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# **Estimating the stability of heartbeat counting in middle childhood: a twin study**

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## Abstract

There is growing interest in interoception, the perception of the body's internal state, and its relevance for health and higher-order cognition across development. To date, most evidence linking interoception to health and cognition has used the heartbeat counting task. However, the stability of the measure across time, particularly during childhood, and the etiological factors that underlie individual differences in stability remain largely unexamined. Using data from the ECHO twin sample (N=204 twin pairs), we estimated the magnitude of genetic and environmental influences on the stability of heartbeat counting across a two-year period (8-10 years), the longest time-frame examined. We found a relatively modest correlation between heartbeat counting accuracy across time ( $r=.35$ ), with accuracy on the heartbeat counting task improving with age. In our longitudinal twin analysis, we found that the heritability of heartbeat counting dropped between Time 1 and Time 2 from 30% to 6%. No new genetic influences were observed at Time 2, suggesting that genetic influences across this age-range are entirely stable. In contrast, shared environmental influences increased from 6% to 22%, with most of the influence at Time 2 due to new environmental factors. Of note, nonshared environmental factors accounted for the greatest proportion of variance at both time points, 64% and 73% respectively, and were the main contributors to temporal stability in heartbeat counting accuracy. Future research should seek to identify these non-shared environmental factors and elucidate whether this relatively modest stability reflects variability of interoception across development or unreliability of the heartbeat counting task.

**Keywords:** Interoceptive accuracy; interoception; heartbeat counting; stability; heritability; Twins

## Introduction

In recent years there has been a growing interest in the importance of interoception, the perception of the body's internal state (Craig, 2002; Khalsa et al., 2018), for health and aspects of higher order cognition (Khalsa & Lapidus, 2016; Khalsa et al., 2018). Indeed, atypical interoception (both unusually good or bad interoceptive ability) has been proposed to underlie a number of transdiagnostic and disorder-specific symptoms. For example, high anxiety has been linked to unusually high interoceptive ability, whereas depression is often associated with poor interoceptive ability (Murphy, Brewer, Catmur, & Bird, 2017; Khalsa & Lapidus, 2016; Khalsa et al., 2018). Within typically developing populations, poor interoceptive accuracy has also been linked to atypical cognition in domains as diverse as decision making (e.g., Dunn et al., 2010), theory of mind (e.g., Shah, Catmur, & Bird, 2017) and emotional processing (e.g., Terasawa, Moriguchi, Tochizawa, & Umeda, 2014).

Much of the research that has examined how individual differences in interoception are related to health (e.g., depression, anxiety or sleep problems) and aspects of higher order cognition (e.g., emotion recognition) has utilized the heartbeat counting task as a measure of interoception (Dale & Anderson, 1978; Schandry, 1981). In this task, participants are asked to count their heartbeat over a series of intervals whilst their objective heartbeat is recorded. The participant's count is then compared to the objective measure to determine its accuracy. Despite widespread use of this task for quantifying interoception, in recent years there has been increasing focus on the *validity* of heartbeat counting as a measure of interoception. Indeed, questions have been raised as to the validity of the task as a measure of interoceptive accuracy given evidence that individual differences in physiology, heart rate knowledge, differences in

task administration, and non-interoceptive factors may contribute towards task performance (e.g., Desmedt, Luminet, & Corneille, 2018; Khalsa, Rudrauf, Sandesara, Olshansky, & Tranel, 2009; Murphy, Brewer, Hobson, Catmur, & Bird, 2018; Ring, Brener, Knapp, & Mailloux, 2015; Zamariola, Maurage, Luminet, & Corneille, 2018).

Despite this research focus on the *validity* of the heartbeat counting task as a measure of interoception, surprisingly few studies have examined the *stability* of heartbeat counting across time, and even fewer in childhood. Given increasing focus on interoception across development (e.g., Murphy et al., 2017), understanding the stability of this commonly-used measure is of crucial importance. Indeed, increased understanding of the stability of scores on this measure may shed light on whether heartbeat counting performance can be considered an enduring trait or whether scores are state specific (Wittkamp, Bertsch, Vögele, & Schulz, 2018). Such data will also be informative in understanding the relationship between heartbeat counting performance and psychopathology across development. In adulthood, estimates of the stability of heartbeat counting performance range from approximately  $r = .58$  to  $.81$  depending on the time period examined, intervention (e.g., meditative training) and participant group employed (Ehlers, Breuer, Dohn, & Fiegenbaum, 1995; Bornemann & Singer, 2017; Ferentzi, Drew, Tihanyi, & Köteles, 2018; Herbert, Herbert, & Pollatos, 2011; Mussgay, Klinkenberg, & Rüddel, 1999; Parkin et al., 2014; for an overview see Ferentzi et al., 2018). However, few studies have examined the stability of heartbeat counting across long time periods (e.g., >6 months). Indeed, in adulthood it appears that 9 months is the longest time period that stability has been assessed, with estimated stability approximately  $r = .70$  (Bornemann & Singer, 2017). To our knowledge only one study has examined the stability of heartbeat counting in childhood. In a large sample ( $N = 1350$ ) of children aged between 6-11 years, stability of only  $r = .33$  was observed across a 1-

year period (Koch & Pollatos, 2014). Such evidence suggests that heartbeat counting performance may be less stable in childhood than adulthood.

The factors underlying stability of heartbeat counting, particularly in childhood where performance appears to be less stable, remain unknown. Furthermore, there is little research into the etiology of heartbeat counting at any developmental period. Twin studies enable the disentangling and estimation of genetic and environmental influences on traits, by comparing the similarity of monozygotic (identical) and dizygotic (non-identical) twins. Longitudinal twin studies can identify the extent to which genes and the environment influence stability and change of traits over time. For example, such studies indicate that the moderate stability of anxiety (and depression) from childhood through to adulthood is predominantly influenced by stability of genetic influences (Nivard et al., 2015; Waszczuk, Zavos, Gregory, & Eley, 2014). In contrast, environmental effects are primarily time specific, and are thus associated with change. To our knowledge only one twin study has examined the etiological factors underlying performance on the heartbeat counting task (Eley, Gregory, Clark, & Ehlers, 2007). The authors observed a moderate genetic influence (~30%) on heartbeat counting at age 8. Non-shared environmental influences were substantial. However, no studies have used longitudinal data to assess whether these etiological influences remain stable over time.

In the present study we aimed to investigate the stability of the etiological influences on heartbeat counting across time. In addition to elucidating the factors underlying the stability of heartbeat counting, we capitalised on the large sample to explore associations with other traits previously shown to covary with heartbeat counting. We aimed to estimate the magnitude of shared genetic and environmental influences between heartbeat counting and other traits associated with heartbeat counting in adulthood, such as anxiety and depression (see Khalsa &

Lapidus, 2016), sleep problems (e.g., reduced sleep quality and insomnia; Ewing et al., 2017; Wei et al., 2016), and aspects of higher order cognition (e.g., emotion recognition; Terasawa et al., 2014) in this sample of children. Whilst few studies have examined these relationships in childhood, those that have typically observe similar associations with anxiety (e.g., Eley et al., 2007; Eley, Stirling, Ehlers, Gregory, & Clark, 2004). Whether other relationships observed in adulthood (e.g., with sleep problems and emotion recognition) can be replicated in childhood remains an outstanding question. Crucially, however, to our knowledge only one study has examined the etiology of these observed associations between heartbeat counting and health (e.g., depression, anxiety, sleep problems) or higher order cognition. In the only twin study of heartbeat counting described above, Eley et al. (2007) observed that higher panic/somatic anxiety ratings were associated with lower error on the heartbeat counting task ( $r = -.13$ ). This relationship was partly explained by genetic factors (genetic correlation =  $-.46$  (95% CI:  $-1.00$ - $1.00$ )) though this did not reach statistical significance. Whether the etiology of this relationship remains stable over time, and can be observed for other factors previously associated with heartbeat counting in adulthood (e.g., sleep problems, emotion recognition, anxiety or depression), and childhood (e.g., anxiety) remains unknown.

This study first aimed to test the stability of heartbeat counting in childhood across a two-year period to examine whether the etiological factors change over time, and to estimate to what extent genetic and environmental factors drive any observed stability. To this end, we revisited data reported in Eley et al. (2007), and previously unexamined data collected in the same twin sample two years later. This is the longest time period across which the stability of heartbeat counting has been assessed at any developmental stage. It is also the only study to examine the etiology of the stability of heartbeat counting. Finally, we examined previously-reported

associations between heartbeat counting and aspects of health (anxiety, depression, sleep problems) and higher order cognition (emotion recognition) to see whether 1) associations observed in adulthood would be observed in childhood; and 2) these associations were stable over time. Where significant relationships were observed, etiological factors underlying these relationships could be assessed. In line with previous reports, we expected that the stability of heartbeat counting would be low in childhood ( $\sim r=.30$ ), with heartbeat counting expected to be associated with anxiety, depression, sleep problems and emotion recognition.

## **Methods**

### **Participants**

The ECHO study consists of 300 twin pairs from the Twins' Early Development Study, which recruited over 215,000 twin pairs born in England and Wales during 1994–96 (TEDS; Trouton, Spinath, & Plomin, 2002). Data were collected at the Institute of Psychiatry, London apart from a few families who were visited at home. Ethical approval was granted by the Maudsley Hospital Ethics Committee, London, UK. Informed consent from parents was obtained via postal methods in advance.

A selected extremes design was used when identifying the ECHO sample, to increase statistical power. This involved the selection of 247 twin pairs who scored high on parent-rated anxiety at age 7, plus 53 pairs of controls. Following the selection of these pairs, 11 were removed due to mental or physical impairments. Zygosity was diagnosed using reported physical similarity plus DNA in uncertain cases. In the final sample, 193 twin pairs completed both time points.

At Time point 1, twins were approximately 8 years of age ( $M = 8.47$ ,  $SD = 0.18$ ). Data for Time point 2 was collected approximately 2 years later. Of the ECHO sample, at baseline 57% was female, and 33% were MZ twins.

## **Measures**

### ***The Heartbeat Counting Task***

Participants were asked to silently count the heartbeats they could feel during three intervals (of 35, 25 and 45 seconds) following a 10 second practice trial that was not analyzed (see Eley et al., 2007 for further details). During each trial, the electrocardiogram (ECG) was recorded and a computer program scored the number of R-waves (the largest peak of the ECG QRS complex, with the amount of peaks representing the actual number of heartbeats).

Participants were explicitly instructed not to take their pulse or to use any other strategies such as holding their breath, which was visually checked by the researcher (trained psychology graduates). At the start of each trial, participants heard a warning stimulus (800 Hz, 65 dB, 100 ms) to prepare them for the task (as in Ehlers & Breuer, 1992). This warning was given 500 ms after an R wave was recorded on the participants ECG. The start signal (1000 Hz, 65 dB, 50 ms) was triggered immediately after the third R wave that followed the warning stimulus. The tone signaling the end of the counting period (1000 Hz, 65 dB, 50 ms) was given after the time interval for that trial was complete and 300 ms after the last R wave had elapsed. At the end of each trial, the child told the researcher how many heartbeats they had counted. To prevent distraction and remove the possibility of cheating, children were seated so that they could not see the computer screen or ECG during the task.



For each trial, percentage error scores were calculated by taking the absolute difference between the actual number of heartbeats recorded by the ECG (AB) and the number of heartbeats counted by the child (CB), as a percentage of the number of actual heartbeats (i.e.,  $((AB-CB)/AB)*100$ ) as in previous work (Ehlers & Breuer, 1992)). An average score was then taken across the three trials completed. As such, a score of zero reflects totally accurate performance, whereas a score of 100 reflects totally inaccurate performance (e.g., feeling no heartbeats at all)<sup>1</sup>.

### ***Questionnaire measures***

At both time points, self-report and parent-report data were available for a number of measures of health and wellbeing that have previously been linked to cardiac interoception in either children or adults. For anxiety, data were available from the Screen for Childhood Anxiety Related Emotional Disorders (SCARED; Birmaher et al., 1999) and the Children's Anxiety Sensitivity Index (CASI; Silverman, Fleisig, Rabian, & Peterson, 1991). Total scores for these measures were computed, with high scores on both measures reflective of higher anxiety/ greater sensitivity to the physical symptoms of anxiety. For depression, data were available from the Children's Depression Inventory (CDI; Kovacs, 1985). Total scores for this measure were computed with high scores reflecting higher depressive symptoms. For sleep problems, data were available from the Sleep Self-Report (SSR; Owens, Maxim, Nobile, McGuinn, & Msall, 2000) and the Children's Sleep Habits Questionnaire (CSHQ; Owens, Spirito, & McGuinn, 2000). Again, total scores for these measures were computed with high scores reflecting greater sleep problems.

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<sup>1</sup> Note that in this sample no children overestimated the amount of actual heartbeats and therefore scores fit within the range of 0-100.

### ***Emotion recognition***

At Time point 2, data for emotion recognition ability were also available. The emotion recognition task consisted of 160 trials (for further details see Lau et al., 2009). On each trial, participants were presented with a facial image that morphed from a neutral expression into one of 5 basic expressions (angry, fear, sad, disgust, happy). All facial expressions were taken from a standard set of pictures of facial affect. Facial expression morphs were displayed as animations changing from neutral to one of four levels of intensity (25%, 50%, 75%, 100%) with intensity adjusted for happy expressions (to 10%, 25%, 50%, 75%) given that they were easier to identify. Head orientation (facing towards or away from the camera) and gaze direction (towards and away from the camera) were also manipulated resulting in 4 different trial types.

Prior to the task, participants were read standardised instructions and were asked to provide a definition of each emotion to ensure they were familiar with the emotion labels. On each trial participants were instructed to name the expression using one of five labels corresponding to the different emotions, with five practice trials completed before commencement of the task.

Accuracy scores were summed across all trials for each of the 5 expressions. These scores were then averaged across the 5 expressions to create an overall score. This variable was taken as a measure of emotion recognition ability, with high scores representing better performance.

## **Statistical analyses**

### ***Selection variable***

All twin analyses were conducted jointly with the 7-year anxiety screening variable from TEDS, allowing us to control for selection bias. Specifically, we linked our heartbeat counting data to the original distribution of the selection variable in the entire sample, and used maximum likelihood to estimate the corrected distributions, variances and covariances of the heartbeat counting variables. This increases the statistical power and generalisability of the analyses.

### ***Twin model-fitting***

The classic twin study design capitalizes on the fact that ‘identical’ monozygotic (MZ) twins in principle share 100% of their genes, whereas ‘non-identical’ dizygotic (DZ) twins share on average 50% of their segregating genes. However, these types of twins are equally similar in terms of their environment. The degree of genetic contribution to variation in a particular phenotype in a population is estimated by comparing monozygotic to dizygotic resemblance. The extent to which members of monozygotic pairs are more similar to one another than members of dizygotic pairs indicates the degree of genetic influence on the trait of interest, because degree of genetic sharing correlates with degree of phenotypic similarity. Specifically, the comparison between MZ and DZ twins is used to estimate the contribution of genetic (A), shared environmental (C), and non-shared environmental (E) influences to variation in the phenotype. The heritability of a trait (A) is the proportion of phenotypic variance that can be explained by genetic variation in the population under study. Shared environment refers to environmental influences that result in family members resembling one another. Non-shared environment refers to environmental influences that make family members different from one another. Here we are

not talking about whether the environmental experiences are shared, but whether their *effects* are shared.

To investigate the influences on the covariance between heartbeat counting performance across time, we fitted a longitudinal twin model, the Cholesky decomposition (see Figure 1). The Cholesky decomposition allows the investigation of stability and innovation in the genetic and environmental influences on our measure of heartbeat counting across the two time points. The first genetic factor (A1) represents genetic influences on heartbeat counting at Time 1. The extent to which these same genes also influence heartbeat counting at Time 2 is also estimated and is represented by the diagonal pathway from A1 to Time 2. The second genetic factor (A2) represents genetic influences on heartbeat counting at Time 2 that are independent of those influencing Time 1. The Cholesky model allows the A, C and E factors underlying the first measured variable to influence the second variable, but not vice versa. The same decomposition is done for the shared environmental and non-shared environmental influences (C1–2 and E1–2, respectively).

### ***Relationships with other measures***

We assessed correlations between heartbeat counting, health variables (e.g., anxiety, depression, sleep) and emotion recognition ability for the data available at both time points. We also conducted prospective analyses, by examining the correlation between heartbeat counting at Time 1 and health (e.g., anxiety, depression, sleep) and emotion recognition ability at Time 2. Differences in accuracy across the two time points was also assessed using a paired samples t-test.

### *Sensitivity analysis*

We tested whether results changed when body mass index (BMI) and sex were regressed out of the heartbeat counting scores given evidence that both body composition and sex are related to heartbeat perception ability (e.g., Murphy et al., 2018; Grabauskaitė, Baranauskas & Griškova-Bulanova, 2017; Rouse, Jones & Jones, 1988). As this had little influence on the pattern of results obtained these data are reported in the Supplementary Information. Analyses in the main text feature the original phenotypes, since the sample size and thus statistical power, was higher than for the phenotypes residualised for BMI.

## **Results**

### **Phenotypic descriptive statistics**

See Table 1 for descriptive statistics of the heartbeat counting measures (for one randomly selected twin from each twin pair). The full sample used for longitudinal twin modelling was 5579, including individuals with ECHO and/or TEDS data, and zygosity data. Note that the selection variable represents case/control anxiety status, based on maternal ratings when the children were aged 7. At both Time 1 and Time 2 heartbeat counting error scores were high with very few children meeting the cut off to be considered good perceivers (defined as <20% error; see Eley et al., 2007). At Time 1, 31 children met cut off (5.6% of the total sample). At Time 2, 39 children met cut off (9.5% of the total sample).

### **Phenotypic correlations across time and across twins**

Error scores for heartbeat counting were significantly lower at Time 2 ( $M = 57.35$ ,  $SD = 27.40$ ) in comparison to those at Time 1 ( $M = 69.09$ ,  $SD = 27.17$ ),  $t(196) = -4.87$ ,  $p < .001$ . The overall phenotypic correlation between heartbeat counting scores across the two time points was  $r = 0.35$ , indicating moderate stability in task performance. Initial inspection of twin correlations for heartbeat counting performance suggested a heritability of ~42% at time-point 1 and ~4% at time-point 2.

### **Phenotypic correlations with other measures**

Heartbeat counting performance was not significantly correlated with any of the examined variables (symptoms of anxiety, depression or sleep quality) at Time 1 nor with any of the examined variables (symptoms of anxiety, depression, sleep problems or emotion recognition) at Time 2 (Table 2). When considering prospective analyses between heartbeat counting at Time 1 and aspects of health (anxiety, depression and sleep) and higher order cognition at Time 2, only emotion recognition performance at Time 2 was predicted by earlier heartbeat counting performance. Specifically, higher error of heartbeat counting at Time 1 negatively predicted subsequent emotion recognition ability (Table 2). However, this relationship was not significant after correction for multiple comparisons. Where subscales were available, we also examined the relationship between heartbeat counting and subscales for these measures. As reported in Eley et al., (2007) at Time 1 heartbeat counting was associated with panic/somatic anxiety symptoms. At Time 2, heartbeat counting was associated with social phobia. However, none of these relationships were significant after correction for multiple comparisons (see supplementary information). Given that no significant relationships were observed, it was not

possible to examine the etiological factors underlying the predicted overlap between these factors and heartbeat counting.

### ***Longitudinal twin model-fitting results***

Figure 1 presents the results of the model-fitting analyses. First considering the total genetic, shared environmental and non-shared environmental influences at each time-point (represented on the horizontal lines for heartbeat counting task at Time 1, and by the addition of the horizontal and vertical lines for Time 2), there was a moderate heritability of heartbeat counting at Time 1 (30%). At Time 2, heritability was much lower, at 6%. In contrast, shared environmental influences increased from 6% to 22% from Time 1 to Time 2. The nonshared environmental contributions were more similar across timepoints, being 64% and 73% at Time 1 and Time 2, respectively. It is important to note that the majority of these parameter estimates are non-significant (Figure 1). This is because, although the sample is well-powered for phenotypic correlation analyses, power for twin model-fitting, especially for distinguishing genetic from shared environmental influences, is low. In sensitivity analyses we tested whether all familial influences (i.e. genetic and shared environmental factors) on stability across time could be dropped from the model. We found that simultaneously removing both genetic and shared environmental factors significantly reduces model fit (Supplementary Table 1), indicating the presence of familial influences on the longitudinal association.

Focusing on the level of innovation in genetic and environmental influences at Time 2 (represented by the vertical lines coming from A2, C2 and E2), there were no new genetic influences on heartbeat counting error scores at Time 2, indicating that genetic influences across this age-range were entirely stable (albeit low). In contrast, for both shared and non-shared

environment there were new influences at Time 2. For example, at Time 2, 22% of the variance is accounted for by the shared environment, of which 82% (18 as a proportion of 22) is new variance specific to this time-point. Non-shared environmental influences were primarily time-specific, and were the strongest contributor to change from Time 1 to Time 2.

Considering the diagonal lines running from A1, C1 and E1 to heartbeat counting task performance at Time 2, the data suggest that some of the shared environmental influence on heartbeat counting performance at Time 1 also influences the trait at Time 2 (4% out of the total 22% shared environmental influence). Non-shared environmental influence from Time 1 also influences trait variation at Time 2 (4% out of the 73%). In sum, genetic influences are largely stable and environmental influences are largely time-specific. Indeed, of the phenotypic stability (0.35), the percentages due to A, C and E were 37% [-36%-103%], 15% [-31%-69%] and 48% [12%-89%]. Here, E contributes most to the sharing of influences between the two time points because non-shared environments are the major sources of variation at both time-points, even if only 4% out of 73% of the non-shared environmental influences at Time 2 affected heartbeat counting at Time 1.

Our sensitivity analyses showed that results did not differ when controlling for BMI (see Supplementary Figure 2).

## **Discussion**

This study aimed to quantify the stability of heartbeat counting across a 2-year period in childhood and examine the etiological factors underlying stability of performance. Additionally, we also examined the relationship between heartbeat counting and aspects of health (anxiety, depression and sleep) and higher-order cognition (emotion recognition) that have previously been associated with heartbeat counting in adulthood, but not previously examined in childhood.



First, considering the longitudinal stability of the heartbeat counting phenotype, results revealed a small but significant correlation between heartbeat counting at Time point 1 and Time point 2, with a reduction in error observed with age. In line with previous estimates across a 1-year period (Koch & Pollatos, 2014), these data suggest that the stability of heartbeat counting in childhood is relatively low ( $\sim r=.35$ ) in comparison to stability estimates in adulthood ( $r=\sim .58-.80$ ; see Ferentzi et al., 2018). One explanation for this discrepancy is differences in the time period examined (to our knowledge 9 months is the longest time period across which heartbeat counting stability has been assessed in adulthood; Bornemann & Singer, 2017). However, as estimates of stability in childhood over a 1-year period ( $\sim r=.33$ ; Koch & Pollatos, 2014) are also lower than estimates in adulthood, this is an unlikely explanation for the pattern of results obtained. An alternative explanation is that heartbeat counting performance changes to a greater extent in childhood than in adulthood. Indeed, in the present study a reduction in error was observed from Time 1 to Time 2 suggesting that (in general), changes in heartbeat counting performance in this sample were driven by an improvement in performance with age. It is therefore possible that in childhood, heartbeat counting ability may not be fully developed or it may not have reached adult levels of stability. For example, anxiety is only moderately stable across childhood: correlations between measures at different time points are only  $\sim r=0.30$  (e.g., Cheesman et al., 2018). In contrast, stability in adulthood is greater, in the region of  $\sim r=.50$  (e.g., Nes, Røysamb, Reichborn-Kjennerud, Harris, & Tambs, 2007) with some evidence that stability increases with age (from around  $r=0.6$  in adolescence to  $r=.80$  in adulthood; e.g., Nivard et al., 2015). As such, whilst the stability of heartbeat counting is lower in childhood than adulthood, this is consistent with a body of literature that indicates that the stability of a number of traits is lower in childhood (a time of great developmental change) as compared to adulthood. Of course,

we must also acknowledge that, in the context of questions over the validity of this measure (e.g., Desmedt et al., 2018; Khalsa et al., 2009; Murphy et al., 2018; Ring et al., 2015; Zamariola et al., 2018), it is also possible that that low stability of heartbeat counting reflects unreliability of the measure, particularly across middle childhood. Alternatively, however, it may be that performance on the heartbeat counting task is largely state-dependent. Given some evidence that time-specific person-situational factors are related to performance in adulthood (e.g., Wittkamp et al., 2018), it may be that state-specific effects have a greater influence on performance in middle-childhood. Future research, using multiple measures of interoception, may help to disentangle these possibilities.

Turning to our twin model-fitting results, our main finding is that the primary factor influencing variation in heartbeat counting at each time-point is the non-shared environment. Such individual-specific environments accounted for the greatest proportion of variance at both time points, 64% and 73% at Time 1 and Time 2, respectively. In terms of the etiological factors underlying performance, some caution is required given that the sample size employed here is small for a twin study. As a result, we have limited statistical power to test the significance of genetic and shared environmental parameters. We therefore discuss the results for the genetic and shared environment components of variance with caution, as most estimates have confidence intervals that cross zero. The heritability of heartbeat counting dropped between Time 1 and Time 2 from 30% to 6%. No new genetic influences were observed at Time 2, suggesting that genetic influences across this age-range are entirely stable. In contrast, shared environmental influences increased from 6% to 22%, with most of the influence at Time 2 due to new environmental factors.

In terms of the etiological factors underlying stability, results from genetic analyses suggest that the small amount of stability of heartbeat counting observed ( $r=.35$ ) is driven by genetic, shared environmental and non-shared environmental factors (37%, 15% and 48%, respectively), with only non-shared environmental factors making a significant contribution to stability. Although measurement error is captured in the non-shared environment component in twin studies, the large non-shared environment estimates are unlikely to be a product of measurement error alone, as this is unlikely to be stable across years. As such, it appears that time-specific non-shared environmental factors contribute significantly towards performance at both time points, but a large proportion of the observed stability is explained by the stable non-shared environmental factors. Such a pattern indicates that heartbeat counting performance, both performance at each time point and stability, is driven largely by child-specific factors – factors that make individuals in the same family different. Whilst these data cannot elucidate what factors these may be, various non-shared factors are likely to contribute to both time-specific performance and stability. This may include factors related to the administration of the heartbeat counting task (e.g., Wittkamp et al., 2018), experience-based factors that may influence performance (e.g., heart rate knowledge; Ring et al., 2015), as well as factors that shape individual differences in the ability to perceive one's heartbeat (e.g. chance or environmentally-driven changes in blood pressure or resting heart rate; see Murphy et al., 2018). Future research should seek to replicate this finding of the primary importance of non-shared environmental influences, and to identify the specific physiological and psychological factors that contribute to this component of variance.

As noted above, several other findings emerge from the data pertaining to genetic and shared environmental influences, but statistical power is too low to have high confidence in these

results. Whilst genetic and shared-environmental factors did not significantly contribute towards time-specific performance or stability, it is still notable that genetic factors remained stable over time – all of the genetic variance at Time 2 was explained by variance at Time 1 – though in comparison to Time 1 (reported in Eley et al., 2007) the genetic influence at Time 2 was lower. In contrast, shared environmental factors were largely time-specific and showed an increase from Time 1 to Time 2. This observation must be treated with some caution: when power is low, it is difficult to distinguish genetic from shared environmental influences. As such, we cannot be certain which makes a greater contribution towards stability. Nevertheless, these data suggest some role of genetic and shared-environmental factors to heartbeat counting performance and highlight a need for further research into the etiology of heartbeat counting performance, and the etiology of individual differences in interoception more broadly, in larger samples.

In addition to answering questions regarding the factors underlying stability, a secondary question concerned the relationship between heartbeat counting and health/cognition during middle childhood. Contrary to predictions based on previously-reported associations in adulthood (e.g., Ewing et al., 2017; Khalsa & Lapidus, 2016; Terasawa et al., 2014), no significant relationship between heartbeat counting and anxiety, depression, sleep problems or emotion recognition was observed in this sample of children at either Time 1 or Time 2 after correction for multiple comparisons. There are several possible explanations for this lack of significant correlations. First, this sample was comprised mostly of highly-anxious individuals and it is possible that the relationship between heartbeat counting and the factors examined here may differ as a function of anxiety levels. However, as low-anxiety control participants were also studied, and no relationship was observed between heartbeat counting and anxiety, it is unlikely that this provides a full explanation of these findings. Second, it is of course possible that these

relationships emerge over the course of development. If, as has been suggested by Murphy et al., (2017), adolescence is a sensitive period for interoceptive development (and the heartbeat counting task can be considered a measure of interoception), it is possible that these relationships emerge at a later stage of development. Third, it must also be noted that in this sample very few children met criteria to be considered a good heartbeat perceiver. As such, it is possible that this also contributes towards the absence of previously reported relationships between heartbeat counting and aspects of health and higher order cognition. Of course, a final possibility is that unreliability of the heartbeat counting task may also contribute towards the lack of significant associations between scores on this task and other measures. Indeed, as highlighted throughout, the validity of this measure remains under question with various non-interoceptive factors thought to contribute towards task performance (e.g., Desmedt et al., 2018; Khalsa et al., 2009; Murphy et al., 2018; Zamariola et al., 2018). As a number of these possible confounds (e.g., heart rate knowledge, systolic blood pressure) were not controlled for here (given that the data were collected prior to the outlining of appropriate controls), it is not possible to conclude that associations between heartbeat counting and aspects of health and cognition would not be found in childhood if the full range of controls were employed. However, given that associations in adulthood have been reported without using these controls, this again is unlikely to fully explain the pattern of results unless it is assumed that these confounds are likely to have a larger impact in childhood than adulthood. These data highlight an urgent need to examine the relevance of interoception to health and cognition across development using valid measures of interoception.

Despite the absence of significant relationships between heartbeat counting and aspects of health (anxiety, depression or sleep) and higher order cognition after correction for multiple comparisons, it is notable that when considering the relationship between heartbeat counting at

Time 1 and emotion recognition ability at Time 2 a significant relationship emerged. Better heartbeat counting performance at Time 1 was marginally associated with better emotion recognition performance at Time 2. Likewise, certain anxiety subscales (e.g., social phobia) were associated with heartbeat counting. Although these relationships did not survive correction for multiple comparisons, and therefore some caution is required, they are consistent with the proposed relationship between interoception and social abilities (emotion recognition and social phobia; e.g., Terasawa et al., 2014; Clark & Wells, 1995). As such, further research into the relationship between social cognition and interoception in childhood is warranted and would benefit from the use of multiple measures of interoceptive ability.

Notwithstanding the importance of these data we must acknowledge certain limitations. First, as this study involved reexamination of historical data, a number of factors were not controlled for (e.g., differences in heart rate physiology, knowledge of resting heart rate or physical activity), and a control task was not utilized. However, given that few studies employ the full range of control variables advocated by Murphy et al., (2018), these data provide a crucial understanding of the etiological factors underlying stability of the heartbeat counting task, and the relationship between the measure and health and cognition, as the task is routinely administered. Second, it is important to acknowledge that given low statistical power the results of the genetic analyses must be treated with some caution. Nevertheless, this one of the largest samples available in studies of heartbeat counting performance in childhood, and is a rare genetically-sensitive resource. We were able to draw several conclusions with confidence, particularly that non-shared environmental factors are the driver of stability and time-specific differences in performance. These data provide the first description of the etiological factors

underlying stability of performance on the heartbeat counting task, highlighting that individual-specific factors play a fundamental role over time.

In conclusion, this study sought to examine the stability of heartbeat counting over a 2-year period in childhood and the etiological factors underlying stability. Results revealed low stability in childhood, with non-shared environmental factors substantially contributing to both time-specific performance and stability. Contrary to predictions, heartbeat counting was not associated with health or higher order cognition in this sample. These data contribute towards the growing debate surrounding the heartbeat counting task, suggesting that low stability may reflect either the unreliability of the measure, or that heartbeat counting is not a stable trait. Identification of the individual-specific factors contributing to stability of performance may shed light on the validity of the measure for quantifying individual differences in interoception.

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**Disclosures:**

Alice Gregory is an advisor for a project sponsored by Johnson's Baby. She has written a book *Nodding Off* (Bloomsbury Sigma, 2018) and has a contract for a second book *Sleepy Pebble* (Nobrow). She is a regular contributor to BBC Focus magazine and has contributed to other outlets (such as *The Conversation*, *The Guardian* and *Balance Magazine*). She occasionally receives sample products related to sleep (e.g. blue light blocking glasses) and has given a paid talk to a business.

**Data Availability Statement:**

The data that support the findings of this study are available from the corresponding author upon reasonable request.



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*Table 1. Descriptive statistics*

|                                  | <b>N</b> | <b>Mean</b> | <b>SD</b> | <b>Min</b> | <b>Max</b> |
|----------------------------------|----------|-------------|-----------|------------|------------|
| Time 1. Heartbeat counting error | 279      | 69.01       | 26.3      | 4.34       | 100        |
| Time 2. Heartbeat counting error | 204      | 57.93       | 27.29     | 5.22       | 100        |
| Anxiety selection variable       | 5345     | 0.17        | 0.37      | 0          | 1          |

**Table 2. Correlations between heartbeat counting and other measures at Time 1 and Time 2**

|                     |                | Anxiety<br>(SCARED) | Anxiety<br>(CASI) | Depression<br>(CDI) | Sleep<br>problems<br>(SSR) | Sleep<br>Problems<br>(CSHQ) | Emotion<br>Recognition |
|---------------------|----------------|---------------------|-------------------|---------------------|----------------------------|-----------------------------|------------------------|
| Time 1              | Heartbeat      | -.028               | -.104             | -.021               | -.011                      | -.015                       | N/A                    |
|                     | counting error | N = 279             | N = 279           | N = 277             | N=277                      | N=269                       |                        |
| Time 2              | Heartbeat      | -.115               | .029              | .043                | -.007                      | .044                        | .079                   |
|                     | counting error | N = 204             | N = 203           | N = 204             | N = 203                    | N = 198                     | N = 203                |
| Time 1 -><br>Time 2 | Heartbeat      | .069                | .045              | .030                | -.041                      | -.109                       | -.152*                 |
|                     | counting error | N = 240             | N = 239           | N = 240             | N = 237                    | N = 231                     | N = 238                |

*Note. Time 1 -> Time 2 refers to phenotypic correlations between heartbeat counting error scores at Time 1 and questionnaire/cognition measures at Time 2. As shown, only emotion recognition scores at Time 2 were predicted by heartbeat perception at Time 1. \*denotes significant at  $p < .05$ .*

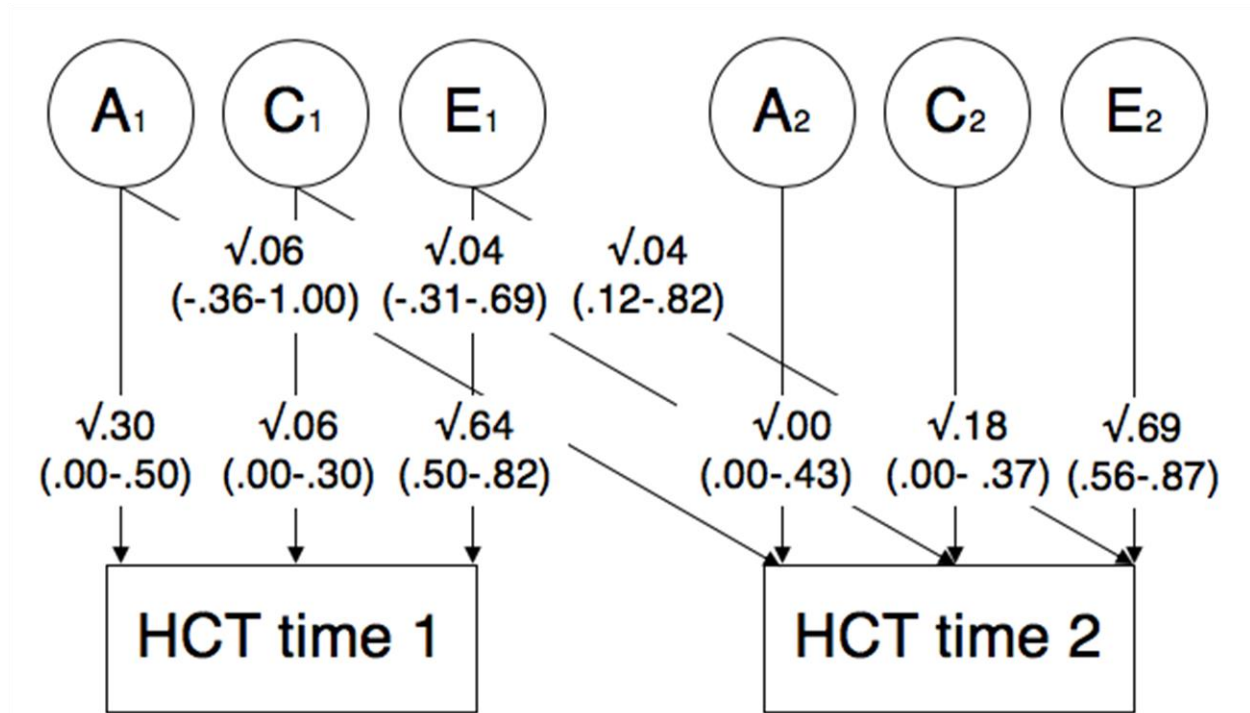


Figure 1: Standardised path estimates from the Cholesky decomposition, plus 95% confidence intervals. Note that  $A_1$ ,  $C_1$  and  $E_1$  represent the proportion of variance in heartbeat counting performance at Time 1 explained by genetic, shared environmental and non-shared environmental influences, respectively.  $A_1$ ,  $C_1$  and  $E_1$  sum to 100%. The diagonal paths show how much  $A_1$ ,  $C_1$  and  $E_1$  influence heartbeat counting at Time 2. These are added to  $A_2$ ,  $C_2$  and  $E_2$ , respectively, to give the overall variance explained by genetic, shared environmental and non-shared environmental factors at Time 2 (the estimates in the figure add to 101% due to rounding). For example, the heritability is 6% at Time 2 (6% + 0%). The selection variable has not been represented, but was accounted for in analyses. See Supplementary Figure 1 for unstandardised estimates and Supplementary Table 1 for model fit statistics.