

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

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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 how they evolve with
18 increasing waiting time. Such information can change key conclusions about psychological
19 processes and can be revealed by using distributional measures that portray the detailed
20 shape of time-to-event distributions.

21 Here we provide a set of tutorials on how to implement one particular distributional
22 method known as discrete-time event history analysis, a.k.a. hazard analysis, duration
23 analysis, failure-time analysis, survival analysis, and transition analysis. We illustrate how
24 one can calculate the descriptive statistics, and how one can implement Bayesian and
25 frequentist regression models, using the R packages tidyverse, brms, and lme4. The R code
26 is publicly available on Github and OSF, and can easily be adapted for other data sets. We
27 discuss possible link functions and prior distributions, how to manage inter-individual
28 differences, implications for experimental design, the advantages of a hazard analysis over
29 other distributional methods available in the literature, limitations, and extensions. Our
30 ultimate goal is to convince readers to start using hazard analysis more often when dealing
31 with time-to-event data.

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

33 Word count: X

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

36 **Introduction**

37 **Means versus distributional shapes**

38 In experimental psychology, it is still standard practice to analyse response times
39 (RTs), saccade latencies, and fixation durations by calculating average performance across
40 a series of trials. However, differences in mean RT conceal when an experimental effect
41 starts, how long it lasts, how it evolves over increasing waiting time, and whether its onset
42 is time-locked to other events. Such information is useful not only for interpretation of the
43 effects, but also for cognitive psychophysiology and computational model selection (Panis,
44 Schmidt, Wolkersdorfer, & Schmidt, 2020).

45 As a simple illustration, Figure 1 shows three examples of how an observed difference
46 in mean response times (RTs) between two experimental conditions conceals differences in
47 the shapes of the underlying RT distributions. In each example, the mean RT is lower in
48 condition 2 compared to condition 1. However, the distributions in the first example show
49 that the effect starts around 200 ms and is gone by 600 ms. In the second example, the
50 effect starts around 400 ms and is gone by 800 ms. And in the third example, the effect
51 reverses around 550 ms.

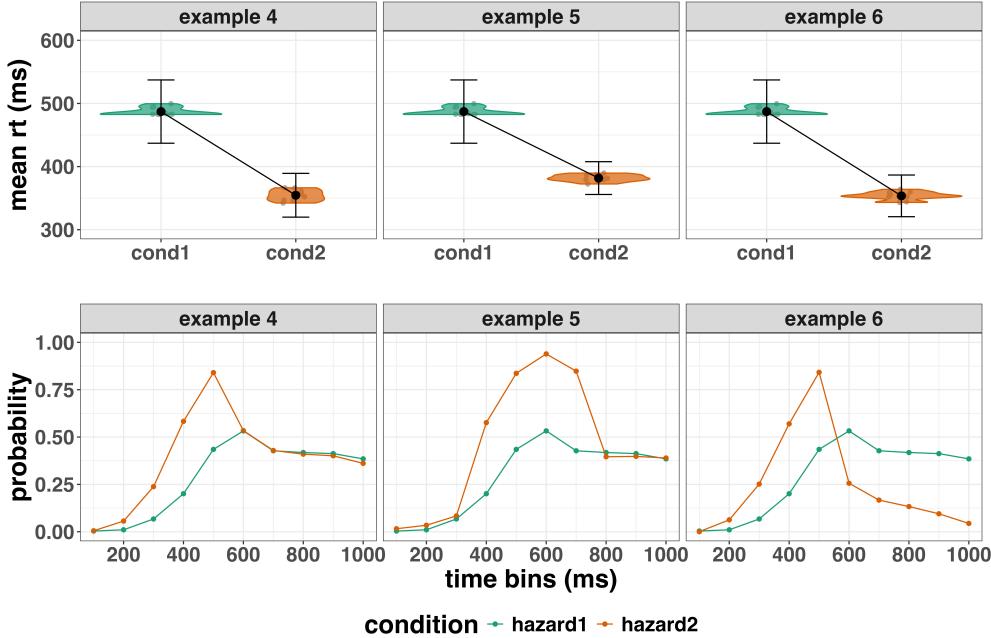


Figure 1. Means versus distributional shapes.

52 Outline of the paper

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

59 We then provide four different tutorials, each of which is written in R code and
 60 publicly available on Github and the Open Science Framework (OSF). The tutorials
 61 provide hands-on, concrete examples of key parts of the analytical process, so that others
 62 can apply the analyses to their own time-to-event data sets. In Tutorial 1 we illustrate how
 63 to calculate the descriptive statistics for a published data set when there is one
 64 independent variable. The descriptive statistics are plotted, and we comment on their

65 interpretation. In Tutorial 2 we illustrate how one can fit Bayesian hazard models to the
66 data. We discuss possible link functions, and plot the model-based effects of our predictors
67 of interest. In Tutorial 3 we illustrate how to fit hazard models in a frequentist framework.
68 Even though both frameworks generate similar parameter estimates, we note that model
69 convergence is often not obtained in the frequentist framework. In Tutorial 4 we illustrate
70 how to calculate the descriptive statistics when there are two independent variables.

71 **Overview of hazard analysis**

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

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

86 Similarly, after dividing time in discrete, contiguous time bins indexed by $t,$ let RT be
87 a discrete random variable denoting the rank of the time bin in which a particular person's
88 response occurs in a particular experimental condition. Discrete-time EHA focuses on the
89 discrete-time hazard function $h(t) = P(RT = t | RT \geq t)$ and the discrete-time survivor

90 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
91 probability mass function $p(t) = h(t).S(t-1)$ and the cumulative distribution function $F(t)$
92 $= 1-S(t)$. The discrete-time hazard probability function gives you the probability that the
93 event occurs (sometime) in bin t , given that the event has not occurred yet in previous
94 bins. Unlike the discrete-time hazard function, which assesses the unique risk associated
95 with each time bin, the discrete-time survivor function cumulates the bin-by-bin risks of
96 event *nonoccurrence*.

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

102 We focus on factorial within-subject designs in which a large number of observations
103 are made on a relatively small number of participants (small- N designs). This approach
104 emphasizes the precision and reproducibility of data patterns at the individual participant
105 level to increase the inferential validity of the design (Baker et al., 2021; Smith & Little,
106 2018). In contrast to the large- N design that averages across many participants without
107 being able to scrutinize individual data patterns, small- N designs retain crucial
108 information about the data patterns of individual observers. This is of great advantage
109 whenever participants differ systematically in their strategies or in the time-courses of their
110 effects, so that blindly averaging them would lead to misleading data patterns. Indeed,
111 Smith and Little (2018) argue that, “if psychology is to be a mature quantitative science,
112 then its primary theoretical aim should be to investigate systematic functional
113 relationships as they are manifested at the individual participant level” (p. 2083). Note
114 that because statistical power derives both from the number of participants and from the
115 number of repeated measures per participant and condition, small- N designs can have
116 excellent power (Baker et al., 2021; Smith & Little, 2018).

We used R (Version 4.4.0; R Core Team, 2024)¹ for all reported analyses. Web links printed in bold. The content of the tutorials is mainly based on Allison (2010), Singer 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, in 800 ms after target onset. Response time and accuracy were recorded on each trial.

128 Prime type (blank, congruent, incongruent) and mask type were manipulated. Here we
 129 focus on the subset of trials in which no mask was presented. The 13-ms prime stimulus
 130 was a double arrow with onset at -187 ms for the congruent (same direction as target) and
 131 incongruent (opposite direction as target) prime conditions.

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

- 135 • “pid”, indicating unique participant IDs;
- 136 • “trial”, indicating each unique trial per participant;
- 137 • “condition”, a factor indicating the levels of the independent variable (1, 2, . . .) and
 the corresponding labels;
- 138 • “rt”, indicating the response times in ms;
- 139 • “acc”, indicating the accuracies (1/0).

141 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")))
```

142 To set up the life tables and plots of the discrete-time functions $h(t)$, $S(t)$ and $ca(t)$
 143 using functional programming, one has to nest the data within participants using the
 144 `group_nest()` function, and supply a user-defined censoring time and bin width to our
 145 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

```

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

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

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

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

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

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

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

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

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

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

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

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

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

159 `(geom_line())`.

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

161 `(geom_point())`.

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

163 `(geom_segment())`.

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

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

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

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

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

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

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

171 estimates of $h(t)$, $S(t)$, $ca(t)$ and their estimated standard errors (se). At time point zero,

172 no events can occur and therefore $h(t)$ and $ca(t)$ are undefined.

173 Figure 2 displays the discrete-time hazard, survivor, and conditional accuracy

174 functions for each prime condition for participant 6. By using discrete-time $h(t)$ functions

175 of event occurrence - in combination with $ca(t)$ functions for two-choice tasks - one can

176 provide an unbiased, time-varying, and probabilistic description of the latency and

177 accuracy of responses based on all trials of any data set.

178 For example, for participant 6, the estimated hazard values in bin (240,280] are 0.03,

179 0.17, and 0.20 for the blank, congruent, and incongruent prime conditions, respectively. In

180 other words, when the waiting time has increased until 240 ms after target onset, then the

181 conditional probability of response occurrence in the next 40 ms is more than five times

182 larger for both prime-present conditions, compared to the blank prime condition.

183 Furthermore, the estimated conditional accuracy values in bin (240,280] are 0.29, 1,

184 and 0 for the blank, congruent, and incongruent prime conditions, respectively. In other

185 words, if a response is emitted in bin (240,280], then the probability that it is correct is

186 estimated to be 0.29, 1, and 0 for the blank, congruent, and incongruent prime conditions,

187 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

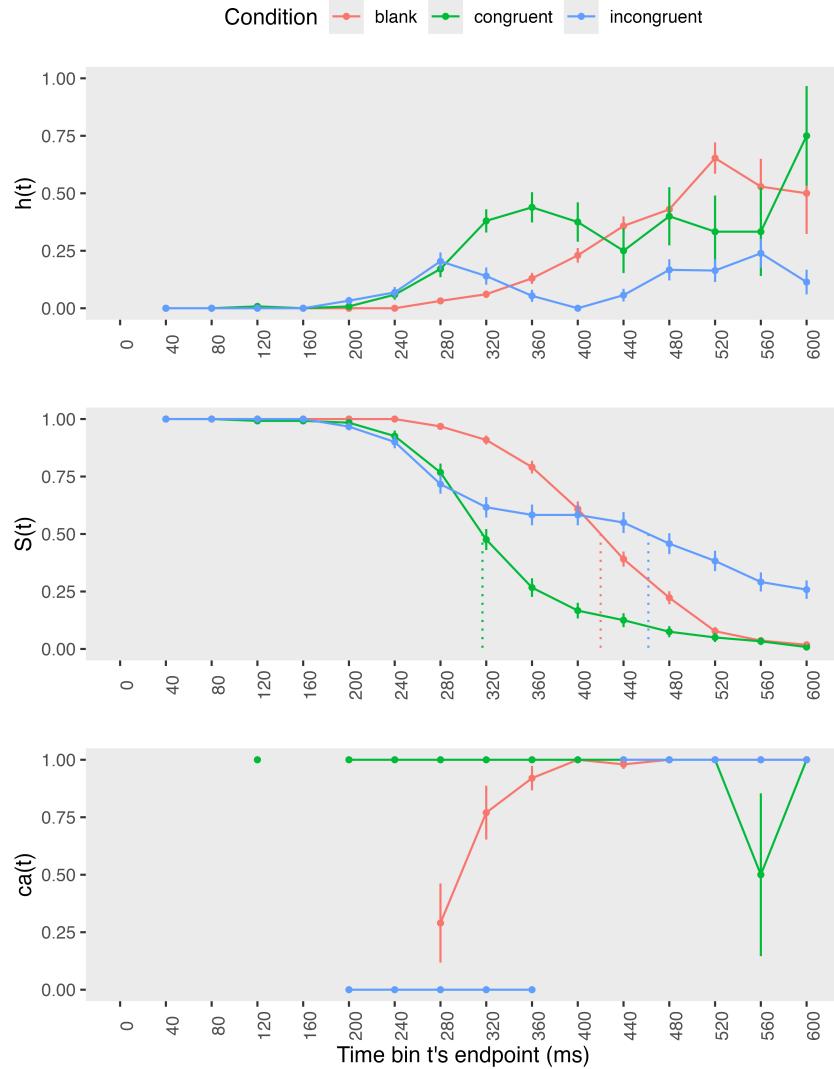


Figure 2. Estimated discrete-time hazard, survivor, and conditional accuracy functions for participant 6, as a function of the passage of discrete waiting time.

188 However, when the waiting time has increased until 400 ms after target onset, then
 189 the conditional probability of response occurrence in the next 40 ms is estimated to be
 190 0.36, 0.25, and 0.06 for the blank, congruent, and incongruent prime conditions,
 191 respectively. And when a response does occur in bin (400,440], then the probability that it
 192 is correct is estimated to be 0.98, 1, and 1 for the blank, congruent, and incongruent prime

193 conditions, respectively.

194 These results show that this participant is initially responding to the prime even
195 though (s)he was instructed to only respond to the target, that response competition
196 emerges in the incongruent prime condition around 300 ms, and that only later response
197 are fully controlled by the target stimulus. Qualitatively similar results were obtained for
198 the other five participants. These results go against the (often implicit) assumption that all
199 observed responses are primed responses to the target stimulus.

200 Also, in their second Experiment, Panis and Schmidt (2016) showed that the negative
201 compatibility effect in the mask-present conditions (see Tutorial 4) is time-locked to mask
202 onset. This example shows that a simple difference between two means fails to reveal the
203 dynamic behavior people display in many experimental paradigms (Panis, 2020; Panis,
204 Moran, Wolkersdorfer, & Schmidt, 2020; Panis & Schmidt, 2022; Panis, Torfs, Gillebert,
205 Wagemans, & Humphreys, 2017; Panis & Wagemans, 2009; Schmidt, Panis, Wolkersdorfer,
206 & Vorberg, 2022). In other words, statistically controlling for the passage of time during
207 data analysis is equally important as experimental control during the design of an
208 experiment, to better understand human behavior in experimental paradigms. As we will
209 show in Tutorials 2 and 3, statistical models for $h(t)$ can be implemented as generalized
210 linear mixed regression models predicting event occurrence (1/0) in each bin of a selected
211 time range.

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

213 In this second tutorial we illustrate how to fit Bayesian hazard regression models to
214 the masked response priming data set used in the first tutorial. Fitting (Bayesian or
215 non-Bayesian) regression models to the data is important when you want to study how the
216 shape of the hazard function depends on various predictors (Singer & Willett, 2003).

217 There are several analytic decisions one has to make when fitting a hazard model.

218 First, one has to select an analysis time window, i.e., a contiguous set of bins for which
 219 there is enough data for each participant. Second, given that the dependent variable is
 220 binary, one has to select a link function (see Figure 3).

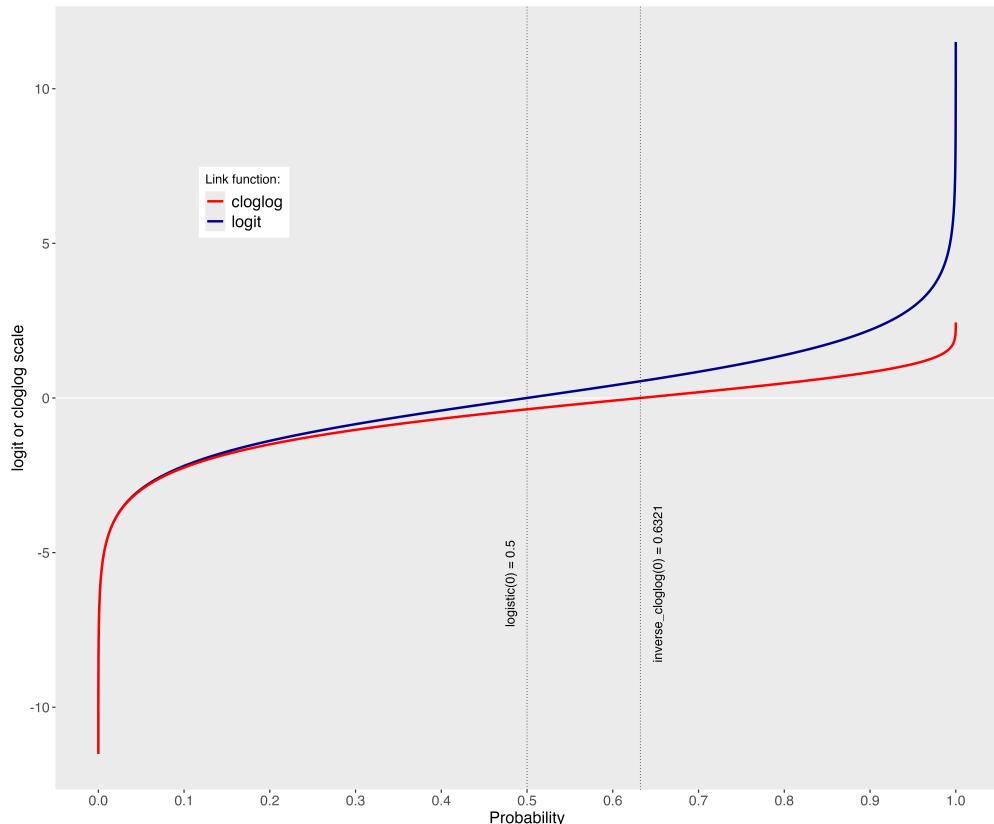


Figure 3. The logit and cloglog link functions.

221 The symmetric logit link function transforms a (hazard) probability into the log of
 222 the odds ratio. The asymmetric complementary log-log (cloglog) link function transforms
 223 hazard into the logarithm of the negated logarithm of the probability of event
 224 *nonoccurrence*. An important difference between these two link functions is that cloglog
 225 provides a discrete-time hazard model that has a built-in proportional hazards assumption,
 226 while logit provides a proportional odds assumption (see below). The cloglog link is
 227 preferred over the logit link when events can occur in principle at any time point within a
 228 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 (compare model 1 with models 2, 3, and 4 below).

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

$$\text{cloglog}[h(t)] = \ln(-\ln[1-h(t)]) = [\alpha_1 \text{ONE} + \alpha_2(\text{TIME}-9) + \alpha_3(\text{TIME}-9)^2] + [\beta_1 X_1 + \beta_2 X_2 + \beta_3 X_2(\text{TIME}-9)].$$

The main predictor variable TIME is the time bin index t that is centered on value 9 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 variable. 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 β_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 9 by β_2 cloglog-hazard units (when $\text{TIME}-9 = 0$), in bin 10 by $\beta_2 + \beta_3$ cloglog-hazard units (when $\text{TIME}-9 = 1$), and so forth. To interpret the effects of a predictor, its β parameter is exponentiated, resulting in a hazard ratio (due to the use of the cloglog link). When using the logit link, exponentiating a β parameter results in an odds ratio.

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; Allison (2010)). When there is clustering in the data, as in the case of a small- N design with repeated measurements, the parameters of a discrete-time hazard model can be

255 estimated using population-averaged methods (e.g., Generalized Estimating Equations),
 256 and Bayesian or frequentist generalized linear mixed models (Allison, 2010).

257 In general, there are three assumptions one can make or relax when adding
 258 experimental predictor variables: The linearity assumption for continuous predictors (the
 259 effect of a 1 unit change is the same anywhere on the scale), the additivity assumption
 260 (predictors do not interact), and the proportionality assumption (predictors do not interact
 261 with TIME).

262 In this tutorial we will fit four Bayesian multilevel models (i.e., generalized linear
 263 mixed models) to the person-trial-bin oriented data set that we created in Tutorial 1. We
 264 select the analysis range (200,600] and the cloglog link. The 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))
```

265 **Prior distributions**

266 To get the posterior distribution of each parameters given the data, we need to
 267 specify the prior distribution of each parameter. The middle column of Figure 4 shows
 268 seven examples of prior distributions on the logit and/or cloglog scales.

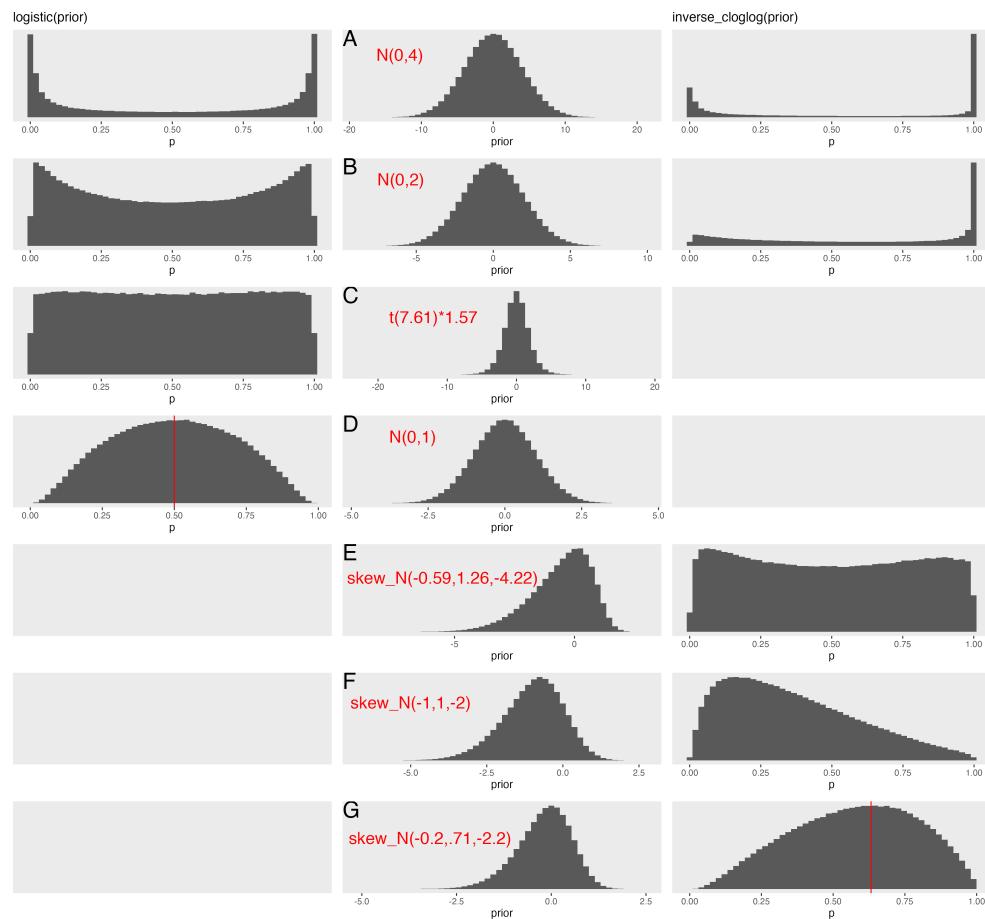


Figure 4. Prior distributions on the logit and/or cloglog scales (middle column), and their implications on the probability scale after applying the inverse-logit (or logistic) transformation (left column), and the inverse-cloglog transformation (right column).

269 While a normal distribution with relatively large variance is often used as a weakly
 270 informative prior for continuous dependent variables, rows A and B in Figure 3 show that
 271 specifying such distributions on the logit and cloglog scales leads to rather informative

272 distributions on the original probability (i.e., discrete-time hazard) scale, as most mass is
273 pushed to probabilities of 0 and 1.

274 To gain a sense of what prior *logit* values would approximate a uniform distribution
275 on the probability scale, Kurz (2023a) simulated a large number of draws from the
276 Uniform(0,1) distribution, converted those draws to the log-odds metric, and fitted a
277 Student's t distribution. Row C in Figure 4 shows that using a t-distribution with 7.61
278 degrees of freedom and a scale parameter of 1.57 as a prior on the logit scale, approximates
279 a uniform distribution on the probability scale. According to Kurz (2023a), such a prior
280 might be a good prior for the intercept(s) in a logit-hazard model, while the N(0,1) prior in
281 row D might be a good prior for the beta parameters in a logit-hazard model, as it gently
282 regularizes p towards .5 (i.e., a zero effect on the logit scale).

283 To gain a sense of what prior *cloglog* values would approximate a uniform distribution
284 on the hazard probability scale, we followed Kurz's approach and simulated a large number
285 of draws from the Uniform(0,1) distribution, converted them to the cloglog metric, and
286 fitted a skew-normal model (due to the asymmetry of the cloglog link function). Row E
287 shows that using a skew-normal distribution with a mean of -0.59, a standard deviation of
288 1.26, and a skewness of -4.22 as a prior on the cloglog scale, approximates a uniform
289 distribution on the probability scale. However, because hazard values below .5 are more
290 likely in RT studies, using a skew-normal distribution with a mean of -1, a standard
291 deviation of 1, and a skewness of -2 as a prior on the cloglog scale (row F), might be a good
292 weakly informative prior for the intercept(s) in a cloglog-hazard model. A skew-normal
293 distribution with a mean of -0.2, a standard deviation of 0.71, and a skewness of -2.2 might
294 be a good weakly informative prior for the beta parameters in a cloglog-hazard model as it
295 gently regularizes p towards .6321 (i.e., a zero effect on the cloglog scale).

296 Model 1: A general specification of TIME, and main effects of congruency and
 297 trial number

298 For the first model, we use a general specification of TIME (i.e., one intercept per
 299 time bin) for the baseline condition (blank prime), and assume that the effects of
 300 prime-target congruency and trial number are proportional and additive, and that the
 301 effect of trial number is linear. Before we fit model 1, we remove unnecessary columns from
 302 the data, and specify our priors. In the code of Tutorial 2, this is accomplished as follows.

```
# remove unnecessary columns before fitting a model
M1_data <- ptb_data %>% select(-c(bl,tr,trial,period, period_9,d9))

# Specify priors
priors_M1 <- c(
  set_prior("skew_normal(-0.2,0.71,-2.2)", class = "b"),
  set_prior("skew_normal(-1,1,-2)", class = "b", coef = "d6"),
  set_prior("skew_normal(-1,1,-2)", class = "b", coef = "d7"),
  set_prior("skew_normal(-1,1,-2)", class = "b", coef = "d8"),
  set_prior("skew_normal(-1,1,-2)", class = "b", coef = "d10"),
  set_prior("skew_normal(-1,1,-2)", class = "b", coef = "d11"),
  set_prior("skew_normal(-1,1,-2)", class = "b", coef = "d12"),
  set_prior("skew_normal(-1,1,-2)", class = "b", coef = "d13"),
  set_prior("skew_normal(-1,1,-2)", class = "b", coef = "d14"),
  set_prior("skew_normal(-1,1,-2)", class = "b", coef = "d15"),
  set_prior("skew_normal(-1,1,-2)", class = "b", coef = "Intercept"),
  set_prior("normal(0, 1)", class = "sd"),
  set_prior("lkj(2)", class = "cor")
)
```

303 We can now estimate our first Bayesian regression model, 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")
```

304 Estimating model M1 took about 70 minutes on a MacBook Pro (Sonoma 14.6.1 OS,
 305 18GB Memory, M3 Pro Chip).

306 **Model 2: A polynomial specification of TIME, and main effects of congruency
 307 and trial number**

308 For the second model, we use a third-order polynomial specification of TIME for the
 309 baseline condition (blank prime), and again assume that the effects of prime-target
 310 congruency and trial number are proportional and additive, and that the effect of trial
 311 number is linear. We first remove unnecessary columns and specify our priors.

```
# remove unnecessary columns
M2_data <- ptb_data %>% select(-c(bl,tr,trial,period, d6, d7, d8, d9, d10, d11, d12, d13, d14, d15))

# Specify priors
priors_M2 <- c(
  set_prior("skew_normal(-0.2,0.71,-2.2)", class = "b"),
  set_prior("skew_normal(-1,1,-2)", class = "b", coef = "Intercept"),
  set_prior("normal(0, 1)", class = "sd"),
  set_prior("lkj(2)", class = "cor")
)
```

312 Now we can fit model 2.

```
plan(multicore)

model_M2 <-
  brm(data = M2_data,
    family = binomial(link="cloglog"),
```

```

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),
prior = priors_M2,
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_M2")

```

313 Estimating model M2 took about 144 minutes.

314 **Model 3: A polynomial specification of TIME, and relaxing the proportionality
315 assumption**

316 For the third model, we use a third-order polynomial specification of TIME for the
317 baseline condition (blank prime), and relax the proportionality assumption for the
318 predictor variables congruency (variable “condition”) and trial number (variable “trial_c”).
319 We use the same data set and priors as for model 2.

```

M3_data <- M2_data
priors_M3 <- priors_M2
plan(multicore)

model_M3 <-
  brm(data = M3_data,
       family = binomial(link="cloglog"),
       event | trials(1) ~ 0 + Intercept + # Note that duplicate terms in the model formula are ignored
              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),
prior = priors_M3,
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_M3")

```

320 Estimating model M3 took about 268 minutes.

321 **Model 4: A polynomial specification of TIME, and relaxing all three
322 assumptions**

323 Based on previous work (Panis, 2020; Panis, Moran, et al., 2020; Panis & Schmidt,
324 2022; Panis et al., 2017; Panis & Wagemans, 2009) and because cognition is likely the
325 behavior of a non-linear dynamical system [ref], we relax all three assumptions in model 4.
326 We use the same data set and priors as for model 2.

```

M4_data <- M2_data
priors_M4 <- priors_M2
plan(multicore)

model_M4 <-
  brm(data = M4_data,
       family = binomial(link="cloglog"),
       event | trials(1) ~ 0 + Intercept + # Note that duplicate terms in the model formula are ignored
              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),
prior = priors_M4,
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_M4")

```

327 Estimating model M4 took about 8 hours.

328 **Compare the models.**

329 We can compare the four models using the Widely Applicable Information Criterion
 330 (WAIC) and Leave-One-Out (LOO) cross-validation, and look at model weights (Kurz,
 331 2023a; McElreath, 2018).

```

model_M1 <- readRDS("../Tutorial_2_Bayesian/models/model_M1.rds")
model_M2 <- readRDS("../Tutorial_2_Bayesian/models/model_M2.rds")
model_M3 <- readRDS("../Tutorial_2_Bayesian/models/model_M3.rds")
model_M4 <- readRDS("../Tutorial_2_Bayesian/models/model_M4.rds")

#model_M1 <- add_criterion(model_M1, c("loo", "waic"))
#model_M2 <- add_criterion(model_M2, c("loo", "waic"))
#model_M3 <- add_criterion(model_M3, c("loo", "waic"))
#model_M4 <- add_criterion(model_M4, c("loo", "waic"))

loo_compare(model_M1, model_M2, model_M3, model_M4, criterion = "loo") %>% print(simplify = F)

```

	elpd_diff	se_diff	elpd_loo	se_elpd_loo	p_loo	se_p_loo	looic
332 ##	0.0	0.0	-5094.4	62.9	127.6	4.2	10188.9
333 ## model_M4	-21.0	10.1	-5115.4	62.5	77.3	2.5	10230.8
334 ## model_M3	-254.9	24.4	-5349.3	64.8	72.7	1.7	10698.6
335 ## model_M1	-256.8	23.6	-5351.2	64.8	39.7	1.0	10702.5
336 ## model_M2	125.8						
337 ##	125.1						
338 ## model_M4	129.6						
339 ## model_M3	129.7						
340 ## model_M1							
341 ## model_M2							

```
loo_compare(model_M1, model_M2, model_M3, model_M4, criterion = "waic") %>% print(simplify = F)
```

```
342 ##          elpd_diff se_diff elpd_waic se_elpd_waic p_waic  se_p_waic waic
343 ## model_M4      0.0     0.0 -5092.8    62.9     125.9    4.0   10185.5
344 ## model_M3   -22.1    10.0 -5114.9    62.5     76.8     2.4   10229.7
345 ## model_M1  -256.3    24.3 -5349.1    64.8     72.5     1.6   10698.2
346 ## model_M2  -258.4    23.6 -5351.2    64.8     39.6     1.0   10702.4
347 ##          se_waic
348 ## model_M4   125.8
349 ## model_M3   125.1
350 ## model_M1   129.6
351 ## model_M2   129.7
```

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

```
352 ## model_M1 model_M2 model_M3 model_M4
353 ##          0     0     0     1
```

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

```
354 ## model_M1 model_M2 model_M3 model_M4
355 ##          0     0     0     1
```

356 Clearly, both weighting schemes prefer model M4.

357 **Plot congruency effects and subject-specific fits for model M4.**

358 Figure 5 shows the effects of congruent and incongruent primes relative to neutral
 359 primes, for each time bin in trial number 1000 for the selected model.

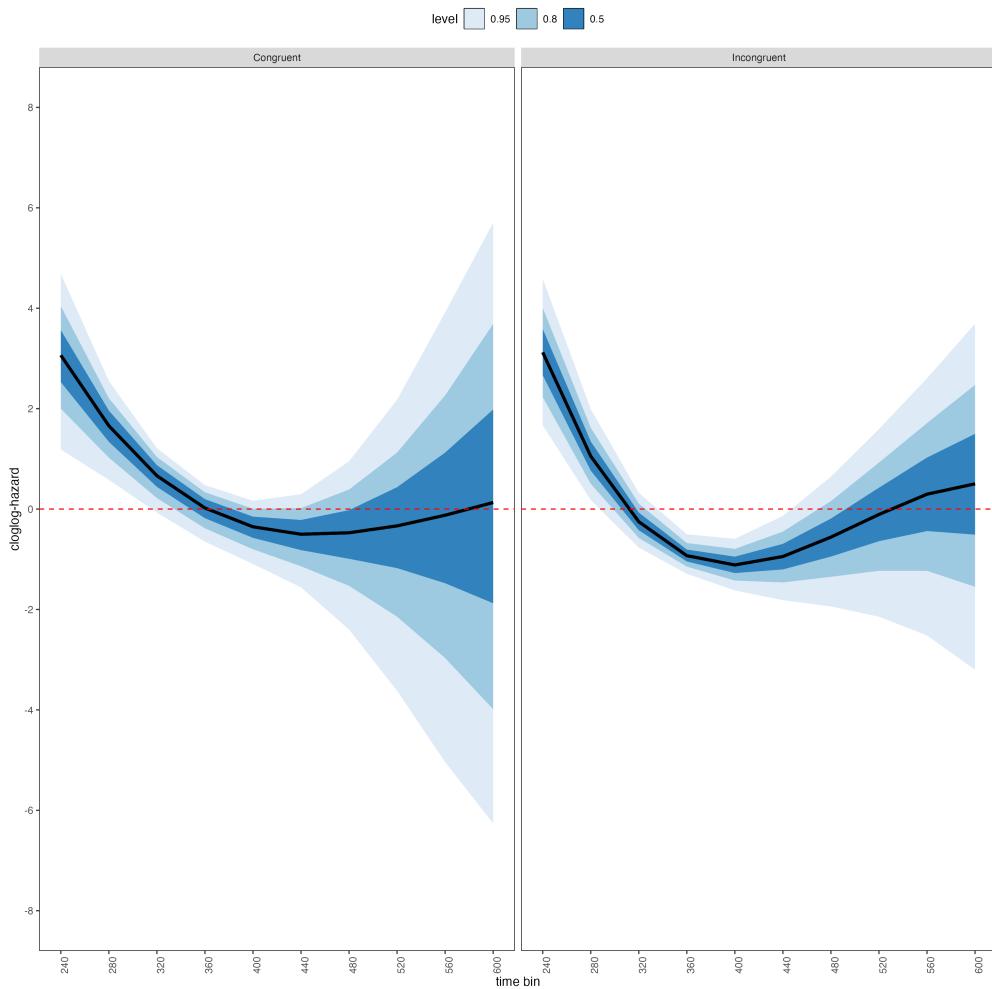


Figure 5. 50/80/95 percentile intervals of the draws from the posterior distributions representing the effect of congruent and incongruent primes relative to neutral primes in trial number 1000.

360 Figure 6 shows the model-based hazard functions for each prime type for participant
 361 6, in trial 500, 1000, and 1500.

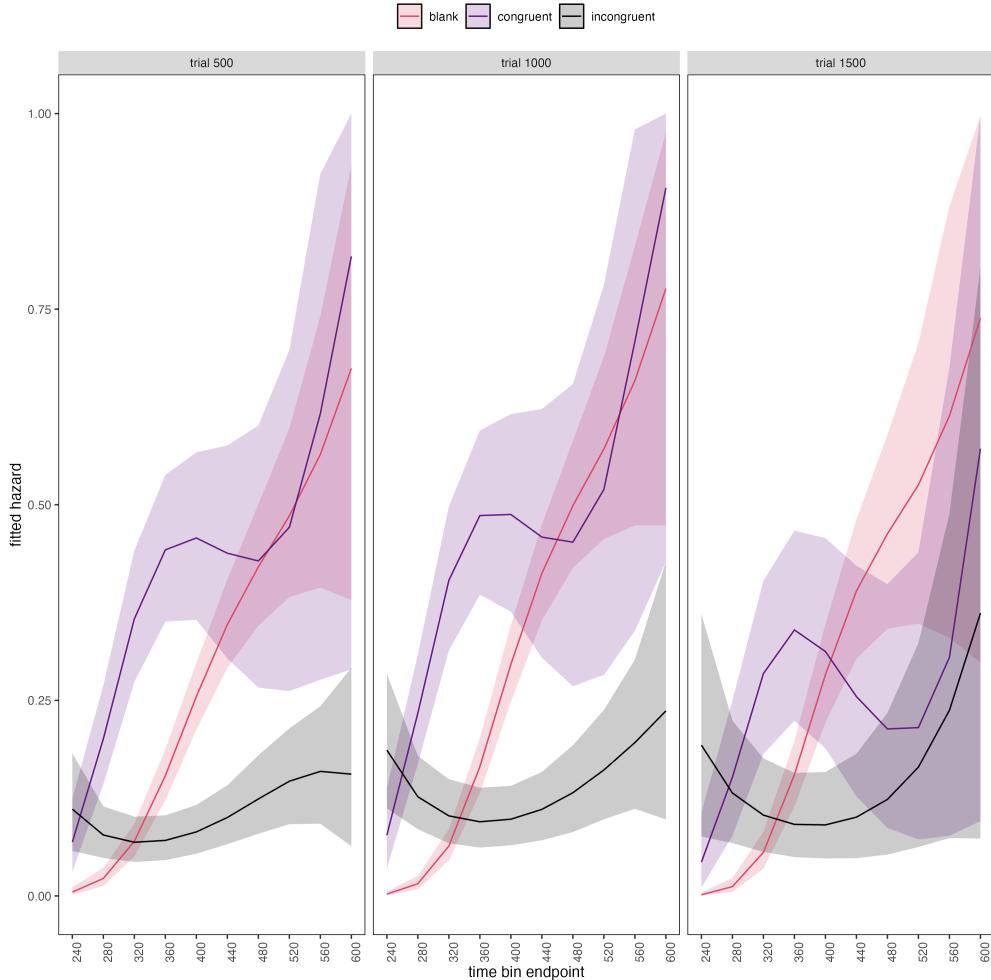


Figure 6. Model-based hazard functions for participant 6 in trial 500, 1000, and 1500.

362

Tutorial 3: Fitting Frequentist hazard models

363

In this third tutorial we illustrate how to fit a multilevel hazard regression model in

364

the frequentist framework, for the data set used in the first tutorial. For illustration

365

purposes, we only fitted model M3 using the function `glmer()` from the package `lme4`.

```
model_M3_f <- glmer(event ~ 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) +
```

```

(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),

# control parameters, data set, and complementary log-log link function
control = glmerControl(optimizer = c("nlminbwrap"),
                        optCtrl = list(maxfun=10000000)),
data=M3_data,
family=binomial(link="cloglog"))

```

366 In Figure 7 we compare the parameter estimates of model M3 from brms() with those
 367 of glmer().

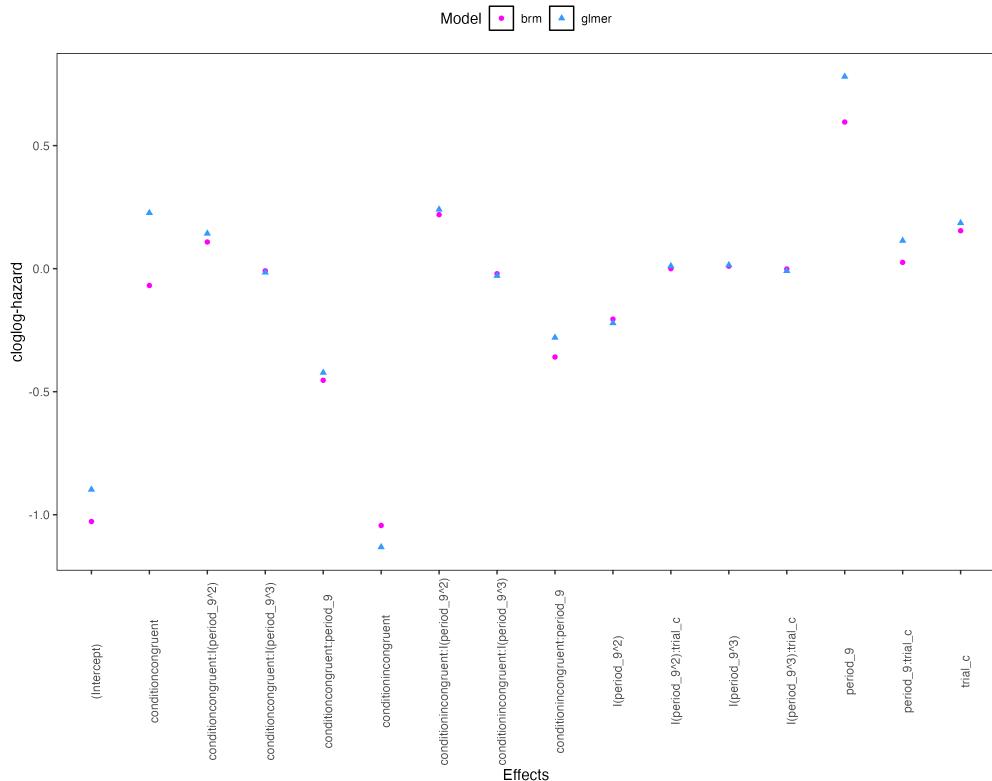


Figure 7. Parameter estimates for model M3 from brms() and glmer().

368 Figure 7 confirms that the parameter estimates from both Bayesian and frequentist

369 models are pretty similar. However, the random effects structure of model M3 was already
370 too complex for the frequentist model as it did not converge and resulted in a singular fit.
371 This is of course one of the reasons why Bayesian modeling has become so popular in the
372 last years.

373 **Tutorial 4: Calculating descriptive statistics when there are two independent
374 variables**

375 In this final tutorial we illustrate how to calculate and plot the descriptive statistics
376 for the full data set of Experiment 1 of Panis and Schmidt (2016), which includes two
377 independent variables: mask type and prime type. As we use the same functional
378 programming approach as in tutorial 1, we simply present the sample-based functions for
379 participant 6 in Figure 8. Note the negative compatibility effect in the hazard and
380 conditional accuracy functions when a (relevant, irrelevant, or lines) mask is present.



Figure 8. Estimated 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).

381

Discussion

382 We noticed that many researchers are still reluctant to abandon analysis-of-variance
 383 and switch to event history analysis when analyzing time-to-event data. By providing this
 384 tutorial, we hope that researchers will start using hazard analysis more often, due to the
 385 many advantages described below. While we focus here on within-subject, factorial,
 386 small- N designs, it is important to realize that event history analysis can be applied to
 387 other designs as well (large- N designs with only one measurement per subject,
 388 between-subject designs, etc.).

389 **Advantages of hazard analysis**

390 Statisticians and mathematical psychologists recommend focusing on the hazard
391 function when analyzing time-to-event data for various reasons. First, as discussed by
392 Holden, Van Orden, and Turvey (2009), “probability density functions can appear nearly
393 identical, both statistically and to the naked eye, and yet are clearly different on the basis
394 of their hazard functions (but not vice versa). Hazard functions are thus more diagnostic
395 than density functions” (p. 331) when one is interested in studying the detailed shape of a
396 RT distribution (see also Figure 1 in . . .).

397 Second, because RT distributions may differ from one another in multiple ways,
398 Townsend (1990) developed a dominance hierarchy of statistical differences between two
399 arbitrary distributions A and B. For example, if $F_A(t) > F_B(t)$ for all t , then both
400 cumulative distribution functions are said to show a complete ordering. Townsend (1990)
401 showed that a complete ordering on the hazard functions $-\lambda_A(t) > \lambda_B(t)$ for all t —
402 implies a complete ordering on both the cumulative distribution and survivor functions
403 $-F_A(t) > F_B(t)$ and $S_A(t) < S_B(t)$ —which in turn implies an ordering on the mean
404 latencies—mean A < mean B. In contrast, an ordering on two means does *not* imply a
405 complete ordering on the corresponding $F(t)$ and $S(t)$ functions, and a complete ordering
406 on these latter functions does *not* imply a complete ordering on the corresponding hazard
407 functions. This means that stronger conclusions can be drawn from data when comparing
408 the hazard functions using EHA. For example, when mean A < mean B, the hazard
409 functions might show a complete ordering (i.e., for all t), a partial ordering (e.g., only for t
410 > 300 ms, or only for $t < 500$ ms), or they may cross each other one or more times. Thus,
411 because the discrete-time hazard function identifies unique information about event
412 occurrence in each bin, while $F(t)$ accumulates the complements of the hazard estimates
413 from the current and previous bins $-S(t) = P(RT > t) =$
414 $[1-h(t)].[1-h(t-1)].[1-h(t-2)]\dots[1-h(1)] -$, instead of using delta-plots – differences in

415 quantiles from $F(t)^{-1}$ – one can simply plot delta- $h(t)$ functions (see ref).

416 Third, EHA does not discard right-censored observations when estimating hazard
417 functions, that is, trials for which we do not observe a response during the data collection
418 period in a trial so that we only know that the RT must be larger than some value (i.e., the
419 response deadline). This is important because although a few right-censored observations
420 are inevitable in most RT tasks, a lot of right-censored observations are expected in
421 experiments on masking, the attentional blink, and so forth. In other words, by using EHA
422 you can analyze RT data from experiments that typically do not measure response times.
423 As a result, EHA can also deal with long RTs in experiments without a response deadline,
424 which are typically treated as outliers and are discarded before calculating a mean. This
425 orthodox procedure can lead to a sampling bias, however, which results in underestimation
426 of the mean. By introducing a fixed censoring time for all trials at the end of the analysis
427 time window, trials with long RTs are not discarded but contribute to the risk set of each
428 bin.

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

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

⁴⁴¹ Schmidt, 2016).

⁴⁴² Individual differences

⁴⁴³ One important issue is that of possible individual differences in the overall location of
⁴⁴⁴ the distribution, and the time course of psychological effects. For example, when you wait
⁴⁴⁵ for a response of the participant on each trial, you allow the participant to have control
⁴⁴⁶ over the trial duration, and some participants might respond only when they are confident
⁴⁴⁷ that their emitted response will be correct. These issues can be avoided by introducing a
⁴⁴⁸ (relatively short) response deadline in each trial, e.g., 600 ms for simple detection tasks,
⁴⁴⁹ 1000 ms for more difficult discrimination tasks, or 2 s for tasks requiring extended
⁴⁵⁰ high-level processing. Because EHA can deal in a straightforward fashion with
⁴⁵¹ right-censored observations (i.e., trials without an observed response), introducing a
⁴⁵² response deadline is recommended when designing RT experiments. Furthermore,
⁴⁵³ introducing a response deadline and asking participants to respond before the deadline as
⁴⁵⁴ much as possible, will also lead to individual distributions that overlap in time, which is
⁴⁵⁵ important when selecting a common analysis time window when fitting hazard models.
⁴⁵⁶ But even when using a response deadline, participants can differ qualitatively in the effects
⁴⁵⁷ they display (refs). One way to deal with this is to describe and interpret the different
⁴⁵⁸ patterns. Another way is to run a clustering algorithm on the individual hazard estimates
⁴⁵⁹ across all conditions. The obtained dendrogram can then be used to identify (hopefully
⁴⁶⁰ big) clusters of participants that behave similarly, and to identify (hopefully small) clusters
⁴⁶¹ of participants with outlying behavioral patterns. One might then exclude the outlying
⁴⁶² participants before fitting a hazard model.

⁴⁶³ Limitations

⁴⁶⁴ Compared to the orthodox method – comparing means with ANOVA –, the most
⁴⁶⁵ important limitation of multilevel hazard modeling is that it might take a long time to

466 estimate the parameters. Another issue that might initially look as a limitation is that you
467 need a relatively large number of trials per condition to estimate the hazard function with
468 high temporal resolution. However, as nature does not reveal itself easily, obtaining insight
469 into behavioral dynamics simply requires more data per condition than is usually collected
470 under the orthodox method. In general, there is a tradeoff between the number of trials per
471 condition and the temporal resolution (i.e., bin width) of the hazard function; We therefore
472 recommend to design as many trials as possible per experimental condition given the
473 available resources.

474 **Extensions**

475 The hazard models in this tutorial assume that there is one event of interest. For RT
476 data, this event constitutes a single transition between an “idle” state and a “responded”
477 state. However, in certain situations, more than one event of interest might exist. For
478 example, an individual might transition back and forth between a “healthy” state and a
479 “depression” state, before being absorbed in a final “death” state. When you have data on
480 the timing of these transitions, one can apply multi-state models which generalize survival
481 analysis to transitions between three or more states (refs). Also, the predictor variables in
482 this tutorial are time-invariant, i.e., their value did not change over the course of a trial.
483 Another extension is to include time-varying predictors, i.e., predictors whose value can
484 change across the time bins within a trial.

485 **Conclusions**

486 see paper...

487

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