illustrate how  
<sup>60</sup> to calculate the descriptive statistics for a published data set when there is one  
<sup>61</sup> independent variable. The descriptive statistics are plotted, and we comment on their  
<sup>62</sup> interpretation. In Tutorial 2 we illustrate how one can fit Bayesian hazard models to the  
<sup>63</sup> data. After selecting the best of four models, we plot the model-based effects and the  
<sup>64</sup> model fits for a few subjects. In Tutorial 3 we illustrate how to fit hazard models in a  
<sup>65</sup> frequentist framework. We compare the model-based effects between Bayesian and  
<sup>66</sup> frequentist approaches. In Tutorial 4 we illustrate how to calculate the descriptive  
<sup>67</sup> statistics when there are two independent variables.

**68 Overview of hazard analysis**

69 To apply event history analysis (EHA), one must be able to define the event of  
70 interest (any qualitative change that can be situated in time, e.g., a button press, a saccade  
71 onset, a fixation offset, etc.), time point zero (e.g., target stimulus onset, fixation onset),  
72 and measure the passage of time between time point zero and event occurrence in discrete  
73 or continuous time units. Both the definition of hazard and the type of models employed  
74 depend on whether one is using continuous or discrete time units.

75 The shape of a distribution of waiting times can be described in multiple ways (Luce,  
76 1991). Let  $RT$  be a continuous random variable denoting a particular person's response time  
77 in a particular experimental condition. Because waiting times can only increase,  
78 continuous-time EHA does not focus on the cumulative distribution function  $F(t) = P(RT \leq t)$  and its derivative, the probability density function  $f(t) = F(t)'$ , but on the survivor  
79 function  $S(t) = P(RT > t)$  and the hazard rate function  $\lambda(t) = f(t)/S(t)$ . The hazard rate  
80 function gives you the instantaneous rate of event occurrence at time point  $t$ , given that  
81 the event has not occurred yet.  
82

83 Similarly, after dividing time in discrete, contiguous time bins indexed by  $t$ , let  $RT$  be  
84 a discrete random variable denoting the rank of the time bin in which a particular person's  
85 response occurs in a particular experimental condition. Discrete-time EHA focuses on the  
86 discrete-time hazard function  $h(t) = P(RT = t | RT \geq t)$  and the discrete-time survivor  
87 function  $S(t) = P(RT > t) = [1-h(t)].[1-h(t-1)].[1-h(t-2)] \dots [1-h(1)]$ , and not on the  
88 probability mass function  $p(t) = h(t).S(t-1)$  and the cumulative distribution function  $F(t) = 1-S(t)$ . The discrete-time hazard probability function gives you the probability that the  
89 event occurs (sometime) in bin  $t$ , given that the event has not occurred yet in previous  
90 bins. Unlike the discrete-time hazard function, which assesses the unique risk associated  
91 with each time bin, the discrete-time survivor function cumulates the bin-by-bin risks of  
92 event *nonoccurrence*.

94 For two-choice RT data, the discrete-time hazard function can be extended with the  
95 discrete-time conditional accuracy function  $ca(t) = P(\text{correct} \mid RT = t)$ , which gives you  
96 the probability that a response is correct given that it has been emitted in time bin  $t$   
97 (Allison, 2010; Kantowitz & Pachella, 2021; Wickelgren, 1977). This latter function is also  
98 known as the micro-level speed-accuracy tradeoff function.

99 Statisticians and mathematical psychologists recommend focusing on the hazard  
100 function when analyzing time-to-event data for various reasons. First, as discussed by  
101 Holden, Van Orden, and Turvey (2009), “probability density functions can appear nearly  
102 identical, both statistically and to the naked eye, and yet are clearly different on the basis  
103 of their hazard functions (but not vice versa). Hazard functions are thus more diagnostic  
104 than density functions” (p. 331). Second, because RT distributions may differ from one  
105 another in multiple ways, Townsend (1990) developed a dominance hierarchy of statistical  
106 differences between two arbitrary distributions A and B. For example, if  $F_A(t) > F_B(t)$  for  
107 all  $t$ , then both cumulative distribution functions are said to show a complete ordering.  
108 Townsend (1990) showed that a complete ordering on the hazard functions  $-\lambda_A(t) > \lambda_B(t)$   
109 for all  $t$ —implies a complete ordering on both the cumulative distribution and survivor  
110 functions  $-F_A(t) > F_B(t)$  and  $S_A(t) < S_B(t)$ —which in turn implies an ordering on the  
111 mean latencies—mean A < mean B. In contrast, an ordering on two means does not imply  
112 a complete ordering on the corresponding  $F(t)$  and  $S(t)$  functions, and a complete ordering  
113 on these latter functions does not imply a complete ordering on the corresponding hazard  
114 functions. This means that stronger conclusions can be drawn from data when comparing  
115 the hazard functions using EHA. For example, when mean A < mean B, the hazard  
116 functions might show a complete ordering (i.e., for all  $t$ ), a partial ordering (e.g., only for  $t$   
117  $> 300$  ms, or only for  $t < 500$  ms), or they may cross each other one or more times. Third,  
118 EHA does not discard right-censored observations when estimating hazard functions, that  
119 is, trials for which we do not observe a response during the data collection period so that  
120 we only know that the RT must be larger than some value. This is important because

121 although a few right-censored observations are inevitable in most RT tasks, a lot of  
122 right-censored observations are expected in experiments on masking, the attentional blink,  
123 and so forth. Fourth, hazard modeling allows incorporating time-varying explanatory  
124 covariates such as heart rate, electroencephalogram (EEG) signal amplitude, gaze location,  
125 etc. (Allison, 2010) which is useful for cognitive psychophysiology (Meyer, Osman, Irwin, &  
126 Yantis, 1988). Finally, as explained by Kelso, Dumas, and Tognoli (2013), it is crucial to  
127 first have a precise description of the macroscopic behavior of a system (here:  $h(t)$  and  
128  $ca(t)$  functions) in order to know what to derive on the microscopic level. For example,  
129 fitting parametric functions or computational models to data without studying the shape  
130 of the discrete-time  $h(t)$  and  $ca(t)$  functions can miss important features in the data  
131 (Panis, Moran, Wolkersdorfer, & Schmidt, 2020; Panis & Schmidt, 2016).

132 We focus on factorial within-subject designs in which a large number of observations  
133 are made on a relatively small number of participants (small- $N$  designs). This approach  
134 emphasizes the precision and reproducibility of data patterns at the individual participant  
135 level to increase the inferential validity of the design (Baker et al., 2021; Smith & Little,  
136 2018). In contrast to the large- $N$  design that averages across many participants without  
137 being able to scrutinize individual data patterns, small- $N$  designs retain crucial  
138 information about the data patterns of individual observers. This is of great advantage  
139 whenever participants differ systematically in their strategies or in the time-courses of their  
140 effects, so that blindly averaging them would lead to misleading data patterns. Indeed,  
141 Smith and Little (2018) argue that, “if psychology is to be a mature quantitative science,  
142 then its primary theoretical aim should be to investigate systematic functional  
143 relationships as they are manifested at the individual participant level” (p. 2083). Note  
144 that because statistical power derives both from the number of participants and from the  
145 number of repeated measures per participant and condition, small- $N$  designs can have  
146 excellent power (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. Web links are printed in bold. The content of the tutorials is mainly based on Allison (2010), Singer and Willett (2003), McElreath (2018), Kurz (2023a), and Kurz (2023b).

## Tutorial 1: Calculating descriptive statistics using a life table

To illustrate how to quickly set up life tables for calculating the descriptive statistics of discrete time), we use a published data set on masked response priming from Panis and Schmidt (2016), available on **ResearchGate**. In their first experiment, Panis and Schmidt (2016) presented a double arrow for 94 ms that pointed left or right as the target stimulus with an onset at time point zero in each trial. Participants had to indicate the direction in which the double arrow pointed using their corresponding index finger, within 800 ms after target onset. Response time and accuracy were recorded on each trial. Prime type (blank, congruent, incongruent) and mask type were manipulated. Here we focus on the subset of trials in which no mask was presented. The 13-ms prime stimulus

160 was a double arrow with onset at -187 ms for the congruent (same direction as target) and  
 161 incongruent (opposite direction as target) prime conditions.

162 After loading in the data file, one has to (a) supply required column names, and (b)  
 163 specify the factor condition with the correct levels and labels. The required column names  
 164 are as follows:

- 165 • “pid”, indicating unique participant IDs;
- 166 • “trial”, indicating each unique trial per participant;
- 167 • “condition”, a factor indicating the levels of the independent variable (1, 2, ...) and  
 168 the corresponding labels;
- 169 • “rt”, indicating the response times in ms;
- 170 • “acc”, indicating the accuracies (1/0).

171 In the code of Tutorial 1, 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")))
```

172 To set up the life tables and plots of the discrete-time functions  $h(t)$ ,  $S(t)$  and  $ca(t)$   
 173 using functional programming, one has to nest the data within participants using the  
 174 `group_nest()` function, and supply a user-defined censoring time and bin width to our  
 175 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
```

176 Note that the censoring time should be a multiple of the bin width (both in ms). The

177 censoring time should be a time point after which no informative responses are expected

178 anymore. In experiments that implement a response deadline in each trial the censoring

179 time can equal that deadline time point. Trials with a RT larger than the censoring time,

180 or trials in which no response is emitted during the data collection period, are treated as

181 right-censored observations in EHA. In other words, these trials are not discarded, because

182 they contain the information that the event did not occur before the censoring time.

183 Removing such trials before calculating the mean event time can introduce a sampling bias.

184 The person-trial-bin oriented dataset has one row for each time bin of each trial that is at

185 risk for event occurrence. The variable “event” in the person-trial-bin oriented data set

186 indicates whether a response occurs (1) or not (0) for each bin. When creating the plots

187 using our function `plot_eha()`, some warning messages will likely be generated, like these:

- Removed 2 rows containing missing values or values outside the scale range

189 (`geom_line()`).

- Removed 2 rows containing missing values or values outside the scale range

191 (`geom_point()`).

- Removed 2 rows containing missing values or values outside the scale range

193 (`geom_segment()`).

194 The warning messages are generated because some bins have no hazard and  $ca(t)$

195 estimates, and no error bars. They can thus safely be ignored. One can now inspect

196 different aspects, including the life table for a particular condition of a particular subject,

197 and a plot of the different functions for a particular participant.

198 Table 1 shows the life table for condition “blank” (no prime stimulus presented) -

199 compare to Figure 1. A life table includes for each time bin, the risk set (number of trials

200 that are event-free at the start of the bin), the number of observed events, and the

201 estimates of  $h(t)$ ,  $S(t)$ ,  $ca(t)$  and their estimated standard errors (se). At time point zero,  
202 no events can occur and therefore  $h(t)$  and  $ca(t)$  are undefined.

203 Figure 1 displays the discrete-time hazard, survivor, and conditional accuracy  
204 functions for each prime condition for participant 6. By using discrete-time  $h(t)$  functions  
205 of event occurrence - in combination with  $ca(t)$  functions for two-choice tasks - one can  
206 provide an unbiased, time-varying, and probabilistic description of the latency and  
207 accuracy of responses based on all trials of any data set.

208 For example, for participant 6, the estimated hazard values in bin (240,280] are 0.03,  
209 0.17, and 0.20 for the blank, congruent, and incongruent prime conditions, respectively. In  
210 other words, when the waiting time has increased until *240 ms* after target onset, then the  
211 conditional probability of response occurrence in the next 40 ms is more than five times  
212 larger for both prime-present conditions, compared to the blank prime condition.

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

Table 1

*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.

## Subject 6

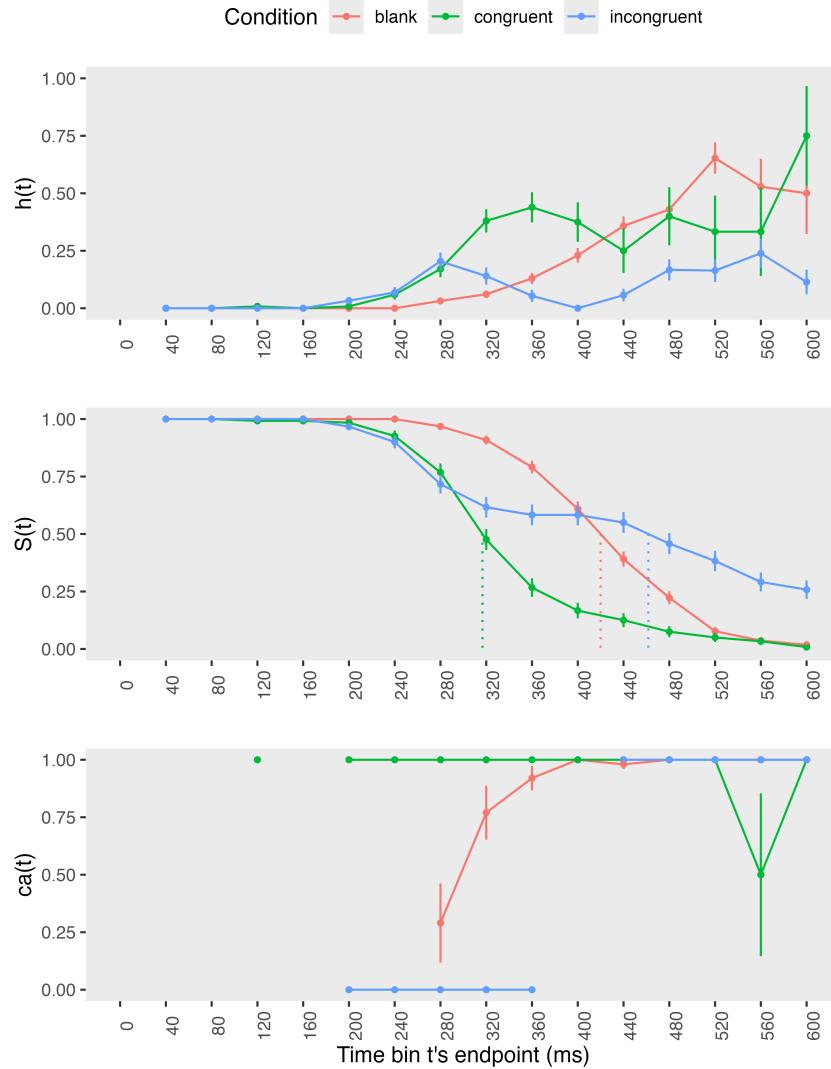


Figure 2. Estimated discrete-time hazard, survivor, and conditional accuracy functions for participant 6, as a function of the passage of discrete waiting time.

218 However, when the waiting time has increased until 400 ms after target onset, then  
 219 the conditional probability of response occurrence in the next 40 ms is estimated to be  
 220 0.36, 0.25, and 0.06 for the blank, congruent, and incongruent prime conditions,  
 221 respectively. And when a response does occur in bin (400,440], then the probability that it  
 222 is correct is estimated to be 0.98, 1, and 1 for the blank, congruent, and incongruent prime

223 conditions, respectively.

224 These results show that this participant is initially responding to the prime even  
225 though (s)he was instructed to only respond to the target, that response competition  
226 emerges in the incongruent prime condition around 300 ms, and that only later response  
227 are fully controlled by the target stimulus. Qualitatively similar results were obtained for  
228 the other five participants. Also, in their second Experiment, Panis and Schmidt (2016)  
229 showed that the negative compatibility effect in the mask-present conditions is time-locked  
230 to mask onset. This example shows that a simple difference between two means fails to  
231 reveal the dynamic behavior people display in many experimental paradigms (Panis, 2020;  
232 Panis, Moran, et al., 2020; Panis & Schmidt, 2022; Panis, Torfs, Gillebert, Wagemans, &  
233 Humphreys, 2017; Panis & Wagemans, 2009; Schmidt, Panis, Wolkersdorfer, & Vorberg,  
234 2022). In other words, statistically controlling for the passage of time during data analysis  
235 is equally important as experimental control during the design of an experiment, to better  
236 understand human behavior in experimental paradigms. As we will show in Tutorials 2 and  
237 3, statistical models for  $h(t)$  can be implemented as generalized linear mixed regression  
238 models predicting event occurrence (1/0) in each bin of a selected time range.

239 **Tutorial 2: Fitting Bayesian hazard models**

240 When you want to study how hazard depends on various predictors, you can fit  
241 regression models to the data (Singer & Willett, 2003). There are two analytic decisions  
242 one has to make. First, one has to select an analysis time range, i.e., a contiguous set of  
243 bins for which there is enough data for each participant. Second, one can choose the logit  
244 link function which transforms a (hazard) probability into the log of the odds ratio, or the  
245 complementary log-log (cloglog) link function, which yields the logarithm of the negated  
246 logarithm of the probability of event *nonoccurrence*. An important difference between  
247 these two link functions is that cloglog provides a discrete-time hazard model that has a  
248 built-in proportional hazards assumption, while logit provides a proportional odds

assumption (see below). The cloglog link is preferred over the logit link when events can occur in principle at any time point within a bin, which is the case for RT data (Singer & Willett, 2003). Third, one has to choose a specification of the effect of discrete TIME (i.e., the time bin index t). One can choose a general specification (one intercept per bin) or a functional specification, such as a polynomial one.

An example discrete-time hazard model with three predictors (TIME, X1, X2) and the cloglog link function can be written as follows:

$$\text{cloglog}[h(t)] = \ln(-\ln[1-h(t)]) = [\alpha_1 \text{ONE} + \alpha_2(\text{TIME}-1) + \alpha_3(\text{TIME}-1)^2] + [\beta_1 X_1 + \beta_2 X_2 + \beta_3 X_2(\text{TIME}-1)].$$

The main predictor variable TIME is the time bin index t that is centered on value 1 in this example. The first set of terms within brackets, the alpha parameters multiplied by their polynomial specifications of (centered) time, represents the shape of the baseline cloglog-hazard function (i.e., when all predictors  $X_i$  take on a value of zero). The second set of terms (the beta parameters) represents the vertical shift in the baseline cloglog-hazard for a 1 unit increase in the respective predictor. Predictors can be discrete, continuous, and time-varying or time-invariant. For example, the effect of a 1 unit increase in  $X_1$  is to vertically shift the whole baseline cloglog-hazard function by  $\beta_1$  cloglog-hazard units. However, if the predictor interacts linearly with time (see  $X_2$  in the example), then the effect of a 1 unit increase in  $X_2$  is to vertically shift the predicted cloglog-hazard in bin 1 by  $\beta_2$  cloglog-hazard units (when  $\text{TIME}-1 = 0$ ), in bin 2 by  $\beta_2 + \beta_3$  cloglog-hazard units (when  $\text{TIME}-1 = 1$ ), and so forth. To interpret the effects of the predictors, the parameter estimates are exponentiated, resulting in a hazard ratio (due to the use of the cloglog link).

In the case of a large- $N$  design without repeated measurements, the parameters of a discrete-time hazard model can be estimated using standard logistic regression software (after expanding the typical person-trial-oriented data set into a person-trial-bin-oriented data set (see Tutorial 1); Allison (2010)). When there is clustering in the data, as in the

275 case of a small- $N$  design with repeated measurements, the parameters of a discrete-time  
 276 hazard model can be estimated using population-averaged methods (e.g., Generalized  
 277 Estimating Equations), and Bayesian or frequentist generalized linear mixed models  
 278 (Allison, 2010).

279 In this second tutorial we illustrate how to fit a Bayesian hazard regression model for  
 280 the masked response priming data set used in the first tutorial. In general, there are three  
 281 assumptions one can make or relax when adding experimental predictor variables: The  
 282 linearity assumption for continuous predictors (the effect of a 1 unit change is the same  
 283 anywhere on the scale), the additivity assumption (predictors do not interact), and the  
 284 proportionality assumption (predictors do not interact with TIME).

285 First, we select the analysis range (200,600] and the cloglog link, and use a  
 286 polynomial to specify the effect of TIME in the “blank” prime condition. Second, based on  
 287 previous work (Panis, 2020; Panis, Moran, et al., 2020; Panis & Schmidt, 2022; Panis et al.,  
 288 2017; Panis & Wagemans, 2009) and because cognition is likely the behavior of a non-linear  
 289 dynamical system [ref], we relax all three assumptions, as follows:

```
# load data
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 discrete TIME (period) on bin 9, and trial on trial 1000
ptb_data <- ptb_data %>% mutate(period_9 = period - 9,
                                    trial_c = (trial - 1000)/1000)
# remove unnecessary columns before fitting a model
ptb_data <- ptb_data %>% select(-c(bl,tr,trial,period)) # 12840 obs. of 5 variables

priors <- c(
  set_prior("normal(0, 1)", class = "b"), # for beta parameters
  set_prior("student_t(7.61, 0, 1.57)", class = "b", coef = "Intercept"), # flat prior for intercept on hazard scale
  set_prior("normal(0, 1)", class = "sd"), # for standard deviation of RE
  set_prior("lkj(2)", class = "cor") # for correlations between RE
)
```

```
#plan(multicore)
#model_full_RE <-
#  brm(data = ptb_data,
#       family = binomial(link="cloglog"),
#       event / trials(1) ~ 0 + Intercept +
#                           condition*period_9*trial_c +
#                           condition*I(period_9^2) +
#                           condition*I(period_9^3) +
#                           (1 + condition*period_9*trial_c +
#                           condition*I(period_9^2) +
#                           condition*I(period_9^3) / pid),
#       prior = priors,
#       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_full_RE")
```

290 To test whether (centered) trial number affects behavior, we fit a model without the  
 291 variable trial\_c.

292 Use WAIC to compare models.

293 Plot the effects of congruent and incongruent for each time bin for the selected model.

294 Plot the model-based hazard and survivor functions.

### 295           **Tutorial 3: Fitting Frequentist hazard models**

296 In this third tutorial we illustrate how to fit a frequentist hazard regression model for  
 297 the data set used in the first tutorial.

### 298           **Tutorial 4: Calculating descriptive statistics when there are two independent 299                                  variables**

300 In this final tutorial we illustrate how to calculate and plot the descriptive statistics  
 301 for the full data set of Experiment 1 of Panis and Schmidt (2016).

302

## Discussion

303 **Individual differences**

- 304     • role of response deadlines, low-level vs. higher-level processes,
- 305     • clustering algorithms based on  $h(t)$  and  $ca(t)$  data
- 306     •

307 **Cognitive psychophysiology and computational model selection**308 **Power analysis**

- 309     • example repo on github

310 **Preregistration**

- 311     • example preregistration for knot data

312

## Conclusions

313

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