**File S1: Clustering and filtering traits**

We initially differentiated as many biological traits as unique names were given to the mapped disease or trait field in the original GWAS Catalog. However, it often occurs that different researchers studying the same trait publish their results using different trait names (*e.g*. “LDL” and “LDL levels”). In order to avoid working with duplicated or redundant traits, we clustered some of them on the basis of their common genetic background.

To do so, we first defined the following measure of similarity (*sim*):

where refers to the number of unique genes names found to be associated with a trait, or to two of them simultaneously. This measure ranges from 0 (no genes in common) to 1 (the same genetic background), as expected in the case where *trait1* equals *trait2*.

This let us build a similarity matrix with the pairwise similarities between traits. We only considered for clustering those traits sharing a high similarity , which represented the 98.85 quantile in similarity terms.

In order to avoid spurious associations, we were very restrictive and only clustered together traits which belonged to graphs where every vertex was connected to more than 50% of the remaining vertex. When this condition was not satisfied, the less connected node (*i.e.* with less edges) was removed recursively until the above condition was met or the graph was completely disconnected. When we found two vertices with the same degree (*i.e*. number of edges), that with the lowest measure of average similarity was removed. This algorithm also allowed disconnected nodes to be reconnected if the above conditions were satisfied. For the purpose of analysis, the new clustered traits replaced the original ones. After this clustering step, traits studied by less than three different PMIDs were also removed. Table S1 shows all traits clustered in this step, along with their *effect type*.

The database after processing and clustering included a total of 11,918 SNPs corresponding to 461 scientific studies for 59 human traits. It is worth mentioning that after the above preprocessing, clustering and filtering steps, the SNP/trait ratio and the PMID/trait ratio had been largely improved, the former from 26.3 to 202 SNP/trait and the later from 1.7 to 9.6 PMID/trait.

To simplify the representation of results, a second higher-level of clustering was carried out, with traits classified within functional domains similar to those used by Polderman *et al.* (2015). The 14 functional domains considered were: Appearance, Cancer, Cardiovascular, Dermatological, Endocrine, Gastrointestinal, Hematological, Immunological, Metabolic, Neurological, Ophthalmological, Psychiatric, Respiratory and Skeletal. Table S1 also shows traits and clusters organized by these functional categories.

**Additional processing steps**

For the analysis of the effect sizes, risk allele frequencies and heritability, we removed from the database scientific studies where the *effect type* (BETA or OR) was ambiguous, corresponding to just six PMIDs. After revision of the publications, a total of 23 studies where the effects were measured in standard units rather than as beta coefficients were also identified and excluded. An additional study for type 2 diabetes contributing only one SNP with an extreme OR value, not reported in any other publication, was also excluded. Traits were pulled out from their clusters if they had different *effect type*. This was the case of the trait “Obesity”, which left the “Obesity-related traits” cluster shown in Fig. S2. SNPs without a measure of the risk allele frequency were also removed in this step.