Cognitive Biases, Mental Illness and Economic Decision-making: A Neuroeconomic investigation into Anhedonia

Depression is a major public health issue, with an Australian Government report identifying the 12-month prevalence for a Depressive Episode at 4.1%, and an estimated lifetime prevalence of 11.6% (Australian Bureau of Statistics, 2008); while recent statistics for New Zealand indicate a higher life-time prevalence rate of 14% (New Zealand Health Survey, 2012). World-wide, it is estimated around 151 million people world-wide suffer from major depression (WHO, 2008). Major depression is the leading cause of disability, in terms of "Years Lost to Disability" (YLD; "measure the equivalent years of healthy life lost through time spent in states of less than full health" (WHO, 2008; p36)) measures, for both men and women, although the burden of depression is around 50% higher for women.

Anhedonia can be defined as a reduced hedonistic response to reward, or a reduced motivation to pursue rewards (Andrews & Thomson, 2009). Anhedonia appears to represent a dysfunction in the brain's reward system. While it is common symptom of depression, it can also feature in non-clinical populations (Hasler, Drevets, Manji, & Charney, 2004). It has been suggested that the anhedonic symptoms associated with clinical depression have an evolutionary basis (Andrews & Thomson Jr, 2009). Recent literature has suggested that there may be two main types of anhedonic symptoms: consummatory anhedonia (which can be broadly considered a loss of "liking") and motivational anhedonia (a loss of "wanting").

While both economics and psychology concern themselves with understanding human behaviour, the advent of neuroeconomics has allowed for a synthesis of these two approaches (Loewenstein, Rick, & Cohen, 2008). As such, neuroeconomics represents a multidisciplinary approach to understanding human behaviour, combining theories and insights from economics, psychology and neuroscience to develop quantitative models of behaviour and the neural processes behind them (Glimcher, 2009).

A Neuroeconomic perspective on Major Depression and Anhedonia.

In recent years, increased attention has been paid to how mental illnesses, such as major depression, affect economic and social decision-making (Hasler, 2011; Kishida, King-Casas, & Montague, 2010). While a neuroeconomic approach to psychiatry is still in its infancy, early results are promising. Many neuroeconomic paradigms have their

roots in game theory, which allows for hard, quantitative and testable models of how a particular disorder can affect valuations and preferences.

As depression and anhedonia influence how individuals respond to both pleasant and unpleasant stimuli (Kaviani et al., 2004), it is plausible that it could also alter their economic choices in more everyday situations. With respect to major depression, Harle, Allen and Sanfey (2010) reported that individuals with major depression were more likely to accept unfair offers in the Ultimatum Game, despite experiencing a stronger negative emotional response (e.g. disgust) to the unfair offers. As a result, the depressed group actually earned significantly more money overall than the control group (around $50 versus $43). The emotional reactions reported were similar to those found in a prior study (Harlé & Sanfey, 2007).

Prior research using tasks with differential rewards have been used to investigate individual differences in motivation or preference towards rewarding stimuli. These tasks have indicated that individuals experiencing depressive symptoms do not show the same motivation or preference towards rewarding stimuli, compared to non-depressed individuals. While healthy, non-depressed participants typically develop a bias towards the more highly rewarded response (or a greater willingness to classify ambiguous stimuli as target), individuals with depressive symptoms do not (Henriques & Davidson, 2000; Henriques, Glowacki, & Davidson, 1994; Pizzagalli, Jahn, & O'Shea, 2005).

EEG correlates of economic and social decision-making

Research studies combining electroencephalography (EEG) with economic games have been used to examine the neural underpinnings of decision-making. For instance, EEG research has identified a neural dissociation in the coding of reward magnitude and valence. Utilising a simple gambling task, researchers have found that the amplitude of one particular brain response (P300) varies in response to reward magnitude but not to reward variance, while the amplitude of the second brain response (FRN) showed the opposite pattern (Sato et al., 2005; Yeung & Sanfey, 2004). Thus it is possible that ERP differences could be a marker for altered decision-making processes in depression or anhedonia.

Study Aims and Rationale

The overall aim of my doctoral research is to demonstrate how a neuroeconomic approach to the study of anhedonia and major depression could offer novel insights into these disorders. The aim of this experiment was to examine how anhedonia affects

economic decision-making and reward learning, at both the behavioural and neurophysiological levels.

Method

Participants

A total of 30 participants were recruited, with 16 participants allocated to the high anhedonia group, and 14 participants to the low anhedonia group. Participants were matched by age and gender. All participants were aged between 18-65 years old, healthy, and able to provide informed consent.

These 30 participants were recruited from an initial sample of 426 participants, recruited from the first year psychology pool at Melbourne and Monash Universities. Participants completed a number of personality questionnaires and received course credit in return for their participation. As part of the consenting process, participants were asked if they wanted to participate in a follow-up study, and those who agreed had their names added to a participant database.

Participants completed computerised versions of 16 personality questionnaire, but with respect to calculating anhedonia scores, only the following scales are of interest: the Chapman Physical Anhedonia scale (Chapman, Chapman, & Raulin, 1976); the Chapman Social Anhedonia scale (Chapman et al., 1976); the Snaith-Hamilton Pleasure Scale (Snaith et al., 1995); the Temporal Experience of Pleasure Scale (Gard, Gard, Kring, & John, 2006). In addition, the Beck Depression Inventory (Beck, Steer, Ball, & Ranieri, 1996) was administrated as a screening tool, to exclude potential participants who may be experiencing depressive symptoms from the anhedonia EEG study.

Five anhedonia scores were obtained across the four questionnaires for each participant. SAS 9.2 (SAS Institute, 2009) was used to run a principal components analysis (PROC FACTOR) to calculate a single anhedonia factor score for each participant (with a mean of zero and a standard deviation of one). Participants with factor scores greater than +0.25 (high anhedonia) or less than -0.50 (low anhedonia), and who also had BDI scores of less than 13 were contacted to ask if they wanted to participate in the follow-up study; the lower threshold for the high anhedonia group was due to the high correlation between anhedonia and BDI depression excluding a large number of participants. However this anhedonia grouping factor proved to be non-significant in subsequent statistical analyses.

Procedure

On arrival, participants were taken to the research laboratory and informed consent was obtained. Participants then underwent a clinical assessment to confirm that they were not suffering from any mental illnesses or conditions that might exclude them from the study. No participants met the criteria for Major Depression, and all participants scored below 6 on the MADRS. Following the clinical assessment, participants were set-up for the EEG. EEG was measured using 64 Ag/Ag Cl electrodes embedded in a stretch lycra cap (Compumedics Quick-Cap), arranged according to the international 10-20 system. Additional electrodes were placed above and below the left eye and next to the outer canthus of each eye. EEG data was acquired using *NeuroScan* software and a *SynAmps 2* amplifier (Compumedics, Melbourne, Australia). Impedences were kept below 5 kΩ at the beginning of each recording and checked between the experimental tasks. Electrodes were referenced to a central in-cap reference (located between Cz and Cpz). The sampling rate was 500 Hz.

Participants then completed the gambling task and signal detection task, with task order counterbalanced across participants. Both tasks were designed by Phillip Hall using *E-Prime 2.0* (Psychology Software Tools, Pittsburgh PA).

For the gambling task, participants were required to choose between a constant low risk gamble (betting 2 cents for a 50% chance to win 8 cents in all trials) against a variable high risk gamble (bet 10c for a 50% chance to win either 0, 10, 20, 30 or 40 cents). Participants were shown a screen displaying the two choices and given an unlimited time to respond; following their response, there was a short delay, after which they were informed as to whether they had won (yellow screen) or lost (red screen), along with a running total of their earnings. Participants completed four blocks of 80 trials.

The signal detection task used a similar procedure as that employed by Tripp and Alsop (1999) and Pizzagalli et al., (2005). Participants were shown a fixation point for 500ms, after which a mouth-less cartoon face is presented; following a 500ms delay, a long or short mouth is briefly shown (100ms), and immediately replaced by the mouth-less face. Participants are tasked with deciding whether they saw a short or long mouth. The key feature of this task is that while short and long faces occurred equally often within each block, one face was rewarded three times more than the other (generating an asymmetrical reinforcer ratio), with the aim to generate a response bias (a preference for one response over another). A controlled reinforcer procedure (Johnstone & Alsop, 2000) was used to ensure that participants did in practice receive reward feedback at the 3:1 ratio specified. Participants completed three blocks of 100 trials. At the

completion of the two tasks, participants were debriefed, thanked for their participation and paid their earnings (AUS$30).

Results – Gambling Task

Behavioural Choice Results

SAS 9.4 (SAS Institute, 2009) was used to analyse the behavioural choice data for 30 participants for the Gambling Task. As the dependent variable ("bet choice") was dichotomous (taking the value "0" for the low-risk choice, and "1" for the high-risk choice), a GEE approach (PROC GENMOD) was used, to run a repeated measures logistic regression. All responses associated with a reaction time of less than 250ms or greater than 3000ms were regarded as errors and removed prior to analysis.

A number of models were tested. Model 1 was a simple main effect plus interaction model, with reward level and anhedonia factor score (inputted as a continuous rather than categorical variable) as independent variables. The main-effects model indicated that while reward level (χ^2 (4, N=30) = 99.82, p <.001) affected the probability of making a risky bet, neither anhedonia (χ^2 (1, N=30) = 0.25, p <.620), nor the anhedonia by level interaction (χ^2 (4, N=30) = 5.80, p =.215) were significant predictors of risky behaviour.

Model 2 was a simple main effects model which used the five anhedonia sub-scales (rather than the anhedonia factor score) as independent variables, along with reward level. As before, reward level was significant (χ^2 (4, N=30) = 102.02, p <.001). Of the anhedonia subscales, anticipatory anhedonia (χ^2 (1, N=30) =10.72, p =.001), physical anhedonia (χ^2 (1, N=30) = 7.78, p =.005) and social anhedonia (χ^2 (1, N=30) = 10.61, p =.001) significantly predicted risky behaviour in the gambling task. However, neither consummatory anhedonia (χ^2 (1, N=30) = 0.37, p =.542, or SHPS anhedonia (χ^2 (1, N=30) = 2.10, p =.148) were significant predictors of risky behaviour.

As the general anhedonia factor (continuous variable) did not appear to be predictive of behaviour (nor did the anhedonia categorical grouping variable), it was decided to utilise the anhedonia sub-scales, and test whether they affected the likelihood of making a risky bet at any specific reward level (i.e. is there a level x anhedonia subscale interaction, for each of the anhedonia subscales).

Model 3 was an interaction model which built on Model 2 by incorporating an interaction between level and each of the anhedonia subscales. The results were similar to Model 2, with the main effects of reward level (χ^2 (4, N=30) = 15.06, p =.005), anticipatory

anhedonia (χ^2 (1, N=30) =10.32, p =.001), physical anhedonia (χ^2 (1, N=30) =7.83, p =.005) and social anhedonia (χ^2 (1, N=30) =7.02, p =.008) remaining significant, and consummatory anhedonia (χ^2 (1, N=30) =0.47, p =.491) and SHPS anhedonia (χ^2 (1, N=30) =0.00, p = .947) remaining non-significant. Regarding the two-way interactions, only the level x physical anhedonia (χ^2 (4, N=30) =9.87, p =.043) and level x social anhedonia (χ^2 (4, N=30) =19.51, p =.001) were significant, indicating that these were the only aspects of anhedonia that moderated betting behaviour as reward level increased.

Reaction Time Results

SAS 9.4 (SAS Institute, 2009) was used to analyse the behavioural reaction time data for 30 participants for the Gambling Task. A Linear Mixed Models approach (PROC MIXED) was used. All responses associated with a reaction time of less than 250ms or greater than 3000ms were regarded as errors and removed prior to analysis. The dependent variable in all analyses was Reaction Time in milliseconds (ms).

As the use of the raw RT data violated statistical assumptions, a range of transformations were compared. In particular, the 1/RT and Log_{10} (RT) provided a substantial improvement over the raw RT distribution, with the Log_{10} (RT) transformation being the best (based on AIC values for direct model comparisons) for the present data-set.

A number of statistical models were tested. The first model was a simple main effects plus interaction analysis, between reward level and general anhedonia factor score. The results of this analysis indicated a significant main effect of Reward level (F(4, 9213) = 115.96, p <.001). However neither anhedonia score (F(1, 9213) = 0.17, p= .680), nor its interaction with level (F(4, 9213) = 1.96, p = .098) were significant predictors of reaction time.

As the general anhedonia factor score did not appear to tap into anything of behavioural significance, the analysis was re-run using level, the five anhedonia sub-scales, and their interactions with the level variable. As with the previous model, a significant effect of reward level was found (F(4,9196) = 7.71, p <.001). With respect to the anhedonia subscales, in general the main-effects for the scales were non-significant, with only Social Anhedonia showing any potential as a predictor (F(1, 9196) = 3.57, p= .059), with Anticipatory Anhedonia, Consummatory Anhedonia, SHPS and Physical Anhedonia subscales all non-significant (all Fs < 1.0). However a different picture emerges when the interactions between the anhedonia subscales with reward level is examined.

A significant interaction between level and anticipatory anhedonia ($F(4, 9196) = 7.04$, $p < .001$), level and consummatory anhedonia ($F(4, 9196) = 4.16$ $p = .002$), and between level and social anhedonia (F (4, 9196) = 12.98, $p < .001$) was observed, while the interaction between level and SHPS (F (4, 9196) = 1.69, $p = .150$) and between level and physical anhedonia (F (4, 9196) = 2.28, $p = .058$) both approached significance.

EEG ERP Results – P300 and FRN

For the current analyses, P300 is defined as mean amplitude 200-400 ms following presentation of the win/loss outcome slide, at electrode Pz; FRN is defined as mean amplitude 200-300 ms following presentation of the win/loss outcome slide, averaged across electrodes Fz and FCz.

Prior to analysis, the individual raw EEG files were re-referenced (to the left and right mastoids) and processed offline. Ocular artefacts were correct with an eye-movement correction algorithm. A band-pass filter (0.1 Hz – 30 Hz) was applied and the EEG data was segmented into separate epochs of 1000ms (200 ms baseline), for each trial type, and these were time locked to stimulus onset (presentation of outcome slide), and baseline corrected to the pre-stimulus interval.

For each epoch, artefacts were automatically detection using a maximum/minimum voltage criterion (±75μV on target channels), and then kept or rejected following visual inspection. Averages were computed if a participant had at least 15 accepted epochs for a particular trial type, and it was these average ERP files that formed the basis for the statistical analysis.

PROC MIXED (SAS 9.4) was used to analyse the averaged ERP data. Two separate analyses were run; the first on P300 amplitude, and the second on FRN amplitude. For both analyses the independent variables of interest were: Outcome (Win/Lose), Bet Choice (Low/High), Anticipatory Anhedonia group (Low/High) and each of their two-way interactions. Both analyses controlled for age and gender effects.

Surprisingly, there was no significant effect of trial outcome on P300 amplitude ($F(1, 28) = 2.03$, $p = .165$), however P300 amplitude was significantly higher following high bet choices, compared to low bet choices, regardless of outcome, ($F(1,28) = 7.67$ $p = .010$). Additionally, while the prior analyses showed no effect of general anhedonia factor score, one of the component subscales, anticipatory anhedonia, did affect P300 amplitude, with average P300 amplitude higher for the high anticipatory anhedonia group compared to the low anticipatory anhedonia group, $F(1,28) = 9.22$, $p = .005$. All of the two-way interactions were non-significant (all Fs < 1.0).

A similar pattern of results was observed for the FRN. No effect of trial outcome on FRN amplitude was observed, $F(1, 28) = 0.05$, $p = .82$; FRN amplitude was higher following high bet choices $F(1,28) = 6.31$, $p = .018$; FRN amplitude was also higher for the high anticipatory anhedonia group, compared to the low group, $F(1, 26) = 5.42$, $p = .028$. All of the two-way interactions were non-significant (all Fs < 2.01, all ps > .168).

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