An investigation into the relationship between visual imagery and stress

Samuel Hasler-Winter

Supervised by: Nils Muhlert

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Abstract

Visual imagery plays a functional role in memory and emotion processing. Similar links between stress, memory and emotion also exist, leading to the proposed hypothesis that there may also be a functional relationship between stress and visual imagery. In 36 students, trait vividness of visual imagery was assessed in relation to two variables: trait stress reactivity and acute stress response. The Trier Social Stress Test (TSST) and a matched control condition were used to induce stress. The Vividness of Visual Imagery Questionaire (VVIQ) and Perceived Stress Reactivity Scale (PSRS) were used as trait measures of visual imagery vividness and stress reactivity respectively. Neither the relationship between vividness of visual imagery and stress reactivity or acute stress response were significant. These findings suggest that stress and visual imagery may not be as closely related as thought. However, significant gender differences seen in the sample and proposed in the literature led to exploratory analyses. These analyses revealed a significant effect of stress-reactive group (high/low) on visual imagery in women, such that low stress reactive individuals experienced more vivid visual imagery and vice versa. Additionally, in women only, the hypothesised inverse relationships: between visual imagery vividness and both trait and acute stress were found to have moderate and strong correlations that were approaching significance, respectively. Given that this was post-hoc analysis, firm conclusions cannot be drawn, although this suggests that the proposed inverse relationship between voluntary visual imagery and stress may exist in women in the healthy population.

Key Words: Visual Imagery, Vividness, Stress, Stress-reactivity

Declaration

The contents of this paper are the students original work unless referenced clearly to the contrary. No portion of the work referred to in the dissertation has been submitted in support of an application for another degree or qualification of this or any other university or other institute of learning.

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Dedication

For my parents, Susan and James, for their unwavering love and support. Also, Liz McManus, for keeping me sane despite my best efforts to the contrary.

Intellectual Property Statement

Introduction

Visual imagery is for many, central to human experience. It has been shown to have a functional role in processes as diverse as memory, regulating emotion, decision-making, and addiction, as well as contributing to the pleasure and persuasiveness of a written narrative (Amit and Greene, 2012; Epstein & Pacini, 2001; Green and Brock, 2002; Kavanagh, Andrade, & May, 2005; Kosslyn et al., 1990; Paivio, 1969; Sparks, Sparks and Gray, 1995).

Visual imagery is commonly understood to be the experience of perceiving an object, scene or person in the mind, in the absence of the relevant external stimuli (de Vito and Bartolemeo, 2016). A phenomena that is comparable to the experience of seeing in typical perception, but often somewhat less defined or vivid (Zeman et al., 2010). Furthering this, Paivio's (1986) dual-coding hypothesis posits that imagery and language are two distinct and additive channels for information processing in the brain, a view upheld by current theorists and supported by evidence of the functional role of visual imagery (Libby and Eibach, 2013). Although there is some debate, visual imagery serves mainly to represent specific, concrete information that is not well represented in language, and doing so serves a function similar to vision (Kosslyn, 2006; Kounios and Holcomb, 1994; Libby and Eibach, 2013; Slee, 1980).

Visual imagination was described by Galton (1880) as a varying trait, suggesting that it's vividness varies between individuals. This suggestion has since been confirmed by neuroscience in more recent years (Pearson, 2014). "Aphantasia", a condition present in 2% of the population involving a complete absence of voluntary mental visual imagery, appears to have both structural and psychogenic causes (de Vito and Bartolomeo, 2016; Zeman, Dewar & Della Sala, 2015; 2016). However, little is known about the cause of variation in visual imagery in the non-clinical population.

Evidence from studies employing a binocular rivalry methodology – in which two differing stimuli are simultaneously shown to the two eyes – demonstrate that the act of perception is altered by imagery priming in much the same way as priming with external stimuli (Pearson, Clifford and Tong, 2008). This effect correlates with subjective reports of vividness in the normal population and in aphantasics (for whom no priming effect is found) (Keogh and Pearson, 2017; Pearson, Rademaker and Tong, 2011). This suggests that in both populations subjectively reported differences in visual imagery vividness reflect genuine differences in phenomenal experience, rather than impaired meta-cognition (Keogh and Pearson, 2017; Pearson et al., 2011). Functional magnetic resonance imaging (fMRI) data shows distinct neural correlates of these subjectively reported and behaviourally measured findings in aphantasics and healthy participants, as well as common neurological underpinnings between visual perception and visual imagery (Cui et al., 2007; Horikawa et al., 2013; Ishai, Haxby and Ungerleider, 2002; Kosslyn et al., 1993; MacKisack et al., 2016; Pearson et al., 2015; Zeman et al., 2010). These data provide strong evidence that visual mental imagery is a visual experience in the phenomenological sense (i.e. resembling perception), and that metacognition and subjective reports of visual imagery are accurate in both healthy and clinical cases. The Vividness of Visual Imagery Questionnaire (VVIQ) is a subjective trait measure

of voluntary visual imagery shown to have high reliability and construct validity, demonstrated partly by meaningful correlation with neural activation (Campos and Pérez-Fabello, 2009; Fulford, 2017; McKelvie, 1995).

Shared cortical activation between visual imagery processing and memory suggest that visual imagery plays a significant role in memory (Aydin, 2018; Farah, 1985). Indeed, a large body of behavioural evidence shows visual imagery plays a functional role in memory, and as such, vivid visual imagery ability, instructions to visualise, or highly imageable and concrete stimuli result in an improved memory performance (Abelson, 1975; Keogh and Pearson, 2011; McKelvie and Demers, 1979; Paivio, 1969; Swann and Miller, 1982; Williams, Healy, & Ellis, 1999). Vividness of visual imagery and memory are also positively correlated in aphantasia, severely deficient autobiographical memory, highly superior autobiographical memory and amnesia (Hassabis et al., 2007; LePort et al., 2012; Palombo, 2015; Watkins, 2017; Zeman et al., 2016). In fact, visual imagery plays such a significant role in memory that vividly imagining fictional autobiographical situations can lead to false memories of them actually occurring (Hyman and Pentland, 1996). The relationship between stress and memory is also well documented (Kuhlmann, Piel and Wolf, 2005; Sauro, Jorgensen and Pedlow, 2003). Transient stress influences both encoding and retrieval memory processes (Domes et al., 2002; Roozendaal, 2002; Schwabe and Wolf, 2010). Although the direction of the effect depends on various factors, crucially, stress can have both enhancing and damaging effects on memory (Cahill, Gorski and Le, 2003; Diamond et al., 2007; Schwabe et al., 2012). In clinical cases, extreme stress can cause amnesia (Freyd, 1994), or memory flashbacks in post-traumatic stress disorder (PTSD) (Ehlers, Hackmann & Michael, 2004). The functional involvement of visual imagery in memory, and the strong influence of stress on memory, taken together, may suggest a link between stress and visual imagery.

The process by which the body responds to stress is referred to as allostasis (McEwen, 2000; 2005). Allostasis is a process of adaptation managed mainly by the Autonomic Nervous System and the Hypothalamic-Pituitary-Adrenal (HPA) axis (McEwen, 1998). This biological reactivity to stressors is evolutionarily selected to prepare the individual for a threat (Ellis, Jackson and Boyce, 2006). However, an excess of these hormones can contribute to or exacerbate disease (McEwen and Seeman, 1999). In times of severe stress, the HPA axis produces excess cortisol, which has a dysregulatory effect on the HPA axis, as well as influencing a number of brain areas including the hippocampus, a core brain region for memory-related processes, including visual imagery (Fortin, Agster and Eichenbaum, 2002; Ishai et al., 2002; Leonard and Myint, 2009; Miller and O'Callaghan, 2002; Sapolsky, 1996). This maladaptation, referred to as allostatic load, describes the dysregulation of stress responses and is one of a variety of factors that can influence how an individual will respond to stress (McEwen and Stellar, 1993). In doing so, the presence of allostatic load results in the negative contributions of stress, including a number of clinical conditions (Dallman, 2010; Seeman et al., 1997; Selye, 1955; Steptoe and Kivimäki, 2012).

Especially powerful in determining the presence of allostatic load are developmental experiences, which impact lifelong physiological reactivity to stress due to increased

susceptibility to environmental influences during this time (Heim and Binder, 2012; Hertzman and Boyce, 2010). Referred to as epigenetics, the interaction between these environmental stressors and genes, particularly at these sensitive points, is responsible for many of the individual differences we see in reactivity to daily stressors (Provençal and Binder, 2015). Evidence shows that individuals who experience severe early life trauma display significantly increased stress reactivity (Flinn and England, 1995; Heim et al., 2000). Psychological processes also mediate the impact of stressful life events, although the formation of these themselves is dependent on early life stress (Hankin, 2005; Young, Klosko and Weishaar, 2003). Other contributing factors are physiological vulnerability: resulting from both genetic predisposition and elements such as diet, exercise, substance use and gender (Claessens et al., 2011; McEwen, 2006; Verma et al., 2011).

Although all forms of stress elicit similar HPA activation, distinctions must be made between physical and social stressors as these impact upon different neural circuits and therefore may have different cognitive effects (Dedovic et al., 2009). Social Self Preservation Theory states that situations which threaten the 'social-self' by involving a social-evaluative component, pose a risk to an individuals self-esteem and in doing so activate the HPA axis (Gruenewald et al., 2004). One method of measuring these individual differences in social stress reactivity is the Perceived Stress Reactivity Scale (PSRS), a subjective tool found to have good stability over time and convergent validity with related constructs (Schlotz et al., 2011).

Longitudinal studies of Cushing's syndrome, which features spontaneous hypercortisolism, show that this is followed in many cases by emotional disturbances (Starkman et al., 1981). This highlights the potential that heightened cortisol levels contribute to dysfunction in emotion regulation and helps imply the direction of causation in an already acknowledged association between cortisol, stress and emotion dysregulation (El-Sheikh et al., 2008; Keenan, 2000). In contrast, visual imagery has been shown to aid interpretation and regulation of emotions, suggesting that visual imagery may in some way mediate this relationship between stress and emotion processing (Dirkx, 2001; Kosslyn et al., 1990). Emotional suppression is also associated with decreased vividness of imagery for both imagined and remembered events, as well as greater stress generally (D'Argembeau and der Linden, 2006; Iwamitsu et al., 2009; Moore, Zoellner & Mollenholt, 2008). This suggests stress may be one mechanism by which visual imagery vividness is inhibited, or that deficits in visual imagery vividness contribute to heightened stress reactivity, or indeed, that the relationship is bi-directional. An alternative explanation is that detrimental individual differences in stress reactivity and visual imagery vividness are both the result of similar maladaptations in the HPA axis although are not functionally related. However, a functional relationship between stress and visual imagery better accounts for the effectiveness of imagery use in therapies for PTSD, social phobia, OCD, depression and a range of other anxious disorders - all disorders characterised by extreme stress reactivity and emotion dysregulation (Cisler and Olatunji, 2012; Etkin and Wagner, 2007; Foa et al., 1980; Hirsch and Holmes, 2007; Holmes, Arntz and Smucker, 2007; Krakow, Hollifield and Johnston, 2001; Speckens et al., 2007).

There are also a number of clinical differences in visual imagery. A key distinction in these differences is between voluntary and involuntary (or intrusive) imagery. Voluntary imagery refers to imagery experienced due to a conscious (although not necessarily effortful) act of intent to picture something, and involuntary imagery being experienced spontaneously without any such intent (Homer and Deeprose, 2017). In clinical cases characterised by involuntary imagery, an excess of this type of imagery is often found (Brewin et al., 2010; Ehlers et al., 2004; Speckens et al., 2007). In contrast, disorders featuring decreases in visual imagery primarily concern voluntary visual imagery (Sierra, 2009). This dissociation is supported by the selective absence of voluntary visual imagery in aphantasia, with the preservation of involuntary imagery (Zeman et al., 2015).

As mentioned previously, PTSD can cause hyper-activation and vivid, involuntary re-experiencing of memories (Ehlers et al., 2004). Eating disorders, which are often greatly exacerbated by stress, also feature highly vivid and distressing involuntary visual imagery (Brewin et al., 2010). Intrusive visual imagery is also sometimes found in Obsessive Compulsive Disorder (OCD), with those who experience visual imagery having more obsessive beliefs, compulsive behaviours and anxiety than those who do not (Speckens et al., 2007). OCD sufferers also exhibit significantly greater cortisol and perceived stress relative to healthy individuals and their perceived stress correlates with the severity of their condition (Morgado et al., 2013). This strongly reinforces an association between stress, cortisol, and visual imagery, and also suggests that psychogenic deletion of visual imagery in such disorders may serve an adaptive function.

In contrast, stress-related disorders such as depression, anxiety and depersonalization are all associated with diminished experience of voluntary, and in particular positive, visual imagery (Holmes et al., 2016; Sierra, 2009). In the context of depression, Paivio's (1985) imagery framework is highly relevant, suggesting that imagery plays a function in managing motivation, arousal and affect, as well as representing goals and activities. Of course, depression is characterised not only by a lack of motivation, but dysregulation of arousal and affect, as well as a loss of interest and pleasure in goals and activities (American Psychiatric Association, 2013). This framework therefore outlines several mechanisms by which a deficit in visual imagery vividness could contribute to the excessive stress characteristic of depression.

Taking the clinical data as a whole, it suggests that stress is related to both hypoactive voluntary visual imagery and hyperactive involuntary visual imagery. Of course, clinical samples are biased towards more extreme levels of stress and visual imagery, and therefore associations are more likely to be found. However, the maladaptations of stress response that underlie many clinical disorders do not only exist within these clinical parameters suggesting that these clinical relationships between individual differences in visual imagery and stress may be mirrored in more subtle ways amongst healthy individuals (Claessens et al., 2011). Additionally, the non-clinical data also suggests that the relationship between stress and voluntary visual imagery is negatively correlated. There is also limited evidence to suggest there is a relationship between stress and hyperactive involuntary imagery in the non-clinical population (Berntsen and Rubin, 2008; Witvliet, 1997). However,

presently there is no empirical research directly investigating the relationship between stress and visual imagery (voluntary or involuntary) within the normal population. Although the topic of involuntary visual imagery requires further understanding, the focus of this investigation is voluntary visual imagery and as such, it is expected that an inverse relationship between stress and visual imagery will be found, if any.

One method of manipulating stress in the non-clinical population is the Trier Social Stress Test (TSST). This task uses a social-evaluative and uncontrollable paradigm to replicate the influence of life's daily social stressors and reliably induce large cortisol responses in healthy participants (Kirschbaum, Pirke and Hellhammer, 1993). Compared to other forms of social stress, some using just public speaking and others using different paradigms, the Trier elicits greater and more robust cortisol elevations making it the obvious choice of task (Dickerson and Kemeny, 2004). Measuring self-report anxiety and salivary cortisol response during this task, coupled with later measures of trait stress reactivity and trait visual imagery, this study aims to explore the relationship between trait visual imagery, trait stress reactivity and acute stress response in a population of undergraduate students. The primary hypothesis of this study predicts that there will be a negative relationship between trait vividness of visual imagery and trait stress reactivity. A secondary hypothesis predicts that there will be a negative relationship between trait vividness of visual imagery and acute stress response.

Method

Participants

39 participants were opportunistically recruited from a pool of undergraduate psychology students at The University of Manchester (UoM). 3 participants withdrew from the study**.** The age of the remaining 36 participants (24 female, 12 male) ranged from 18-26 years old (M =20.66, SD =2.179). Participants were split randomly between two conditions (18 stress, 18 control)**.** Informed consent was received from all participants. Participants were obtained from a non-clinical population and therefore were not at any identified risk from stress induction. Participants were awarded university credits or cash payment for their participation. All data were de-identified to ensure confidentiality. An independent ethics committee approved the experiment.

Design

The experiment used a between-subjects design, with participants randomly allocated between two conditions (18 stress, 18 control). The experimental design was also correlational. Independent variables were condition, voluntary visual imagery vividness and perceived stress reactivity. The dependent variable was acute stress response.

Materials

A modified version of the Trier Social Stress Test (TSST) (Kirschbaum et al., 1993) was used to induce stress. The Vividness of Visual Imagery Questionnaire (VVIQ) (Marks, 1995) was used to subjectively measure trait voluntary visual imagery (see Appendix A). Trait stress reactivity was measured using the Perceived Stress Reactivity Scale (PSRS) (Schlotz et al., 2011) (see Appendix B). Salivary cortisol was measured as a biomarker of psychological stress response (Choi et al., 2014) – this was analysed through ELISA kits, a precise, reliable tool (Shimada et al., 1995). Stress response was also assessed using a Visual Analogue Scale for Anxiety (VAS-A) - shown to be a reliable, valid, sensitive measure with good convergent validity (Cella and Perry, 1976; Facco et al., 2011; 2013; Hornblow and Kidson, 1976) (see Appendix C).

Procedure

Testing was carried out in the afternoon for all participants in order to control for the influences of circadian rhythm on resting cortisol levels (Thuma et al., 1995). Participants were required to abstain from eating and drinking 2-hours prior to arrival. Upon arrival, participants were given an information sheet about the study (see Appendix D), informing them they would be involved in a stress test. They were then required to sign consent forms (see Appendix E). Following this, there was a ten-minute waiting period to allow participant cortisol to return to resting levels. Participants were then questioned briefly regarding that day's smoking habits, exercise, illness and medication (see Appendix F for list of questions), as these factors are all known to influence cortisol levels (Direk et al., 2011; Hibel et al., 2006; Hill et al., 2008; Kirschbaum, Pirke and Hellhammer, 1995). Participants then completed a Visual Analogue Scale for Anxiety (VAS-A) and provided a saliva sample.

Participants in the stress condition were then taken into a second testing room to be introduced to the TSST, where they were introduced to the panel and told they were required to give a 5-minute speech for a job interview for a position at the UoM Psychology department, after which another task would follow. They were informed that the interview was video and voice recorded for analysis and that one panel member was trained in behavioural analysis and was taking notes (see Appendix G for experimenter script). Participants in the control condition were taken into the second testing room and introduced instead to the control task, which required them to speak about a topic of their choice for 5 minutes with friendly peers. In this condition, normal positive social cues from the panel were permitted although the participant was still encouraged to lead the discussion. Following introduction to the stress and control tasks respectively participants were returned to the initial testing room for a ten-minute preparation period where they were allowed to make notes.

After the 10-minute waiting period participants completed a second VAS-A. Control and stress condition participants then returned to the second testing room to have their discussion or give their speech respectively. The stress-condition speech was given unaided (without notes) to a panel of two unresponsive "job interviewers" who offered no positive social cues to the participant. Periods of silence were allowed for 20 seconds before a follow up question was asked (see Appendix H for interviewer script) After the speech/discussion participants completed a third VAS-A. Following

this, participants were instructed regarding and subsequently completed the second task. For the stress condition this was a 5-minute mental arithmetic task that involved counting backwards out loud in 13s from 1687. For the control maths task a booklet of simple addition and subtraction questions was completed by the participant (see Appendix I). Participants then completed a fourth VAS-A and provided a second saliva sample. Following this, both stress and control condition participants were returned to the initial testing room for a 10-minute resting period, after which they completed a final VAS-A and saliva sample. Participants in the stress condition were then debriefed regarding the nature of the stress test, that the job interview and behavioural analyst were fictitious, that the video camera and voice recorder were not functional and that the panel were deliberately unresponsive and abstaining from positive feedback with the purpose of provoking a stress response. Participants returned the following day, signed a second consent form and completed the VVIQ and the PSRS.

Data Analysis

The area under the curve for VAS-A (AUC anxiety) scores was found to estimate each individual's change in anxiety during the TSST or control task. An independent samples t-test was carried out between AUC anxiety for stress and control conditions to test the effectiveness of our modified TSST. A Pearson's correlation co-efficient on PSRS and AUC anxiety scores was used to assess the convergent validity of these two measures in the sample.

A median split was performed on VVIQ scores to form high and low VVIQ groups. PSRS scores were then tested for measures of normality, these were met and so analysis proceeded with parametric tests. An independent samples t-test was conducted with an independent variable of VVIQ group and dependent variable of PSRS. Pearson's correlation co-efficient was also conducted between PSRS and VVIQ scores.

For analysis involving AUC anxiety and cortisol-response scores data were split between stress and control conditions and stress data only used. AUC anxiety (stress) scores were tested for measures of normality. Measures of normality were met so analysis proceeded with parametric tests. An independent samples t-test was performed with an independent variable of VVIQ group (stress) and dependent variable of AUC anxiety (stress). Pearson's correlation co-efficient was then carried out between AUC anxiety scores (stress) and VVIQ scores (stress), and between cortisol-response (stress) and VVIQ scores (stress).

Normality of data was tested in each case by calculating the z-score for skewness (skewness/SEskewness) and kurtosis (excess kurtosis/SEexcess kurtosis), for a sample size < 50, z-scores < 1.96 were judged to be normal (Kim, 2013).

Due to the observed gender differences between conditions, exploratory analysis were carried out to assess the role of gender differences, if any.

Results

The independent samples t-test of AUC anxiety revealed there was a significant difference in the scores for stress (*M* =21.60, *SD* =4.81, SE =1.13) and control (*M* =12.68, *SD* =7.55, SE = 1.78) conditions; *t*(34)=3.76, *p* =.001, SE =2.11 and 95% CI [3.63, 12.21]. Table 1 shows descriptive statistics of number and age of participants in each gender between conditions. Pearson's correlation co-efficient of AUC anxiety and PSRS scores returned a significant moderate correlation between the two variables (r =.376, n =30, *p* =.026).

	Stress			**Control**		
	Females	**Males**	**Total**	**Females**	**Males**	**Total**
N	11	7	18	13	5	18
Age	20.50 (2.07)	20.57 (2.23)	20.53 (2.07)	20.73 (2.45)	21.00 (2.45)	20.80 (2.37)

[illegible]

An independent samples t-test revealed there was a not a significant difference in the PSRS scores for High VVIQ (*M* =23.83, *SD* =7.55, SE =1.78) and Low VVIQ (*M* =23.47, *SD* =7.71, SE =1.87) groups; *t*(33)=-.141, *p* = .731. PSRS was normally distributed, with skewness of -.221 (SE =3.98) and kurtosis of -.168 (SE =.778). Pearson's correlation co-efficient between PSRS and VVIQ was not significant (r =-.163, n =35, *p* =.349).

	[illegible]		[illegible]	
	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]
[illegible]	[illegible]	[illegible]	[illegible]	[illegible]

[illegible] R [illegible]

Table 2. shows the number and percentages of male and female participants in the high and low groups for VVIQ and PSRS scores. There was a disproportionately large number of women in the high scoring participant groups for both PSRS and VVIQ measures.

An independent samples t-test revealed there was no significant difference in the AUC anxiety scores between High VVIQ (*M* =19.94, *SD* =2.55, SE =.96) and Low VVIQ (*M* =21.02, *SD* =5.90, SE =1.78) conditions; *t*(15)=-.535, *p* = .601. Levene's test for equality of variances was significant, F =6.671, *p* =.020. Owing to this violation, a *t* statistic not assuming homogeneity of variance was calculated. AUC anxiety was normally distributed, with skewness of .022 (SE =.393) and kurtosis of -.363 (SE =.768). Pearson's correlation co-efficient between AUC anxiety scores (stress) and VVIQ scores (stress) showed no significant correlation between the two variables (r =-.280, n =18, *p* =.260).

Exploratory analyses were carried out to examine gender differences and findings within each gender separately. An independent samples t-test revealed there was no significant difference between VVIQ scores between men (M =52.08, SD =17.59, SE =5.08) and women (M =56.92, SD =10.44, SE =2.13); *t*(34)=-1.037, *p* =.307. An independent samples t-test of AUC anxiety (stress condition) revealed there was a significant difference between men (M =17.76, SD =2.60, SE =.98) and women (M =22.40, SD =5.10, SE =1.54); *t*(16)=-2.212, *p* =.042. An independent samples t-test revealed there was a significant difference in PSRS scores between men (M =18.08, SD =7.22, SE =2.08) and women (M =26.57, SD =5.95, SE =1.24); *t*(33)=-3.72, *p* =.001.

A pearson's correlation between VVIQ (stress) and AUC anxiety (stress) in women revealed a large negative correlation that was approaching significance (r =-.516, n =13, *p* =.071). The results of this correlation can be found in Figure 1.

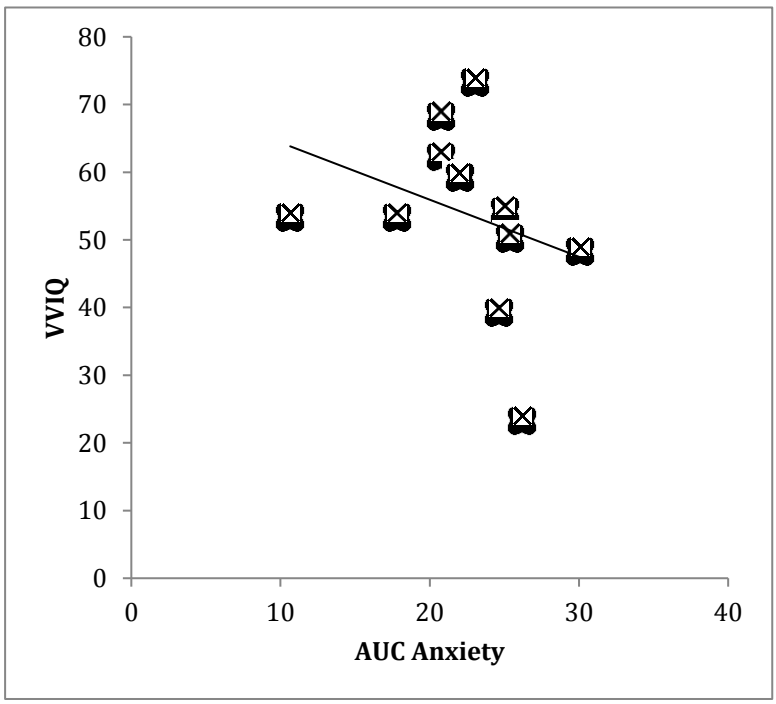

Figure 1. Scatter-graph of VVIQ and AUC anxiety scores in women in the stress condition.

A pearson's correlation between VVIQ and PSRS scores in women revealed a moderate negative correlation that was approaching significance (r =-.389, n =23, *p* =.060). The results of this correlation can be found in Figure 2.

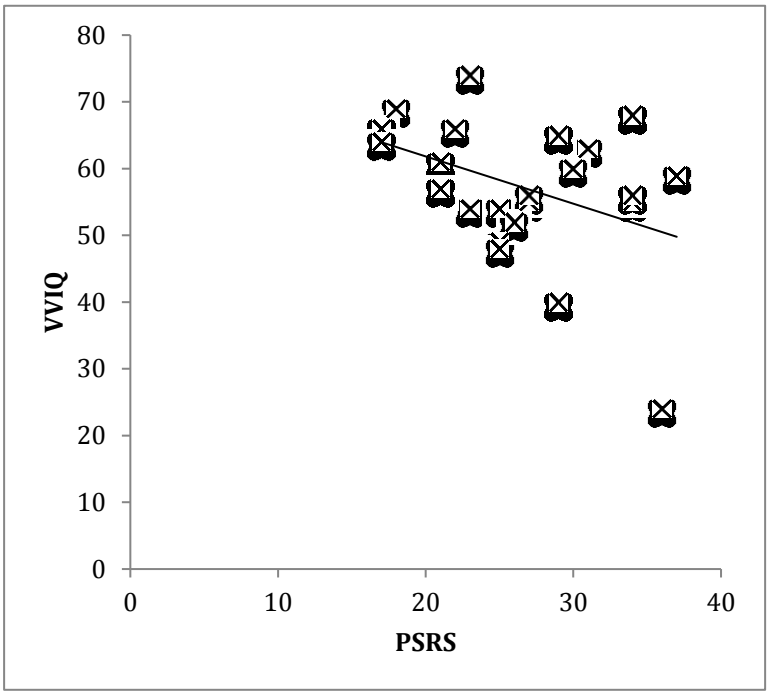

Figure 2. Scatter-graph of VVIQ and PSRS scores in women.

An independent samples t-test was performed on PSRS scores (women) between high VVIQ (women) and low VVIQ (women) groups. Levene's test for equality of variances was significant, F =4.395, *p* =.048. Owing to this violation, a *t* statistic not assuming homogeneity of variance was calculated. The t-test showed no significant difference in PSRS scores for low VVIQ (M =27.78, SD =4.44, SE =1.48) and high VVIQ (M =25.79, SD =6.80, SE =1.82) groups in women; *t*(20.97) =.850, *p* =.405. A median split was performed on PSRS scores to create a high PSRS and low PSRS group. An independent samples t-test revealed that there was a significant difference between VVIQ scores for high PSRS (M =53.44, SD =10.44, SE =2.61) and low PSRS (M =63.88, SD =6.45, SE =2.23) groups in women; *t*(22) =2.577, *p* =.017. Figure 3 shows mean VVIQ scores for high and low PSRS groups in women.

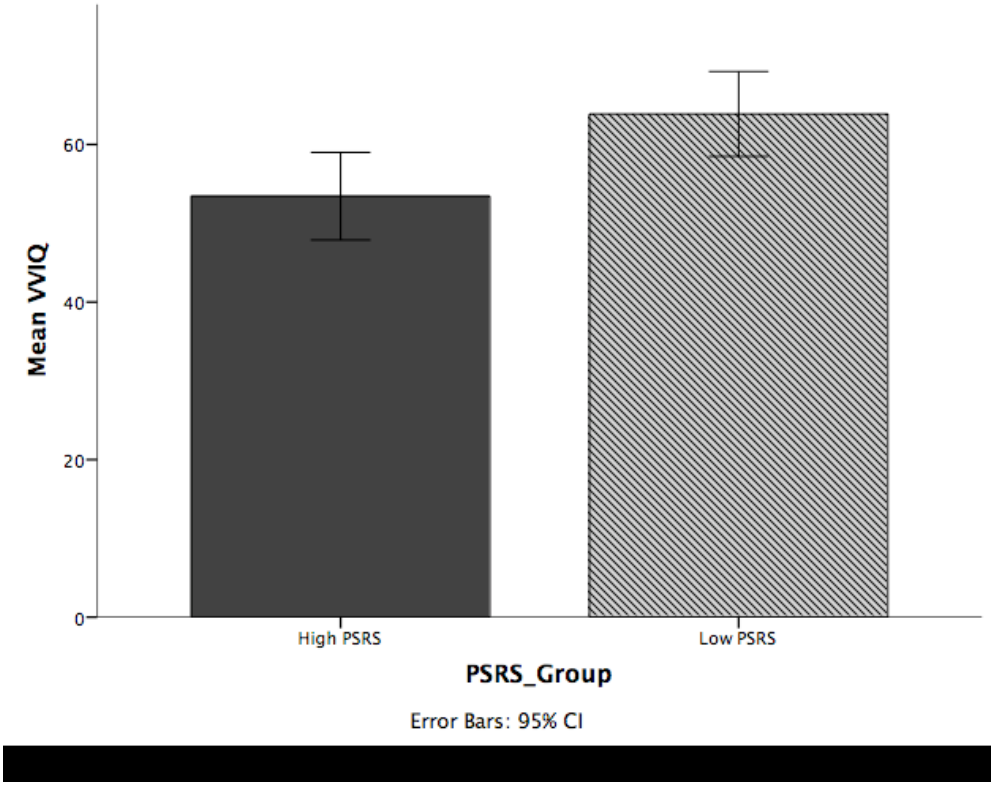

Figure 3. Mean VVIQ scores for high PSRS and low PSRS groups in women.

Discussion

Analyses of the relationship between trait stress reactivity and trait visual imagery were not significant. This finding suggests that there is no relationship between trait stress reactivity and trait voluntary visual imagery. Likewise, tests of the relationship between acute stress response and trait vividness of visual imagery were not significant. This suggests that there is also no relationship between an individual's stress response to an environmental stressor and their trait vividness of visual imagery. The independent samples t-test performed on self-report anxiety between conditions revealed a significant effect of the modified TSST in inducing a stress response in participants. This demonstrates that the lack of a relationship found between acute stress and visual imagery was not the result of inadequate stress induction by our task. The significant moderate correlation between measures of acute stress response and trait stress reactivity attests to the convergent validity of these measures in our sample.

The lack of relationship observed between visual imagery and both acute and trait stress is contrary to the proposed hypothesis devised based on previous research linking both stress and visual imagery to memory and emotion regulation (Ehlers et al., 2004; Foa et al., 1980; Freyd, 1994; Hassabis et al., 2007; Sierra, 2009; Starkman et al., 1981; Zeman et al., 2016). This may be in part due to the inability to apply clinical findings to healthy populations. However, the same rationale can also be made based on non-clinical data (Aydin, 2018; Dirkx, 2001; D'Argembeau and der Linden, 2006; Keogh and Pearson, 2011; Kosslyn et al., 1990; Moore et al.,

2008; Schwabe and Wolf, 2012). Sample size was determined by a power calculation to ensure it was sufficiently powered to detect a moderate effect, so inadequate power cannot be responsible for the finding of no relationship in the sample. Practical limitations meant that cortisol data were unable to be analysed in time for submission of this report, however, cortisol data would be expected to correlate strongly with self-report acute anxiety as obtained by the VAS-A scale (Saliba et al., 2016). As such, the absence of this data does not pose a serious limitation to this study. However, it is possible that gender differences may have obscured any effects between visual imagery and stress. With respect to gender differences, cortisol measurements may have been informative, given that research shows that men exhibit significantly larger salivary cortisol measurements following stress exposure in the TSST (Kudielka et al., 2007).

Gender differences were significant in the case of stress reactivity and acute stress response, with women scoring higher in each case. However, no significant gender differences were observed in visual imagery vividness. These gender differences led to a separate analysis of the relationship between stress and visual imagery in women only (men were a very small part of the sample, and revealed no interesting or significant findings). A significant difference in vividness of visual imagery was found between high and low stress reactive groups. This suggests that the hypothesised inverse relationship between voluntary vividness of visual imagery and stress reactivity may exist in women. The negative correlation between vividness of visual imagery and perceived stress reactivity in women was approaching significance, as was the negative correlation between vividness of visual imagery and acute stress response to the TSST in women, supporting this interpretation. Due to the smaller sample size created by splitting data based on gender (and by gender and condition for analysis involving acute stress response), these exploratory analyses are underpowered. Therefore, further investigation in women with a larger sample size is required to confirm whether or not these relationships would each reach significance respectively. However, given that stress response and stress reactivity are related constructs, the approaching significant correlation between both of them and visual imagery, along with the significant difference in visual imagery vividness found between high and low stress reactive groups, implies that this may not be a chance statistical occurrence – and that stress and vividness of voluntary visual imagery may indeed be inversely related in women. No significant difference was found in stress reactivity between high and low vividness groups. This suggests that whereas high or low stress reactivity can be used as a predictor of visual imagery vividness, visual imagery vividness cannot be used as a predictor of stress reactivity.

The finding that women have both a higher acute stress response and greater stress reactivity than men is supported by the consensus in the literature that women have a higher self-report anxiety than men (Milani et al., 2004; Piccinelli and Wilkinson, 2000; Schlotz et al., 2011). In contrast, previous findings show that women also exhibit more vivid visual imagery, however our data found no significant difference between genders in visual imagery (Campos and Sueiro, 1993; Richardson, 1995). This may be the result of these analyses being underpowered due to the gender split rather than a genuine absence of effect. The finding that women's VVIQ scores were

on average higher than men's supports this interpretation. These analyses generally support the understanding that our data is representative of the wider population.

Future research could investigate the relationship between stress and vividness of visual imagery in women specifically, with a larger sample, to enable firm conclusions to be drawn about the relationship between these two variables in this population. Investigation into these effects in men is also required but crucially this must be done separately, due to clear gender differences. Future research could also employ a state measure of visual imagery vividness such as a trial-by-trial subjective report as this has been shown to have significantly larger effect size estimates in correlations with neurological and behavioural measures than the VVIQ (Runge et al., 2017). A trial-by-trial methodology would also be capable of identifying transient changes in visual imagery vividness due to stress and help to infer causality. Alternatively, behavioural measures of imagery such as binocular rivalry would offer the benefit of not being susceptible to socially desirable responding, as the VVIQ has been shown to be (Allbutt et al., 2008; 2011). It is widely acknowledged in the literature that visual imagery is not an entirely unified construct and can be split into object and spatial imagery (Aydin, 2018; Blajenkova, Kozhevnikov and Motes, 2006a; 2006b; Vannucci and Mazzoni, 2009). Another potential direction for future research in this field may then be for the analysis of the relationship between stress and each of these distinct subtypes of visual imagery.

This investigation is not adequate to make such claims, but if it were found that low visual imagery vivdness is reliably associated with heightened stress reactivity, then low visual imagery vividness could represent a risk factor for stress-related disorders such as depression or anxiety. Indeed, one potential method for reducing stress in individuals and helping them avoid entering clinical cycles of stress could be the training of visual imagery to enhance vividness. One study attempted to train increased vividness of visual imagery and failed, however this study had only a 5-day time frame, which could be a serious limitation (Rademaker and Pearson, 2012). A study by Aleman et al. (2000) found that musically trained participants had superior auditory imagery ability for both musical and non-musical auditory imagery. Whilst evidence suggests these effects are not cross-modal, it does seem likely that if training of imagery is possible in one modality it could be in others (Aleman et al., 2000). However, this study was correlational and therefore cannot infer causality. It is possible that people with superior auditory imagery ability are more inclined to acquire musical training, for instance. Future research could investigate this question in the context of visual imagery; perhaps with photographic or visual art training. Blackwell et al. (2015) found that in depressives, practising generating positive imagery in response to ambiguous scenarios lead to an increase in positive involuntary imagery experienced in daily life, with beneficial behavioural outcomes and reduction in anhedonic symptoms of depression. Crucially, imagery vividness during the first session of the intervention was a strong predictor of the degree in ultimate reduction of depressive symptoms (Blackwell et al., 2015). Therefore, if a viable method of training increased vividness of visual imagery was achieved, this could be used in conjunction with a mode of therapy such as this to significantly enhance efficacy.

In conclusion, this research contributes to the study of visual imagery by being the first of its kind as a direct investigation into the relationship between visual imagery and stress in a healthy population. In addition, this study helps to clarify that research in this area should be gender specific. This is because there are significant differences between genders with regards to trait stress, acute stress and trait vividness of visual imagery, which may obscure effects if left uncontrolled for. This study demonstrated that no relationship between either acute or trait stress and vividness of visual imagery is found unless controlling for gender. Controlling for gender, however, indicates a possible negative relationship of vividness of visual imagery with both acute stress and trait stress reactivity in women only. This may have valuable theoretical and therapeutic implications for clinical psychology and the study of psychological wellbeing, as well as the continued understanding of both visual imagery and stress fields in their own right.

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