Positive (But Not Negative) Punishment Predicts Anxiety and Depression Among Prostate Cancer Patients: An Exploration of the Behaviour Analytic Model of Depression

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The relative power of Positive and Negative Punishment as predictors of anxiety and depression was investigated within the gender-specific population of Prostate Cancer patients. As well as being a more powerful predictor of total test scores, Positive Punishment was also a stronger predictor of the presence of clinical levels of anxiety and depression. Examination of the particular Positive Punishment events that were significantly associated with clinical anxiety and depression showed considerable overlap, supporting the concept of a combined anxiety-depression disorder. Suggestions for behavioural interventions with this patient group are made.

Keywords: punishment, anxiety, depression, prostate cancer

In his description of depression as a series of self-induced avoidance behaviours emitted in the face of either the onset of aversive environmental events or loss of previously available social reinforcers, Ferster (1973) argued that the primary withdrawal behaviour that underlies depression manifested itself via (for example) crying, psychomotor retardation, cognitive slowing, complaints and reduced social interactions with others. By highlighting the key role that disturbances in positive social relationships played within depression, Barnett and Gotlib (1988), emphasised the *withdrawal-as-adaptation* construct suggested by Ferster (1973). The functionality of depression-based withdrawal behaviours has been further explicated by Dougher and Hackbert (1994), who expanded the behaviour-analytic model of depression to include the symptomatology that currently comprises the more common models of depression (e.g., DSM-IV-TR: APA, 2000).

The presence of adaptation responses within psychopathologies has been described by a series of papers. For example, Gilbert (1998, 2006) conceptualised psychological disorders as arising because of compromises between competing needs within the individual that could not be resolved, some of which included withdrawal from threat. Dixon (1998) referred to the notion of 'arrested flight', seen among other animals as a potential explanatory model for the withdrawal and anhe-

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donia responses shown in depression. Price (1998) also described depression as adaptive because it assisted the individual to de-escalate danger, exemplified in 'playing dead', shown in many mammals when confronted with an inescapable predator. In humans, this 'playing dead' response provided time for the threatened individual to rethink the situation and their responses to it, as well as allowing others to take control and 'save' the individual. Nesse (1998: pp. 402–406) described ten ways in which withdrawal can provide a benefit after the loss of a source of valued social reinforcement. Perhaps most graphic is Maskowitz's (2004) description of catatonia as an evolutionary-based fear response, not dissimilar to that shown in depressive behaviour.

A more recent development in understanding depression from a behaviour analytic perspective was presented by Kanter, Busch, Weeks and Landes (2008: p. 9), who updated the traditional ABA model of depression by construing it as 'a maladaptive dysregulation or extension of this adaptive experience'. That is, the normal adaptive (withdrawal) response from a particular aversive stimulus becomes abnormal as it is maintained or extended to other (nonaversive) environmental stimuli or events. In this way, the depressed individual generalises their withdrawal response from the specific unpleasant event (either the onset of a noxious stimulus or the nonavailability of a previously available reinforcer) to other more widespread environmental stimuli.

This progression from initial (adaptive) withdrawal to maladaptive generalisation may, according to Kanter et al., (2008) pass through an affective state in which the individual exhibits excessive escape and avoidance behaviour and which may be described by the symptomatology of anxiety. Anxiety is comprised of many symptoms that are the outcome of exaggerated sympathetic nervous system (SNS) activation (e.g., cold sweating, rapid heart beat, increased blood pressure, psychomotor agitation, shivering), thus linking the individual's initial awareness of the noxious environmental stimulus with SNS arousal (escape, avoidance) and (as the escape and avoidance mechanisms fail to protect the individual from the noxious stimulus) depression or adaptive withdrawal from the general environment (Kanter et al., 2008). The overlap between anxiety and depression symptoms has been previously commented upon (Zinbarg, Barlow, Liebowitz, Street, Broadhead, Katon, et al., 1994), and there are suggestions that a Mixed Anxiety and Depression category should be included in a revised DSM series (Barlow & Durand, 2005), thus verifying the links between these two disorders.

An issue that has not received much attention is the *nature* of the aversive environmental stimuli that engender the adaptive anxiety and depression responses described above. Such aversive stimuli may be the onset of previously absent negative events (i.e., Positive Punishment/Punishment Type I) or the cessation of previously available social reinforcing events (i.e., Negative Punishment/Punishment Type II), but there are no reports of the comparison of the effects of these two types of aversive stimuli upon anxiety and depression responses.

One group of people who may be seen to suffer both of these kinds of aversive events are Prostate Cancer patients. Prostate Cancer (PCa) affects only men and usually later in their lives (Arnold-Reed, et al., 2008; Sharpley & Christie, 2007a) and, as such, offers a gender-specific population within which to study the relative effects of Positive and Negative Punishment upon the development of anxiety and depression. The progression from initial diagnosis to subsequent treatment for PCa brings with it many life-changing challenges that can cause patients to become

depressed and anxious (Sharpley, Bitsika & Christie, 2008; Sharpley & Christie, 2007b). For example, PCa patients experience numerous aversive environmental events that might be described as Positive Punishment (e.g., receiving their diagnosis, radiotherapy and antiandrogen therapy, treatment-induced fatigue and nausea) resulting in loss of previously available pleasant events (e.g., sexual function, restricted access to hobbies, disturbed social relationships: Incocci, Slop & Levendag, 2002; Vale, 2000), which could constitute Negative Punishment. In this study the focus was on distinguishing between these two types of aversive events in order to investigate the relative impact of them on patient distress.

As well as investigating the possible causal processes underlying development of anxiety and depression among PCa patients by comparing the two types of punishment, the results of this study could also hold implications for the application of behavioural interventions with this patient group. That is, while the symptom clusters required for diagnosis of these mental disorders are well delineated in the literature and identification of key deficit areas goes some way in guiding development of psychological interventions (APA, 2000), the effectiveness of such interventions can be limited if they focus on remediating symptoms with minimal understanding of the life events (i.e., causes) that contribute to patient distress. In applying the behavioural analytic model for depression to patients with PCa, it is suggested that they are exposed to numerous, intense and uncontrollable disease-based events that predispose them to adopting an adaptive withdrawal behaviour pattern that can lead to depression if it generalises to nondisease events in their lives. Describing these aversive events and comparing the two types of punishment may also provide directions for more focused behavioural treatment of the psychological distress experienced by these men.

This study was therefore designed to collect data from PCa patients regarding their anxiety and depressive symptomatology, plus the kinds of events that had happened to them and that might be categorised as producing conditions of Positive and/or Negative Punishment.

Methods

Sample

The sample comprised 381 PCa patients with a mean time since diagnosis of 13.9 months, ranging from 1 to 96 months. All participants had cancers limited to the primary site and regional draining lymph nodes diagnosed using conventional staging investigations. All had received radiotherapy, plus hormone therapy and surgery when required.

Measures

Data collection included a background questionnaire to gather information on age, living situation, month and year of first diagnosis, treatments received and continuing, and present status of their cancer.

Patients then completed the following three questionnaires:

Self-Rating Anxiety Scale (SAS; Zung, 1971)

The 20-item SAS is based on DSM (APA, 2000) definitions of anxiety and drawn from 'the most commonly found characteristics of an anxiety disorder' (Zung, 1971: p. 371). Positively- and negatively-worded items reduce response bias and reversed

items act as a lie scale. Respondents are asked to indicate how they have felt during the last week according to: 'None or a little of the time' (scored as 1), 'Some of the time' (2), 'Most of the time' (3) or 'All of the time' (4). Total raw scores range from 20 to 80, with higher scores indicative of greater anxiety. The SAS correlates .75 with the Hamilton Anxiety Scale (Zung, 1971) and significantly discriminates between nonanxious adults and patients with anxiety disorders (Zung, 1971). Reliability data are .71 (split half: Zung, 1971) and .79 (coefficient alpha) in an Australian sample of 552 noncancer participants (Sharpley & Rogers, 1985) and between .74 and .77 for two samples of Australian PCa patients (n = 195, 150) (Sharpley & Christie, 2007b; Sharpley, Bitsika, & Christie, 2009). Zung stated that raw scores above 36 indicated that participants had 'clinically significant' anxiety (Zung, 1980: p. 18).

Self-Rating Depression Scale (SDS; Zung, 1965)

The SDS has 20 items that were identified in factor analytic studies of the syndrome of depression and which underlie the DSM definition (APA, 2000). Positively- and negatively-worded items reduce response bias, plus several reversed items act as lie scales. Respondents are asked to use the same four criteria as described above for the SAS. Raw scores range from 20 to 80, with higher scores indicative of more severe depression. The SDS has split-half reliabilities of .81 (Zung, 1965), .79 (DeJonge & Baneke, 1989) and .94 (Gabrys & Peters, 1985). Internal consistency (alpha) has been reported as .88 for depressed patients and .93 for nondepressed patients (Gabrys & Peters, 1985), and as .84 and .83 for previous Australian PCa samples (Sharpley & Christie, 2007b; Sharpley, Bitsika, & Christie, 2009). The SDS has been shown to be superior to the MMPI Depression Scale and the Beck Depression Inventory for assessing depression in male psychiatric inpatients (Scheaffer et al., 1985: p. 335). Zung (1973) recommended a cutoff score of 40, above which respondents could be described as having 'clinically significant depression'. SDS and SAS raw scores were used in this study.

The Effects of Prostate Cancer on Lifestyle Questionnaire (EPCLQ) is a 36-item measure developed from 50 events indicated by PCa participants in an interview study as causing them major psychological distress (Sharpley, Bitsika, & Christie, 2007) and later refined to 36 items (Sharpley, Bitsika, & Christie, 2009). Participants in the current study were invited to complete this latter version of the EPCLQ using the same four-point scale as for the SAS and SDS. Total scores ranged from 36 to 144. All procedures were approved by the Uniting Health Care Human Research Ethics Committee.

Procedure

Eight hundred PCa patients living in Brisbane, Australia, were invited by letter mailed out by the Data Manager, Premion, to participate in a survey of 'how you feel about the things that have happened to you since being diagnosed'. All responses were accepted by the Data Manager who then coded completed questionnaires for anonymity before sending them to the researchers for analysis.

Results

From the 47.6% of PCa patients surveyed who completed the questionnaires, reliability (Cronbach's alpha) was satisfactory for the SAS (.78), SDS (.84) and the

EPCLQ (.88), justifying further examination of the data from these scales (Anastasi, 1982). The mean SAS score was 32.23 (*SD* = 6.93), median = 32.23, ranging from 20 to 64/80. The 5% trimmed mean was 31.93, only 0.30 below the sample mean, indicating negligible effects on the mean score from outliers in the sample. Skewness was positive (.76), suggesting a clustering at the lower end of the possible range of scores, to be expected within a non clinical sample. Kurtosis was 1.29, indicating a peaked distribution, confirmed by examination of the histogram. The advice of Tabachnik and Fidell (1996: pp. 73–75) that skewness and kurtosis will 'not make a substantive difference' to further analysis when the sample size is large (i.e., from 100 to 200+) was followed here. Inspection of the Boxplot revealed three outliers, but all these scores were valid, very high SAS scores. Using Zung's cut-off score of 36, 90 patients (23.9% of the sample) were experiencing 'clinically significant' anxiety (Zung, 1980). Although the Kolmogorov-Smirnov statistic was significant, this is 'quite common in larger samples' (Pallant, 2001) and was countered by the shape of the histogram (normally distributed) and the Normal Q-Q Plot, which was almost a completely straight line. In addition, the Detrended Normal Q-Q Plot showed that most points collected around the zero line. Thus, the SAS data may be accepted as satisfying the requirements of normality for further analyses.

The mean SDS score was 34.08 (*SD* = 8.831), median = 34.0, ranging from 20 to 66/80. The 5% trimmed mean was 34.68, only 0.60 greater than the sample mean and allowing outlier effects to be discounted. Skewness was positive (.68) showing minor clustering at the lower end of the SDS scale and kurtosis was .09, indicating a relatively flat distribution, both confirmed by the histogram. Boxplot inspection showed three outliers, all valid SDS scores, two of which were also the outliers in the SAS, suggesting that these two PCa patients were extremely highly anxious and depressed. Using Zung's (1973) cut-off score of 40, 98 (26.0%) of the sample were clinically depressed. The incidence of clinically significant anxiety and depression among this sample was within the overall range from the wider literature on PCa patients (Sharpley, Bitsika, & Christie, 2008). Again, the Kolmogorov-Smirnov statistic was significant but the histogram was normally distributed, the Normal Q-Q Plot was almost a completely straight line and the Detrended Normal Q-Q Plot showed a collection of points around zero, arguing for the normality of the SDS data.

The mean total score for the EPCLQ (EPCLQTOT) was 65.69 (*SD* = 13.12, *SEM* = 0.678), median = 63.5, mode = 56.0, with scores ranging from 38 to 134 from a possible range of 34 to 144. The 5% trimmed mean was 64.76, only .92 less than the sample mean, indicating that outlier effects were minimal. Skewness (1.34) indicated a clustering of scores at the lower end of the distribution and kurtosis (3.15) suggested that the distribution was markedly peaked, both confirmed by inspection of the histogram. Inspection of the Boxplot showed two outliers (i.e., more than 1.5 box-length from the edge of the box). Examination of the raw data from these two participants failed to show any errors and therefore they were included. As for the SAS and SDS, the Kolmogorov-Smirnov significant result was outweighed by the histogram, the almost completely straight line of the Normal Q-Q Plot and the clustering of the Detrended Normal Q-Q Plot around zero.

Blind ratings by the first two authors were used to categorise the EPCLQ items into two subscales. In the first subscale, those items that described the loss of a previously available and valued event or environmental stimulus (n = 19) were grouped as 'Lost Positives' and constituted a Negative Punishment stimulus (e.g., withdrawing from others, swimming less). In the second subscale, those items that

described the gaining of an unpleasant event or environmental stimulus (n = 17) were grouped as 'Gained Negatives' and constituted a Positive Punishment stimulus (e.g., difficulty in bowel motions, feeling angry). A full table of these two subscales is available from the first author.

As a first step in investigating the relative power of the two subscales of EPCLQ items in predicting anxiety or depression, regression analysis was performed using total SAS and SDS scores as the dependant variable. All assumptions were met satisfactorily. For the SAS, R^2 was .514, indicating that just over half of the SAS total score was explained by the Gained negatives and Lost positives model, and this result was significant, $F(2, 370) = 194, 386$, $p < .001$. Examination of the Beta weights (standardised coefficients) identified Gained Negative as making a strong unique contribution ($\beta = .703$, $t = 16.352$, $p < .001$) to SAS total score, but that the contribution made by Lost Positives ($\beta = .026$, $t = 0.606$, ns) was not significant. Inspection of the Normal probability Plot (P-P) and the Scatterplot confirmed that the assumptions of multicollinearity, normality, linearity, homoscedasticity and independence of residuals were met. The same Regression procedures were run for SDS scores (all assumptions were met satisfactorily). R^2 was .451 ($F = 151.073$, $p < .001$), and the Beta weight for Gained Negatives was .625 ($t = 13.697$, $p < .001$) but the corresponding value for Lost Positives was nonsignificant ($\beta = .081$, $t = 1.783$, ns).

However, these results describe the range of anxiety and depression scores and do not directly describe the relative power of the two EPCLQ subscales in predicting clinical anxiety or depression. Therefore, Logistic Regression was used in this second step of the data analysis, using the same two EPCLQ subsets of items as predictor variables and SAS or SDS clinicity (i.e., whether patients had SAS or SDS scores that were above the cutoff levels stipulated by Zung (1965, 1980) as indicating 'clinically significant' anxiety or depression). For SAS, the model was statistically significant, χ^2 (2) = 105.694, $p < .001$, showing that it was able to distinguish between clinically anxious and nonclinical patients and the Hosmer-Lemeshow Goodness of Fit Test further supported the model. The entire model explained between 24.8% (Cox and Snell R^2) and 37.1% (Nagelkerke R^2) of the variance in clinical anxiety status and correctly classified 82.5% of the cases. Examination of the Wald Tests for the variables in the equation indicated that only Gained Negatives had a significant value (55.191, $p < .001$), with Lost Positives showing a very low and nonsignificant value of 0.136, supporting the finding above from the first regression equation using the total SAS score. When classified into clinically significant versus nonclinical depression categories via SDS scores, the model including Gained Negative and Lost Positive EPCLQ subsets of items produced a statistically significant result, χ^2 (2) = 110.501, $p < .001$, and the Hosmer-Lemeshow Goodness of Fit Test confirmed this. The model explained between 25.6% (Cox and Snell R square) and 37.4% (Nagelkerke R squared) of the variance in clinical depression status and correctly classified 84.0% of the cases. Examination of the variables in the equation indicated that the Gained Negative subset of EPCLQ items had a significant value from the Wald Test (55.611, $p < .001$), but that the Wald Test for Lost Positives (0.047, ns) failed to predict clinically significant depression as it had failed to predict clinically significant anxiety.

These analyses refer to EPCLQ subscales. The kinds of events/stimuli that befell PCa patients and which contributed most to the development of anxiety and depression were also considered to be of value to this investigation. Therefore, a further analysis was conducted to determine which of the particular items contained

within the two EPCLQ subscales were most strongly associated with clinically significant anxiety and depression. MANOVA was performed on the two EPCLQ subscales separately, including all the items within the Gained Negatives subscale (that alone had been shown to significantly predict SAS and SDS 'clinicity' categories). Although this kind of analysis has often been done via Discriminant Analysis, Pallant (2001: p. 515) recommended that MANOVA 'has some features unobtainable in any other' Discriminant Analysis programs. There were significant main effects for SAS and SDS but not for the interaction of SAS and SDS. Table 1 shows the EPCLQ Gained Negatives subscale items ranked according to the strength of their particular *F* value across anxiety and depression clinicity. In all cases, mean values for clinically significant patients were greater than for those patients whose SAS/SDS scores did not reach Zung's cut off levels for clinicity. Items were accepted as significant discriminators of the presence of clinicity only if their *F* value reached a significance value of $p < .002$, established via Bonferroni correction based upon the numbers of EPCLQ items in the various subsets.

From Table 1, it is apparent that there was considerable overlap in the EPCLQ items that discriminated between clinically significant anxiety and clinically significant depression, which may be due to the overlap of the symptomatology between these two disorders (DSM, 2000; Zinbarg, et al., 1994). As might be expected, further Regression analyses of the Gained Negative and Lost Positives EPCLQ subscales against the combined SAS and SDS scores confirmed the previous findings that anxiety-depression was most powerfully predicted by Gained Negatives ($\beta = .697$, $p < .001$) rather than Lost Positives ($\beta = .060$. ns).

Discussion

As well as significantly predicting total anxiety and depression scores, the Gained Negatives subscale of the EPCLQ significantly predicted clinical and nonclinical anxiety and depression, suggesting that patients were most distressed and reactive to

TABLE 1

EPCLQ Gained Negatives Subscale Items From MANOVA Ranked in Size of Univariate Effect for Anxiety and Depression Clinicity (all $p < .002$)

Subscale items	
Gained negatives	
Anxiety	Depression
Tired	Nauseous
Feeling depressed	Sadder
More anxious	Feeling depressed
Nauseous	More anxious
Angry	Angry
Less tolerant of others	Getting on worse with friends
Feeling worse overall	'Fuzzy' about organisation
Increased pain	Feeling worse overall
Poorer sleeping	
'Fuzzy' about organisation	

exposure to the new aversive events associated with their disease compared to the Positive Loss subscale of the EPCLQ (that did not differentiate between clinically and nonclinically anxious and depressed patients). These data support previous findings of the relative powerfulness of Positive Punishment over Negative Punishment as instigator of escalations in anger, frustration and confusion that lead to escape and avoidance behaviours (Spiegler & Guevremont, 2003).

The overlap in the EPCLQ items that discriminated between clinically and nonclinically depressed and anxious PCa patients reflects the intersection of symptomatology of these two disorders. For instance, experiences of clinical depression and clinical anxiety were both associated with: nausea; elevated feelings of sadness, nervousness and anger; getting on worse with friends; being 'fuzzy' about organisation; and feeling worse overall. These findings corroborate the commonalities between anxiety and depression and support a model of mental disorder that conceptualises these two conditions as stages along a continuum of mental distress rather than separate disorders.

These findings have several implications for clinical behavioural practice targeting PCa patients. It is clear that this disease exposes patients to numerous cancer-based events that are intensely aversive and uncontrollable and that patient responses to these events will differ and fluctuate as they progress from diagnosis through to treatment termination (Arnold-Reed, et al., 2008). Although a proportion of physiological and psychological symptoms arising from treatment must simply be endured, there is scope to assist patients in the amelioration of the responses that can exacerbate their difficulties. The results of this study suggest that Negative Gains (or Positive Punishers) play a substantial role in the development of depression and anxiety in PCa patients and that the aversiveness of disease-based events may derive (at least in part) from the newness and uncontrollability of these events. PCa patients might be assisted to understand and anticipate the effects of the disease- and treatment-based events they will experience by receiving a pre-treatment map of key aversive events and counselling on their likely responses to these. Patients might also be guided to identify those aspects of their life and their responses that can remain under their direct control and continue to be shaped by them. For instance, a patient undergoing chemotherapy treatment can be taught systematic muscle relaxation and visualisation to reduce (i.e., exert some control over) nausea effects. In addition, the presence of a possible sequential relationship between clinical anxiety to depression allows anxiety to be conceptualised as SNS-arousal responses that are more likely to predominate when PCa patients have received their diagnosis and begin to feel intense fear and worry about their future. Following this stage, depression may become the principal area of impairment when the patient is immersed in coping with aversive treatment effects. Therefore, PCa patients might be best assisted by receiving targeted treatments for anxiety first and then depression, with an acknowledgement that particular responses might be caused by one or other disorder depending on where the client is in the diagnosis-treatment sequence. Further, investigation of possible triggers for adverse responses arising from day to day life would assist in addressing the factors that exacerbate such responses. A final avenue for application of these findings within a behaviour analytic model for depression would be to explain that depression is a natural and adaptive withdrawal response to sustained exposure to adverse life events. However, withdrawal responses may also be viewed by PCa patients and their families as being indicators of emotional dysfunction, leading to exacerbation of distress and social

disconnection. This process could be addressed directly via counselling that emphasised the adaptive value of withdrawal, plus further investigation of the specific ways in which this response facilitates the patient's day to day coping. Continued monitoring of client withdrawal could identify if this distancing response has generalised from PCa-related unpleasant events to nonaversive life events. The client might then be trained to use basic social behaviours (e.g., skills for responding positively during interactions with family and friends) to reduce this generalisation of withdrawal behaviours.

In conclusion, the relative power of Positive Punishment over Negative Punishment as a predictor of anxiety and distress in PCa patients confirms previous suggestions regarding these two sets of aversive stimuli upon behaviour within a population that has not been examined in this way before. By studying these effects within a gender-specific group of patients who experience a homogenous set of symptoms and treatments (that carry negative sequelae), these data provide a more focused test of the effects of two types of punishment upon anxiety and depression than possible within more heterogeneous samples. Data regarding the effects of Positive and Negative Punishment upon total anxiety and depression scores were extended by classification of the sample into those who met Zung's (1965, 1980) criteria for clinically significant symptomatology, therefore enabling suggestions for behavioural treatments of these disorders with these patients to be based upon a firmer foundation.

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