

# When heritability within groups is informative about differences among groups: a comment on Schraiber and Edge (2024)

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Author’s note: We submitted this 500-word comment on Schraiber and Edge (2024) to PNAS. The editor-in-chief decided not to publish the comment on the grounds that the “Editorial Board has determined that it does not contribute substantially to the discussion of the original article.” Usually, as per COPE guidelines, authors of criticised material are given an opportunity to respond. In our view, Schraiber and Edge’s (2024) title statement that “Heritability within groups is uninformative about differences among groups” is misleading, since within-group heritability is informative about group differences under the common causation assumption; under this empirically testable assumption, between-group heritability is a linearly increasing function of within-group heritability. Moreover, Schraiber and Edge (2024) err in affirming Lewontin’s (1970) claim that “the genetic basis of the difference between two populations bears no logical or empirical relation to the heritability within populations.” In this brief comment, we show the logical, conditional relation connecting within- and between-group heritabilities.

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Schraiber and Edge (1) claim that “[h]eritability within groups is uninformative about differences among groups.” This is surprising as within-group genetic variance is related to between-group differences in  $Q_{st}$ - $F_{st}$  comparisons (2) and because behavioral genetics methods can leverage within-group variance components to decompose sources of group differences (3, 4). Within-group heritability ( $h_W^2$ ) must, at least conditionally, be informative about the sources of group differences.

After their provocative title claim, Schraiber and Edge (1) qualify many statements, noting that  $h_W^2$  provides no information “on its own,” potentially rendering their claim little different from those of researchers like Arthur Jensen whom they critique. Unfortunately, they don’t elaborate.

To clarify the issue, Figure 1 – see similarly: Jensen [5] -- depicts the relationships between variances and terms from Defries’ (6) formula, which Schraiber and Edge (1) criticize. In this,  $\sigma G_w^2$ ,  $\sigma G_b^2$ ,  $\sigma E_w^2$ , and  $\sigma E_b^2$  represent the within- and between-group genetic and environmental variances,  $r$  and  $t$  (shaded) represent, respectively, the between-group genetic and phenotypic intraclass correlations, and  $h^2$  represents the total heritability.

We can write  $h_B^2$  as:

$$h_B^2 = \frac{\sigma^2 G_b}{\sigma^2 G_b + \sigma^2 E_b} \cong \frac{r \cdot h^2}{t}$$

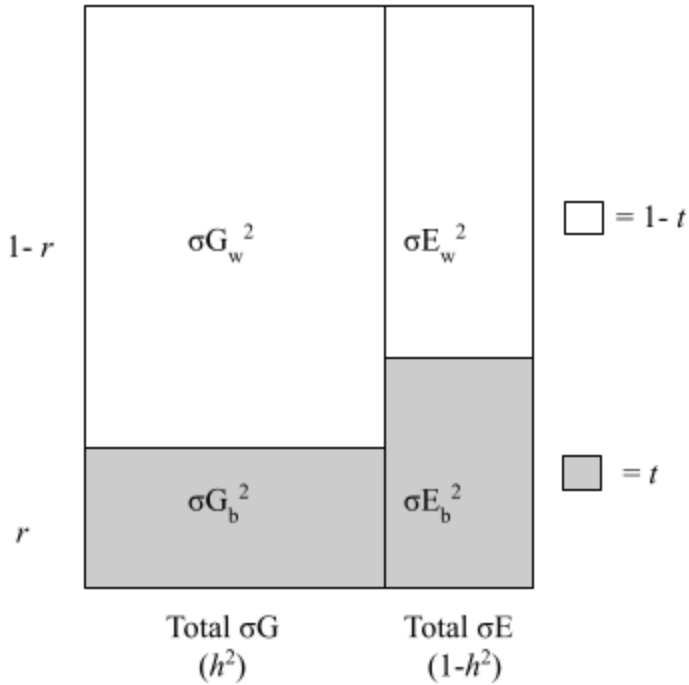


Figure 1. Relation between variances and heritability

Since  $h_w^2$  is a component of  $h^2$ ,  $h_w^2$  and  $h_B^2$  are logically related, yet  $h_w^2$  doesn't constrain  $h_B^2$  unconditionally. The constraint occurs when, following Defries (6), we relate  $h_w^2$  and  $h_B^2$  through  $h^2$  using the equation for  $h_w^2$ :

$$h_w^2 = \frac{\sigma^2 G_w}{\sigma^2 G_w + \sigma^2 E_w} = \frac{(1-r) \cdot h^2}{(1-t)}$$

Thus,

$$h^2 = h_w^2 * \frac{(1-t)}{(1-r)}, \text{ and so } h_B^2 \cong h_w^2 * \frac{(1-t)r}{(1-r)t}$$

This derivation of  $h^2$  makes the common causation assumption, that the sources of between-group variance are a subset of the sources of variance within-groups. When so,  $h_B^2$  is a linear function of  $h_w^2$  and naturally becomes undefined when  $\sigma^2 G_w = 0$ . Recognizing this assumption, we can integrate Schraiber and Edge's (1) and Defries's (6) formulas by distinguishing between common and uncommon sources of variance:

$$h_B^2 = \frac{\sigma^2 GC_b + \sigma^2 GU_b}{\sigma^2 GC_b + \sigma^2 GU_b + \sigma^2 EC_b + \sigma^2 EU_b} \cong \frac{h_w^2 * \frac{(1-t)r}{(1-r)t} * \sigma^2 P_b + \sigma^2 GU_b}{\sigma^2 GC_b + \sigma^2 GU_b + \sigma^2 EC_b + \sigma^2 EU_b},$$

where  $\sigma^2 GC_b$  represents variance between groups due to genes causing variability within groups, while  $\sigma^2 GU_b$  represents variance between groups due to genes not causing variability within groups; mutis mundus for  $\sigma^2 EC_b$  and  $\sigma^2 EU_b$ , replacing "genes" with "environmental

factors”;  $\sigma^2P$  represents the phenotypic variance between groups. Under the common causation assumption, this equation reduces to Defries’s (6).

In this expanded formulation,  $h_B^2$  is a function of  $h_W^2$ , except when  $\sigma^2GC_b = 0$ , and  $h_W^2$  can constrain  $h_B^2$  when  $\sigma^2GU_b = 0$ .  $h_W^2$  can be used to decompose the sources of group differences (3, 4) because, under the common causation assumption, mean differences on trait indicators will be a linear function of  $h_W^2$  in a biometric factor model.

Both Jensen (5) and Warne (7) argued that  $h_W^2$  can be informative about group differences. Both noted that  $h_W^2$  is *not necessarily* related to group differences because these *could be* due to uncommon sources of variance (i.e., X-factors). Schraiber and Edge (1) do not address the substance of these arguments, but primarily reiterate the previously articulated conditional relation between  $h_B^2$  and  $h_W^2$  (8).

### References

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