Figure 1. Schematic representation of the decision tree (upper part) and Markov model (lower part). The decision tree shows that patients initiating coumarin anticoagulant therapy could be treated by one of the two dosing algorithms with different chances of developing adverse events in each genotype group. For each genotype group a Markov model (M) was applied, which included the following health states: no event, thromboembolic event (stroke or transient ischemic attack), haemorrhagic event (intracranial or extra cranial haemorrhage), sequelae and death. After a thromboembolic or haemorrhagic event patients move to either 'no event (i.e., no sequelae)', 'sequelae' or 'death'.

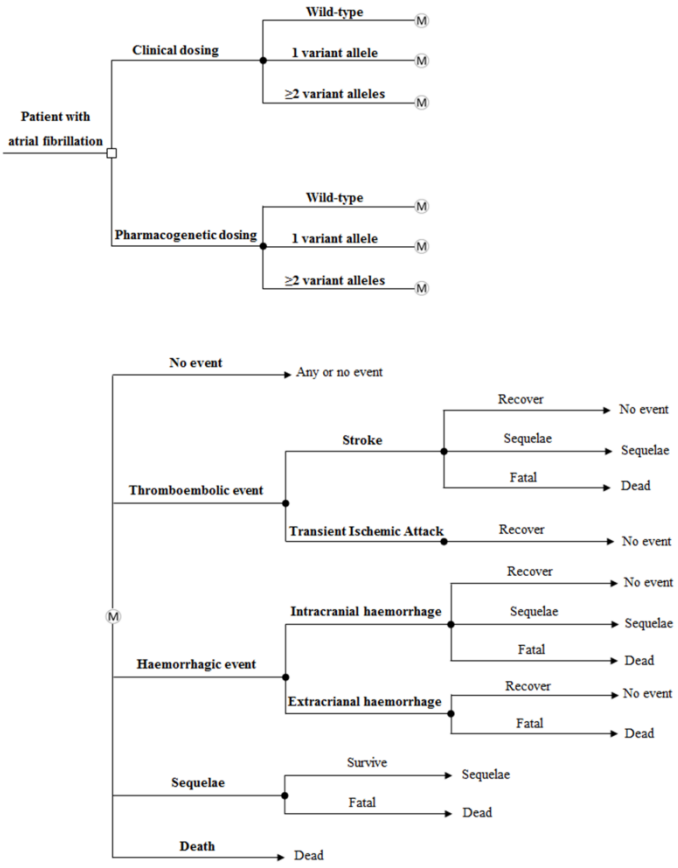

Figure 2. Mean difference (and 95% confidence intervals) in percentage time spent in different INR ranges during the first month between the pharmacogenetic arm (PGx) and clinical arm. a: phenprocoumon, b: acenocoumarol.

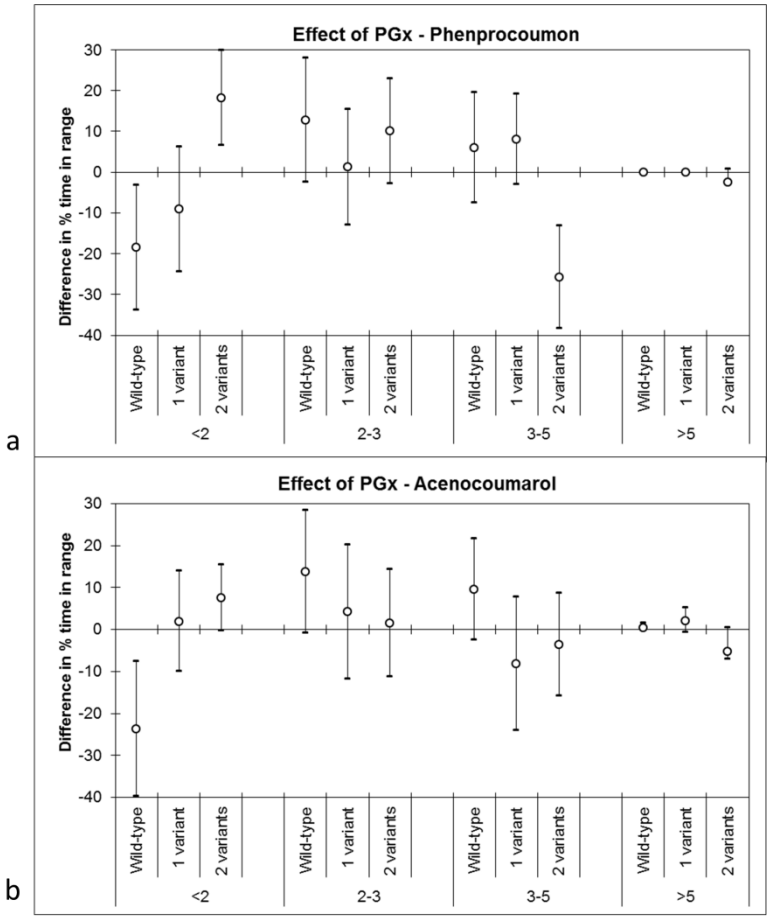

Figure 3. Tornado diagrams of the incremental cost-effectiveness ratios of pharmacogenetic dosing versus clinical dosing (excluding parameters regarding the effect of genotyping). a: phenprocoumon pharmacogenetic algorithm versus clinical algorithm, b: acenocoumarol pharmacogenetic algorithm versus clinical algorithm.

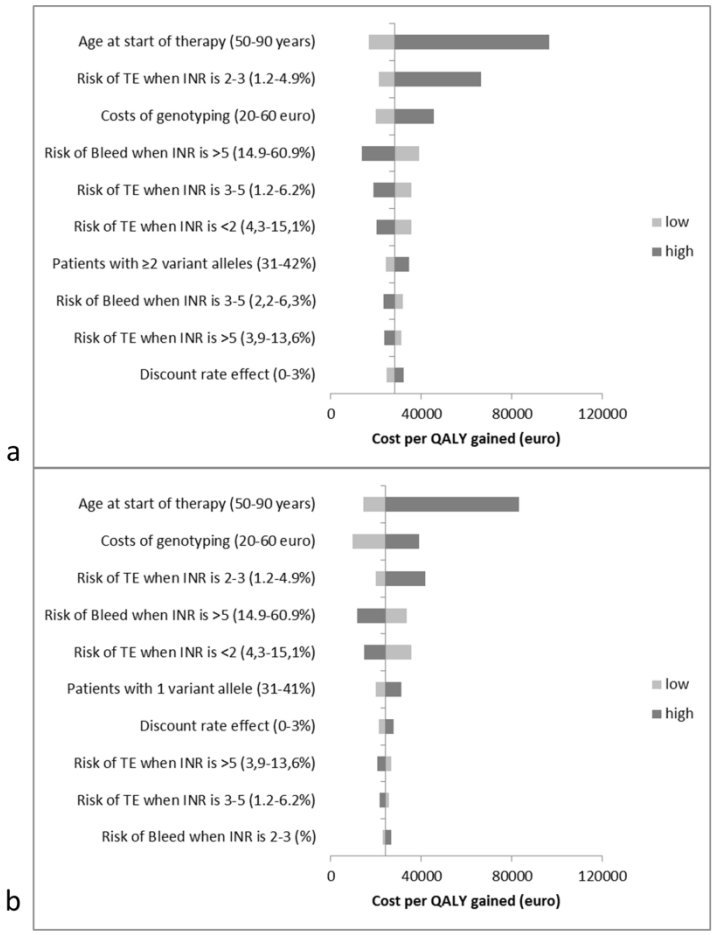

Figure 4. Scatter plot reflecting the uncertainty in the differences in costs and effectiveness between pharmacogenetic dosing and clinical dosing (based on probabilistic sensitivity analysis).

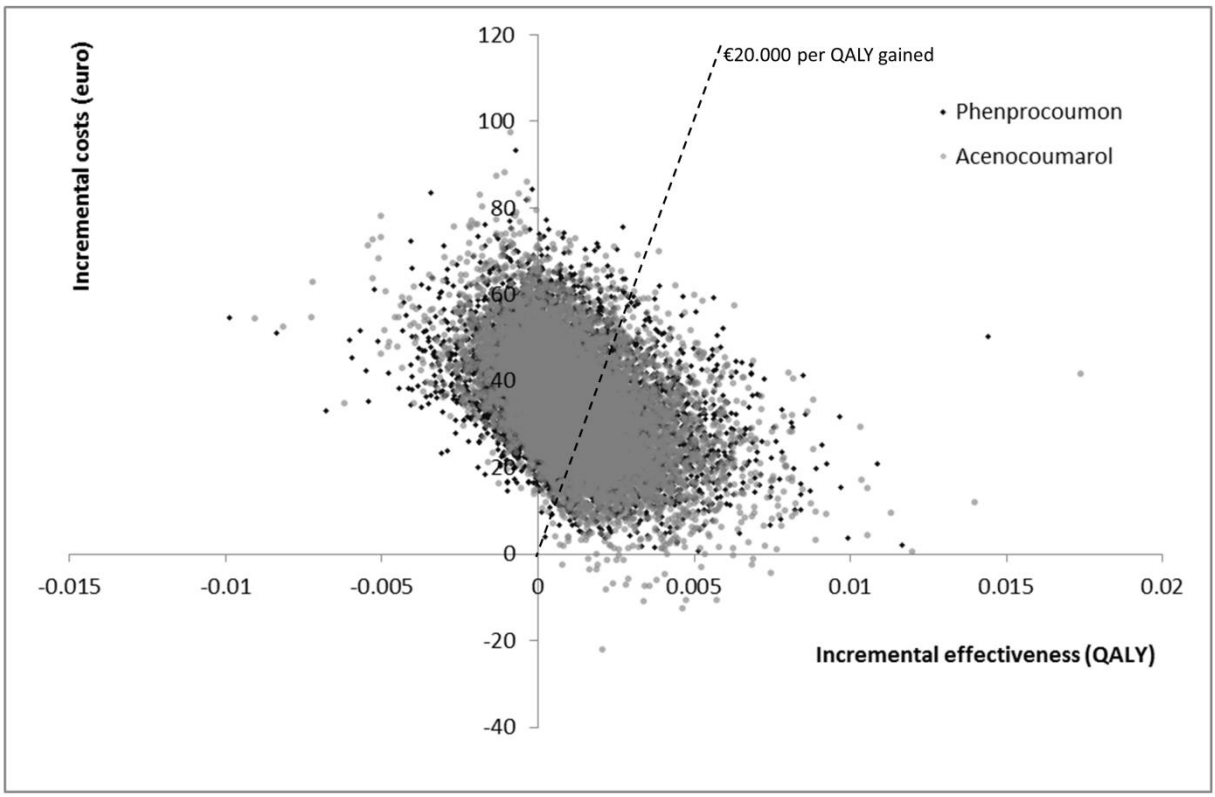

Figure 5. Cost-effectiveness acceptability curve for phenprocoumon pharmacogenetic dosing versus clinical dosing (a) and acenocoumarol pharmacogenetic dosing versus clinical dosing (b). This graph shows the chance that genotyping would be cost-effective given different willingness-to-pay thresholds.