

Supplementary Results

Cortisol

The range of average participant ages in the cortisol analyses was somewhat restricted, with the exception of a few outliers. Namely, 73 of the studies in the analysis had an average age between 18 and 30. In the remaining four studies of the cortisol analyses, one had an average age of 39.9, the second outlying study's average was 47.3, the third outlying study's average age was 62.1, and the fourth outlying study's average age was 64.6. However, the effect of age on cortisol responses did not appear different these four outlying studies. Excluding those four studies, the effect of age on cortisol responses was positive and significant, $B=.096$, $t(69)=2.11$, $p=.038$. When examining only the four outlying studies, the direction and magnitude of the moderating effect of age on cortisol responses were similar to the other studies, $B=.066$, and although this was not significant, $p=.338$, this lack of significance was due to low statistical power (i.e., only four studies were included in this analysis). Thus, older participants appear to have greater cortisol responses to stress than do younger participants.

Restriction of range was not an issue for any other moderator analysis, such as percentage of male participants.

Encoding

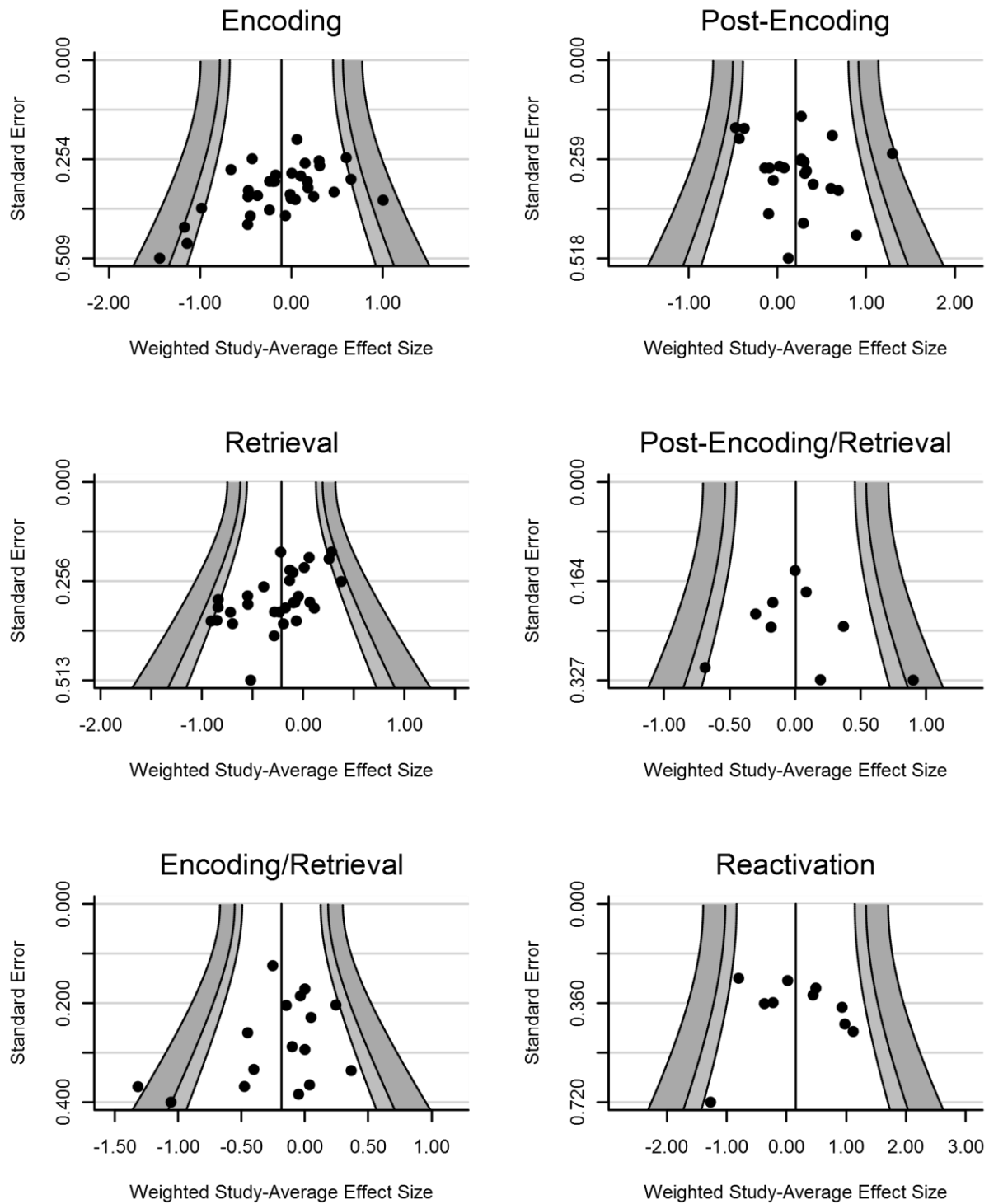
Although we grouped studies of stress during encoding with studies of stress prior to encoding, it is possible that these effects are categorically different, rather than being quantitatively different as a function of the stress-encoding delay. As such, we present analyses here grouping studies of stress during encoding separately from studies of stress prior to encoding.

Stress during encoding. We defined stress during encoding as an encoding paradigm that occurred after the stressor began but prior to the ending of the stressor. There were five studies examining effects of stress during encoding on memory, with 24 effects within those five studies. The overall effect of stress during encoding on memory was not significant, $g^+ = .219$, $t(3.9) = 1.42$, $p = .231$, 95% CI_g $[-.214, .652]$. There was low heterogeneity in this analysis, $\tau^2 = 0.15$. An examination of all moderators revealed that only the relevance of the items to the stressor emerged as significant, $t(3.8) = 4.40$, $p = .013$, such that memory for items related to the stressor was enhanced, $g^+ = .472$, $t(2.9) = 3.84$, $p = .032$, whereas memory for items unrelated to the stressor was not affected, $g^+ = -.152$, $t(3.0) = -0.79$, $p = .490$. These results are in agreement with our primary analyses of encoding, which found that memory for items related to the stressor was enhanced when the stress-encoding delay was low. However, it should be noted that in this analysis, df was less than four, so there is a twofold greater risk of making a Type I error. In all these analyses, we likely lacked power to detect significant moderating effects due to the small study set size.

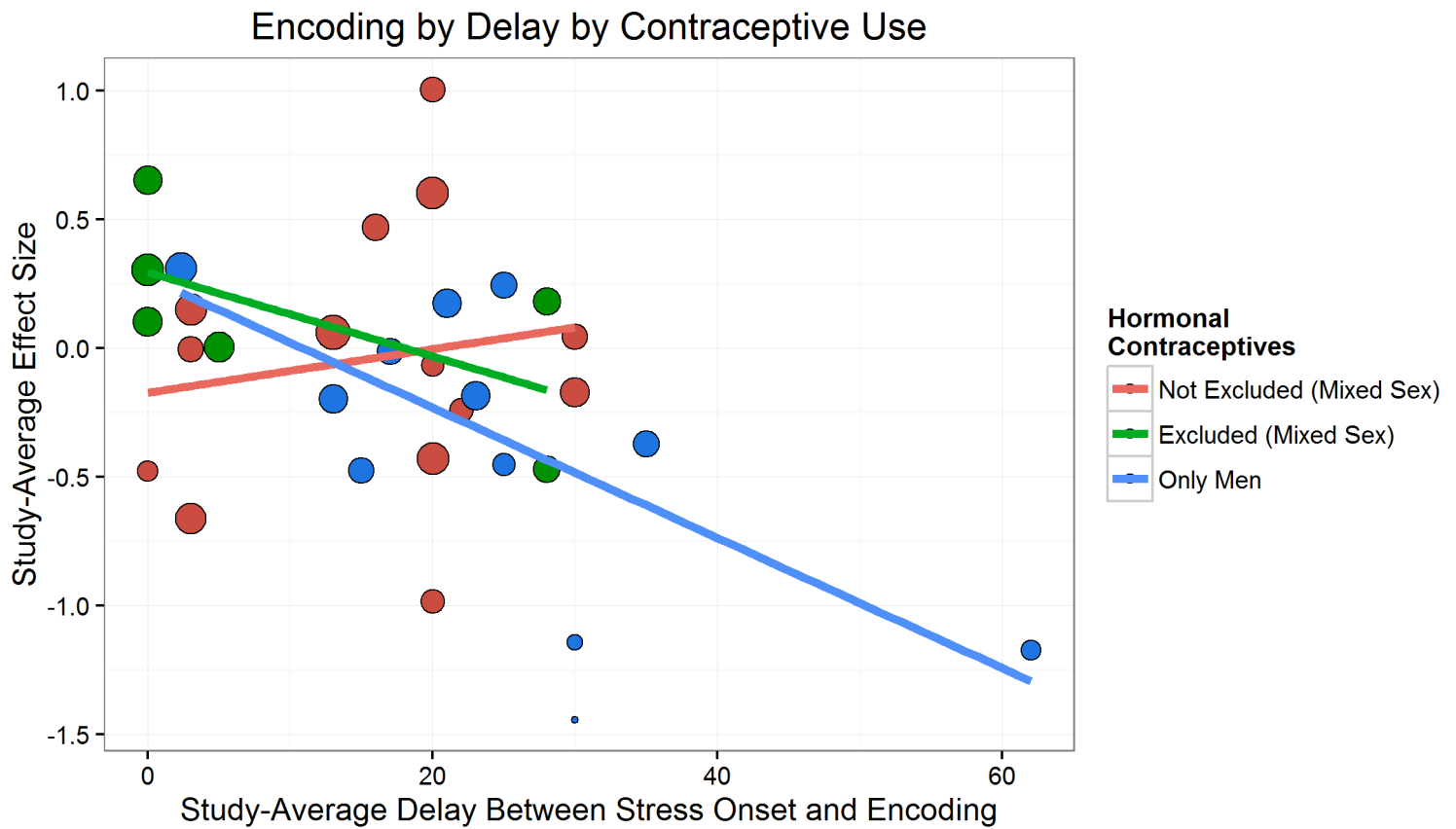
Stress prior to encoding. We defined stress prior to encoding as an encoding paradigm that occurred after the stressor finished. There were 28 studies examining effects of stress during encoding on memory, with 107 effects within those five studies. The overall effect of stress prior to encoding on memory was marginal, $g^+ = -.169$, $t(26.5) = -1.82$, $p = .080$, 95% CI_g $[-.360, .021]$. There was low heterogeneity in this analysis, $\tau^2 = 0.21$. An examination of all moderators revealed the stress-encoding delay was a marginal moderator of effects of stress prior to encoding on memory, $B = -.016$, $t(4.6) = -2.19$, $p = .086$. Notably, the magnitude of this slope was nearly identical to its magnitude in the primary analyses (that is, including stress during encoding, where the slope was $B = -.0167$), indicating that the moderating effect of the stress-encoding

delay does not differ when removing studies of stress during encoding. With only two studies of stress prior to encoding including items relevant to the stressor, we were unable to examine whether the relevance of the item to the stressor moderated effects of stress prior to encoding on memory. The remainder of the moderator analyses produced results virtually identical to the primary analyses reported within the manuscript, with the exception that the interaction of contraceptive exclusion and stress-encoding delay became marginal rather than significant.

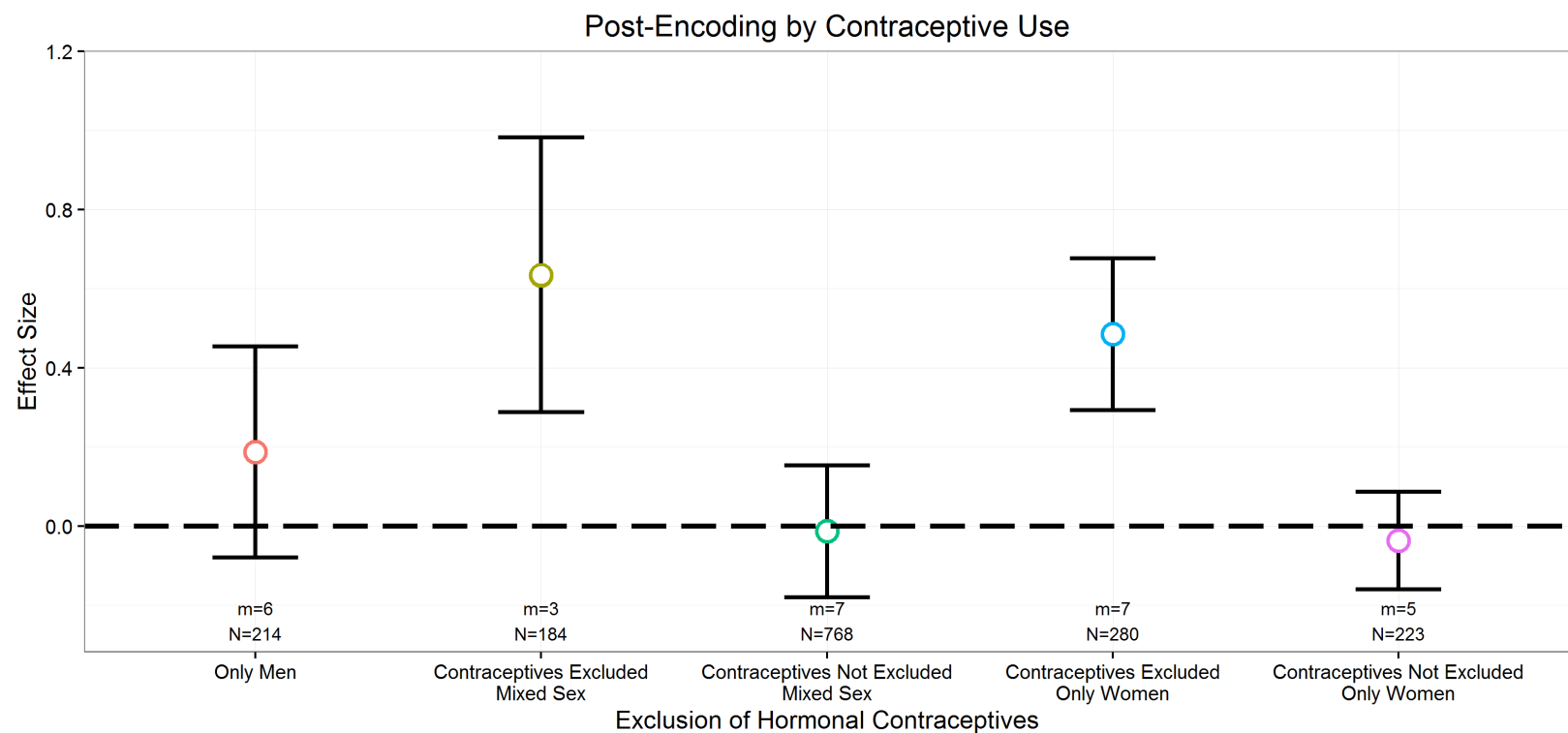
Summary. Because of the confound that studies including items related to the stressor were almost entirely studies of stress during encoding, it is not possible to disentangle whether the relevance of items to the stressor has any lasting effects on memory in studies of stress prior to encoding. Similarly, the small study set size precluded us from conducting any well-powered analyses of effects of stress during encoding on memory. However, the relative lack of change in the magnitude of the stress-encoding delay moderator when excluding studies of stress during encoding indicates that studies of stress during encoding appear to differ quantitatively (as a function of the stress-encoding delay) from studies of stress prior to encoding rather than qualitatively.



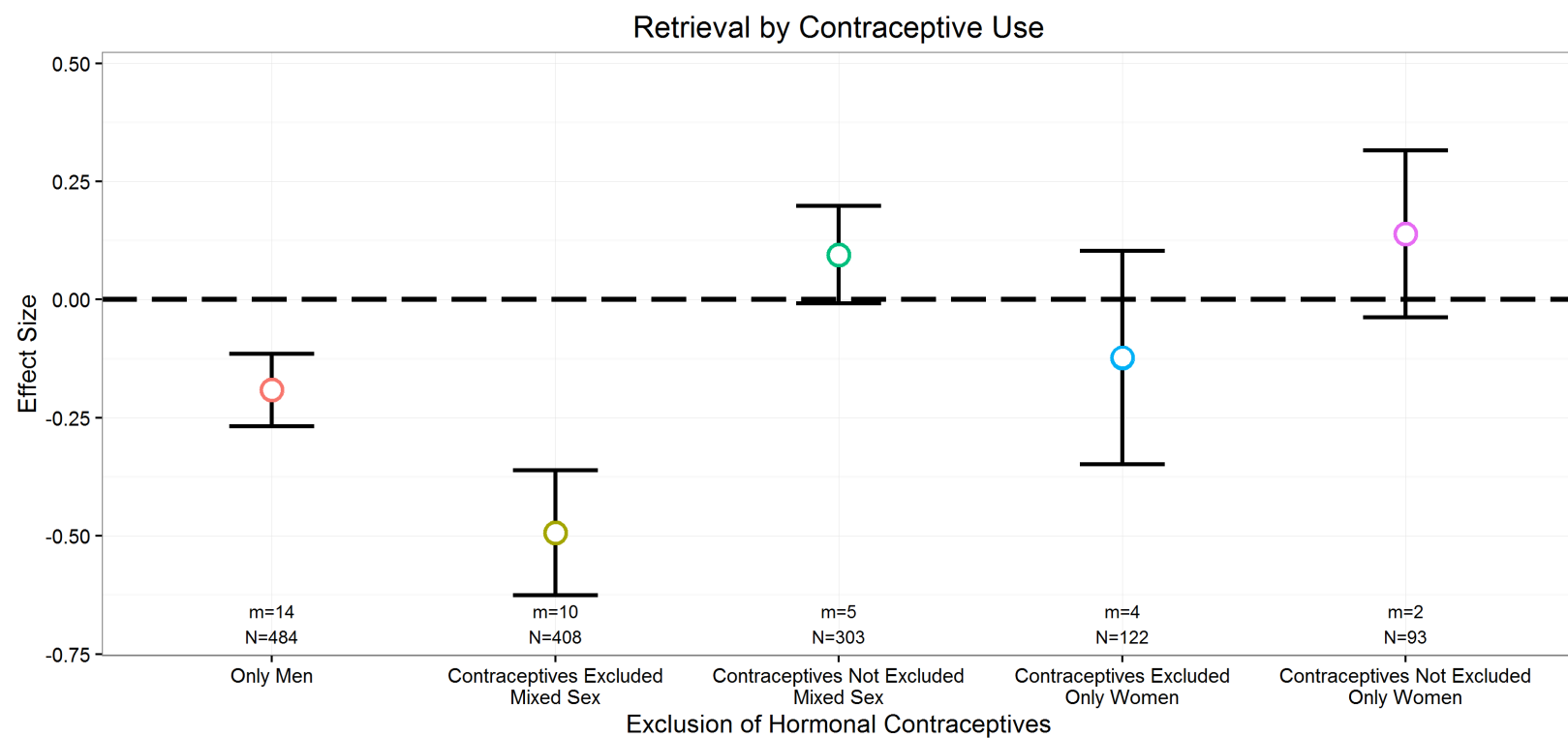
Supplementary Figure 1. Funnel plots assessing publication bias in our observed effects. Asymmetry of studies (the dots) around the grand effect (the middle line) indicates publication bias. Light gray indicates 95% confidence intervals, dark gray indicates 99% confidence intervals. Encoding showed marginal evidence for publication bias, and retrieval showed significant evidence.



Supplementary Figure 2. Breakdown of contraceptive exclusion by delay effects for studies of stress at encoding. Studies that excluded women on hormonal contraceptives and studies of only men both showed the expected moderating effect of delay on stress at encoding, whereas studies that did not exclude women on hormonal contraceptives did not show the expected moderating effect of delay. There were no studies of only women for stress at encoding experiments.



Supplementary Figure 3. Breakdown of contraceptive exclusion for studies of post-encoding stress. Studies of only men and studies that excluded women taking hormonal contraceptives showed enhancing effects of post-encoding stress on memory, whereas studies that did not exclude women taking hormonal contraceptives did not demonstrate an enhancing effect of post-encoding stress on memory.



Supplementary Figure 4. Breakdown of contraceptive exclusion for studies of stress at retrieval. Studies of only men and studies that excluded women taking hormonal contraceptives showed impairing effects of stress on retrieval, whereas studies that did not exclude women taking hormonal contraceptives did not demonstrate an impairing effect of stress on retrieval.