



Anger, psychopathology and cognitive inhibition: a study of UK servicemen

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Abstract

A link between anger and psychopathology, and aggression and cognitive impairment has been postulated. This study examined the relationships between anger, psychopathology, and neurocognitive function in a military sample of 136 men without overt brain disease. A battery of neurocognitive tests was administered, including the State-Trait Anger Expression Inventory (STAXI), which recognises different experience and expression components of anger. Significant positive correlations between anger and psychopathological measures were revealed, with the exception of anger control which exhibited a negative relationship. Correlations between anger measures and neuropsychological variables were weaker, few were significant, and no specific pattern emerged. Structural equation modelling indicated that psychopathological variables and their relationship with neurocognitive variables, rather than anger, contributed significantly to the model. We propose that anger appears to be a manifestation of non-specific psychopathology (anxiety and depression), and that any relationship between anger and cognitive function is likely to be mediated through depression.

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Anger is probably latent in all individuals and cultures (Kovacs, 2000). A large body of evidence exists to suggest that anger is related to a number of negative health outcomes (House, 2002; Peters, 2001; Tennant & McLean, 2001).

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1. Anger, depression and post-traumatic stress disorder (PTSD)

Psychodynamic formulations associate depression with anger turned inwards (Freud 1917[1958]) and this notion finds some support in recent studies (e.g. Biaggio & Godwin, 1987; Tschannen, Ducko, Margolis, & Tomazic, 1992). Indeed, Clay, Anderson, and Dixon, (1993) found inwardly directed anger to be a significant predictor of depression, while Kellner, Hennanders, and Pattak (1992) found depression to be a significant predictor of self reported anger inhibition. A number of studies also support the theory of catharsis, that the release of anger reduces depression (e.g. Wadsworth & Barker, 1976), although doubt has been cast on the effectiveness of therapeutic approaches employing catharsis (see Lewis & Bucher, 1992). Goldman and Haaga (1995) found that despite depressed people reporting higher levels of anger experience and suppression than their non-depressed counterparts, they often report lower or equivalent levels of anger expression. Indeed most studies show a rather complex association between depression and various expressions of anger (see Culbertson & Spielberger, 1996; Folkman & Lazarus, 1980; Thomas & Atakan, 1993).

Post-traumatic stress disorder (PTSD) is associated with depression and anxiety, yet the clinical picture is often characterised by irritability and anger (American Psychiatric Association, 1994). Riley, Treibel, and Woods (1989) investigated the relationship between anger/hostility and depression in normal people, depressed, and PTSD patients. They reported that both clinical groups scored more highly on anger experience than normals, with the PTSD group exhibiting the highest level of anger experience and expression, findings corroborated by Pitman, Orr, Forgue, de Jong, and Clairborn (1987) and Chemtob, Hamada, Roitblat, and Muraoka (1994) and most recently by Biddle, Elliott, Creamer, Forbes, and Devilly (2002). Riley et al also reported that the depressed group showed higher levels of anger suppression than the other two groups, who did not differ on this measure.

Summarising the above studies, it appears that there is a general association between anger and certain types of psychopathology including post-traumatic stress disorder, anxiety and depression, rather than a specific link between types of anger expression or experience and depressed mood.

2. Anger and neuropsychology

It is well documented that depression is associated with impaired neuropsychological performance (Hartlage, Alloy, Vasquez, & Dykman, 1993; Weingartner, Cohen, & Murphy, 1981). A similar association between anger and cognition is less well established. The research that addresses this issue comes from studies of aggression rather than anger per se. The clinical literature is replete with reports of increased aggression, lability of mood including anger, and impulsivity following frontal lobe lesions (Stuss & Benson, 1984; Damasio, 1994; Lishman, 1998;). Seguin, Phil, Tremblay, and Boulerice (1995) followed a cohort of boys from the age of 6–18, divided into one of three groups; stable aggressive, unstable aggressive, and non-aggressive. They found that the former group performed most poorly on a series of neuropsychological tests, and that the strongest association was with tests of “executive cognitive functioning”, presumed to reflect frontal lobe activity. Aggressive behaviour and executive function was investigated by Giancola, Martin, Tarter, Pelham, and Moss (1996) in a sample of 10–12 year old boys. The authors found that under circumstances of high provocation, poor executive function was associated with

aggressive behaviours, even after controlling for IQ. Lau and Pihl (1996) reported that, low executive functioning individuals failed to inhibit aggression in order to receive a reward. They also found that their high executive functioning group was able to successfully inhibit aggression which followed intoxication. Finally, Best, Williams, and Coccaro, (2002) showed that impulsive responding to an experimental gambling task characterised patients diagnosed with ‘intermittent explosive disorder’ in comparison to healthy controls.

Anger research has been facilitated by the development of a psychometric instrument which operationalises and quantifies different aspects of anger. The State-Trait Anger Expression Inventory (STAXI) developed by Spielberger (1996) distinguishes anger from other related concepts, and separates the experience of anger, conceptualised as comprising ‘state’ and ‘trait’ components, from the expression of anger, viewed as having three components, anger-in, anger-out, and anger-control.

The aim of the current study was to examine in more detail the relationships between anger, using the STAXI, and, on the one hand, psychopathology (symptoms of depression and PTSD), and on the other, specific neurocognitive functions in the absence of overt brain disease. It was hypothesised that (a) higher levels of anger would be positively associated with both elevated depression and PTSD scores; and (b) higher levels of anger would be associated with poorer cognitive performance on tasks which appear to be related to such executive processes as inhibition of inappropriate responses, freedom from distraction and maintenance of set.

3. Method

3.1. *Subjects and subject selection*

Subjects were selected from a cohort comprising of 8195 individuals who had returned a completed health questionnaire during the first phase of a study into the health of UK servicemen (Unwin et al., 1999). The sample was drawn from three groups: veterans of the 1990–1991 Persian Gulf War, a group who were serving at the same time, but who were not deployed to the Gulf, and group who had served in Bosnia during peacekeeping operations after April 1992. A case definition for ill health was defined as a score in the range of the lowest centile of the non-deployed group on the physical functioning subscale of the SF-36 (Ware, Snow, Kosinski, & Gandek, 1993). The current study comprised a random sample of 136 male subjects, “cases” and “non-cases”, from all three groups who agreed to undergo a physical and psychological assessment as part of Phase 2 of the study.

Participants were given a battery of tests designed to measure overall intellectual competence as well as aspects related to response control. Socio-demographic data were also gathered, including information regarding alcohol and cigarette consumption. The battery took approximately 2–3 h, and was administered in a standardised order, the research assistant being blind as to the cohort status of the participants. The following tests were used:

3.2. *Psychopathology instruments*

- *STAXI* (Spielberger, 1996)—a 44 item self-report questionnaire which produces six scales and two subscales. Each item is rated on a four-point scale from ‘almost never’ to ‘always’. State anger refers to the current feelings experienced by an individual, such as “I am

furious”, and “I feel like breaking things”. Trait anger refers to an individual’s disposition to experience anger and comprises items like “When I get mad I say nasty things”, and “I am quick tempered”. Trait anger items are used to produce two subscales: trait anger-T, which measures an individual’s disposition to experience anger without provocation (“I am a hotheaded person”), whereas trait anger-R, refers to the disposition to experience anger when provoked (“I get angry when I am slowed down by other’s mistakes”).

- The expression of anger is viewed as having three components; Anger in, measures the tendency for an individual to suppress angry feelings, e.g., “I am angrier than I am willing to admit”. Anger out, measures the tendency to outwardly express anger towards people or objects, such as “I lose my temper”. Anger control, refers to attempts to control angry feelings (e.g. “I calm down faster than other people”). The anger expression scales are then used in the following calculation (anger-in + anger-out – anger control + 16) to produce AX-EX, an index of anger-expression regardless of anger-expression style.
- *Beck Depression Inventory* (BDI; Beck & Steer, 1993)—range 0–63; a self-completion questionnaire designed to measure the severity of depression.
- *Mississippi Scale for Combat Related Posttraumatic Stress Disorder* (Keane, Cadell, & Taylor, 1988)—39 item self-report instrument adapted to pertain to the three groups under study.

3.3. Cognitive measures

3.3.1. General

- *Wechsler Adult Intelligence Scale—Revised* (WAIS-R; Wechsler, 1981)—an estimate of current IQ. Verbal subtests: Vocabulary, Digit Span, Arithmetic, Similarities; Performance subtests: Picture Arrangement, Block Design, Object Assembly; Digit Symbol.
- *National Adult Reading Test* (2nd ed.) (NART; Nelson, 1991)—a reading test of ‘irregular’ words, which gives a stable estimate of pre-morbid IQ.
- *Letter Number Sequencing task* (WAIS III; Wechsler, 1997)—a sequence of letters and numbers are presented verbally and the subject is required to reorder the sequence, giving numbers first in numerical order, and then letters in alphabetical order.
- *Wechsler Memory Scales* (WMS-R; Wechsler, 1987): Logical memory Immediate and Delayed Recall; Verbal Paired Associates Immediate and Delayed Recall; Visual Paired Associates Immediate and Delayed Recall. Raw scores were entered in to the analysis.
- *Camden Recognition Memory Tests* (Warrington, 1996): Faces and words, each scored out of 25.
- *Cognitive Failures Questionnaire* (CFQ—Broadbent, Cooper, Fitzgerald, & Parkes, 1982)—range 0–100; a 25 item self-completion instrument, to assess the frequency of self-reported errors in everyday cognition.

3.3.2. Specific

- *Paced Auditory Serial Addition Task* (PASAT; Gronwall, 1977)—60 single digits are presented auditorily (1 digit per 2 seconds.). Subjects are asked to add each digit to the one that comes before it and to give their answer aloud before the next digit has been auditorily presented. The score is the number of correct responses given (max. 60).

- *Sustained Attention to Response Task* (SART; Robertson, Manly, Andrade, Baddeley, & Yiend, 1997)—a computer administered vigilance task where 225 single digits are presented visually over 4.3 min. A score representing the number of errors of commission (max. 25) is calculated, i.e. the number of ‘3s’ mistakenly responded to. Also, mean reaction time (RT) for correct responses is calculated. (i.e. pressing the mouse for numbers other than 3).
- *Stroop Neuropsychological Screening Test* (Trenerry, Crosson, Deboe, & Leber, 1989)—a colour-word naming test. A Stroop effect was calculated, based on the difference in time to name the colour of ink of incongruent colour-word stimuli minus the reading of words written in congruent ink.
- *Trail Making Test, Parts A and B* (Reitan & Wolfson, 1985)—a test of motor sequencing and set shifting. The time (s) to complete the simple A trail and the alternating B trail were recorded, and the difference (B–A) was also taken as a dependent measure.

4. Results

4.1. Subject characteristics

The sample comprised 136 males, age ranging between 22 and 58 years; mean (SD) 36.4 (7.85) years. The means and standard deviations for the study sample and published norms are reported in Table 1. Means were compared using *t*-tests in STATA (STATA Corp. 1997). Results indicated the study and normative samples were significantly different for each of the anger scales, with the exception of state and trait anger. The study sample had significantly higher trait anger T, anger in, anger out, and AX-EX, and lower trait anger R, and anger control than the normative sample.

The Pearson’s correlations between each of the anger scales themselves are reported in Table 2. The statistics indicate moderate to strong positive correlations ($r = 0.27–0.89$), with the exception

Table 1
Means, standard deviations, and *t*-test statistics for the study and normative samples

STAXI scales	Sample					
	Study ^a		Normative ^b		<i>t</i> -Test ^c	
	<i>M</i>	SD	<i>M</i>	SD	<i>T</i>	<i>P</i>
State	10.0	2.5	11.3	3.2	–1.16	0.25
Trait	18.8	5.0	18.7	4.8	0.42	0.67
Trait-T	6.7	2.0	6.2	2.5	1.96	0.05
Trait-R	8.7	2.7	9.3	2.6	–2.67	0.008
Anger-in	17.9	4.6	15.4	3.9	7.21	<0.001
Anger-out	15.2	4.1	14.4	3.3	2.69	0.007
Anger-control	23.4	5.4	26.2	4.3	–7.33	<0.001
AX-EX	25.5	10.3	19.4	7.4	9.20	<0.001

^a $n = 136$ males.

^b $n = 2880$ adult males, mean age 40 years (18–67 years).

^c *t*-Test figures are for two-tailed tests.

Table 2
STAXI scale intercorrelations (Pearson's r) for the sample

	1	2	3	4	5	6
1. Trait	–					
2. Trait-T	0.89	–				
3. Trait-R	0.80	0.49	–			
4. Anger-in	0.46	0.31	0.52	–		
5. Anger-out	0.74	0.76	0.45	0.27	–	
6. Anger-control	–0.55	–0.56	–0.32	–0.14	–0.58	–
7. AX-EX	0.75	0.71	0.53	0.62	0.79	–0.78

All $P < 0.01$.

Table 3
Sample means and standard deviations for selected tests^a

Test Variable	Mean	SD
BDI Total Score	10.1	8.8
CFQ Total Score	46.7	16.7
Mississippi Total Score	81.4	24.8
WAIS FSIQ	102.3	11.2
Letter Number Sequencing	12.0	2.5
NART Predicted FSIQ	101.9	10.3
Camden Memory Test;		
Faces	24.5	0.9
Words	24.3	1.1
Wechsler Memory Scale;		
Logical Memory 1	25.4	5.7
Logical Memory 2	20.9	6.5
Verbal Associates 1	19.5	3.5
Verbal Associates 2	7.6	0.8
Visual Associates 1	14.3	3.2
Visual Associates 2	5.6	1.0
Stroop Colour Task ^b ;		
Correct responses	111.9	0.4
Time on colour task	54.5	9.4
Stroop Colour-Word Task ^b ;		
Correct responses	104.9	11.0
Time on colour task	111.7	11.1
SART		
False Presses	9.9	6.9
Trail Making Test;		
Total time on part A	27.5	8.0
Total time on part B	58.6	16.6

BDI, Beck Depression Inventory; CFQ, Cognitive Failures Questionnaire; WAIS FSIQ, Wechsler Adult Intelligence Scale Full Scale IQ; NART, National Adult Reading Test; SART, Sustained Attention to Response Task.

^a $N = 136$ or less (not all subjects completed the full battery).

^b Number of correct responses and time within 120 s.

of anger control, which is negatively correlated ($r = -0.14$ to -0.58) to each of the other anger scales.

Table 3 gives the means and standard deviations for administered and self-completion scales. With regard to BDI scores, 41.9% ($n = 54$) of respondents scored above the authors' suggested cut-off of 9 for mild depression. However of these, nearly half (46.3%, $n = 25$) were categorised as having mild depression and only 5.6% ($n = 3$) fell within the category of severe depression. Of the 112 respondents who completed the Mississippi scale (Keane et al., 1988), 16 (14.3%) scored above the cut-off of 106 for a probable PTSD diagnosis.

4.2. Anger and psychopathology

The results of Pearson's correlations between BDI and Mississippi for each of the STAXI scales are presented in Table 4. State anger was excluded from analysis due to the lack of variance. Correlation coefficients were found to be moderate, ranging from -0.36 to 0.56 for BDI, and from -0.45 to 0.60 for the Mississippi. AX-EX and trait anger had the strongest correlations with BDI and Mississippi scores (r 's > 0.56). All anger scales were positively correlated with both the BDI and the Mississippi, with the exception of anger control, which was negatively correlated. Therefore, increased levels of trait anger, anger in, anger out, and overall anger expression, are associated with higher levels of depression and PTSD scores, apparently supporting our first hypothesis

Correlations between each of the STAXI scales with alcohol and cigarette consumption did not reach statistical significance.

4.3. Anger and cognitive performance

Table 5 reports correlation coefficients for each of the STAXI sub-scales, and the cognitive performance variables. A number of significant correlations were found. For example, each STAXI scale correlated significantly with WAIS FSIQ and Performance IQ, with the exception of anger-in, which was not significantly correlated to either. Overall, anger-control correlated with superior cognitive performance compared to the other anger parameters which in general correlated negatively with performance. Most of the correlations were however, modest.

Regarding measures which weighted more heavily on attention and set shifting/maintenance, all correlations were negative (i.e. more anger, worse attention), with the exception of anger-control.

Table 4
Pearson's correlation coefficients between the STAXI scales and the BDI, and Mississippi

STAXI variable	BDI score ($n = 129$)	Mississippi score ($n = 111$)
Trait anger	0.58	0.60
Trait anger-T	0.56	0.60
Trait anger-R	0.37	0.34
Anger-in	0.50	0.40
Anger-out	0.44	0.50
Anger-control	-0.36	-0.45
AX-EX	0.56	0.59

All $P < 0.01$ for a one tailed test.

Table 5

Pearson correlation coefficients for selected neuropsychological variables and each anger scale

Test variable	<i>n</i>	Trait	Trait-T	Trait-R	Ang-In	Ang-out	Ang-con	AX-EX
CFQ	133	0.53**	0.49**	0.33**	0.39**	0.41**	-0.28**	0.45**
WAIS-R FSIQ	135	-0.34**	-0.33**	-0.19*	-0.08	-0.27**	0.29**	-0.26**
NART FSIQ	135	-0.15*	-0.23**	-0.05	0.05	-0.16*	0.25**	-0.17*
PASAT ^a	131							
no correct		-0.19*	-0.16*	-0.10	-0.14	-0.15*	0.13	-0.19*
longest string		-0.17*	-0.13	-0.11	-0.21**	-0.10	0.09	-0.18*
no missed		0.16*	0.14	0.06	0.12	0.13	-0.12	0.17*
SART	130							
No correct		0.06	-0.01	0.11	-0.03	-0.00	-0.03	-0.10
False presses		0.21**	0.18*	0.19*	0.23**	-0.00	-0.17*	0.19*
Stroop ^b	134							
Total time		0.20*	0.18*	0.19*	0.24**	0.07	-0.16*	0.20**
Stroop ^c	132							
no correct	^d	-0.22*	-0.20*	-0.17*	-0.18*	-0.18*	0.09	-0.17*
total time		0.20*	0.18*	0.18	0.18	0.12	-0.07	0.15
TMT	135							
Time (Part A)		0.09	0.12	0.06	0.15*	0.13	-0.12	0.16*
Time (Part B)		0.06	0.15*	-0.10	0.05	0.06	-0.03	0.08

CFQ, Cognitive Failures Questionnaire; WAIS-R FSIQ, Wechsler Adult Intelligence Scale-Revised Full Scale IQ; NART FSIQ, National Adult Reading Test Full Scale IQ; PASAT, Paced Auditory Serial Addition Task, SART, Sustained Attention to response Task; TMT, Trail Making Test.

^a Two second trial.

^b Word task

^c Colour word task.

^d Number correct within 120 s

* $P < 0.05$, one-tailed.

** $P < 0.01$, one-tailed.

No significant correlations were found between the STAXI scales and Trail Making Test B, or the increased time taken to complete Trails B in comparison to Trails A. In addition, the STAXI scales, with the exception of anger-out, correlated significantly with SART false presses, and total time taken to complete the Stroop task. However, no significant correlations were found between each of the STAXI scales and Stroop effect (time taken to complete colour-word task minus the time taken to complete the word task), although they did correlate with number of correct responses, also a measure of cognitive inhibition. Significant moderate correlations were found between each of the STAXI scales and the CFQ. All correlations were positive, apart from the correlation with anger control, which was negative.

4.4. Structural equation modelling (SEM)

To further investigate the relationships between anger, depression and the other variables measured in the study, a number of structural equation models were fitted. Such models are described in detail in Dunn, Everitt, and Pickles (1993). In brief, they involve postulating a number of latent variables thought to be responsible for generating the associations between the

manifest variables. In addition, the investigator needs to specify how the latent variables themselves are related. The parameters of the postulated model, i.e. the loadings of observed variables on latent variables, error variances, correlations and regression coefficients amongst the latent variables, are then estimated by a procedure which makes the correlations between the manifest variables, as predicted by the model, as close as possible to the observed correlations between these variables, where “closeness” can be measured by a variety of criteria that give rise to different estimation procedures. Here maximum likelihood is used. Standard errors of the parameter estimates also result from the fitting process as do a number of measures of fit of the model under consideration (see Dunn et al., 1993). The correlation matrices on which the model fitting is based (from 96 subjects who had data on all 24 variables) are available from the authors on request. We are well aware that a larger sample size is generally recommended for SEM, but felt that it was worthwhile carrying out the model fitting process to assess what insights it might give to the structure of the data. Nevertheless all the results need to be interpreted cautiously given the small sample size.

The structural equation model of interest can often be usefully displayed in the form of a path diagram, indicating the postulated links between latent variables and between latent variables and manifest variables. Here the path diagram of the first model to be considered, based on a priori assumptions from the psychology literature, is shown in Fig. 1. The latent variables, anger and depression are assumed to be correlated, and the other latent variables, cog1 (attention), cog2 (memory) and disinhibition are regressed on both. The observed variables assumed to be indicators of each latent variable are shown in Fig. 1.

The parameter estimates and their standard errors are shown in Table 6. (The loading of one of the observed variables on each latent variable is set at one to fix the scale of the variable; see Dunn et al., 1993, for details). The various measures of fit of the model [chi-squared = 361.15, $df = 180$, $P < 0.001$, Bentler-Bonett Normal Fit Index (BBNFI) = 0.657, Bentler-Bonett Non Normal Fit Index (BBNNFI) = 0.749, Comparative Fit Index (CFI) = 0.785] (all of which are described in detail in Dunn et al., 1993) suggest that the model does not account for the observed correlations at all well (the fit indices, for example, need to be 0.9 or above to claim a reasonable fit). The regression coefficients linking cog1, cog2 and disinhibition to anger and depression are not impressive, particularly those for anger. The estimated correlation for anger and depression, 0.41 is moderate.

If the regression paths between anger and cog1, cog2 and disinhibition are removed from the model, the revised goodness-of-fit statistics are as follows: chi-squared = 362.60 ($df = 183$, $P < 0.001$), BBNFI = 0.656, BBNNFI = 0.756, CFI = 0.787. Dropping those three paths causes no significant deterioration in fit. But if the regression paths between depression and cog1, cog2 and disinhibition are dropped from the original model, the new goodness-of-fit information suggests that the fit gets substantially worse (chi-squared = 377.00 ($df = 183$, $P < 0.001$), BBNFI = 0.642, BBNNFI = 0.736, CFI = 0.770). Table 7 provides the regression coefficients estimated for both the original (model 1) and this new model (model 2).

5. Discussion

The purpose of this study was to investigate the relationship between anger and measures of depression, PTSD, and cognitive performance within a male military sample. Although the

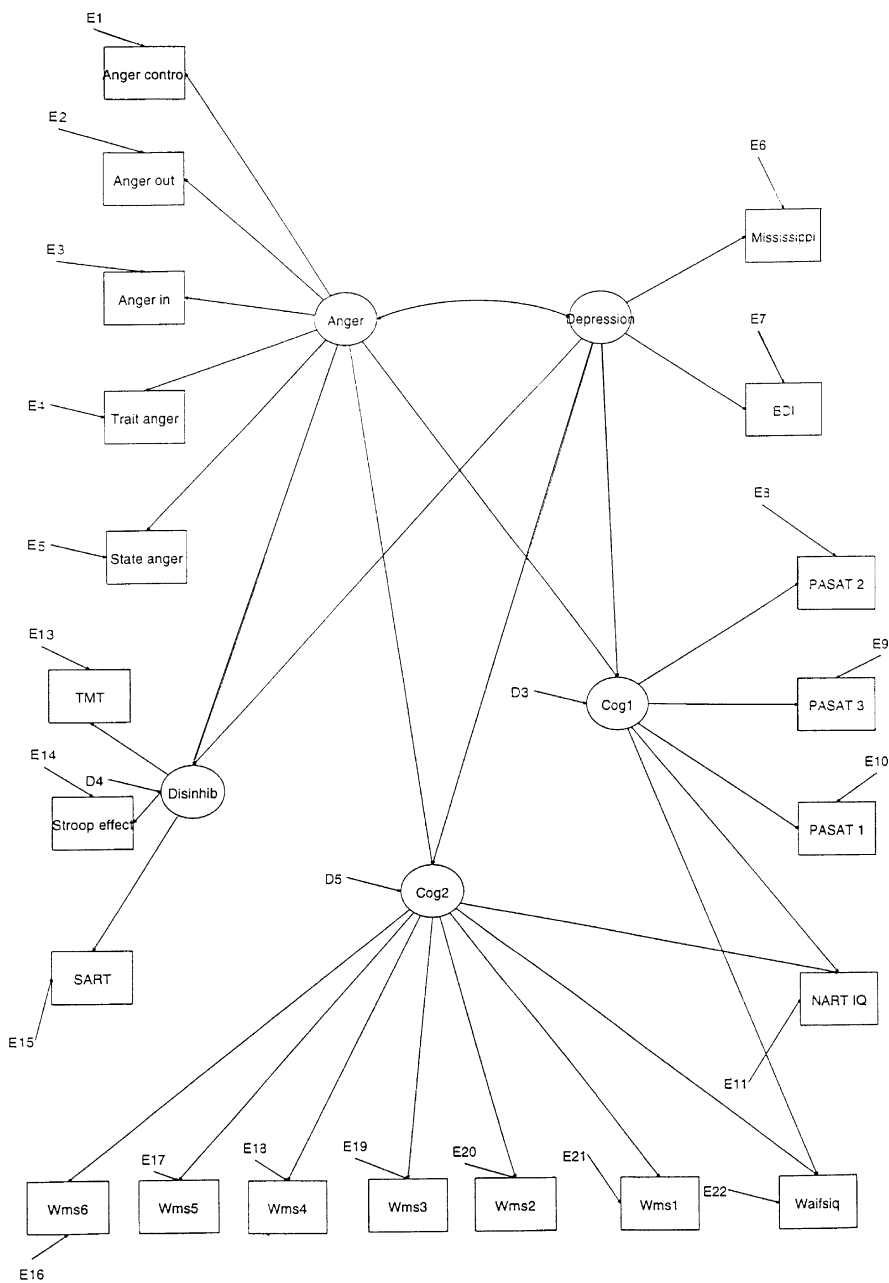


Fig. 1. Path diagram for anger, disinhibition, depression and cognition. *Notes:* Mississippi, Mississippi Scale for Combat Related Posttraumatic Stress Disorder (total score); BDI, Beck Depression Inventory (total score); PASAT Paced Auditory Serial Addition Task (2 second trial), PASAT 1, longest unbroken string, PASAT 2, number of errors, PASAT 3, number missed; NART FSIQ, National Adult Reading Test predicted full scale IQ; WAIS FSIQ, Wechsler Adult Intelligence Scale full scale IQ; WMS, Wechsler Memory Scale, WMS 1, Logical Memory Immediate Recall, WMS 2, Logical Memory Delayed Recall, WMS 3, Verbal Associates Immediate Recall, WMS 4, Verbal Associates Delayed Recall, WMS 5, Visual Associates Immediate Recall, WMS 6 Visual Associates Delayed Recall, TMT, Trail Making Test (time B–time A). Rectangles: observed variables. Circles: latent variables.

Table 6
Parameter estimates for the model represented in Fig. 1

Latent variable	Observed variable	Estimated loading	S.E.	Estimate/S.E.
Anger	Anger-control	1.00 (fixed)	–	–
	Anger-out	1.19	0.19	6.32
	Anger-in	0.79	0.18	4.47
	Trait	1.41	0.20	6.92
	State	0.74	0.18	4.20
Depression	Mississippi	1.00 (fixed)	–	–
	BDI	0.92	0.09	10.44
Cog1	Pasat 3	1.00 (fixed)	–	–
	Pasat 2	–0.04	0.13	–0.35
	Pasat 1	0.95	0.13	7.30
	Nart IQ	0.51	0.12	4.33
	WAIS IQ	0.55	0.11	4.95
Cog2	WMS 6	1.00 (fixed)	–	–
	WMS 5	0.67	0.32	2.09
	WMS 4	1.18	0.39	3.00
	WMS 3	1.16	0.39	3.00
	WMS 2	2.63	0.69	3.83
	WMS 1	2.44	0.64	3.82
	Nart IQ	0.66	0.30	2.23
	WAIS IQ	0.95	0.33	2.86
Disinhibition	TMT	1.00 (fixed)	–	–
	Stroop Effect	1.03	0.66	1.52
	SART	1.72	0.95	1.81

Refer to notes to Fig. 1 for explanation of test variables.

nature of the sample may limit generalisation, the concept of “controlled anger” would appear to be highly salient in a military context.

In support of our first hypothesis, anger experience and expression were found to be significantly and positively correlated with depression. This was true using simple correlations and in the structural equation modelling. The strongest correlation was with “anger-in”, supporting previous research which identifies an association between the suppression of anger and depression (i.e. Kellner et al., 1992; Riley et al., 1989; Clay et al., 1993). However, in contrast to studies which have found support for the theory of catharsis, and those which suggest that depressed individuals report lower levels of anger expression, anger-out was also found to positively correlate with depression (see also Thomas & Atakan, 1993). PTSD was also found to positively correlate with anger experience and expression, corroborating previous research (Chemtob et al., 1994; see also Biddle et al., 2002). The strength of the associations between PTSD score and each of the anger expression styles were similar, indicating that no one anger style stood out as more important than the others.

Table 7
Regression coefficients for two models

	Latent variables		
	Cog 1	Cog 2	Disinhibition
<i>Model 1^a</i>			
Anger	−0.12	0.09	−0.04
S.E.	0.23	0.09	0.12
Parameter/S.E.	−0.054	0.99	−0.37
Depression	−0.18	−0.18	0.23
S.E.	0.15	0.08	0.13
Parameter/S.E.	−1.17	−2.30	1.80
<i>Model 2^b</i>			
Depression	−0.34	−0.11	0.18
S.E.	0.16	0.07	0.15
Parameter/S.E.	−2.10	−1.53	1.22

^a Refers to the initial model shown in Fig. 1.

^b Refers to the model in which the regression paths between depression and cog1, cog2 and disinhibition have been dropped.

Correlational analyses provided minimal support for our second hypothesis, predicting that higher levels of anger would be associated with poorer cognitive performance on specific tasks related to response control and inhibition. Despite the direction of the majority of correlations between the anger scales and cognitive performance variables being indicative of a relationship, only a few were found to be significant, and there was no specific pattern. The coefficients were also much weaker than for the psychopathological measures, with the exception of the CFQ, which is not a direct measure of cognitive performance and has previously been found to be strongly associated with measures of anxiety and depression (Binder, Storzbach, Anger, Kent, Campbell, & Rohlman, 1999; Gass & Apple, 1997; Wagle, Berrios, & He, 1999).

A consistent finding which contradicts the hypotheses, and the findings of Culbertson and Spielberger (1996), is that, unlike most anger measures, higher anger control scores were associated with lower levels of depression, PTSD, and improved cognitive performance. This could be due to a number of reasons. Firstly, it may reflect the nature of the questions making up the anger control item, which may add up to an effective strategy for dealing with anger resulting in the reduction of anger experience, rather than an anger expression style per se. Second, it is possible that anger dealt with effectively may in fact be functional. Indeed, a distinction between “healthy” and “unhealthy” anger has been made (Ellis, 1973). Thirdly, those scoring high on anger control may actually be experiencing very little anger in the first instance.

The anger scales were found to be more strongly correlated with other psychopathology measures than with measures of cognitive functioning and inhibition. It is, therefore, not surprising that the structural equation modelling found anger and its relationship to the cognitive variables contributed very little to the goodness of fit of the overall model. Indeed, omitting these from the analysis failed to cause a significant deterioration of fit. Rather, what was found to be central to the goodness of fit was the relationship between depression (including PTSD) and the cognitive

variables, both general and specific. Indeed, we would expect that depression and cognitive functioning are inversely associated (Hartlage et al., 1993; Weingartner et al., 1981). Given previous research and results from the current study supporting an association between depression and anger, it is reasonable to suggest that any relationship between anger and cognitive function, if it exists, is likely to be mediated through depression. Anger may serve to amplify the effects of depression on cognition, but alone is associated with minimal cognitive dysfunction.

However, there may be methodological factors which contribute to lack of support for a direct relationship between anger and cognitive measures, particularly inhibition. The measures put forward as being specific to cognitive control, response inhibition and maintenance of set, were limited in this regard. The Trails (especially the B version) confounds set maintenance and set shifting and could be regarded as primarily a motor sequencing task. It could be argued that the SART involves aspects of response inhibition, requiring the subject to withhold a pre-potent manual response. The anger scales were found to significantly correlate albeit modestly, with SART false presses. However, Robertson et al., (1997) proposed that the SART relies more heavily on the ability to continuously sustain attention rather than the capacity to inhibit a response. The Stroop colour-word test, appears at face value to require inhibition of task-irrelevant information. Length of time taken to complete the Stroop word task as well as accuracy were found to be associated with anger, variables which are perhaps indirect measures of inhibition.

The use of the Stroop, however, as a measure of cognitive inhibition may also be problematic. Visser, Das-smaal, and Kwakman (1996) investigated the relationship between social and cognitive impulsivity (rather than anger) with lack of cognitive inhibition in children, using both the Stroop interference effect and negative priming paradigms. Although they found reduced negative priming for social impulsives, the two groups were not distinguishable on the basis of the Stroop test. The authors suggested that the Stroop is an insufficiently sensitive measure of cognitive inhibition, at least in children.

The relationship between impulsivity and anger is another area where confounding could be a problem. Chemtob et al. (1994) investigated the idea that PTSD symptoms overlap with anti-social tendencies reflecting impulsivity (Helzer, Robins, & McEvoy, 1987). Despite finding that the groups under investigation reported differing levels of anger, Chemtob et al. claimed that groups did not differ in terms of impulsivity. Furthermore, anger and impulsivity measures were not found to be positively correlated.

Other studies on anger and impulsivity used different “executive function tests”, such as non-spatial conditional association, self-ordered pointing, and strategic problem solving (Seguin et al., 1995) and this could explain study differences. Also, it appears that adolescent boys (Giancola et al., 1996) and girls (Giancola, Mazzich, & Tarter, 1998) with, or at high risk of developing psychosocial difficulties, may demonstrate such relationships more than adults for executive functions. The latter may have since developed more effective mechanisms for inhibiting anger and aggression. Having said that, normal adults selected for aggressive traits have been shown to exhibit impairment on specific cognitive tasks (Giancola & Zeichner, 1994) and our results offer modest support for this general notion..

Another explanation for our inability to discern a clear association between anger and neuropsychological measures using simple or sophisticated statistical techniques could lie in the psychopathology measures used. These were self-report instruments which are prone to social desirability effects, especially in the context of anger research. However, the overall scores were in

line with published norms and differences in our sample (all well within one standard deviation from the mean) tended to be in the direction of greater anger expression and less control. Such differences could reflect the nature of the military sample, many of whom were selected on the basis of poor physical functioning. Although the sample displayed a wide range of anxiety, depression and PTSD scores, their overall levels of depression and PTSD were low. So, even with more refined tests of executive function, the deficits in question are likely to be subtle and quantitative unlike the deficits seen in patients with overt brain lesions or major psychiatric disorder. While we do not think this undermines the validity of the study of individual differences, extrapolations to clinically affected groups should be made with caution. Finally, inferences about causality are constrained by the cross sectional nature of the study.

In conclusion, we have found that in young and middle aged men in the military, anger appears to be a manifestation or mode of expression of associated with certain kinds of psychopathology, which includes depressed mood and symptoms of post traumatic stress (as measured by the BDI and Mississippi Scales, respectively). There is little hint that the various aspects or modes of expression of anger behave differently in this respect with the exception of anger control which correlates negatively with psychopathology and cognitive performance. In addition we have started to explore the neuropsychological correlates of anger and a specific inverse relationship between response control or cognitive inhibition was sought, but this failed to emerge unequivocally. Hence at present there is no sound basis to view such laboratory indices of *cognitive* control such as freedom from distraction, maintenance of set, and inhibition of pre-potent responses, as equivalent to *behavioural* control. Instead, depressed mood and anxiety symptoms are related to both general and specific neurocognitive measures so that any relationship between these and anger appears to be mediated through depressed mood.

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