1. **Random-effects Model**

As presented in Ouwens et al. (2010) [17] the random-effects model was defined as follows:

 (1)

In this formula, represents the underlying hazard rate in study for treatment at time point . The vectors are treatment-specific and reflect the parameters and of the “baseline” treatment in study . In our case study, everolimus was the “baseline” treatment in METEOR, CheckMate025, and RECORD-1; placebo was the “baseline” treatment in TARGET and sorafenib was the “baseline” treatment in AXIS. The vector reflects the study-specific difference in scale and shape of the log-hazard curve for treatment relative to the “baseline” treatment in study . In our case study, treatment corresponded to cabozantinib in METEOR, nivolumab in CheckMate025, placebo in RECORD-1, sorafenib in TARGET and axitinib in AXIS.

Estimation of model parameters of interest – baseline and effect vectors – was performed in Bayesian framework. The prior distributions as used for the parameters of the random-effects model were chosen non-informative as follows:

 was sampled from a non-informative bivariate normal distribution with 0 correlation. The same holded for the vector of treatment effect. A Wishart prior distribution was specified for the variance matrix , with the scale matrix and 2 the smallest degrees of freedom. In our experiment, the values of have been changed from to to accelerate the convergence rate. In fact, with the smaller variance, the algorithm did not converge with 100 000 iterations but we got a good convergence with 80 000 iterations once we increased the prior mean of . On the contrary, we did not meet any convergence issue with the fixed-effects model.