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Bias in the measurement of bias. Letter regarding 'Citation bias and selective focus on positive findings in the literature on the serotonin transporter gene (5-HTTLPR), life stress and depression'

de Vries *et al.* (2016) argue that discussion of the 5-HTTLPR-stress gene–environment interaction (G × E) (Caspi *et al.* 2003) is more positive than merited because authors often cast negative results as positive in abstracts, and negative papers with positive focus are differentially cited. These bold claims deserve careful scrutiny. Four methodological choices we highlight bias their primary results; the vast majority of papers disclose mixed and negative results in their abstracts (Table 1). Further, even if positive focus *was* prevalent, it could not bias meta-analytic results. The field can best move forward by ameliorating environmental measurement.

Methodological concerns

de Vries *et al.* (2016) coded papers' full results sections as positive or negative, then compared this with abstract conclusion sentences' positivity. Four choices that lead to errant conclusions contrast decisions reflecting care not to bias results – selecting the smallest p value when both traditional and triallelic results were available, and when both adjusted and unadjusted results were available. Similarly, sensitivity analyses using the lowest p value should address several issues, but still provide 'positive focus' results that contradict disclosures we extracted from abstracts. We focus our comments on their primary approach, which informs their conclusions.

Averaging p *values*

When papers included multiple G × E p values, the authors averaged them in their primary analyses, an approach biased toward negative conclusions. For a hypothetical paper with three findings at the $p = 0.001$ level and one finding at the $p = 0.300$ level, the average of the four is non-significant by traditional standards, $p = 0.076$. But who would conclude such a paper was negative overall? Although the most inclusive 5-HTTLPR and life stress G × E meta-analysis took a similar approach (Sharpley *et al.* 2014), a bias for negative conclusions could be entirely appropriate for a meta-analysis that ultimately has positive conclusions. However, the negative bias favors the perspective of de Vries *et al.* (2016).

Dichotomizing averaged p *values*

The authors imposed a false negative/positive dichotomy on averaged p values. For example, Jenness *et al.* (2011) reported a significant interaction for 5-HTTLPR with family chronic stress ($p = 0.02$) but not with recent stressful life events ($p = 0.88$), leading to a negative classification by de Vries *et al.* (2016) (average $p = 0.46$). Despite disclosure of mixed findings in their abstract (Table 1), de Vries *et al.* (2016) labeled their work as having partially positive focus relative to 'negative' findings. An alternative if imperfect approach is to classify papers across at least three categories (positive, negative and mixed), then evaluate abstracts for fidelity to actual findings.

Unbiased or atheoretical?

The primary approach assumes each of the averaged p values are equally valid, an approach which runs roughshod over theory. Several papers specifically hypothesized that one of their tests was more valid than another – sensitivity testing that refines G × E research and ought to be highly cited – accordingly presented the results of both approaches, and found support for their hypothesis. Uher *et al.* (2011) found support for Brown & Harris's (2008) hypothesis that the childhood adversity G × E predicts persisting depression, $p = 0.003$, but not single-episode depression, $p = 0.231$ (a finding replicated elsewhere; Brown *et al.* 2013). These results transparently appear in their abstract (Table 1), yet the faulty assumption that these tests are equally valid leads de Vries *et al.* (2016) to classify Uher *et al.*'s (2011) and Brown *et al.*'s (2013) papers as negative with positive focus. Although sensitivity analyses selecting the lowest p value ought to allow for theory to favor a particular test, we identified abstract sentences disclosing results for more papers than these analyses suggest.

Evaluation of abstract conclusion sentences not full abstracts

To determine whether abstracts had overly positive focus, the authors rated the conclusion sentence(s), not the full abstract. Such a selective approach

Table 1. *Transparent sentences from abstracts of papers that de Vries* et al. *(2016) classified as having (partially) positive focus*

First author (year)	Quote from abstract results disclosing results
Brown (2013)	'The short alleles of 5-HTTLPR moderated the relationship between childhood maltreatment and chronic depression in adulthood, reflected in a significant gene–environment interaction (RD = 0.226, 95% CI: 0.076–0.376, $p = 0.0032$). 5-HTTLPR did not moderate the effects of either childhood maltreatment or severe life events on new depressive onsets'
Cichetti (2007)	None
Cichetti (2011)[a]	None. Test of G × G × E[b]
Eley (2004)	'In addition, there was a trend for an effect of 5HTTLPR, which was significant in female subjects. Furthermore, there was a significant genotype–environmental risk interaction for 5HTTLPR in female subjects only. . ..'
Goldman (2010)	'Although the gene–environment (G × E) interaction with recent major life events is not significant, our results suggest that trauma has a worse effect on depressive symptoms for those with S/S or S/L genotype than for those who do not carry the S allele ($p < 0.05$)'
Grabe (2012)[a]	'Tobit regression analyses revealed a three-way-interaction between the three genotypes of 5-HTTLPR and the BDNF genotypes and overall childhood abuse for the BDI-II score ($p = 0.02$). . . . The s/s genotype of the 5-HTTLPR exerted its negative impact on mental health after childhood abuse only in the presence of the BDNF Val/Val genotype but not in the presence of the BDNF Met allele. In contrast, the l allele of the 5-HTTLPR also emerged as a genetic risk factor for depression in carriers of one or two Met alleles'
Hankin (2011)	'Lagged hierarchical linear modeling analyses showed 5-HTTLPR interacted with idiographic stressors (increases relative to the child's own average level over time), but not nomothetic stressors (higher stress exposure relative to the sample), to predict prospective elevations in depressive, but not anxious, symptoms'
Jenness (2011)	'A significant G × E showed that chronic family stress predicted prospective increases in depressive symptoms over 6 months among youth possessing the high-risk S allele. This G × E was not found for episodic stressors occurring in the last 6 months. . . . This is the first study to show that chronic family stress, but not episodic stressors, when ascertained by rigorous stress interview, interacts with 5-HTTLPR to prospectively predict depressive symptoms among children and adolescents'
Mitchell (2011)[a]	None. Test of (G + G) x E[b]
Quinn (2012)	'The results support a role for genetic factors in the development of non-melancholia. The lack of findings in melancholia indicates that other mechanisms may underlie the subtype'
Ritchie (2009)	'Interactions were observed between the 5-HTTLPR long (L) allele, poverty, and excessive sharing of parental problems'
Scheid (2007)	'The relationship between exposure to abuse and elevated depressive symptoms was more pronounced in the s/s group (OR 24.5) than in the s/l group (OR 3.0) and the l/l group (OR 7.7), but this significant interaction was detected only after excluding 73 (13%) women with recent use of psychotropic medications'
Scheid (2011)	'The relation between stressful life events and "elevated" depressive symptoms was stronger in S/S compared with LA/LA genotype (interaction $p = 0.11$). Of the six subconstructs, only abuse showed a statistically significant gene–environment interaction'
Sjöberg (2006)	'First, boys and girls carrying the short 5-HTTLPR allele react to different kinds of environmental factors. Whereas males were affected by living in public housing rather than in own owned homes and by living with separated parents, females were affected by traumatic conflicts within the family. Second, the responses of males and females carrying the short 5-HTTLPR allele to environmental stress factors go in opposite directions'
Stefanis (2011)[a]	'Homozygous for the 5-HTTLPR S allele reported significantly higher scores for paranoid ideation as compared with L-allele carriers. Slight effects on other subscales were observed, but were not significant after correction for multiple testing. . . . In particular, variation within this gene may confer risk for paranoid/defensive reactions under conditions of environmental stress associated with military induction'
Sugden (2010)	None
Uher (2011)	'In both cohorts, statistical tests of gene–environment interactions showed positive results for persistent depression but not single-episode depression. Individuals with two short 5-HTTLPR alleles and childhood maltreatment had elevated risk of persistent but not single-episode depression'
Wichers (2008)[a]	None. Test of G × G × E[b]
Wilhelm (2006)	None
Wilhelm (2012)	'The 5-HTTLPR low-expression genotype group (S or L_G allele carriers) had significantly higher psychological distress (K10) scores ($n = 234$, $p = 0.047$). Subsequent analysis revealed that the effect of genotype was related to anxiety symptoms rather than depression symptoms. Furthermore, the main effect of genotype was not observed when the modification of the SNP polymorphism was not taken into account' [Exposure only design]

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Table 1 (*cont.*)

First author (year)	Quote from abstract results disclosing results
Zalsman (2006)	None
Zhang (2009*a*)	'In addition, the individuals carrying the L/L genotype of 5-HTTLPR could be susceptible to MDD when exposed to negative life events and MDD in the Chinese population'

5-HTTLPR, Serotonin transporter gene; RD, risk difference; CI, confidence interval; BDNF, brain-derived neurotrophic factor; OR, odds ratio; SNP, single nucleotide polymorphism; MDD, major depressive disorder; G × E, gene–environment interaction.

[a] The primary focus of the paper was something other than 5-HTTLPR G × E for depression.

[b] We debated whether to expect papers with a focus other than the 5-HTTLPR G × E (but which included it as an ancillary test) to report on this G × E in their abstracts. These include tests of G × G × E effects and one additive (G + G) × E test. To be conservative, we report results both ways. In each noted case, a paper tests a more complex effect but does not fully characterize the ancillary 5-HTTLPR G × E in the abstract.

disregards an abstract's 'gestalt' without any rationale for doing so. Where is the evidence that researchers cite papers based on abstract conclusion sentences? In contrast to the authors' assertions, we were able to identify very clear acknowledgement of mixed results in all but seven of the 22 abstracts characterized as having (partially) positive focus (Table 1).

Results of alternative rating approach

To estimate these decisions' impact on the positive focus ratings of de Vries *et al.* (2016), we rated the 38 'negative' papers. Two raters examined results, assigning negative, or mixed classifications, and examined the full abstract to determine whether negative or mixed results were not disclosed (ratings appear in online Supplementary Table S1). We extracted sentences demonstrating disclosure (Table 1). We deemed it unfair to papers with a primary focus other than the 5-HTTLPR G × E (e.g. focus on a G × G × E), but which included it as an ancillary test, to expect they report G × E results in their abstract; to be conservative, we present results both ways. Group discussion adjudicated non-matching ratings. Of these 38 'negative' studies, we characterized them as 58% ($n = 22$) negative and 42% ($n = 16$) mixed. We assigned (partially) positive focus ratings to four to seven of the 22 articles that de Vries *et al.* (2016) characterized as having (partially) positive focus (depending on treatment of papers with a focus other than the 5-HTTLPR G × E). We conclude that the ratings of de Vries *et al.* (2016), which form the basis for evaluation of citation bias, are fundamentally flawed.

Sensitivity analyses using the lowest p value still do not square with evidence that authors disclosed results (Table 1): these indicate 12 have (partially) positive focus relative to our four to seven. Moreover, the authors suggested that sensitivity analyses did not markedly influence their findings (for citation bias), but their effect size of (partially) positive focus drops by 26% relative to their negative ratings (22/38 to 12/28) and by 45% relative to the population of 73 studies. Their procedures have a marked impact on estimating the prevalence of positive focus. We observe that this is not reported in their abstract.

Biased conclusions

A conclusion the authors draw in their own abstract is noteworthy: 'discussion of the 5-HTTLPR–stress interaction is more positive than warranted'. How positive *should* the discussion be? Clearly, this is controversial. On the one hand, there have been two negative meta-analyses that included a small number of reports to use homogeneous designs ($k = 5$ and 14, respectively; Munafò *et al.* 2009; Risch *et al.* 2009), many G × E investigations are under-powered (Duncan & Keller, 2011), and we observed some questionable research practices as we read. On the other hand, inclusive meta-analyses from Karg *et al.* (2011) ($k = 54$) and Sharpley *et al.* (2014) ($k = 81$) both reach positive conclusions, with Sharpley *et al.* (2014) showing that the meta-analytic effect emerges across four separate design subtypes. Karg *et al.* (2011) show that differences between the negative meta-analyses and theirs are due to paper selection, not meta-analytic technique. Papers selected for their statistically homogeneous designs tend to have methodological flaws including retrospective lifetime stress and depression assessment (Moffitt & Caspi, 2014) leading to confounding (Uher & McGuffin, 2010). Moreover, Karg *et al.* (2011) show that reports with more robust measures of stress (interview and

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objective measures) possess a more robust meta-analytic effect, so much that others observe an almost 1:1 relationship between stress measurement quality and likelihood of at least partial G × E effect replication (Uher & McGuffin, 2010). Neither positive focus nor citation bias influences this evidence. There is at least a reasonable basis for concluding that this is a legitimate G × E effect. Thus, when papers characterize the results of the 5-HTTLPR G × E literature positively and cite positive studies, how is this 'more positive than warranted?'

Where to go from here?

There is a much larger problem – and opportunity for progress – in G × E depression research. The unique environment contributes roughly 60% of risk to depression (Sullivan *et al.* 2000), but in G × E research we often fail to invest in environmental measurement. Many G × E researchers measure the environment with insufficiently valid measures (for discussion, see Monroe & Reid, 2008; Uher & McGuffin, 2010; Karg *et al.* 2011; Sharpley *et al.* 2014). But in addition, we must all more carefully conceptualize the 'candidate environment'.

Recent work supports that chronic stress and major severity interpersonal stress were consistent unique predictors of depressive episode onset across two samples of emerging adults, whereas minor stressors were never unique predictors and non-interpersonal stressors were rarely so (Vrshek-Schallhorn *et al.* 2015). Early evidence indicates that these distinctions matter for G × E tests: Whereas no G × E effect emerged for minor events, consistent with expectations, an overall G × E effect between 5-HTTLPR and major events was accounted for exclusively by major *interpersonal* events and not non-interpersonal ones (Vrshek-Schallhorn *et al.* 2014). All forms and severities of stress are not created equal. As G × E research moves beyond 5-HTTLPR, we hope the field will work toward large-scale G × E research with valid, thoughtfully conceptualized environmental measures.

Conclusions

Although positive focus sometimes occurs in G × E research, as we expect it unfortunately does throughout science, through their methodological choices, the paper of de Vries *et al.* (2016) exemplifies bias. Four choices including classifying abstracts by only their conclusion sentence bias the primary results. Sensitivity tests do not overcome these issues. Ultimately, depression–genetics research enterprise aims to enhance prediction and intervention for depression. It is time we all renewed our 'positive focus' on that goal.

Supplementary material

The supplementary material for this article can be found at http://dx.doi.org/10.1017/S0033291716002178

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Declaration of Interest

None.

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