

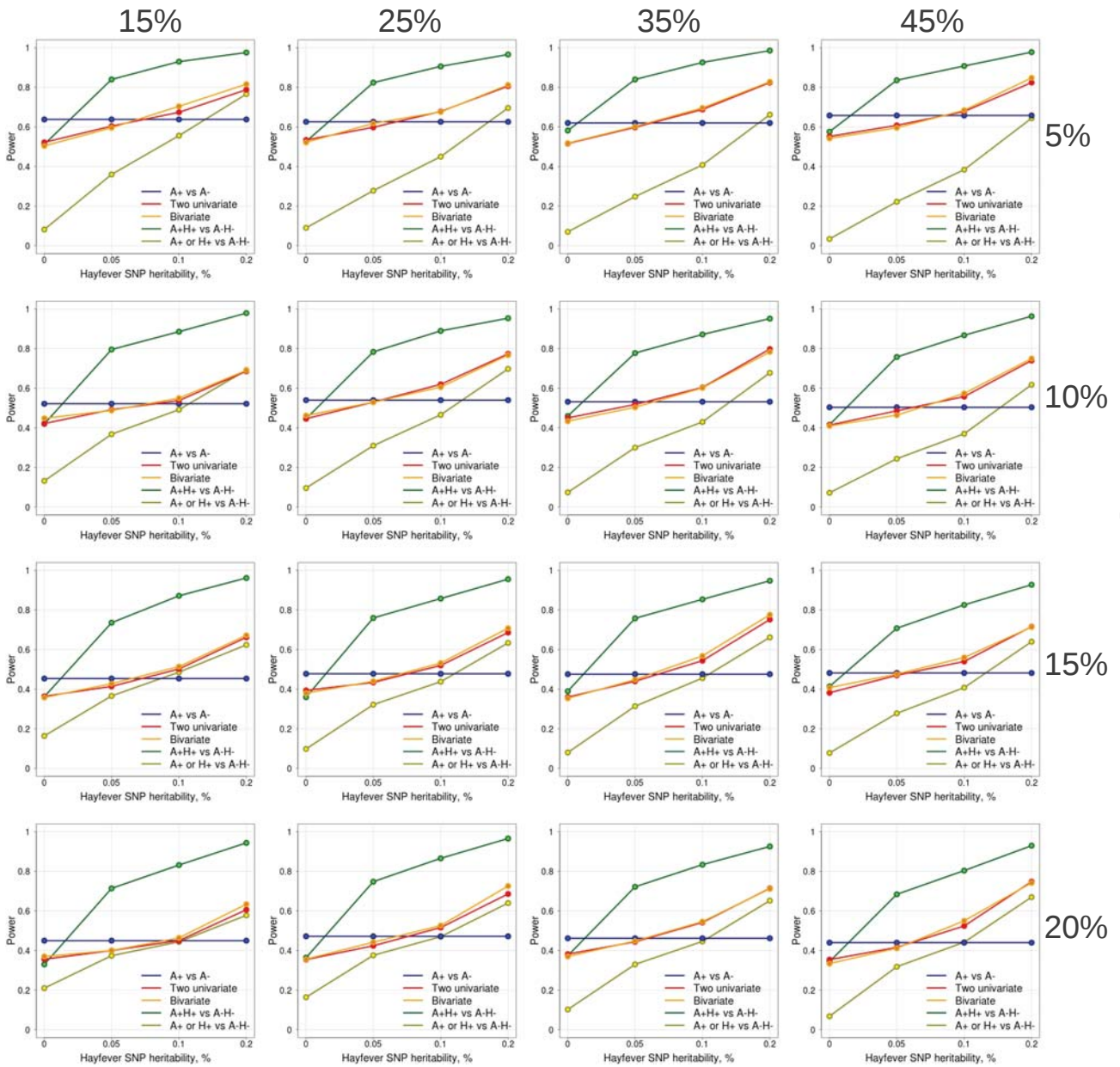
*Twin Research and Human Genetics*

**Improving the power to detect risk variants for allergic disease by defining case-control status based on both asthma and hay fever**

*Supplementary Material*

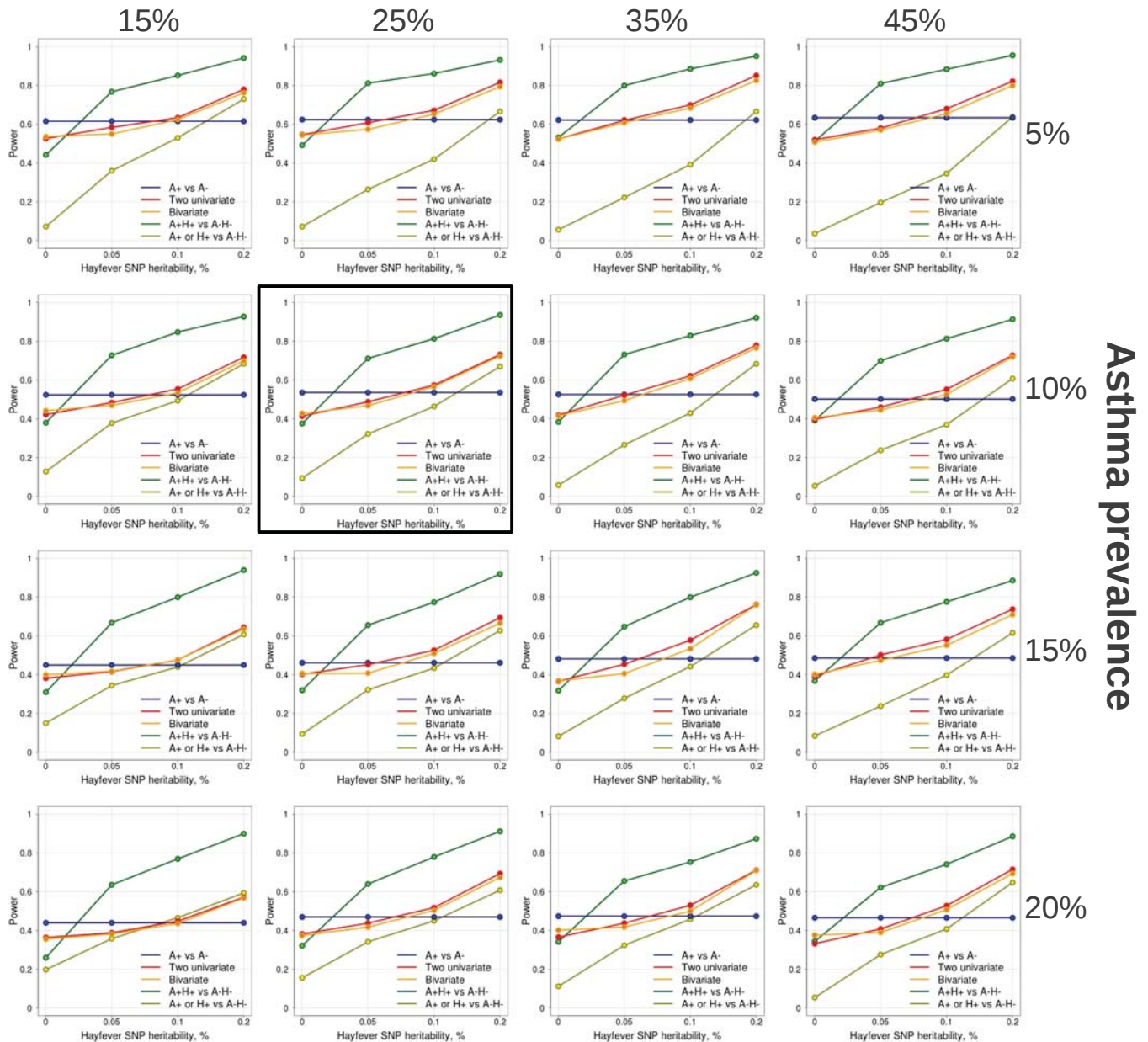
Manuel A.R. Ferreira

# Hay fever prevalence



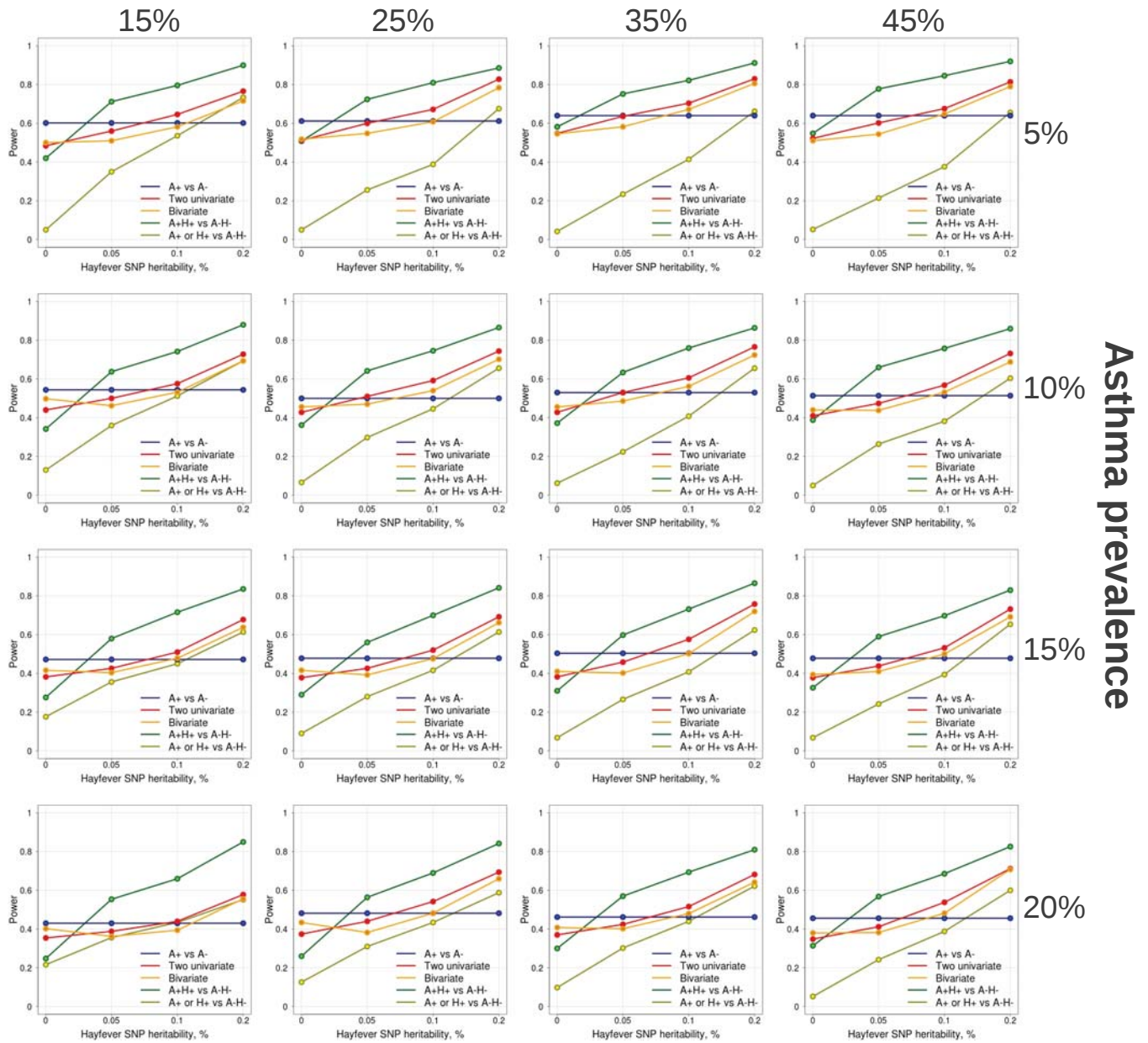
**Supplementary Figure S1. Impact of ascertainment strategy on the power to detect a risk variant shared between asthma and hay fever when the genetic correlation was 0.3. Power according to the study design used to ascertain samples for genotyping, hay fever prevalence and asthma prevalence.**

## Hay fever prevalence

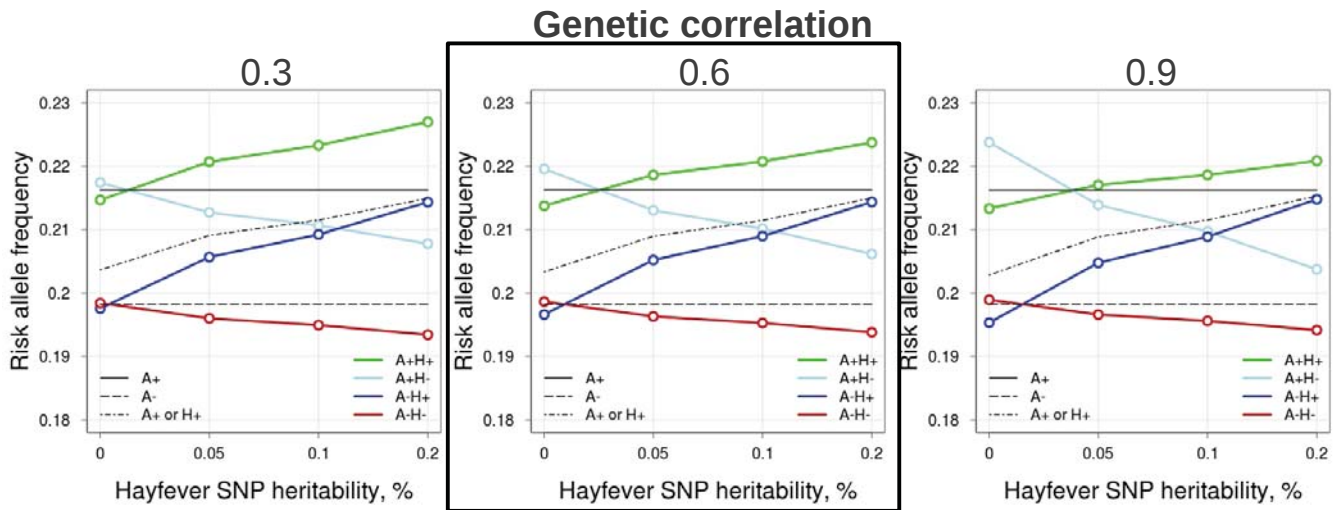


**Supplementary Figure S2. Impact of ascertainment strategy on the power to detect a risk variant shared between asthma and hay fever when the genetic correlation was 0.6. Power according to the study design used to ascertain samples for genotyping, hay fever prevalence and asthma prevalence. Plot shown in Figure 1A is highlighted by the black box.**

## Hay fever prevalence



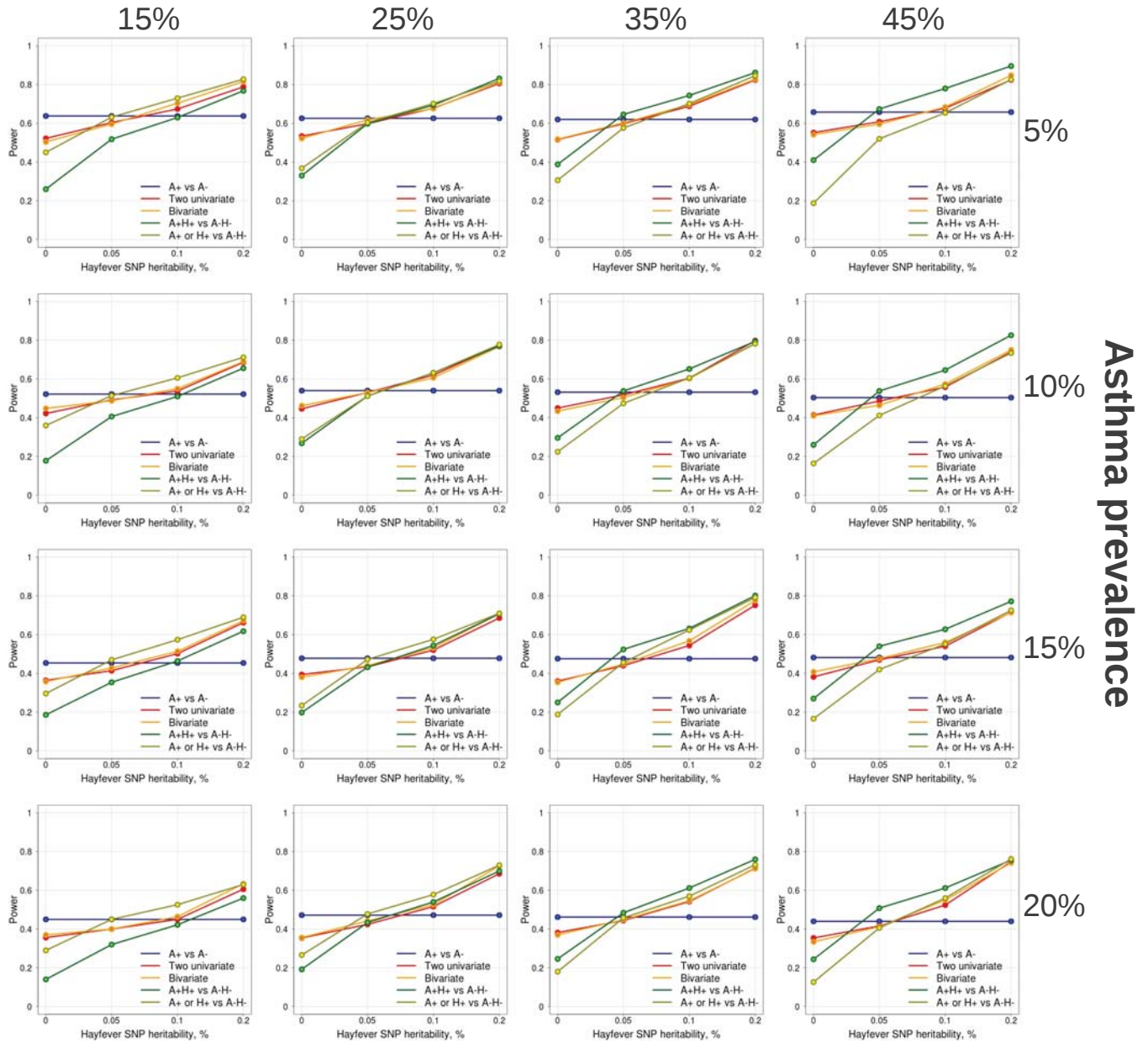
**Supplementary Figure S3. Impact of ascertainment strategy on the power to detect a risk variant shared between asthma and hay fever when the genetic correlation was 0.9. Power according to the study design used to ascertain samples for genotyping, hay fever prevalence and asthma prevalence.**



**Supplementary Figure S4.** Frequency of the SNP risk allele in the overall population in sub-groups of individuals defined by asthma and/or hay fever status, according to the genetic correlation between asthma and hay fever. Plot shown in Figure 1B is highlighted by the black box.

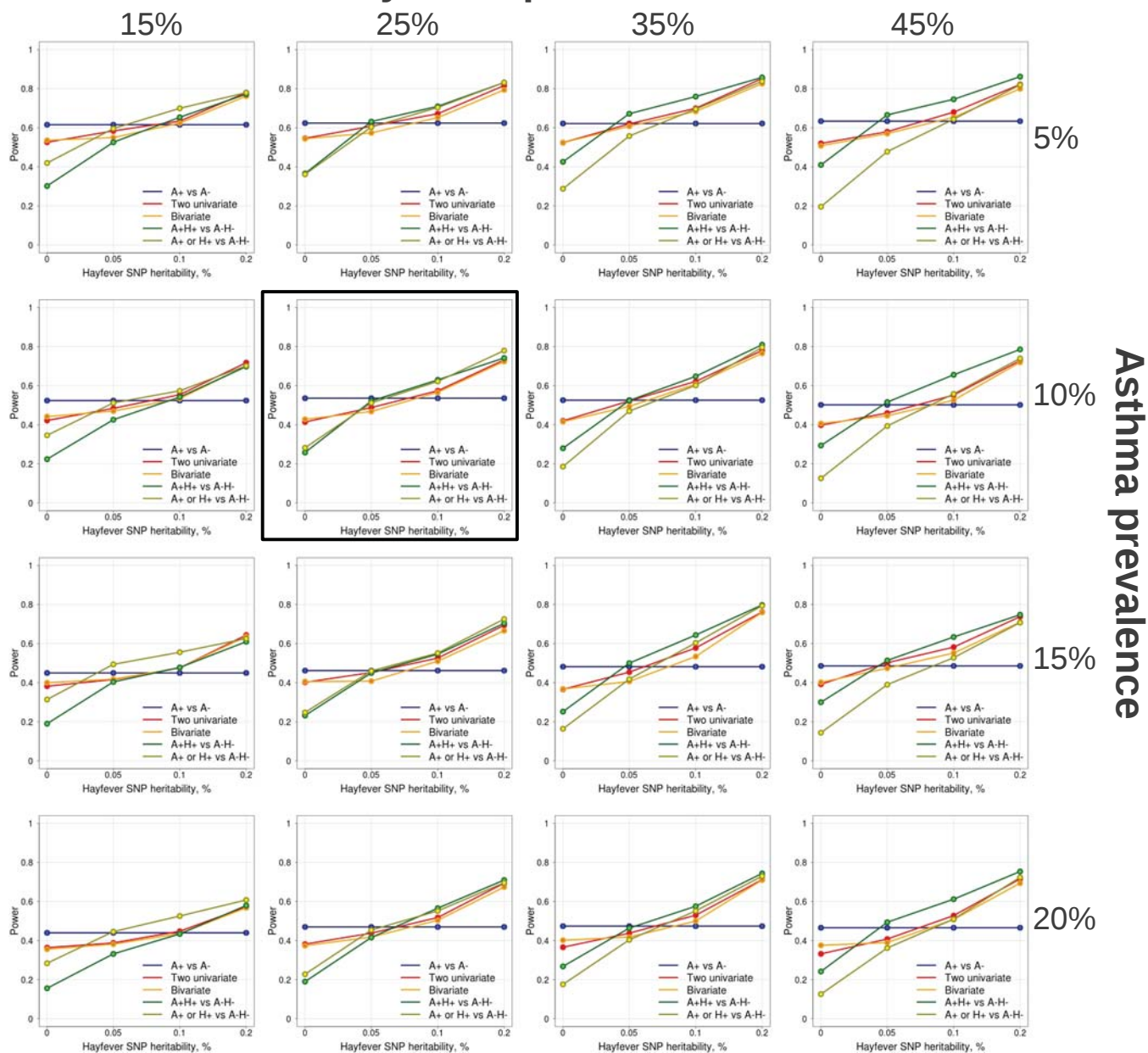


# Hay fever prevalence



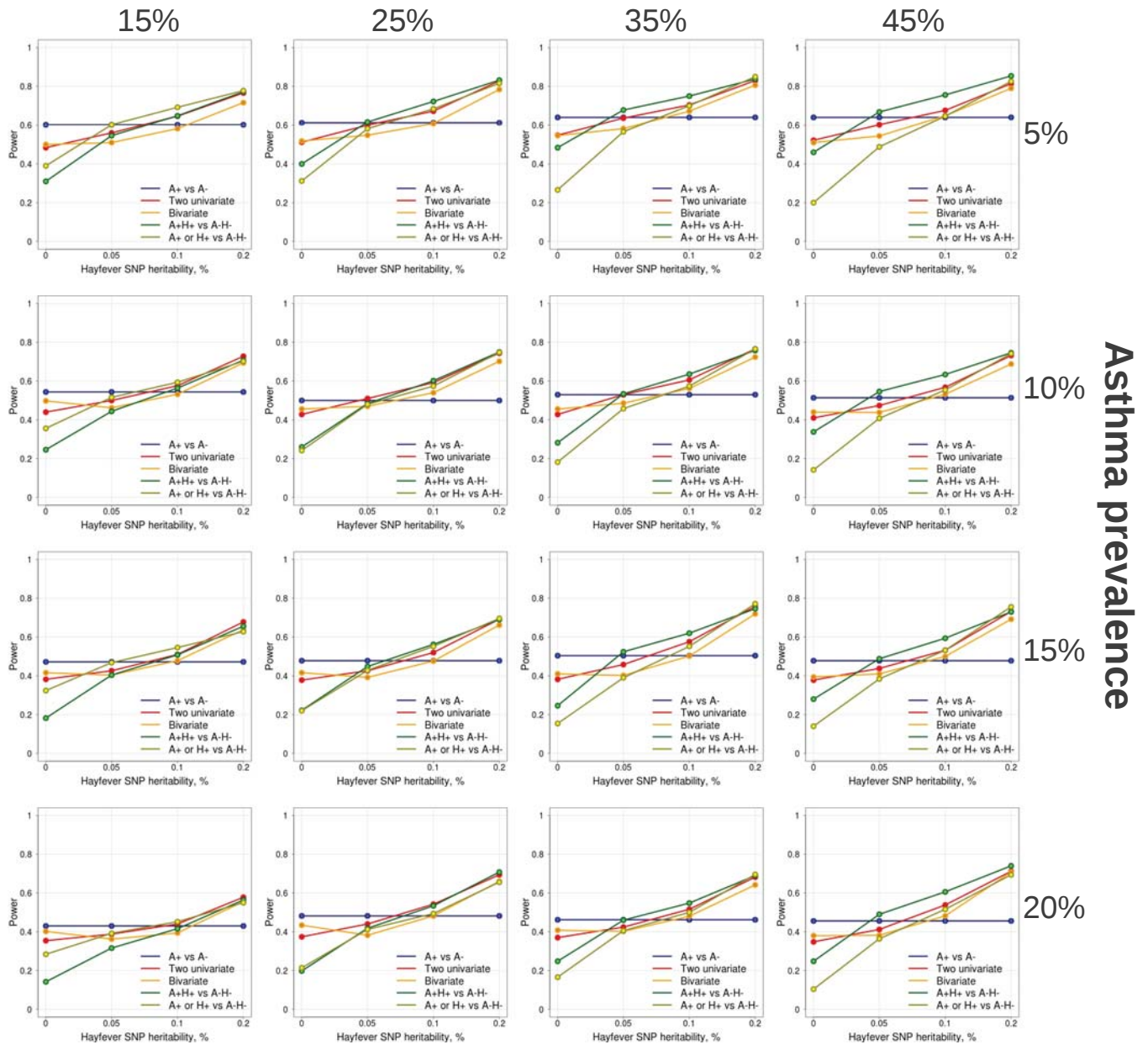
**Supplementary Figure S5. Impact of analytical strategy on the power to detect a risk variant shared between asthma and hay fever in an existing case-control GWAS of asthma when the genetic correlation was 0.3. Power according to the phenotype classification used to define case-control status, hay fever prevalence and asthma prevalence.**

## Hay fever prevalence



**Supplementary Figure S6. Impact of analytical strategy on the power to detect a risk variant shared between asthma and hay fever in an existing case-control GWAS of asthma when the genetic correlation was 0.6.** Power according to the phenotype classification used to define case-control status, hay fever prevalence and asthma prevalence. Plot shown in Figure 2A is highlighted by the black box.

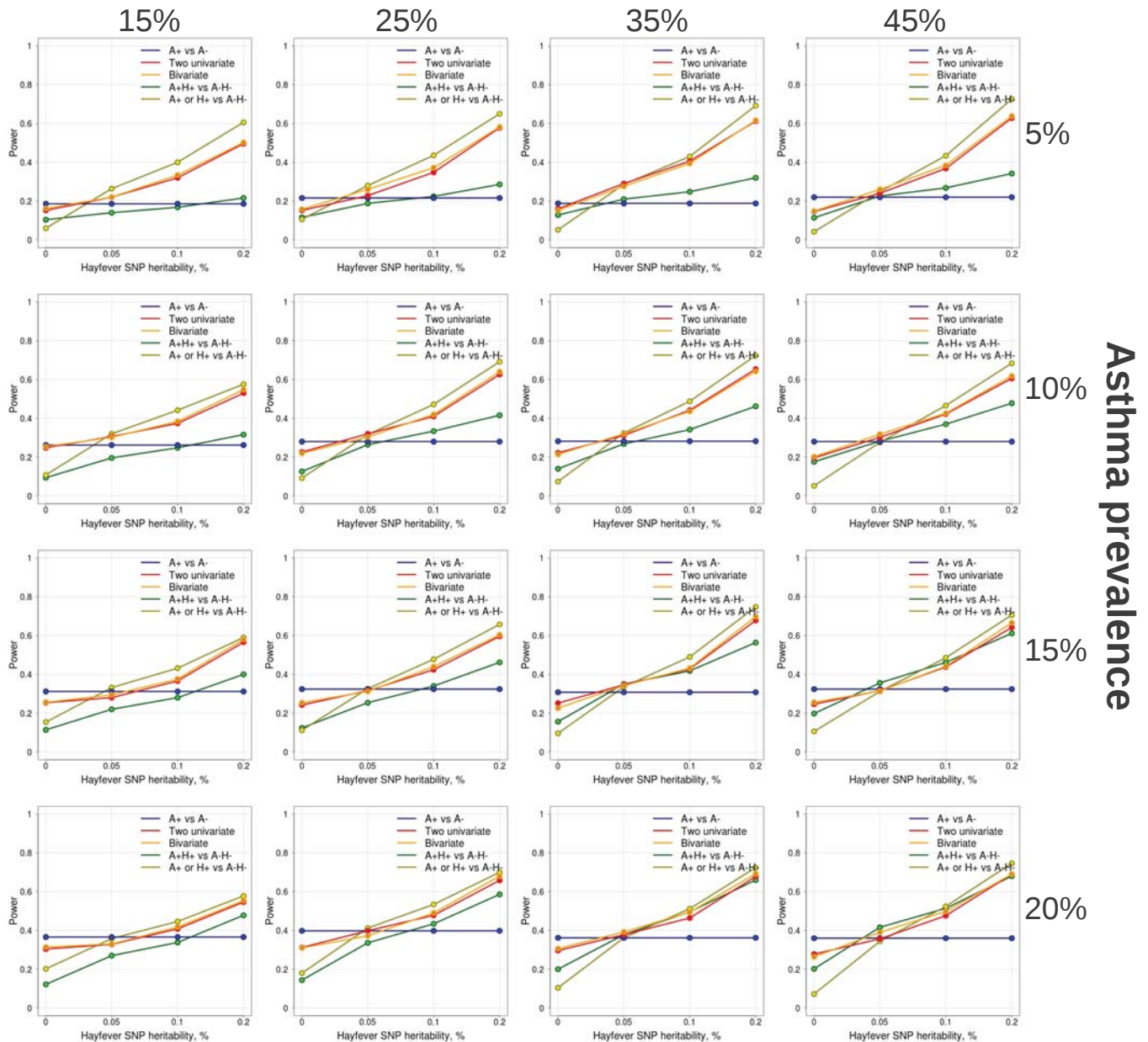
## Hay fever prevalence



**Supplementary Figure S7. Impact of analytical strategy on the power to detect a risk variant shared between asthma and hay fever in an existing case-control GWAS of asthma when the genetic correlation was 0.9. Power according to the phenotype classification used to define case-control status, hay fever prevalence and asthma prevalence.**

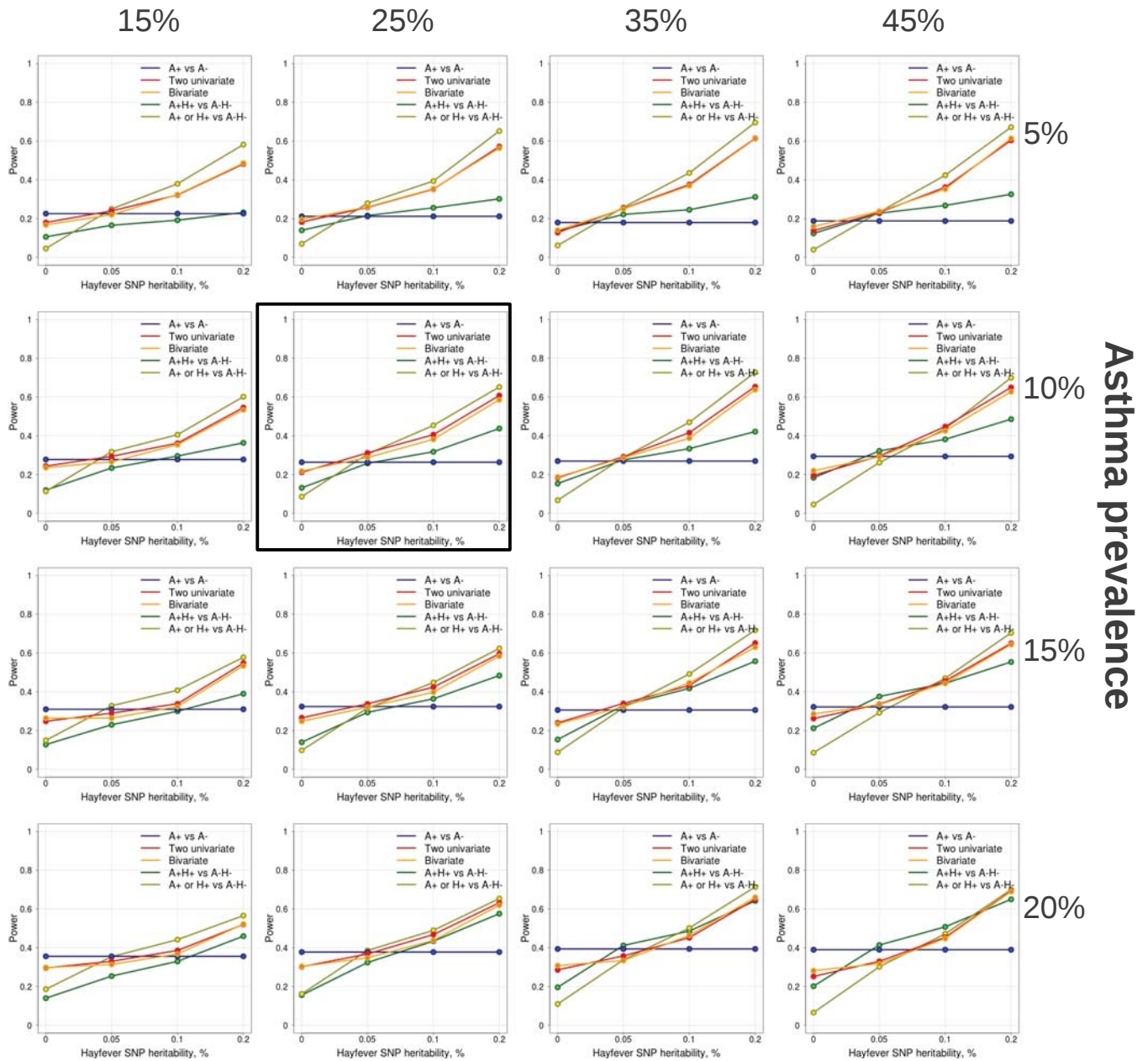


# Hay fever prevalence



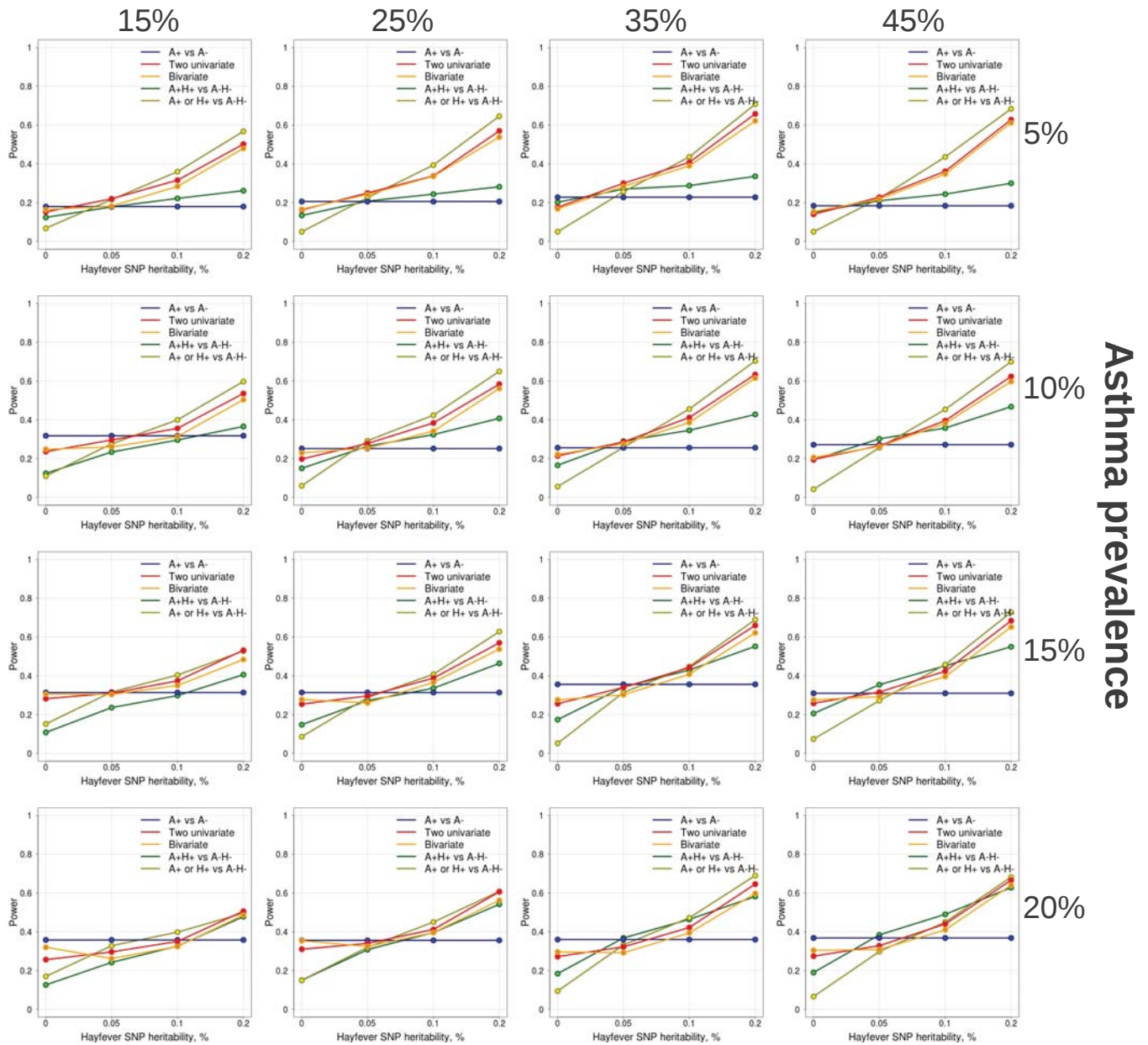
Supplementary Figure S8. Impact of analytical strategy on the power to detect a risk variant shared between asthma and hayfever in an existing cross-sectional GWAS when the genetic correlation was 0.3. Power according to the phenotype classification used to define case-control status, hay fever prevalence and asthma prevalence.

# Hay fever prevalence



**Supplementary Figure S9. Impact of analytical strategy on the power to detect a risk variant shared between asthma and hay fever in an existing cross-sectional GWAS when the genetic correlation was 0.6. Power according to the phenotype classification used to define case-control status, hay fever prevalence and asthma prevalence. Plot shown in Figure 3 is highlighted by the black box.**

# Hay fever prevalence



**Supplementary Figure S10. Impact of analytical strategy on the power to detect a risk variant shared between asthma and hay fever in an existing cross-sectional GWAS when the genetic correlation was 0.9. Power according to the phenotype classification used to define case-control status, hay fever prevalence and asthma prevalence.**