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Abstract

A controversial finding in the field of causal learning is that mood contributes to perceptions of uncorrelated relationships. When people are asked to report the degree of control they have, people with dysphoria or depression are more realistic than others in recognising non-contingency between their actions and outcomes (Alloy & Abramson, 1979). The strongest evidence for this depressive realism (DR) effect is based on an experimental procedure in which the dependent variables are verbal or written ratings of contingency or cause. In order to address the possible confounds that such ratings may introduce, we used a two response free-operant causal learning task and performance based dependent measures. Participants were required to respond to maximize the occurrence of a temporally contiguous outcome that was programmed with different and temporally varying probabilities across two responses. Dysphoric participants were more sensitive to the changing outcome contingencies than controls even thought they responded at a similar rate. During probe trials, in which the outcome was masked, their performance recovered quicker than that of the control group. These data provide unexpected support for the depressive realism hypothesis suggesting that dysphoria is associated with heightened sensitivity to temporal shifts in contingency.

Keywords: Causality, contingency, reinforcement, contingency, learning, response rate, time, dysphoria, depression, depressive realism.

Introduction

Experiences related to the effectiveness of our behaviour in producing outcomes shape our perceptions of volition (Neuringer and Jensen, 2010) and personal agency (Bandura, 1982) and importantly may play a part in mental health. A controversial finding suggests that depressed people, possessing symptoms that resemble personal helplessness, are in fact more sensitive to the causal consequences of their behaviour (Alloy and Abramson, 1979;Martin et al., 1984;Alloy et al., 1985;Benassi and Mahler, 1985;Vasquez, 1987). The strongest evidence for this effect, which has been labelled depressive realism, comes from the contingency judgement task (Dobson and Franche, 1989). However, there are possible problems with this method as an objective measure of causal relation understanding. Here, we test mood related differences in causal learning using a less biased behavioural test of contingency and contiguity sensitivity measured over time, a method often used to study contingency sensitivity (e.g., Thomas, 1981;Dickinson et al., 1992) and timing sensitivity (Chiang et al., 2000) in animals. First, we briefly review depressive realism studies before describing how the present experiment will address interpretative issues with extant measures.

The term depressive realism originally stems from a series of studies carried out by Alloy and Abramson (1979). Student participants were given opportunities to press or not to press a button and observe whether an outcome (light) was temporally contingent upon their actions (key press). The programmed contingency between the action and the outcome can be formally described by the delta P measure (DP: Allan, 1980). DP expresses the strength of the relationship in terms of a number between -1 and +1, allowing for negative relationships. It is calculated as the difference between the conditional probabilities of the outcome following an action [*p*(light|press)] and following no action [*p*(light|no press)]. In most similar experiments, participants' numeric judgements of their control over the outcome are then analysed for consistency with the programed contingency. Indeed, much research has been conducted to determine the extent to which DP, as a formal model of contingency learning, was an accurate predictor of judgements (e.g., Jenkins and Ward, 1965;Allan and Jenkins, 1980;Chatlosh et al., 1985).

However, Alloy and Abramson's (1979) aim was not so much to test the model but to check the relative accuracy of judgements made by student participants who they categorised as mildly depressed or not depressed. A range of conditions and manipulations were tested across a series of experiments, though it was two critical conditions that engendered differences between the two mood groups. These were conditions where the frequency of outcomes was varied (25% versus 75% of trials included outcomes) but the difference between the two conditional probabilities and degree of control was always zero ($\Delta P = 0$). Judgements made by the depressed participants reflected this contingency and in both conditions were close to zero, suggesting that they recognised their lack of control. Judgements made by the non-depressed participants, although low in the 25% condition, were higher in the 75% condition and were consistent with the perception of a moderate degree of control.

Based on these findings (Alloy and Abramson, 1979), and subsequent replications of the effect (Martin et al., 1984;Alloy et al., 1985;Benassi and Mahler, 1985;Vasquez, 1987), the general conclusions were that depressed people were realistic about control whereas the non-depressed were optimistic in their perceptions of causal efficacy. This evidence is considered to be strong largely because DP is regarded as an accurate objective measure of control against which to assess people's ratings (Dobson and Franche, 1989;Ackermann and DeRubeis, 1991;Haaga and Beck, 1995).

The interpretation of findings as indicating realism is based on the assumption that the programmed DP and the DP experienced by the participant (Experienced DP: EDP) are very similar. However, this may not be the case (Msetfi et al., 2005). DP is defined by two conditional probabilities. The first, *p*(light|press), is clear as it is defined by the participants' responses. However, the second probability, *p*(light|no press), is ambiguous to manipulate experimentally because it is determined by the frequency of not responding, in other words non-events (Msetfi et al., 2005;Msetfi et al., 2007). A stronger test of depressive realism might involve conditions in which the experienced conditional probabilities and EDP were more under experimenter control.

Furthermore, response rate variability can influence the EDP. EDP is determined, to some extent, by the relative tendency to respond and to withhold responding. For instance, during 20 possible action opportunities if a participant responds 18 times and withholds responding 2 times, then this sets limits on the range of possible contingencies that might be experienced. Some participants do tend to respond a lot while others respond less when instructed to sample both situations. In extreme cases, the participant might experience only the *p*(light|press) or the *p*(light|no press) rather than ΔP (Matute, 1996) or, in less extreme cases, a skewed ΔP depending on the programming method used (and Matute, 1996;see also Hannah and Beneteau, 2009). In fact, Matute (1996) has argued that the depressive realism effect might occur simply because the depressed respond less than the non-depressed who respond at high rates and experience a more positive contingency.

Equally important is how the perception of control is actually measured. DR studies usually require participants to make explicit verbal or written judgements about their perception of control using scales provided by the experimenter. An alternative method is to examine participants' behaviours or performance in response to a contingency situation (see Hannah and Beneteau, 2009). Such measures may be differentially sensitive to contingency manipulations but also differentially reflective of causal learning. For instance, some experimental manipulations that affect verbal judgements, such as the overall density of reinforcement, do not influence performance measures (Allan et al., 2005). Thus verbal judgements may be biased and representative of people's willingness to predict that an outcome will occur rather than their perception of the contingency itself (Allan et al., 2007). Measurements of people's behaviour, when they are required to produce outcomes, may be a more accurate reflection of causal learning.

The current study was designed to examine the effects of depressed mood on contingency perception in conditions where EDP is less ambiguous. Like previous studies, participants were students where levels of dysphoria were measured using a depression inventory. The procedure was freeoperant and participants were instructed to cause a light to flash as many times as they could. They could do this by pressing one of two buttons at any one time and as many times as they wanted during each 50s trial. During the first half of each trial, 85% of presses on one button and 15% of responses on the other result in a light flash. During the second half of each trial, the outcome contingencies reversed. The task involves deciding which of two actions is more contingent with the outcome with the outcome never occurring in the absence of the response (DP = .85, DP = .15). Thus, no matter what the response rate, the *p*(light|no press) was always zero. Probe trials, during which the light is masked but the task goal remains the same, are introduced to measure acquisition or sensitivity to the point during the trial at which the reversal occurs in the absence of feedback. This task also tests timing of behaviour and shifts in the predictability of behaviour. Sensitivity to shifts in the temporal predictiveness of actions for individual cues has recently been shown to be an important cue to causality (Greville and Buehner, 2010). In the present work, participants were required to learn when a particular action changed in effectiveness.

This task was also chosen because it involves performance measures thought not to involve conscious cognitive bias. Previous research indicates that under similar conditions, people tend to 'match' their responses to the outcome contingency rather than using the more effective all or nothing maximisation strategy (e.g., Chatlosh et al., 1985;Koehler and James, 2009) consistent with Herrnstein's (1961) Matching Law in which the relative probability of response on one of the two behavioural choices should 'match' the probability of reinforcement in relation to total reinforcement available.

Therefore, if realistic, dysphoric participants' response rate probabilities will be more similar to the programmed contingencies than those of the control group, that is they will show greater response matching. Although, based on Matute and her colleagues work on the link between response rates and depressive realism (Matute, 1996;Blanco et al., 2009), we also predict that controls will respond at higher rates and, consequently, experience more light flashes than the dysphoric group. Finally, non-reinforced probe trials might be considered as extinction trials and should therefore produce a decrement in performance. Here this will result in a decrease in how closely response probabilities match reinforcement probabilities, as well as the effectiveness of responses in maximising the occurrence of the light flash. Therefore**,** between group differences in contingency matching and the effectiveness of responding will increase during probe trials.

Material and Methods

Participants

University students completed the Beck Depression Inventory (BDI: Beck et al., 1961) before being invited to participate and again during participation to ensure a stable score. All participants gave informed consent to taking part in this study. The final sample comprised forty-eight participants who were assigned to the dysphoric (n = 24) or control groups (n = 24) on the basis of their BDI scores. As in the majority of depressive realism research (e.g., Alloy & Abramson, 1979; Msetfi et al., 2005), scores of 9 or above indicated dysphoric mood and scores of 8 or below indicated no depression and membership of the control group. The groups were matched on various demographic variables, such as gender, age, years of education, pre-morbid IQ measured by the National Adult Reading Test (NART: Nelson, 1982) and short term memory capacity (Digit span: Lezak, 1995). All between group tests on demographics were not reliable (all $t < 1.14$). As expected, the dysphoric group had significantly higher BDI scores ($M = 15.6$, $SE = 1.6$) than the control group ($M = 4.4$, $SE = 0.5$: $t(46) = 6.56$, $p < .001$).

Procedure

Participants were briefed and given a written information sheet. After giving consent, participants completed the digit span test, the NART and the BDI. Instructions for the task were then presented on the computer screen. Participants were asked to maximise the occurrence of a brief light flash on the computer screen by pressing two on-screen buttons. The button on the left could be pressed using the 'tab' key and the button on the right using the 'return' key on the computer keyboard. Buttons were not to be pressed simultaneously or held in the on position. Each trial was 50-s long and separated by a 10-s inter-trial interval. During the first 25-s of each trial, 85% of presses on the 'early' button were reinforced with a light flash, while 15% of presses on the 'late' button were reinforced. Half the participants in each experimental group experienced the early button on the left, while the other half experienced the early button on the right. The outcome contingencies were switched after 25-s. Dependent measures were response rates and the probability of pressing the late button during each 5-s time segment of every experimental trial [*p*(late) = F(late)/(F(early) + F(late)].

There were a total of 18 trials. However, participants were told that there would be some probe trials where the light would be hidden from them, but that they should use what they had already learned in order to make the light flash as many times as possible (trials: 9, 12, 15 & 18). An onscreen message at the end of each trial recorded the number of light flashes during that trial. Finally, participants were debriefed, and paid a nominal fee for their participation.

Results

Reinforcement probabilities shifted from 15% to 85% on the late button after 25s. The probability of pressing the 'late' button was calculated for every 5s time segment for each participant, across reinforced learning trials and also across masked probe trials.

Reinforced learning trials

The *p*(late) was compared to the DP programmed at the same time points. As participants had been instructed to *maximise* the occurrence of the light flash, response probabilities were also compared to values consistent with a maximisation strategy, 0 and 1 (see Table1).

Table 1:
Mean probability of responding on the late button [p(late)] for the control and dysphoric groups during each 5-s time segment averaged over 14 experimental trials

Control group			Contingency comparison .15 or .85		Maximisation comparison 0 or 1	
Time	*M*	*SE*	*t*	*p*	*t*	*p*
5s	0.173	0.028	0.819	0.421	6.182	<.001
10s	0.135	0.027	-0.563	0.579	4.921	<.001
15s	0.126	0.027	-0.874	0.391	4.65	<.001
20s	0.112	0.027	-1.379	0.181	4.128	<.001
25s	0.119	0.026	-1.194	0.245	4.513	<.001
30s	0.574	0.019	-14.605	<.001	-22.532	<.001
35s	0.861	0.027	0.42	0.678	-5.094	<.001

40s	0.879	0.027	1.062	0.299	-4.448	<.001
45s	0.89	0.025	1.579	0.128	-4.326	<.001
50s	0.896	0.023	1.964	0.062	-4.477	<.001
Dysphoric group			Contingency comparison .15 or .85		Maximisation comparison 0 or 1	
Time	*M*	*SE*	*t*	*p*	*t*	*p*
5s	0.211	0.04	1.538	0.138	5.291	<.001
10s	0.177	0.035	0.753	0.459	5.016	<.001
15s	0.154	0.036	0.125	0.902	4.463	<.001
20s	0.158	0.033	0.252	0.803	4.789	<.001
25s	0.171	0.035	0.587	0.563	4.779	<.001
30s	0.627	0.015	-15.246	<.001 -	25.487	<.001
35s	0.874	0.026	0.942	0.356	-4.918	<.001
40s	0.882	0.027	1.178	0.251	-4.403	<.001
45s	0.884	0.025	1.404	0.174	-4.716	<.001
50s	0.868	0.025	0.716	0.481	-5.34	<.001

NB: These comparisons were made using single sample t-tests and the alpha level was ameliorated to $\alpha = .00125$ for 40 comparisons.

For both groups, response probabilities during 9 of the 10 time segments were not significantly different from the programmed contingencies but significantly different from maximisation probabilities. This shows that all participants' responses matched contingencies rather than being consistent with the more effective maximisation strategy.

The 30-s segment did not fit this pattern as participants changed their response probabilities during that segment in response to the change in contingency. Improved contingency sensitivity would be indicated by a more rapid switch in response probabilities between the two buttons at 25-s. Therefore, the *p*(late) for each 5-s time segment averaged over the 14 reinforced trials was analysed using mixed analysis of variance, with time segment as the repeated measures factor. Mood and counterbalancing were between subjects factors.

Response probabilities did change across time segments, $F(9, 36) = 50.56$, $p < .001$. However, dysphoric participants *p*(late) was higher ($M = .50$, $SE = .01$) than controls ($M = .48$, $SE = .01$) throughout the experimental trial, $F(1, 44) = 4.25$, $p = .045$, $\eta^2 = .088$, $MSE = .016$. This higher probability in the dysphoric group is indicative of responses that are more consistent with the programmed contingency than the control group. Consider that if responses are distributed across buttons in a manner consistent with the programmed contingency (.15 and .85), then the response probability should average out at .50 over the course of each experimental trial. As dysphoric participants responded on the late button at a probability of .50 and this was significantly higher than controls, who responded at a probability of .476, this is evidence of increased contingency sensitivity in the dysphoric group. Inspection of Table 1 suggests that dysphoric participants responded to changes in the programmed contingency more rapidly than controls and did not perseverate by continuing to respond on the early button once the programmed contingency had changed.

Finally, we calculated a Pearson's *r* correlation coefficient for each participant to describe the strength of the relationship between response probabilities and the programmed reinforcement contingency during every time segment. Higher values for *r* would indicate more consistency between response probabilities and the programmed contingencies. Indeed, correlations were at a very high level but not significantly different between the dysphoric (M = .93, SE = .03) and control groups (M = .94, SE = .01) where, $F < 1$.

Response frequencies on reinforced trials were explored with rates increasing rapidly from an average of 140.5 (SE = 10.9) on Trial 1 to 279.5 (SE = 11.1) on Trial 14, $F(13, 32) = 13.31$, $p <.001$, $\eta^2 = .844$, MSE = 1538.94. Dysphoric participants responded on average 257.1 times during each trial (SE = 15.1), while controls seemed to make fewer responses (M = 229.1, SE = 15.1). However, the mood effect was not significant, $F(1, 44) = 1.73$, $p = .195$, MSE = 761.58, nor was the mood by trials interaction, $F(13, 32) = 1.82$, $p = .08$, MSE = 1538.94.

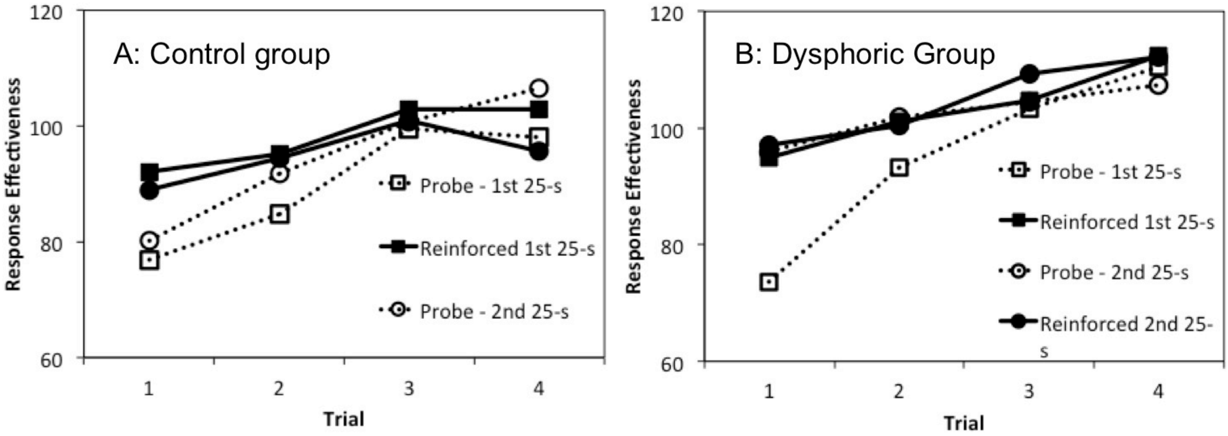
Response frequency data was used to calculate a measure of the effectiveness of responding over reinforced trials [in the first half of the trial (F Early × .85) + (F Late × .15); in the second half of the trial (F Early × .15) + (F Late × .85)]. These data were analysed using a mixed analysis of variance, with trials (14) and trial half (first 25-s, second 25-s) as the repeated measures factors. Mood and counterbalancing were between subjects factors. Response effectiveness improved over trials, $F(13, 32) = 19.35$, $p < .001$. Although the dysphoric group received more flashes (M = 93.72, SE = 5.1) than controls (M = 84.9, SE = 5.1), the mood effect was not significant, $F(1, 44) = 1.47$, $p = .231$, as were all of the interactions involving mood. Controls did not respond at a higher rate or receive more light flashes.

Masked probe trials

In order to make direct comparisons between reinforced and probe trials, we calculated Pearson's correlation coefficients to describe the relationship between response probabilities and programmed reinforcement probabilities averaged over the four reinforced trials preceding each probe and averaged over each of the four probe trials. Correlations were reduced in probe trials, $F(1, 43) = 19.36$, $p <.001$, $\eta^2 = .3\ 1$, MSE = .028. For the control group, the average correlation dropped from .93 (SE = .02) in the preceding trial to .80 (SE = .05) in probe trials. Correlations dropped slightly more in the dysphoric group, reducing from .94 (SE = .07) to .76 (SE = .06) in probe trials. However, the difference was not reliable, $F < 1$.

Response effectiveness scores were also calculated for each probe trial and each preceding probe trial (see Figure 1). These data were analysed with a mixed analysis of variance with Trial type (reinforced, probe), trial half (1st 25s, 2nd 25s), and trial number (1-4) as repeated measures factors. Mood and counterbalancing were between subjects factors. The 4-way interaction, which was of primary interest (all variables excluding counterbalancing), was significant, $F(3, 42) = 4.03$, $p = .013$, $\eta^2 = .224$, and therefore we examined the control and dysphoric groups' data separately.

Figure 1



For the control group (Figure 1, A), the three-way interaction with trial type, trial half and trial number only approached the level of significance, $F(3, 20) = 2.947$, $p = .058$, $\eta^2 = .307$. Further analysis showed that the two-way interactions with trial type and trial half, and with trial type and trial number were reliable ($p = .003$, $p = .002$ respectively). For controls, there was a significant effect of withdrawing reinforcement with a decrement in response effectiveness in the first ($p < .001$) and second probe trials ($p = .006$), although the effect was eliminated by the third probe trial (*ns*). Interestingly, by the second half of the final probe trial, the control group improved their response effectiveness in comparison to the previous reinforced trial ($p = .004$).

The pattern was different for the dysphoric group (Figure 1, B); the three-way interaction with trial type, trial half and trial number was reliable, $F(3, 20) = 3.85$, $p = .025$, $\eta^2 = .366$. There was a large decrease in response effectiveness in the first half of the first probe trial ($p = .001$). This decrement was similar in terms of effect size to the decrement experienced by the control group (controls: $\eta^2 = .419$, dysphoric: $\eta^2 = .415$) but for the dysphoric group effectiveness returned to its formerly high levels and there were no further reliable differences between reinforced and probe trials.

Discussion

Consistent with previous research participants, 'matched' their responding to the programmed reinforcement contingencies (e.g., Chatlosh et al., 1985). Participants did not use what would have been a more effective but more effortful, all or nothing, maximization strategy (Koehler and James, 2009). Making all responses on one operanda in the first half of each trial [p(early) = 1, p(late) = 0] and then switching to the other operanda during the second half [p(early) = 0, p(late) = 1], would have produced more outcomes. However, dysphoric participants distributed their behaviour between the two responses in a manner that more closely matched the different reinforcement probabilities and were able to learn to switch response patterns rapidly at the time point at which the programmed contingencies changed. By contrast, controls perseverated with their initial response pattern significantly longer than controls before making the switch. This effect, although not an obvious one, is suggestive of greater response-outcome contingency sensitivity in the dysphoric group.

Response rate variability related to mood was also a key issue in this study. Specifically, we wanted to check whether controls tended to respond at higher levels and therefore more effectively than the dysphoric group accounting for their apparent optimism. In this study, higher levels of responding would have produced more light flashes. However, we still found no evidence that controls responded at higher levels and received higher levels of reinforcement as has been suggested previously (Blanco et al., 2009). In fact, overall, controls responded at a lower level than the dysphoric group. In reinforced trials, this difference was too subtle to produce a significant effect on response effectiveness.

It was also important to examine responding in the absence of direct exposure to reinforcement. On masked probe trials, both groups responded appropriately and effectively. As, hypothesized, between group differences were amplified in the absence of reinforcement, but not in the predicted direction. We expected that realism, or an improved awareness of the causal effectiveness of actions, would result in less of a decrement in performance when reinforcement was withdrawn. However, although response effectiveness dropped for both groups in early probe trials, the speed of recovery from it depended on mood. The control group experienced a significant reduction in response effectiveness but this steadily improved such that effectiveness had recovered by the third probe and improved by the fourth. The dysphoric group only experienced the decrement in effectiveness in the first half of the first probe trials after which effectiveness recovered to its formerly high levels. Essentially, the dysphoric group required fewer trials to recover from the withdrawal of direct reinforcement. These results have several theoretical implications and suggest avenues that require further exploration.

For example, these data provide no support for the idea that non-depression is consistent with high response levels and increased exposure to reinforcement. In fact, both dysphoric and control groups responded at equally high levels. This finding is inconsistent with Blanco et al., (2009), who found that dysphoric participants, exposed to a zero contingency procedure with a high frequency of outcomes, responded less and made lower contingency ratings than controls.

Moreover, in the present study, dysphoric participants were less affected than controls by the withdrawal of reinforcement in probe trials. Although response effectiveness was reduced similarly to controls, the effect did not last so long in the dysphoric group. One obvious reason for this might be because people with dysphoria are less responsive or sensitive to reinforcement in the first place and less affected by its absence. This suggestion is consistent with negative relationships, reported in the normal population, between mood and reinforcer sensitivity (Glautier et al., 1998). It seems somewhat counterintuitive that decreased sensitivity would produce an improvement in performance in some cases. Although this is not unprecedented given that improved learning due to lack of sensitivity to potentially interfering stimuli in some learning situations (e.g., latent inhibition) is a hallmark of schizophrenia (Gray et al., 1992).

It should be noted however that, rather than evidence of good learning, probability matching as observed in the general population has been characterized as a non-normative tendency (West and Stanovich, 2003). In comparison to a considered and effective maximization strategy, matching could be seen as a 'mistake' based on a rapid response to the situation (Koehler and James, 2009). From this perspective, the current results are not suggestive of improved learning in dysphoria but perhaps a stronger tendency towards less than normative responses.

In summary, we have found dysphoria to be associated with improved response-outcome contingency sensitivity. Dysphoric participants adjusted their behaviour over time to changes in the response outcome contingency more rapidly than controls. The effectiveness of their responses also recovered more rapidly from the withdrawal of reinforcement. There was no evidence for a link between dysphoria and a reduced propensity to respond and experience reinforcement contingencies. The findings from this behavioural task, in which effective performance must involve sensitivity to contingencies which change over time, have been useful in terms of understanding differences in causal learning related to depressed mood. These findings also provide further support for the depressive realism hypothesis.

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Figure caption

Figure 1: Mean number of light flashes received (response effectiveness) during the first and second 25-s of probe trials and preceding reinforced trials.

Figure 1.TIFF