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Emotion and Decision Making: Effects of Anxiety on the Shape of the Decision

Weight Function

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Abstract

Prospect Theory (Tversky & Kahneman, 1992) asserts that people make decisions not on the basis of final states, but in relation to gains and losses experienced from a reference point. Furthermore, when making decisions under uncertainty, the weights assigned to outcomes and their associated utilities are not, as in expected utility theory, equal to the probabilities of those outcomes. Rather, the weights used in expected utility calculations have an “s-shaped” relationship with the underlying probabilities so that people have a tendency to overweight low probability events and underweight high probability events. Currently, there is no clear explanation for why this phenomenon occurs, although scholars suggest it may have something to do with the role that emotion plays in decision making. The goal of this study is to use neurological and psychological research to help create a plausible emotion-based explanation for the shape of the curve, as well as experimentally investigate the conditions under which the curve may change. Focusing on anxiety, I estimate decision weight functions for 62 participants, expose them to video clips which induce anxiety, reduce it, or are neutral (three treatment groups), and then experimentally estimate another decision weight curve for all participants. When controlling for self-reported and measured differences in anxiety levels, we find evidence that the decision weight function seems to shift down when anxiety is induced experimentally.

I. Introduction

Decision sciences are a rapidly growing field searching to understand how and why individuals make the decisions they do. While traditional economic theory of decision making assumes individuals are rational, much experimental evidence suggests otherwise (Kahneman, 2013), calling for a reexamination of decision making paradigms. Prospect theory developed as a leading model to describe the actual behavioral process used to make a decision. The theory proposed that instead of basing decisions on the anticipated final state of an outcome, individuals make decisions based on the gain or loss associated with a decision relative to a reference point (Tversky & Kahneman, 1992).

Faced with uncertainty, individuals place a subjective value on the possibilities of different outcomes. Decision weights are an important determining factor in a decision. Decision weights can be characterized as a function which has a “s-shape”. The reasoning behind this specific shape is not fully understood. While some evidence suggests emotion plays an integral role in the process of formulating decision weights (Fehr-Duda, Epper, Bruhin, & Schubert, 2011; Trepel, Fox, & Poldrack, 2005), their direct role in the process is not fully understood either. Looking to neurological and psychological research and theory may provide an answer.

The goal of this study is to use neurological and psychological research to develop an emotion-based model for the s-shape of the decision weight function and subsequently investigate the conditions under which this shape may change. I

hypothesize that emotion plays an integral role in the process and that specific types of emotional states can change the decision weight function. In particular, I focus on the emotion of anxiety and seek to build a replicable framework for studying the functions or patterns associated with other emotions.

II. Prospect Theory

In the late 1970s and early 1980s, prospect theory emerged as a behavioral model counter to traditional expected utility theory. Expected utility theory presumed that people chose those option that maximized the expected utility associated with final states. It assumes a 1:1 linear weighting function, in which all individuals have decision weights equal to the probability. In contrast, prospect theory claims an individual used their own subjective value, called a decision weight, instead (Tversky & Kahneman, 1992). The decision weight function is assumed to be an s-shaped curve (see Figure 1).

The s-shaped curve produced by prospect theory produces two main effects. For low probability events, there is a general tendency to overweight their value relative to their actual worth. For example, a person plays the lottery even though there is a low probability of winning because they overweight the small chance that they could win. This is called the possibility effect. The certainty effect, on the other hand, shows there is a general tendency to underweight high probability events (Kahneman, 2013). For example, a person may settle a court case even though they are almost certain to win because they underestimate that high probability of winning. Although this pattern is observed across many experiments in the literature (Kahneman & Tversky, 1979; Tversky & Kahneman, 1992; Gonzalez & Wu, 1999;

Neilson & Stowe, J. 2002), little explanation is offered as to why individuals tend to assign subjective weight in such a similar and predictable s-shape.

III. Explanations for the S-Shaped Decision Weight Function

Diminishing sensitivity

One explanation offered for the s-shaped curve is based on the idea of diminishing sensitivity. At the end points of the curve, events are either certain or impossible. Complete certainty or impossibility is simple for our brains to comprehend. However, as we move away from those events, and add degrees of uncertainty or ambiguity, we become less able to process and predict probabilities, causing the curve to be relatively flat in the middle (Tversky & Wakker, 1995).

Moving towards the middle of the curve, we are marginally less sensitive to changes in probability. It is still unclear why this phenomenon occurs or what mechanisms are responsible for this probability distortion. Therefore, there needs to be more investigation into why we have diminishing sensitivity.

Emotion.

An alternate explanation for the s-shape of the decision weight curve is that it is affect dependent. The curve can be thought of as reflecting emotion in and of itself.¹ The possibility effect is often associated with hope or excitement, while the certainty effect is associated with fear or anxiety (Kahneman & Tversky, 1979; Rottenstreich & Hsee, 2001). Psychological research supports this notion. Some studies suggest we use feelings when making decisions about risk, and those in a “hot” emotional state will systematically process risk differently than those who are

¹ Emotion is defined as the physiological body response to a thought or stimuli (Damasio, 1996).

in a “cold” rational state (Loewenstein, Weber., Hsee & Welch, 2001). The more vivid and salient the emotion, the more it affects the decision-making process. Many studies document the effect emotion has on decision making and risk taking attitudes. Inducing affect by using positive or negative words has been shown to impair performance on simple gambling task (Hinson, Whitney, Holben & Wirick, 2006). Others show participants tend to rely more heavily on emotion to assess risk when in time sensitive or stressful situation (Finucane, Alhakami, Slovic & Johnson, 2000), and when risk was framed in a positive emotional way, they perceived events as less risky.

Risk processing in terms of emotion lends itself to the idea that the value we assign to an option is a function of the emotional quality associated with it and quantity. Studies show that when asked to value a set of goods, participants tended to value the goods differently when presented in an affect rich way as opposed to in an affect poor manner (Hsee & Rottenstreich, 2004).² For example, when asked to value a set of CDs, the participant gave one value, but when asked to value the same set of CDs after being told to imagine they belonged to a good friend who was moving away from the country, participants were willing to pay more for them. While this study produced strong evidence for the importance of emotion in decision making, it did specify a function to show how people assign subjective value in a systematic way; it only showed tendencies.

Another study used the same principles to look at the decision weight curve specifically (Fehr-Duda et. al. 2011). The researchers asked participants to rate

² Affect rich refers to a prime which has a high emotional quality, whereas affect poor refers to something generally void of emotional vividness.

their mood at the time of the experiment, and they found that those in a better mood had greater amplitude s-shaped curves than those who were not in a good mood. Although this shows emotion affects the decision weight function, participants were simply categorized into a “good mood” or “not in a good mood”. This binary approach failed to specify what specific emotions were felt, how intense the emotion was, or how variance in emotion and intensity explains changes in decision weight function within and between subjects. It also fails to account for the subjectivity differences in self-reporting across individuals, as one person’s “good mood” could be a “neutral” mood to someone else.

Neural Evidence

Looking to neuroscience, we can see what circuitry is involved in decision making and compare it to the circuitry involved in emotion, in order to develop a clearer understanding of the mechanisms which could be responsible for shaping the decision weight curve. When considering decisions made in a neutral state³, the ventromedial prefrontal cortex (vmPFC) and orbitofrontal cortex, brain regions associated with reasoning and higher level executive function, have been shown to be involved in assigning value to choices (Chib, Rangel, Shimojo & O’Doherty, 2009; Plassmann, O’Doherty & Rangel, 2007). Another pathway, beginning in the cortex and projecting to the striatum, through the globus pallidus and thalamus, and back to the cortex, is shown to have changes in activation associated with assessing risk and the ability to stop taking risk (Meder et.al., 2016). This pathway begins in areas associated with value encoding, and then moves through the

³ Neutral will be defined as the state of an individual when no stimulus or emotional prime is presented.

reward pathway, suggesting that the brain does go through a valuation process and then considers the potential reward associated with that option and its outcome.

The insula, and area associated with many functions including emotional processes, is also highly involved in decision making processes and has shown increased activation in assessing how risky a prospect is, as well as when error has been made after the outcome is revealed (Preuschoff, Quartz, & Bossaerts, 2008). This suggests a complete circuit involving valuation, risk assessment, award assessment, and realization of any risk prediction errors once the outcome is revealed.

The neural decision making process also shows projections from value encoding areas into areas associated with emotion and emotional processing, which suggests these processes are not separate, but instead work together to develop the subjective value assigned to a prospect. The amygdala, an area heavily associated with emotion, shows increased activation when making decisions about risk (Levy, Snell, Nelson, Rustichini, & Glimcher, 2010). The amygdala has circuits connecting not only to the insula, which processes risk and evaluates risk predicting errors, but also to the vmPFC, the value processing area. It is suggested that a feedback loop exists between the vmPFC and amygdala which is crucial for decision making (Bechara & Damasio, 2005). The vmPFC evaluates a thought, then sends this information to the amygdala. The amygdala, which is also involved in regulating physiological responses in the body, receives this info and causes the body to change state. If emotions are defined as physiological changes in the body, then the body produces an emotion, the vmPFC can process this change, it causes

a change in firing in the amygdala, which causes a change in body state and so on in a loop (Damasio & Bishop, 1996). This is supported by clinical evidence showing those with lesions to the amygdala have a tendency to have poor decision making skills, a generally flat affect, and fail to show physiological body responses to stimuli (Gupta, Koscik, Bechara, & Tranel, 2011).

Evolution

It is also possible evolution helped shape the possibility and certainty effects observed in prospect theory. Research in monkeys found they also exhibit the same s-shaped decision weight function as humans (Stauffer, Lak, Bossaerts, & Schultz, 2015), suggesting humans evolved to exhibit this behavior. This would suggest the s-shape function must have some evolutionary advantage. It is possible that humans evolved to seek novelty because without this tendency, humans would miss potentially better outcomes. Thus, the possibility effect could be adaptive. The tendency encourages the undertaking of low probability actions with potential positive consequences and avoidance of low probability events with negative consequences. On the other hand, too much risk could be fatal, leading to the underweighting of highly likely events.

This theory is supported by studies linking neurotransmitters to decision weights. Research has found high correlations between dopamine receptor density and differences in decision weight functions (Takahashi et. al. 2010), which suggests that with varying levels of dopamine present, decision weights vary in a correlated way. The dopamine system may play a huge role in creating the behavior described by prospect theory. Blocking these same receptors leads to consistently

riskier choices in simple gambling tasks, providing more evidence in support of dopamine's role in forming decision weights. If humans have inherent circuitry underlying these behaviors, it provides strong support for the idea that humans evolved to behave in this manner.

Dopamine Reward Prediction and the S-Shape

Previous discussion illuminated brain circuits implicated in the decision-making process. Now we turn to examine a more complete theory as to how specific neurotransmitters could be involved in the creation of the possibility and certainty effects. This section is meant to show that humans are not diminishingly sensitive to probabilities, but rather have an innate understanding of them.

Comparisons of expected outcomes yield overweighting and underweighting. It is a purposeful mechanism.

In building the explanation of the s-shape decision weight curve, we must first understand dopamine. Dopamine (DA) is established in the literature as the neuroeconomic literature as the neural correlate to utility (Caplin & Dean, 2008) and is highly important in motivating and modulating behavior. Studies show DA is released in varying amounts in relation to our expectations. That is, the more certain we are an event will occur, the less marginal DA is released when it does.

Conversely, the less certain we are an event will occur, the greater the marginal DA released will be when the event happens (Schultz, 1997). Furthermore, the more certain we are an event will occur and it does not, the larger the decrease in DA firing. Similarly, there is very little marginal decrease in DA firing if we did not expect the event to occur and it does not. Research suggests this relationship between DA

release and expectations is nonlinear (Glimcher, 2011). Therefore, thinking in terms of utility, this would suggest there is a large utility associated with unexpected outcomes occurring. Also, there is a large disutility associated with highly expected outcomes not occurring. This relationship is illustrated in Figure 2.

Figure 3 shows our ex post reactions to events that occur in our lives, suggesting experiential utility, or the utility we experience in the moment from an outcome, is a function of our expectations. For example, say you are taking a test. Assume you studied vigorously for the test and expect to get an A. When you get your grade back, you get an A and receive utility of point A on the graph above. Now let us suppose you got the test back but you got an F. You would get the disutility of point B. Notice, the marginal utility (MU) you received from getting an A you expected is less than the marginal disutility (MD) you receive from getting an F.

Now suppose you did not study for the test and expect you will get an F. First, assume you get the test back and get an A. You get the utility of point C. Now assume you get a F that you expected and get utility of point D. Notice the MU of getting an A you did not expect is larger than the MD of getting the F you expected. Furthermore, the MU of the unexpected A when you did not study is larger than the MU of the expected A when you did study.

As we can see from this example, our utility is not only a function of gains or losses, but instead is a function of how certain we are that an event will occur and if it does or not. The more certain we are something will occur, the less utility we receive when it does occur and the more disutility we will receive if it does not occur.

This ex-post phenomena influences how we weight potential outcomes in the ex-ante phase. When giving subjective decision weights to potential prospects in a decision, we consider the potential utility and disutility associated with the outcome vs how sure we are it will occur. This interaction causes us to overweight or underweight potential probabilities of outcomes in decisions. If the MU of the event occurring is greater than the MD of an event not occurring, we overweight the possibility. If the MU of an event occurring is less than the MD not occurring, then we underweight the possibility.

For example, say you want to ask someone on a date. Suppose you are certain this person likes you and will say yes. Using this scenario, we can examine the decision process. Assume you are 75% sure they will say yes. The utility you gain if they do say yes is point A, as shown in Figure 4. If they say no, you receive the disutility of point B. As shown, the MD if they say no is greater than the MU if they say yes. Therefore, in your head, you underestimate the probability they will say yes, point D, when point C is the real probability the person will say yes. This underweighting will make you less likely to ask the person out to avoid experiencing the potential disutility of being wrong.

This overweighting of small probabilities causes us to seek out that which maximizes MU of an event occurring is greater than the MD of the event not occurring, and minimize MD of an event not occurring being greater than MU of it occurring.

Dopamine reward prediction error provides a good framework for explaining the s-shape exhibited by the decision weight function. DA is released from areas in

the basal ganglia, which is where the striatum of the reward circuit is situated. It therefore fits well into the overall decision pathways outlined. However, this explanation is only suitable to describe differences between individual's decision weight curves, and fails to account for differences within one person. Are there conditions under which one individual's decision weight function will change? Many of the aforementioned studies show evidence that in different emotional states, individuals make decisions in varying ways. Therefore, emotion must still play an integral role in the decision-making process. To further investigate this, I will extensively examine anxiety in order to determine how it could interact with the decision-making process. Emotional states could be responsible for changes in the curve, for example shifting it up or down, as well as changing the curvature.

IV. Anxiety

A large body of literature studies anxiety and its effects on individual's social wellbeing as well as their decision-making patterns. As a symptom of many psychiatric disorders, much research has been conducted to understand how and why anxiety has the effects it does (Maner et al, 2007). Since anxiety is a relatively well understood emotion, physiologically and neurologically speaking, it is a good emotion for use in this study. Using anxiety, we can build a framework for understanding how emotion interacts to create subjective decision weights in any instantaneous state.

Anxiety as an emotion can be thought of in two ways. It is usually invoked in response to threats, causing heightened physiological responses and heightened alert in detecting the environment (Engelmann, Meyer, Fehr, & Ruff, 2015). This

was an evolutionary important emotion, as heightened alertness in response to a novel stimulus in the environment increased an organism's chances of survival. Another way to interpret anxiety is the cognitive attention given to potential outcomes when the resolution is uncertain (Wu, 1999). Anxiety, therefore, is an unease surrounding the unknown, which is essential for allowing us to identify threats in an environment or situation.

Anxiety and Risk

The effects of anxiety on risk attitudes have been extensively studied. Many studies find evidence supporting the notion that anxiety causes increased risk aversion (Raghunathan & Pham, 1999; Maner et al, 2007; Robinson, Vytal, Cornwell, & Grillon, 2013). Studies have examined anxiety and risk attitudes in two ways. The first method looks for correlations in *trait anxiety* and differences in risk attitudes. Trait anxiety typically is measured by an anxiety survey to determine one's level of general anxiety as a part of their personality (Leon & Revelle, 1985). These studies then use simple gambling tasks, such as the Iowa Gambling Task (IGT) in which players choose between decks of cards with different probabilities of winning, or choosing between binary prospects such as winning a lottery or taking a certain amount (Raghunathan & Pham, 1999). These studies find that high anxious individuals are more risk averse than low anxious individuals (Charpentier et al, 2015). However, these studies, ignore the role of *state anxiety*, or the anxiety felt in the moment. In these experiments, there is no way to know if anxiety is being felt in the moment of the experiment and is indeed the reason for differences among individuals, or if other factors contribute to differences in results.

Another method for studying anxiety is to look specifically state anxiety, and see how the state influences risk attitudes. Examining state instead of trait anxiety can be a more effective way to study anxiety (Robinson et al 2013; Clark et al, 2012). While trait anxiety may reflect personality, in controlled experiments looking at state anxiety, participants can be manipulated to ensure they are feeling anxiety, which isolates the effects of anxiety on the decision-making process. Studies examining state anxiety use a variety of priming and induction methods, such as time pressure situations, threats of electric shocks, movie clips, and many other methods (Clark et al, 2012; Heilman et al, 2010; van Marle, Hermans, Qin, & Fernández, 2009). Many of these studies also find evidence supporting the notion that anxiety causes increased risk aversion (Clark et al 2012, Engelman et al 2015; Charpentier et al 2015). However, these studies only look at relative risk taking attitudes, and do not seek to see how anxiety specifically plays a role in the valuation process and creation of decision weights.

Neural Circuits Implicated in Anxiety

To gain a clearer picture of how anxiety can influence decision making, we can examine which pathways are affected by it, and look at how this interacts with neural pathways used to make decisions when not in an anxious state. Evidence suggests anxiety leads to decreased activity in the vmPFC and the orbitofrontal cortex, both areas associated with value making in the brain (Engelman et al 2015; Park, Wood, Bondi, Del Arco, & Moghaddam, 2016). If anxiety interferes with the ability to create an initial valuation, then this may lead to a change in the shape of the decision weight curve.

Anxiety has also been shown to interfere with the reward processing pathway (Charpentier et al 2015, Engelman et al 2015). Anxiety decreases communication between the vmPFC and striatum, which implies that under an anxious state, the brain is having difficulty making an initial valuation and then assessing the predicted utility associated with the outcome. Furthermore, evidence also suggests anxiety leads to less communication between the vmPFC and insula (Nitschke, Sarinopoulos, Mackiewicz, Schaefer, & Davidson, 2006). As the insula is involved in risk prediction and error correction, with decreased communication, the insula is not receiving proper inputs in order to accurately assess risk. This interference in the valuation, reward, and risk predicting pathway would change the ability of the brain to assign a decision weight to a prospect. Therefore, we would expect the decision weight of a prospect to differ when in an anxious state and a neutral state.

Anxiety is also highly associated with increased activation of the amygdala, which as was discussed previously, is an integral part of the decision-making process (Bechara, Damasio, & Damasio, 2003; Bechara, Damasio, Damasio, & Lee, 1999; Gupta, Koscik, Bechara, & Tranel, 2011). When under threat or stress, the amygdala increases activation (Hartley & Phelps, 2012). This causes hyper responsivity and attention to environment. Furthermore, physiological arousal is connected to anxiety. As the amygdala regulates these body states, information about these body states is sent to the vmPFC, heightening the feedback loop proposed by the SMH (Bechara & Damasio, 2005). This suggests that the heightened amygdala activation may lead to exaggeration of decision weights assigned to prospects.

Evidence from the literature suggests we should see a change in the decision weight function when in an anxious state instead of a neutral one. If there is a change, then we can conclude that emotion is a critical part in the formation of decision weights, and an integral part in forming the subjective value we give to choices in our lives. Furthermore, the heightened feedback loop suggests the intensity with which anxiety is felt should explain some of the variation in the decision weight curves when in a neutral vs anxious state. To test this theory, we will first estimate a decision weight curve of the participant in a neutral state. For the entirety of the experiment, heart rate will be recorded as a proxy for anxiety. Then we can induce an anxious state using a movie clip, and then estimate another curve while in an anxious state.

V. Methods

For this study, 62 undergraduate students from Macalester College participated (21 male, 40 female, 1 other), who in compensation for their efforts were paid \$10 for participating. The methods and procedures used in this study were approved by the Macalester IRB, and all participants gave informed written consent before engaging in the experiment.

Materials

Heart rate variability (HRV) was measured using a Polar H7 heart rate monitor (Polar Electro, Kempele, Finland). The monitor wirelessly collected HRV data from the monitors and sent the data to an application (Heart Rate Variability Logger, Marco Altini). State trait anxiety was measured using a six-item short form of the state trait anxiety inventory (STAI) (Marteau & Bekker, 1992). All prospects

were displayed on a computer, which collected data of all choices using the program Qualtrics. The chosen binary prospects were adapted from the original Cumulative Prospect Theory experiment (Tversky & Kahneman, 1992). The prospect pairings used were 250-0, 100-0, 75-0, 50-0, and 25-0. These prospects were spread out amongst the probability levels of 0.05, 0.10, 0.25, 0.50, 0.75, 0.90 and 0.95.

Procedure

Each participant had their HRV recorded throughout the duration of the experiment. To get a baseline measure of anxiety inherent in each participant, they answered the short form STAI. They were then asked to choose between a series of binary prospects, for example: 25% chance to win \$150 or \$50 for certain. The series of 35 prospects were logarithmically spaced across a spectrum of probabilities (Tversky & Kahneman, 1992). Each choice was recorded individually. At each probability level, a series of six descending sure outcomes were displayed, and the participant chose between the sure amount and the same risky prospect. Then, based on the chosen values, the program displayed an additional three prospects spaced equally between the 25% more than highest certain amount rejected and 25% less than the lowest certain amount accepted, in order to narrow down the certainty equivalent, which is the amount making an individual indifferent between a certain amount of money or a lottery. This process was repeated at each of the seven probability levels, using prospect pairs of 25-0, 50-0, 75-0, 100-0, and 250-0, for a total of 315 questions. At the end of the 315 questions, 3 questions were repeated to check for consistency.

Participants were then randomly separated into one of three groups--an anxiety inducing, neutral, and anxiety reducing--which were shown then shown three different movie clips. The neutral group was shown a movie clip which lacked any emotional vividness, a video of how a pencil is made. The anxiety reducing group was shown a video of soothing scenes of nature with calming music. The anxiety inducing group was shown a video clip, a scene from the movie "Marathon Man" (1976), where a man is tortured via a dental procedure. This clip has been shown to significantly change pain perception in an unpublished study done at Macalester College. Studies have shown that using violent or unsettling movie clips increased activation in the amygdala, an important structure implicated in anxiety (van Marle, Hermans, Qin, & Fernández, 2009; Heilman, Crişan, Houser, Miclea, & Miu, 2010). As anxiety is an emotional response to threats or the unknown, it is important that the prime induces this sense of threat or fear of the unknown to be sure our participants feel anxiety. Other studies have shown that these types of movie clips induce a significantly similar pattern of activation in the amygdala as studies done where participants were under constant threat of a potential electric shock (Clark et al, 2012). As fear of an electric shock is an acceptable paradigm for inducing anxiety in participants, and certain kinds of movie clips can induce the same brain activity, I believe this is a good method of anxiety induction for this study.

The participants then chose between a set of 35 binary prospects again, which were at the same probability levels. The pairings used in this phase were 25-0, 75-0, 50-0, 100-0, and 250-0. Like in the previous phase, at each probability

level, three additional questions were presented in order to narrow down the certainty equivalent. At the end of the second phase, another three questions from the second phase were repeated to check for consistency. During this period of the experiment, an image from or related to the movie scene they watched would randomly display on the screen. The random reminder of the prime was intended to keep the participants in the emotional state equivalent to their assigned group and prevent them from habituating to the stimulus.

At the end of the experiment, the participants were asked to answer a short self-reflective survey, a slight variation of the Short Form STAI, to indicate the level of anxiety, discomfort, or other emotion evoked by the movie clip.

VI. Definitions of Variables and Empirical Models

Definitions

For each individual, the certainty equivalent was calculated for each binary prospect and its matching probability level. The certainty equivalent, CE , was found by calculating the midpoint of the highest certain amount rejected and lowest certain amount accepted. Then, to calculate the decision weight, I calculated the ratio of CE/x , where x is the non-zero prospect (Tversky & Kahneman, 1992). To plot the decision weight curve, I graphed CE/x as a function of probability. The decision weight, w , is equal to the ratio CE/x . For example, suppose a participant is asked to choose between a 25% chance to win \$100 or \$40 for certain, and then asked if what they would choose the same lottery or \$30 for certain. If they say they will take \$40 for certain, but choose the bet over \$30 for certain, then their certainty equivalent equals \$35. Their decision weight, then, is 0.35.

The completed Short Form STAI produced a score measuring baseline anxiety on a scale of 20-80. This variable will be referred to as the *AScore*. After completion of the experiment, the participants then answered another Short Form STAI in relation to how the movie made them feel, which also produced a score on a scale of 20-80 and will be referred to as the *MScore*.

HRV was recorded throughout the entire experiment. A large body of literature connects HRV to anxiety and emotional regulation (Dulleck, Ristl, Schaffner, & Torgler, 2011; Cacioppo, Berntson, Larsen, Poehlmann, & Ito, 2000; Malik & Camm, 1990), specifically in stressful situations which are mediated by the vmPFC and amygdala pathway (Thayer, Åhs, Fredrikson, Sollers, & Wager, 2012). As both are important pathways in anxiety, I found HRV to be an appropriate physiological proxy for anxiety felt by the participant. The initial phase, used to calibrate the monitors, consisted of the period from which the participant put on the monitor, until the participant finished reading instructions. No heart rate data from this period was analyzed. The next phase, the “before” phase, began once the participant finished with all instructions. This phase continued until they have chosen between all 350 prospect pairs. The next period, called the “during” phase, began once they finish choosing prospects, and lasted until they finish viewing the movie clip. The final period, called the “after” phase, began after the movie clip finished, and lasted until they finished choosing between the last 315 prospects. As each participant completed the experiment in varying amounts of time, the most appropriate measure of HRV is the percentage of variable beats (pNN50).

Empirical Models

If I observe behavior consistent with Prospect Theory, there should be an obvious probability and certainty effect, meaning the resulting decision weight function should be nonlinear. The function I estimated is the following polynomial:

$$w(p) = \beta_0 + \beta_1 p + \beta_2 p^2 + \beta_3 p^3 \quad (1)$$

Where p is the probability, $w(p)$ is the decision weight, and the betas are coefficients.

Under expected utility theory, we would expect that $\beta_0 = 0, \beta_1 = 1, \beta_2 = 0,$ and $\beta_3 = 0$. However, under prospect theory, we expect that $\beta_0 \neq 0, \beta_1 \neq 1, \beta_2 \neq 0,$ and $\beta_3 \neq 0$.

In order to test the effects of the prime, within each treatment group I will test to see if there is a difference in the two curves before and after the priming video, as shown in the equation below:

$$w(p) = \beta_0 + \beta_1 p + \beta_2 p^2 + \beta_3 p^3 + \gamma_0 B + \gamma_1 p B + \gamma_2 p^2 B + \gamma_3 p^3 B + controls \quad (2)$$

where B is a dummy variable which equals zero before the prime and one after. In the anxiety group, I expect $\gamma_1 \neq \gamma_2 \neq \gamma_3 \neq 0$, with the direction of the change being ambiguous. In the reducing group, I also expect $\gamma_1 \neq \gamma_2 \neq \gamma_3 \neq 0$, and the curve should move in a direction opposite of the anxiety group, when controlling for baseline anxiety and physiological differences. Within the neutral group, we should see there is no change before and after the prime, so I expect $\gamma_1 = \gamma_2 = \gamma_3 = 0$.

Lastly, looking between groups, I expect to see that before the priming, all curves across all three groups should be relatively the same. Using Equation 3, the dummy variable G represents the specific control group. There should be no

difference between groups before the prime, which means $\gamma = \gamma_1 = \gamma_2 = \gamma_3 = 0 = 0$.

$$w(p) = \beta_0 + \beta_1 p + \beta_2 p^2 + \beta_3 p^3 + \gamma_0 G + \gamma_1 pG + \gamma_2 p^2 G + \gamma_3 p^3 G + \text{controls} \quad (3)$$

After the prime, also using Equation 3, there should be a difference across groups. In comparing the anxiety group to the neutral group $\gamma_1 \neq 0$, or $\gamma_2 \neq 0$, or $\gamma_3 \neq 0$. Furthermore, I expect $\gamma_1 \neq 0$, or $\gamma_2 \neq 0$, or $\gamma_3 \neq 0$ does not equal zero when comparing anxiety to the reducing group.

VII. Results

Summary Statistics

Examining the certainty equivalents collected before the experimental prime was introduced, I find there is not initially a large difference between groups. Tables 1, 2, and 3, show the median cash equivalents of the anxiety inducing, reducing, and neutral groups respectively⁴.

The priming method did indeed make a difference in anxiety levels across groups. In looking at the variable AScore, which is the composite score of the Short Form STAI, the mean base level anxiety for each treatment group is relatively the same. Table 4 shows these differences. The control groups are relatively similar in their baseline anxiety, which affirms that participants with differing levels of innate anxious participants were randomly distributed throughout the three treatment groups, and therefore should not have any significant impact on group level results. The MScore, the self-reported anxiety the participant claimed their respective

⁴ See Appendix A for presentations of mean cash equivalents.

treatment clip made them feel, differed across groups. Those in the anxiety group saw a nearly 20-point increase in anxiety levels, and also had higher anxiety levels than the neutral and reducing treatment groups. The reducing group saw a small decrease in anxiety levels. The neutral group also reported decreasing anxiety levels, but this is likely not because the movie made them less anxious, but rather due to the wording of the question. The MScore was based on how the movie made them *feel* and it is likely that the movie clip induced no anxiety, effectively lowering their MScore.

To help eliminate any speculation about discrepancies in self-reported data versus actual emotion felt, I measured HRV to approximate the inner activation of the sympathetic nervous system. Research shows lower HRV is correlated with higher levels of anxiety and aggression (Cacioppo et al, 2000). Table 5 shows a decrease in median pNN50 after the viewing of the movie clip in the anxiety group, indicating the movie did in fact make the participants anxious. Furthermore, we see an increase in pNN50 for those in the reducing group, showing that although they self-reported small decreases in anxiety, their physiological response showed greater reduction in anxiety. The neutral group showed no significant decrease in median pNN50, therefore, even though the group reported less anxiety their body state did not change. Collectively, these results show the priming method had effects on each group, and should be effective in creating any differences in the results.

Median certainty equivalents for post-treatment are shown in Table 6, 7, and 8. Figure 5 shows the median CEs for each treatment group both before and after

treatment. There is a small difference before and after the prime in the anxiety group, but there does not appear to be one in the neutral or reducing group. It is important to note the graphs in Figure 5 show median CE's fitted with a loess smoother, and no conclusions can be drawn from them.

Analysis

To estimate equations 1-3 and control for differences between individuals, I clustered data by participant ID number. All data was transformed by a factor of 100. Table 9 summarizes the findings of the within anxiety group analysis⁵. Model 1 shows that with no other factors considered, the anxiety group shows a nonlinear relationship between decision weights and probabilities, consistent with the expectations of prospect theory. Within group analysis shows that in the anxiety group there is no significant effect of the treatment ($p=0.141$) as suggested by Model 3, however, this does seem to trend towards significance when controlling for baseline anxiety levels, as shown in Model 4 ($p=0.1310$). When interacting the treatment with each variable, Model 3 shows there likely is not a difference in curvature of the decision weight function before and after treatment, suggesting that the results of the other models are more indicative of a downward shift of the decision weight curve when in an anxious state rather than not.

As shown in Table 10, within the reducing group there are no significant effects of the treatment. The cluster analysis shows no shifting of the curve or change in curvature before and after exposure to the priming video⁶. Table 11

⁵ See Appendix A for graphs of fitted regression values.

⁶ See Appendix B for graphs of fitted regression values.

summarizes the analysis within the neutral group⁷. Interestingly, there is no significant effect of the prime in models 2 and 4 ($p=0.22$, $p=0.227$), however, when interacting the treatment with the curvature, we find there is a significant difference before and after the treatment video ($p=0.03$). Therefore, there appears to be an upward shift in the decision weight curves before and after treatment in the neutral group.

There also did not appear to be significant differences between treatment groups after the presentation of the priming video, as shown in Table 12. Looking between the anxiety and neutral group⁸, the anxiety group has a decision weight function which is shifted downward from the neutral group, though this relationship is not statistically significant across any of the models presented in Table 12. Furthermore, there does not seem to be a difference in curvature between the two groups. The anxiety group also showed no significant differences from the reducing group post treatment, with the regressions in Table 13 suggesting the anxiety group had a curve shifted upward from the reducing group, and that the anxiety group had a more extreme curvature⁹. Between the reducing group and neutral group¹⁰, the reducing group shows significant downward shift from the neutral group when controlling for differences in curvature between groups ($p=0.005$), although the differences in curvature do not appear to be significant themselves¹¹.

⁷ See Appendix C for graphs of fitted regression values.

⁸ See Appendix D for graphs of fitted regression values.

⁹ See Appendix E for graphs of fitted regression values.

¹⁰ See Appendix F for graphs of fitted regression values.

¹¹ See Table 14.

The curves are estimated along seven different probability levels. In order to look at the differences between the curve, I analyzed the differences along each point of the curve before and after treatment or across treatment group using two sample t-tests. Within the anxiety group, presented in Table 15, there is no significant difference between the points at low probabilities before and after treatment. However, at probability=0.75 and probability=0.95 there is a significant difference between the mean decision weight. Looking between the anxiety group and reducing group post treatment, shown in Table 16, we see that there is no significant difference at high or low probabilities, which is likely because both groups showed a downward shift from their respective baselines. However, when looking at the difference between the anxiety and neutral group, shown in Table 17, we see a significant difference between the mean decision weight at almost all estimated points along the curve.

VIII. Discussion

The first notable finding of this study is that across all treatment groups, both before and after treatment, I find significant evidence of an s-shaped curve. Furthermore, the s-shape described by prospect theory holds under varying levels of state anxiety. Prospect theory is robust enough to explain decision making tendencies even when in emotional states such as anxiety. Anxiety does not change the fundamental s-shape of prospect theory into a new function, however it could lead to shifts in the curve or slight changes in amplitude. Regardless, this study supports the notion that the s-shape decision weight function inherently underlies decision making when faced with uncertainty.

Secondly, it is noteworthy that between the neutral and anxiety groups, when comparing the curves on a point by point estimated basis, we see the decision weights are significantly different along the curve. We see that at low probabilities, those less than 0.5, the decision weights were significantly lower than when in a neutral state, and the decision weights at higher probabilities are also significantly lower. The lower decision weights in an anxious state show the participants value the lotteries less than they do in a neutral state and are consequently less likely to take a risk at any given probability. Therefore, in an anxious state, people are more risk averse.

The results of the t-tests support the results of the regression analysis. Although regression analysis shows no significant change in the shape of the decision weight curve when in an anxious state, they do suggest some interactions with anxiety which warrant further investigation. Within the anxiety group, regression analysis shows there is a downward shift of the curve after exposure to the priming video, indicating the participants weighted lotteries less than their expected values. Therefore, in an anxious state, the results suggest participants are more risk averse, consistent with the literature (Raghunathan & Pham, 1999; Maner et al, 2007; Robinson, Vytal, Cornwell, & Grillon, 2013).

Furthermore, when comparing across groups, we see the anxiety group appears slightly more curved than the neutral and reducing group, and the reducing group has a flatter curvature than the neutral group. More convexity of the decision weight curve at high probabilities indicates the participant expects a larger disutility if wrong. Therefore, when in an anxious state, it could be that the body anticipates a

larger decrease in dopamine firing if wrong, leading to the more extreme underweighting found in the anxiety group. Furthermore, the reducing group appears to have a flatter curvature post treatment compared to the anxiety group. As those in the reducing group exhibited more HRV, indicating more relaxation and emotional regulation, the body may not have been creating feedback loops which amplified the excitement or fear of winning and losing, leading to a flatter curve. The anxiety group, with a lower HRV, had poorer regulation and therefore allowed body states to compound (Bechara & Damasio, 2005) and lead to an exaggeration of the curve. However, since the results are not statistically significant, we cannot take these hypotheses as outright conclusions from this study, but instead perhaps as factors to consider or investigate in future research.

It is also interesting to note that both the anxiety and reducing groups showed a downward shift in the decision weight curve relative to the neutral group post treatment. This seems like an unlikely outcome, as we would expect that if inducing anxiety moves the curve in one direction, then reducing anxiety should move it in the opposite direction. Several interpretations could explain this. One is that the prime did not work and did not induce the intended effect. However, we can see in the HRV data and self-reported anxiety scores that this is not the case. It could also mean that the decision weight curve is unaffected by emotion, however, literature linking risky choice to affective quality suggests this is not the case (Hsee & Rottenstreich, 2004). Then it is possible that the decision weight curve is more heavily influenced by baseline anxiety levels and an individual's departure from that baseline. These differences in anxiety levels could be more potent in determining

one's decision weight function than the experimental treatment. This may explain why in the within anxiety group analysis, shown in Table 9, the effect of the *AScore* is significant, but the effect of the treatment is not. Therefore, it may be that each individual has an inherent shape of their decision weight curve which varies based on that individual's trait anxiety. Induction of an anxious state may exacerbate the shape, but does not entirely change it.

The study had several limitations. It is difficult to keep participants in an experimentally induced emotional state for an extended period of time. While during the movie clip, key brain areas involved in anxiety, such as the amygdala, may have been activated (van Marle, Hermans, Qin, & Fernández, 2009; Heilman, Crişan, Houser, Miclea, & Miu, 2010), it is likely that this effect wore off over time. We do see that the mean pNN50 for the anxious group is lower after the prime, but it could be that all the low variance occurred almost immediately after the priming video and increased as the experiment progressed, indicating anxiety decreased later in the experiment. It is also likely that the anxiety felt in the moment of the prime far exceeded the anxiety felt afterwards while answering questions. Although I tried to keep participants primed throughout the second phase of the experiment with random GIF images relating to their movie clip, this may not have been salient enough to produce significant effects. This may be why the results trend towards significant but are not. In the future, it may be best to study this phenomena with better experimental paradigm, such as continued threat of electric shock (Clark et al, 2012).

Another factor which may have limited the results I refer to as the “boredom factor.” To experimentally estimate a curve of 35 points, the participant had to answer 315 questions, both before and after watching the video. While this took most participants only 10-20 minutes, it is possible that they may have gotten bored and thus did not answer questions with honest appraisal, but did so hastily just to finish. In this haste, many participants indicated they held the same certainty equivalent regardless of probability. For example, within the anxiety group post treatment¹², the mean certainty equivalent for a 50% chance and 75% chance to win \$50 was \$31.20. There are several instances of this in the data, or very minute changes in certainty equivalents which are not proportional to the changes in probability. While it is possible that participants may hold the same certainty equivalent across a range of probabilities, which would imply their decision weight function would be flat with steps, this is not likely and has not been found in other studies investigating its shape (Wu, 1999; Tversky and Kahneman, 1992; Fehr-Duda et al, 2011). Therefore, the “boredom” factor may have limited my results by creating functions which are not representative of the participant’s real preferences.

Lastly, this study assumed that decision weights would change depending on physiological state, regardless of the affective quality tied to the question. It could be that if the internal state and emotional value attached to an outcome are independent of each other, there is not an observable effect. However, if the outcomes themselves have an emotional value attached to them, there may be a distinctive difference in how an individual weights decisions. For example, the study

¹² See Table 6.

asked questions with a simple lottery, and these numbers themselves are very neutral as emotional stimuli. However, if the question would have been worded that there is a 10% chance to win a trip to Disney World, or \$50 for certain, this may have changed the value because there is a salient excitement associated with the thought of Disney World which may not have been associated with a numeric lottery of the same value. This effect, found in several studies (Hsee & Rottenstreich, 2004; Rottenstreich & Hsee, 2001), may be more important in determining decision weights. Therefore, it is possible decision weights may vary with the emotional quality induced by a potential outcome, but not with a random emotional state which is independent of the decision at hand.

IX. Conclusion

The emotional interaction with underlying decision weights warrants further investigation. Emotion is integral to the decision-making process, and the more we understand how emotions interact with the decision-making process, the better we will understand human behavior. Furthermore, we should continue to investigate why we have a tendency for probability distortion, as understanding this phenomenon will help to build better models of decision in uncertainty. An interdisciplinary approach, using neurological and psychological research, will continue to be important in the updating and improvement of economic models.

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Decision Weight Function

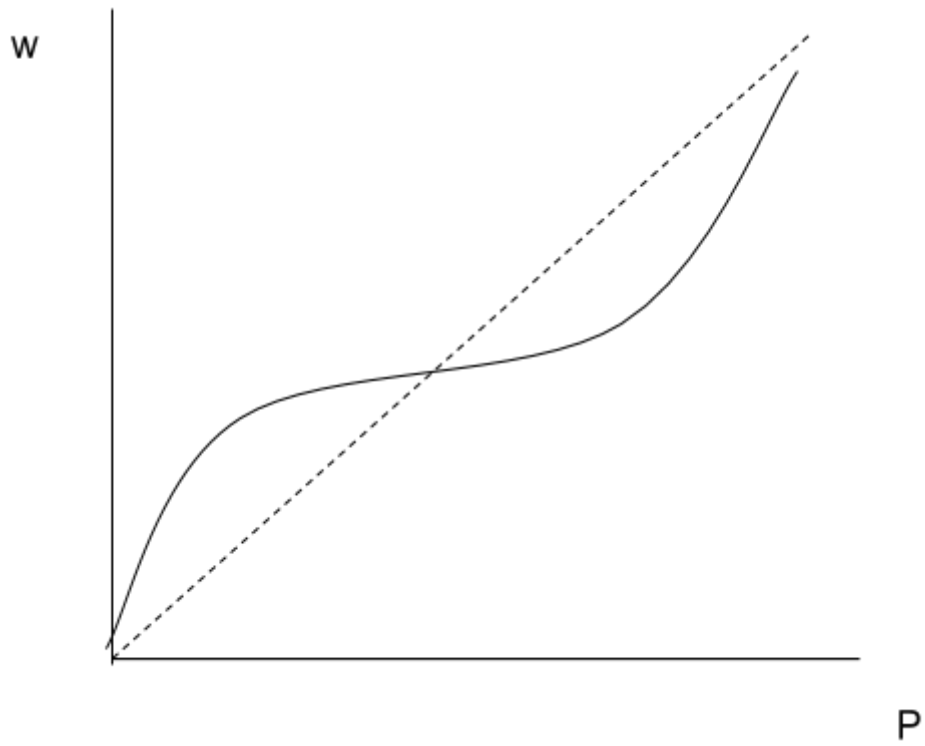
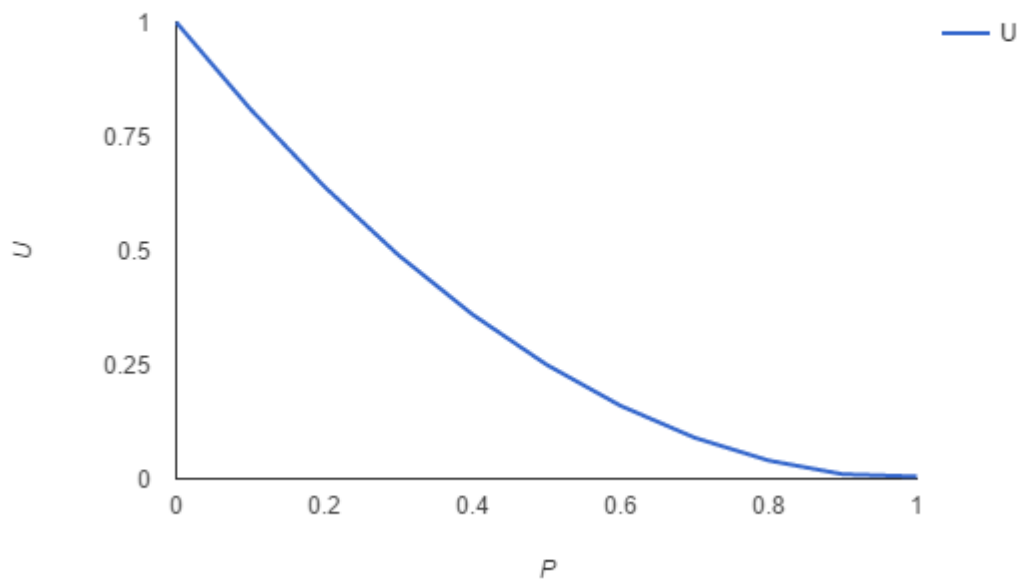


Figure 1. A decision weight curve as proposed by Cumulative Prospect Theory (Tversky & Kahneman, 1992).



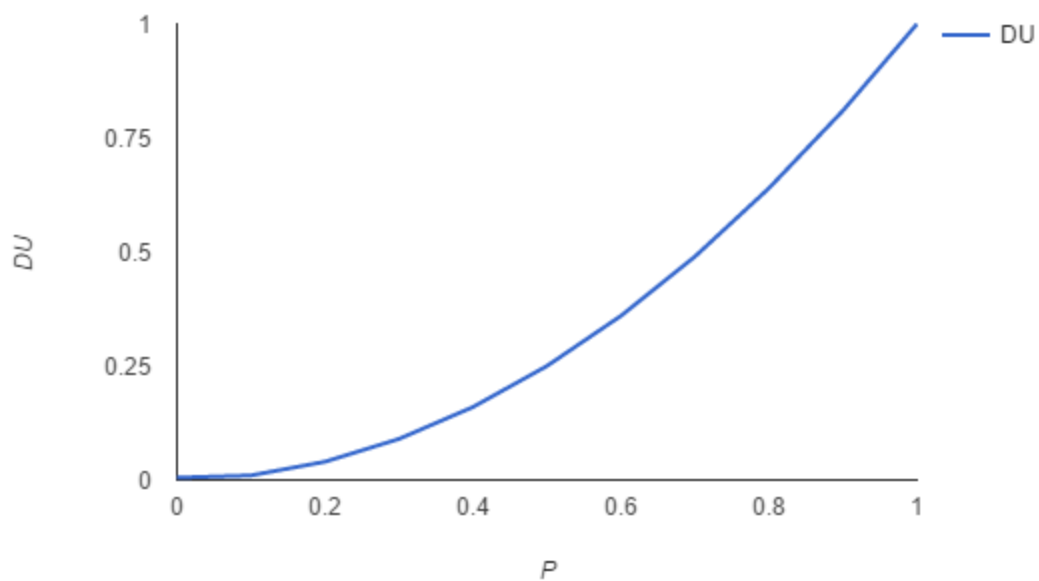


Figure 2. Nonlinear relationship between utility (top), disutility (bottom), and expectations regarding the probability of an event occurring. *DU* is the disutility associated with an event, where p is the individual's own expectation of how probable an event is.

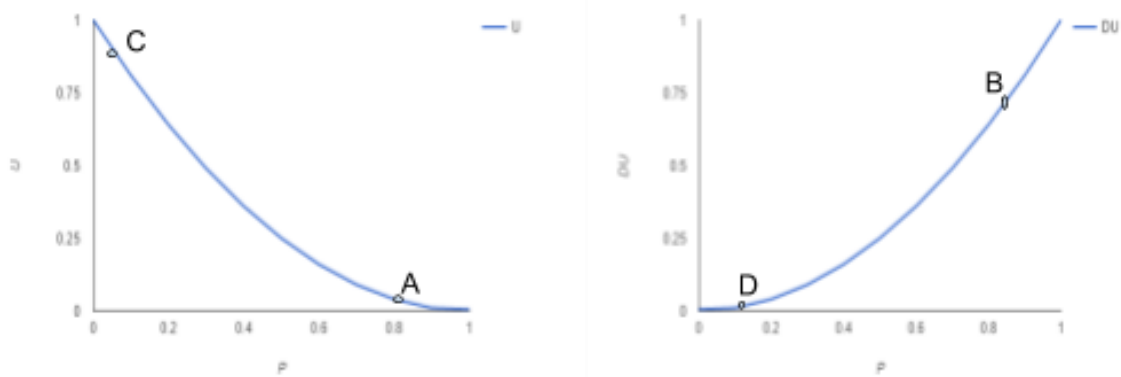


Figure 3. Example illustrating the utility and disutility associated with passing or failing a test based on expectations the individual has about how they will perform on it.

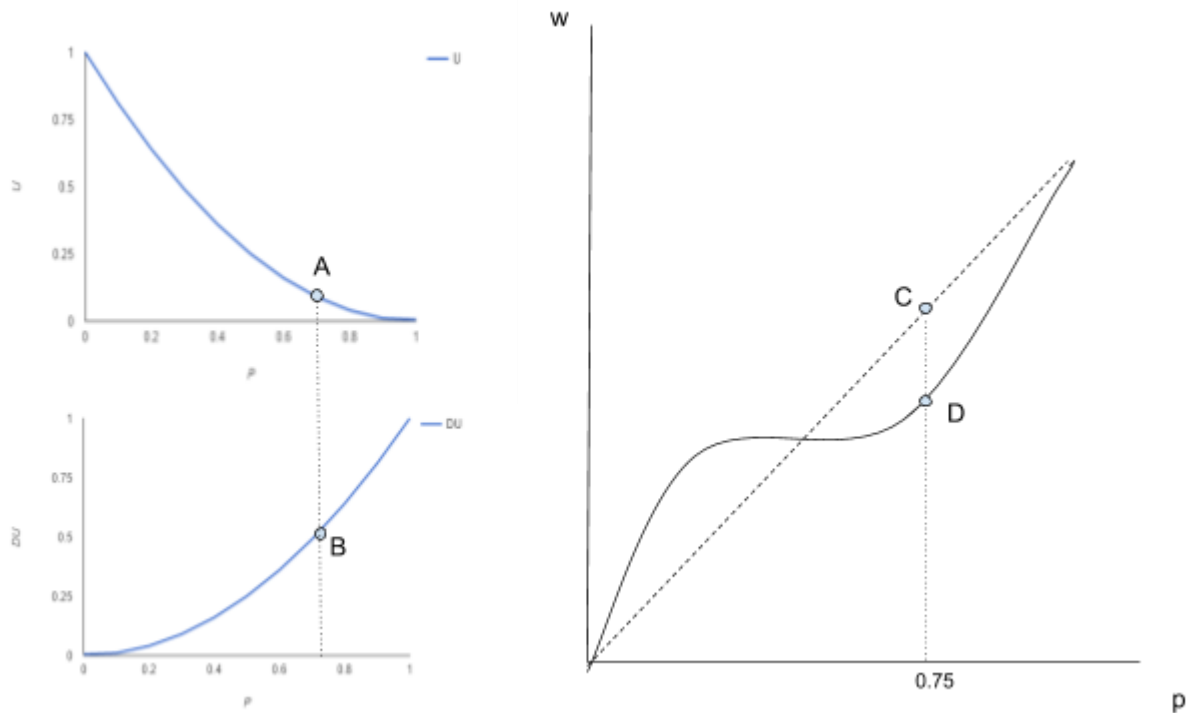


Figure 4. Example about dating used to illustrate how potential utilities associated with an outcome combine to form a decision weight.

	0.05	0.1	0.25	0.5	0.75	0.9	0.95
25-0	4.165	5.625	6.875	13.62	15	16.88	16.88
50-0	6.260	11.67	11.67	21.34	29.17	29.17	40.67
75-0	5.875	9.625	17.5	33.12	40.62	42.19	62.59
100-0	12	12	24.4	39.2	49.04	63.5	81.5
250-0	7.5	25	45	80.2	80.2	162.5	187.5

Table 1. Median CEs in dollars of the anxiety group before exposure to the priming video. Each cell represents the certainty equivalent, or the amount that makes the participant indifferent between the lottery and a certain amount of money. For example, if there is a p probability of winning $\$x$ then at $\$CE$, the participant is indifferent between the lottery and the sure amount $\$x$. For example, the top left cell can be interpreted as the following: given the option of a 5% chance to win $\$25$ or $\$0$ otherwise, or being given $\$4.165$ for certain, the participant is indifferent between the lottery and the sure amount of money.

	0.05	0.1	0.25	0.5	0.75	0.9	0.95
25-0	7.5	4.125	4.585	11.19	13.120	16.88	19.24
50-0	2.34	9.005	13.84	19.34	26.26	37	40.67
75-0	9.625	13.12	17.5	24	41	53.75	55.9
100-0	12	12	24.34	46.68	55.42	73.5	89.25
250-0	25	25	43.95	85.12	129.8	187.5	200.4

Table 2. Median CEs in dollars of the reducing group before exposure to the priming video. Each cell represents the certainty equivalent.

	0.05	0.1	0.25	0.5	0.75	0.9	0.95
25-0	4.125	5.25	6.688	13.12	15	13.62	14.88
50-0	9.005	11.67	19.34	25.51	27.34	37	37
75-0	10.88	13.75	17.5	29.12	40.62	53.75	60.2
100-0	13	19.1	33.6	46.68	52.35	73.5	81.5
250-0	25	32.5	45.4	83.5	96.7	148.0	187.5

Table 3. Median CEs in dollars of the neutral group before exposure to the priming video. Each cell represents the certainty equivalent.

Group	AScore	MScore
Anxiety	35.18 (10.03968)	55.53 (13.36022)
Reducing	34.42 (9.464377)	31.46 (10.21853)
Neutral	34.67 (7.466256)	30.35 (7.270122)

Table 4. Mean AScore and MScore by treatment group. Standard deviations reported in parenthesis.

Group	Before	After
Anxiety	11.770 (21.48216)	8.9280 (19.48098)
Reducing	13.270 (11.37674)	16.7600 (12.66769)
Neutral	18.7800 (16.74832)	19.7400 (11.31822)

Table 5. Median pNN50 before and after viewing of the treatment video by treatment group. Standard deviations reported in parenthesis.

	0.05	0.1	0.25	0.5	0.75	0.9	0.95
25-0	5.2	5.625	6.745	10.52	12.19	16.88	19.14
50-0	6.26	6.26	16	21.34	21.34	37	37.84
75-0	3.625	9.625	17.500	33.12	33.12	43.75	53.75
100-0	12	12	24.4	41.76	41.68	62.5	74.5
250-0	18.5	25	42.5	69.35	106.8	129.8	162.5

Table 6. Median CEs in dollars of the anxiety group after exposure to the priming video. Each cell represents the certainty equivalent.

	0.05	0.1	0.25	0.5	0.75	0.9	0.95
25-0	2.812	4.585	5.875	10.42	13.120	13.12	18.0.1
50-0	6.26	9.005	11.67	19.34	26.26	37	37
75-0	3.625	9.625	17.5	26.62	50	53.75	60.62
100-0	12	12	24.4	46.68	46.68	73.5	81
250-0	7.5	25	42.5	80.2	134.8	162.5	187.5

Table 7. Median CEs in dollars of the reducing group after exposure to the priming video. Each cell represents the certainty equivalent.

	0.05	0.1	0.25	0.5	0.75	0.9	0.95
25-0	5	9.29	8.125	13.12	13.12	13.12	17.56
50-0	9.585	11.67	19.34	22.42	32	37	37
75-0	10.88	13.75	24.12	40.62	46.25	53.75	53.75
100-0	12	19.1	24.4	46.68	46.85	73.5	73.5
250-0	32.5	42.5	52.5	80.2	129.8	129.8	187.5

Table 8. Median CEs in dollars of the neutral group after exposure to the priming video. Each cell represents the certainty equivalent.

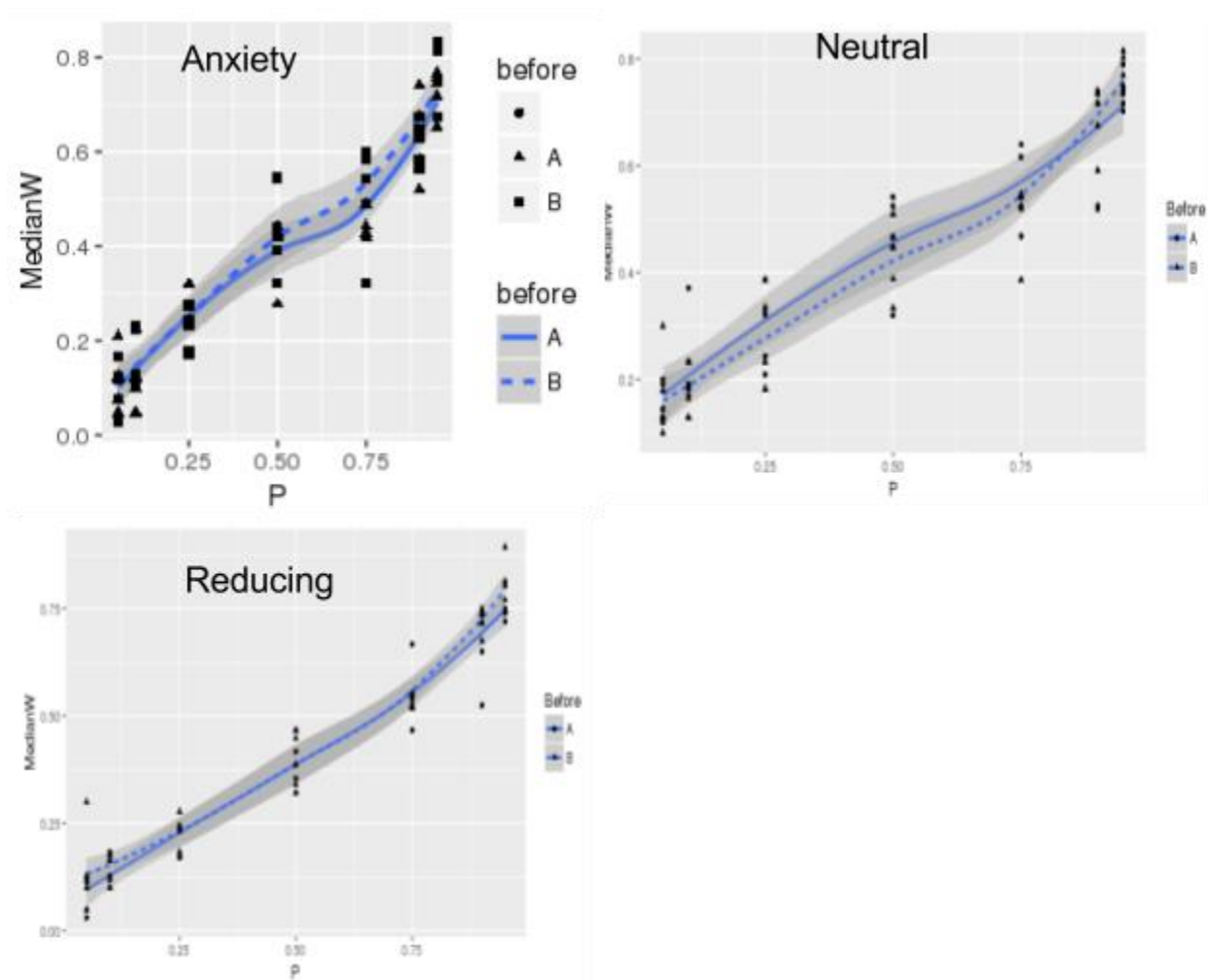


Figure 5. Graphs of median probabilities vs median decision weights for each treatment group, both before and after exposure to the priming video. Lines fitted with a loess smoother.

CLUSTER WITHIN ANALYSIS OF ANXIETY GROUP				
Dependent variable= w (Decision Weight)				
	Model 1	Model 2	Model 3	Model 4
p	1.155	1.164	1.156	1.1617
	9.85***	9.99***	5.55***	9.77***
p^2	-0.0162	-0.0164	-0.0144	-0.0163
	-5.99***	-6.10***	-3.04***	-5.97***
p^3	1.16e-4	1.17e-4	9.98e-5	1.17e-4
	6.36***	6.48***	3.28***	6.37***
Before (B)	-	-1.8218	-0.0054	-1.870
($B=0$ before treatment)		-1.53	0.02	-1.57
$B \times p$	-	-	-0.0036	-
			-0.60	
$B \times p^2$	-	-	-3.67e-3	-
			0.60	
$B \times p^3$	-	-	3.30e-5	-
			0.83	
AScore	-	-	-	-0.5109
				-3.48***
Intercept	7.650	8.461	6.686	26.45
	3.50***	4.18***	2.87***	4.37***
F Stat	84.42	69.42	83.25	79.79
R^2	0.588	0.589	0.592	0.625
N	1440	1439	1439	1439

Table 9. Cluster analysis of anxiety group. T statistics reported underneath coefficient value. *Significant at the 10% level. ** Significant at the 5% level. ***Significant at the 1% level.

CLUSTER WITHIN ANALYSIS OF REDUCING GROUP				
Dependent variable= w (Decision Weight)				
	Model 1	Model 2	Model 3	Model 4
p	0.9647	0.9634	1.026	0.9630
	9.02***	9.05***	7.92***	9.04***
p^2	-0.0115	-0.0115	-0.0128	-0.0115
	-3.40***	-3.43***	-3.52***	-3.43**
p^3	9.07e-5	9.05e-5	1.00e-4	9.04e-5
	3.59***	3.62***	3.85***	3.61***
Before (B)	-	-2.381	0.3856	-22.332
($B=0$ before treatment)		-1.21	0.23	-1.17
$B \times p$	-	-	-0.1388	-
			-0.79	
$B \times p^2$	-	-	2.89e-3	-
			-0.67	
$B \times p^3$	-	-	-2.13e-5	-
			-0.71	
AScore	-	-	-	0.0011
				0.36
Intercept	7.226	8.35	7.06	4.56
	4.78***	4.72***	4.34***	0.45
F Stat	75.29	55.48	43.19	75.99
R^2	0.604	0.6058	0.606	0.607
N	1302	1302	1302	1302

Table 10. Cluster Analysis results of reducing group. T statistics reported underneath coefficient value.

*Significant at the 10% level. ** Significant at the 5% level. ***Significant at the 1% level.

CLUSTER WITHIN ANALYSIS OF NEUTRAL GROUP				
Dependent variable= w (Decision Weight)				
	Model 1	Model 2	Model 3	Model 4
p	0.9701	0.9685	1.047	0.9721
	5.56***	5.55***	5.71***	5.63***
p^2	-0.0122	-0.0122	-0.0135	-0.0123
	-4.01***	-4.00***	-4.07***	4.08***
p^3	8.86e-5	8.84e-5	9.70e-5	8.88e-5
	4.75***	4.73***	4.86***	4.84***
Before (B)	-	1.8324	4.973	1.812
($B=0$ before treatment)		1.26	2.32**	1.25
$B \times p$	-	-	-0.1609	-
			-1.36	
$B \times p^2$	-	-	2.76e-3	-
			0.88	
$B \times p^3$	-	-	-1.74e-05	-
			-0.78	
AScore	-	-	-	-0.2207
				-0.50
Intercept	13.98	13.09	11.54	20.70
	6.62***	6.65***	6.01***	1.33
F Stat	78.31	61.55	38.55	67.67
R^2	0.519	0.520	0.521	0.524
N	1448	1448	1448	1448

Table 11. Cluster Analysis results of reducing group. T statistics reported underneath coefficient value. *Significant at the 10% level. ** Significant at the 5% level. ***Significant at the 1% level.

CLUSTER ANALYSIS BETWEEN ANXIETY AND NEUTRAL GROUP AFTER TREATMENT Dependent variable= w (Decision Weight)				
	Model 1	Model 2	Model 3	Model 4
p	1.0658	1.0655	0.9071	1.0342
	10.67***	10.65***	5.62***	8.35***
p^2	-0.0144	-0.0144	-0.0122	-0.0142
	-6.66***	-6.67***	-4.06***	-6.11***
p^3	1.04e-4	1.04e-4	8.86e-5	1.04e-4
	7.16***	7.17***	4.81***	7.06***
Group (G)	-	-4.351	-5.325	-4.1653
(anxiety=1)		-1.14	-1.51	-1.12
$G \times p$	-	-	0.1808	-
			0.87	
$G \times p^2$	-	-	-5.83e-3	-
			-1.31	
$G \times p^3$	-	-	4.42e-5	-
			1.51	
AScore	-	-	-	-0.3870
				-1.72*
Intercept	10.61	13.51	13.98	27.02
	5.91***	4.37***	6.70***	3.31***
F Stat	130.25	99.25	79.13	67.67
R^2	0.549	0.554	0.528	0.524
N	2161	2161	2157	2157

Table 12. Cluster Analysis results of neutral group. T statistics reported underneath coefficient value.
*Significant at the 10% level. ** Significant at the 5% level. ***Significant at the 1% level.

CLUSTER ANALYSIS BETWEEN ANXIETY AND REDUCING GROUP AFTER TREATMENT				
Dependent variable= w (Decision Weight)				
	Model 1	Model 2	Model 3	Model 4
p	1.074	1.074	0.964	1.074
	11.42***	11.45***	9.15***	11.34***
p^2	-0.014	-0.0143	-0.0115	-0.0143
	-6.18***	-6.20***	-3.44***	-6.13***
p^3	1.05e-4	1.05e-4	9.07e-5	1.05e-4
	6.52***	6.54***	3.64***	6.49***
Group (G)	-	1.628	1.1435	1.941
(anxiety=1)		0.43	0.45	0.51
$G \times p$	-	-	0.1861	-
			1.18	
$G \times p^2$	-	-	-6.51e-3	-
			-1.39	
$G \times p^3$	-	-	4.21e-5	-
			1.25	
AScore	-	-	-	-0.3165
				-2.06**
Intercept	7.59	6.45	7.22	17.27
	4.71***	2.23**	4.85***	2.68**
F Stat	126.60	94.93	81.62	78.00
R^2	0.580	0.581	0.584	0.599
N	2052	2052	2011	2052

Table 13. Cluster Analysis results between anxiety group and neutral group after exposure to priming video. T statistics reported underneath coefficient value.

*Significant at the 10% level. ** Significant at the 5% level. ***Significant at the 1% level.

CLUSTER ANALYSIS BETWEEN NEUTRAL AND REDUCING GROUP AFTER TREATMENT Dependent variable= w (Decision Weight)				
	Model 1	Model 2	Model 3	Model 4
p	0.0094	0.0094	0.0088	0.0094
	7.27***	7.29***	4.84**	7.34***
p^2	-1.15e-4	-1.15e-4	-1.08e-4	-115e-4
	7.27***	-4.60***	-3.10***	-4.63***
p^3	8.51e-7	8.51e-7	7.95e-7	8.51e-7
	5.20***	5.26***	3.43***	5.30***
Group (G)	-	-0.0506	-0.0929	-0.0509
(anxiety=1)		-1.20	-2.99***	-1.22
$G \times p$	-	-	7.81e-4	-
			0.37	
$G \times p^2$	-	-	-7.53e-7	-
			-0.16	
$G \times p^3$	-	-	1.12e-7	-
			0.33	
AScore	-	-	-	-0.0005
				-0.18
F Stat	117.81	88.03	66.66	77.46
R^2	0.529	0.536	0.567	0.536
N	2060	2060	2023	2060

Table 14. Cluster Analysis results between neutral group and reducing group after exposure to priming video. T statistics reported underneath coefficient value.

*Significant at the 10% level. ** Significant at the 5% level. ***Significant at the 1% level.

T-Test Comparisons of Anxiety Group Before and After Treatment				
p	Mean w Before	Mean w After	T stat	p-value
0.05	0.124	0.146	-1.226	0.221
0.10	0.172	0.181	-0.509	0.610
0.25	0.263	0.269	-0.277	0.782
0.5	0.426	0.393	1.367	0.173
0.75	0.545	0.484	2.236	0.026**
0.90	0.629	0.611	0.671	0.503
0.95	0.746	0.697	1.771	0.078*

Table 15. Within anxiety group comparison of decision weights, w , both before and after treatment at each probability level using two sample t-tests. *Significant at the 10% level. **Significant at the 5% level. ***Significant at the 1% level.

T-Test Comparisons of Between Anxiety and Reducing Group After Treatment				
p	Mean w Anxiety	Mean w Reducing	T stat	p-value
0.05	0.146	0.114	1.884	0.061*
0.10	0.181	0.163	0.867	0.386
0.25	0.269	0.231	1.700	0.090*
0.5	0.393	0.382	0.429	0.668
0.75	0.484	0.510	-0.831	0.406
0.90	0.611	0.622	-0.359	0.719
0.95	0.697	0.709	-0.376	0.707

Table 16. Between anxiety group and reducing comparison of decision weights, w , both before and after treatment at each probability level using two sample t-tests. *Significant at the 10% level. **Significant at the 5% level. ***Significant at the 1% level.

T-Test Comparisons of Between Anxiety and Neutral Group After Treatment				
p	Mean w Anxiety	Mean w Neutral	T stat	p-value
0.05	0.146	0.196	-2.476	0.014**
0.10	0.181	0.262	-3.521	0.000***
0.25	0.269	0.320	-2.041	0.042**
0.5	0.393	0.441	-1.779	0.076*
0.75	0.484	0.554	-2.472	0.014**
0.90	0.611	0.664	-1.872	0.062*
0.95	0.697	0.712	-0.500	0.617

Table 17. Between anxiety group and neutral group comparison of decision weights, w , both before and after treatment at each probability level using two sample t-tests.

*Significant at the 10% level. ** Significant at the 5% level. ***Significant at the 1% level.

Appendix A: Graphs of Within Anxiety Group Fitted Polynomial Regression Results

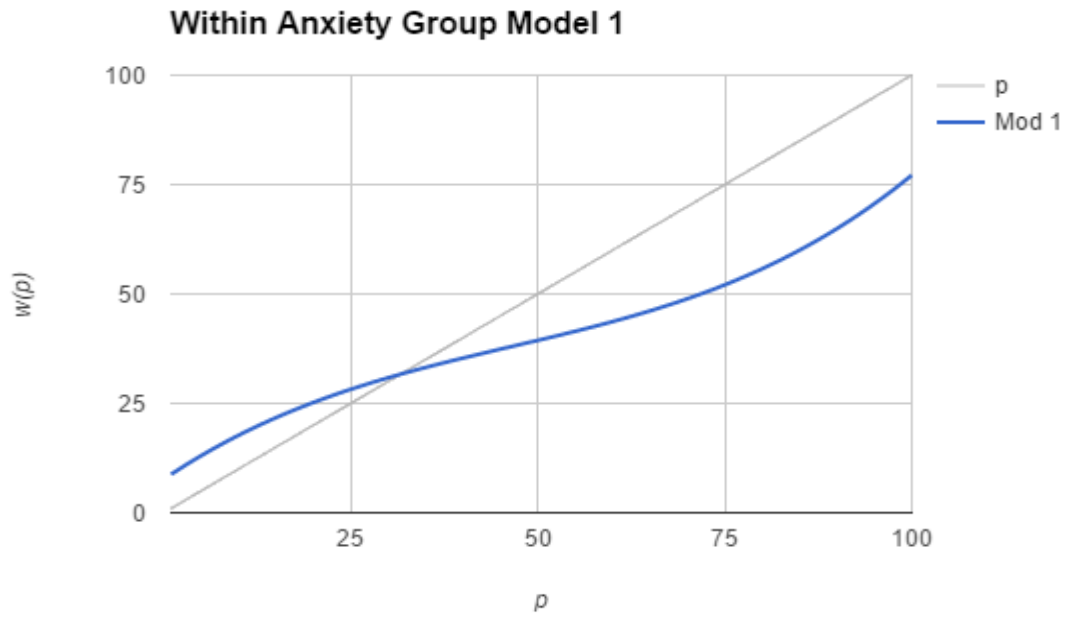


Figure 1. Graph of decision weights versus probability using fitted values of Table 9, Model 1.

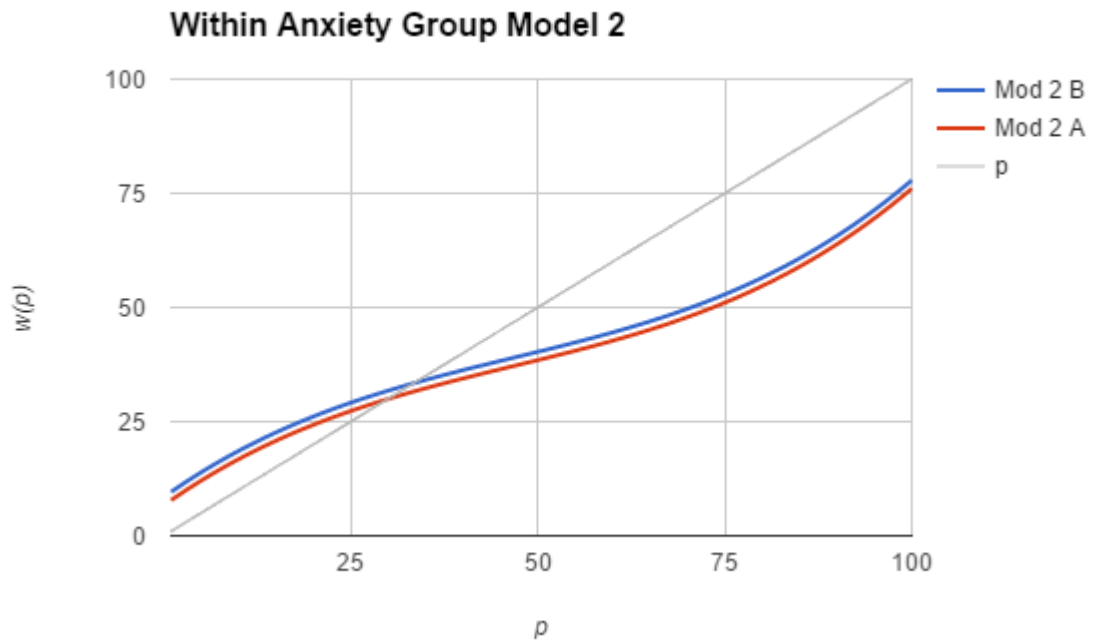


Figure 2. Graph of decision weights versus probability using fitted values of Table 9, Model 2, where B denotes before treatment and A denotes after treatment.

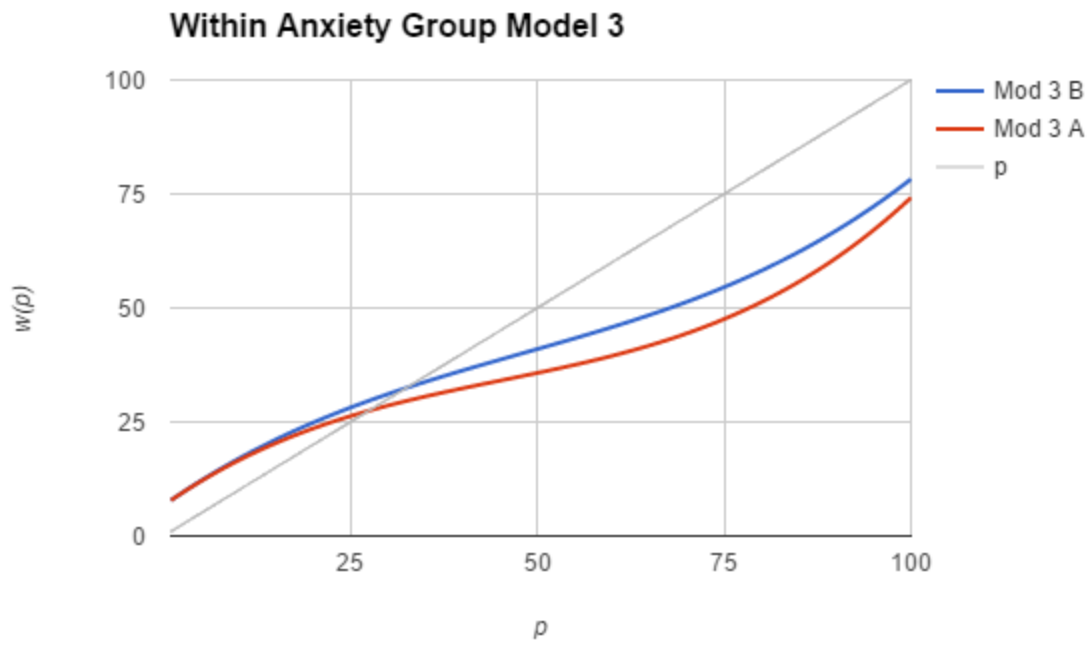


Figure 3. Graph of decision weights versus probability using fitted values of Table 9, Model 3.

Appendix B: Graphs of Within Reducing Group Fitted Polynomial Regression Results

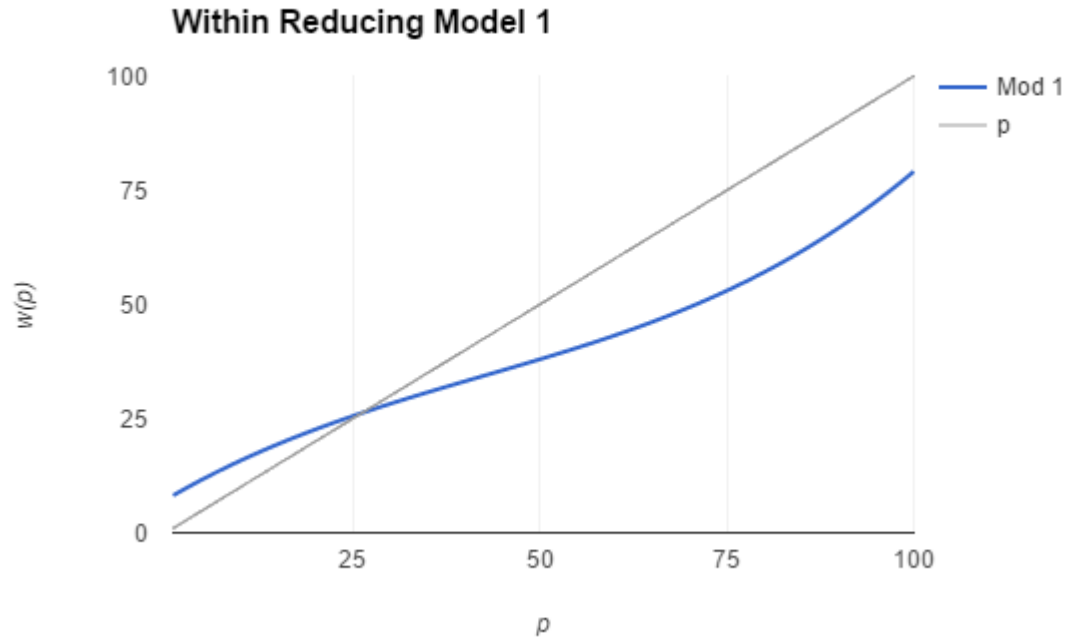


Figure 1. Graph of decision weights versus probability using fitted values of Table 10, Model 1.

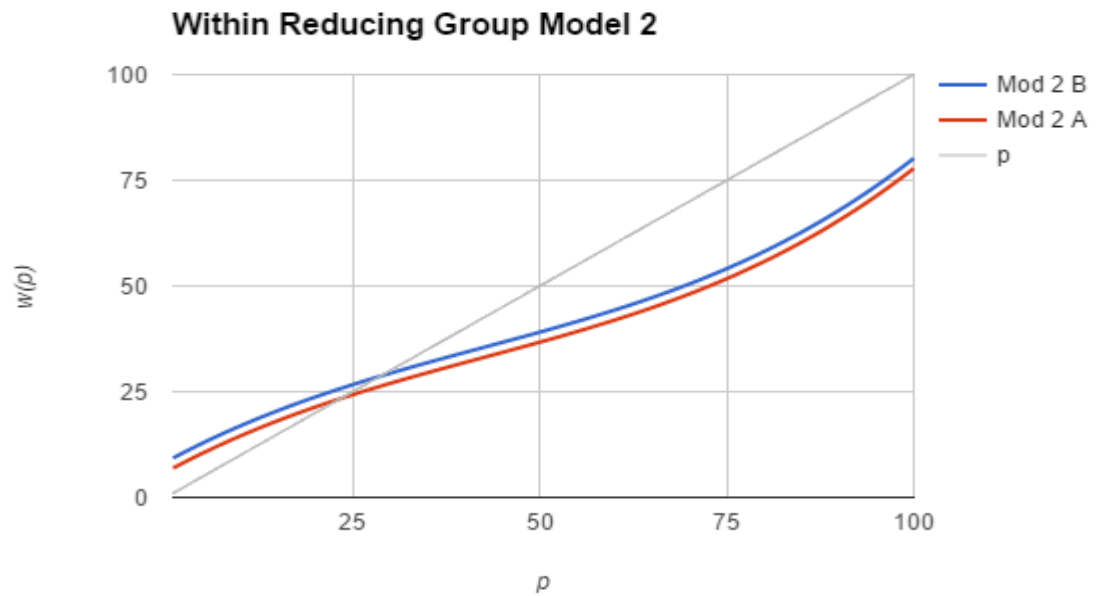


Figure 2. Graph of decision weights versus probability using fitted values of Table 10, Model 2.

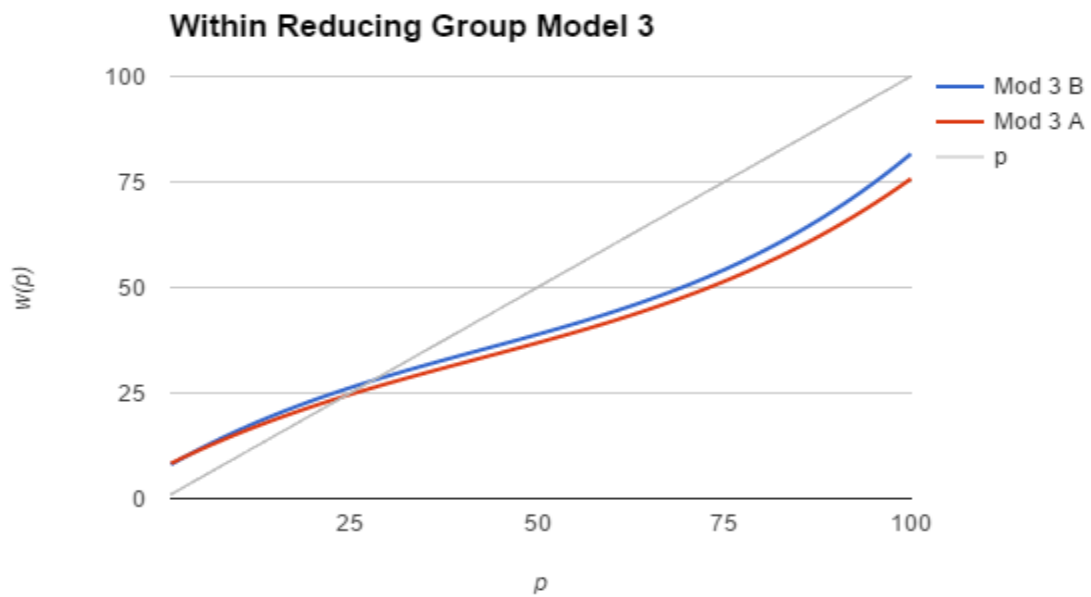


Figure 3. Graph of decision weights versus probability using fitted values of Table 10, Model 3.

Appendix C: Graphs of Within Neutral Group Fitted Polynomial Regression Results

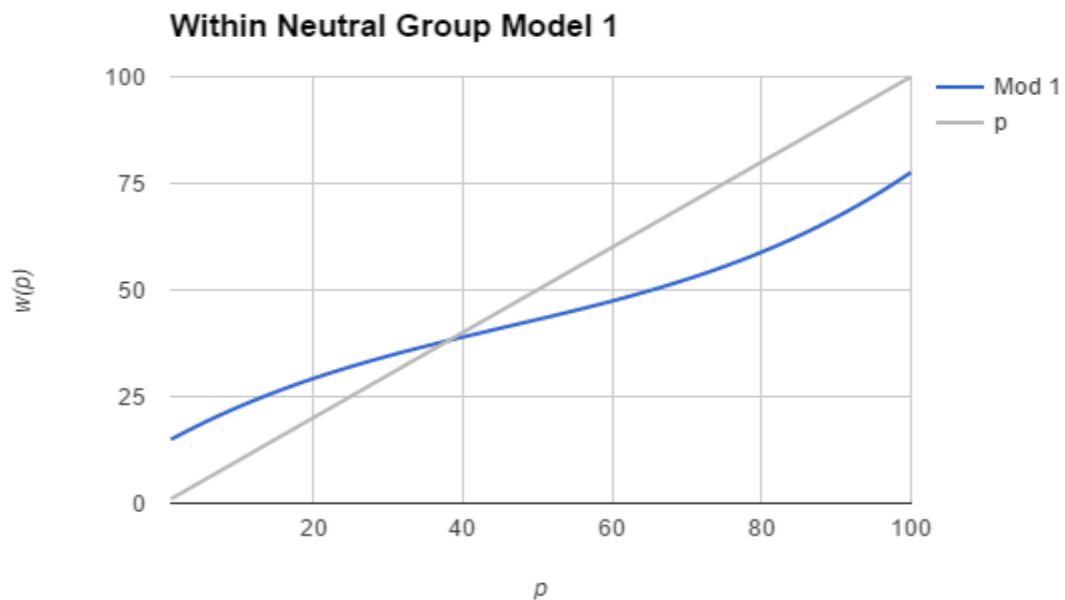


Figure 1. Graph of decision weights versus probability using fitted values of Table 11, Model 1.

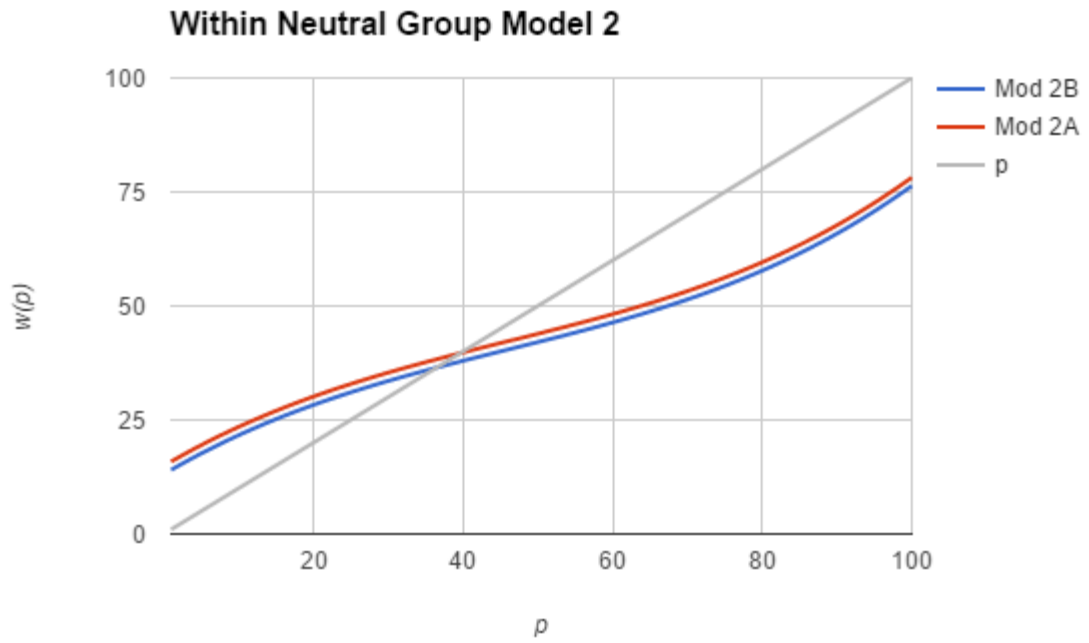


Figure 2. Graph of decision weights versus probability using fitted values of Table 11, Model 2.

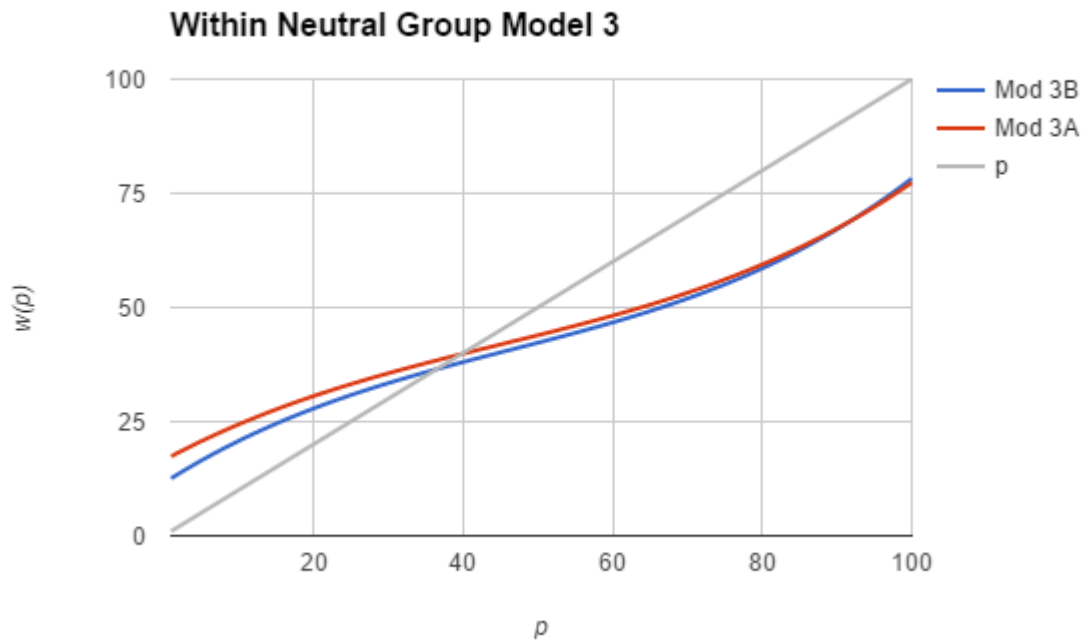


Figure 3. Graph of decision weights versus probability using fitted values of Table 11, Model 3.

Appendix D: Graphs of Between Anxiety and Neutral Group Fitted Polynomial Regression Results

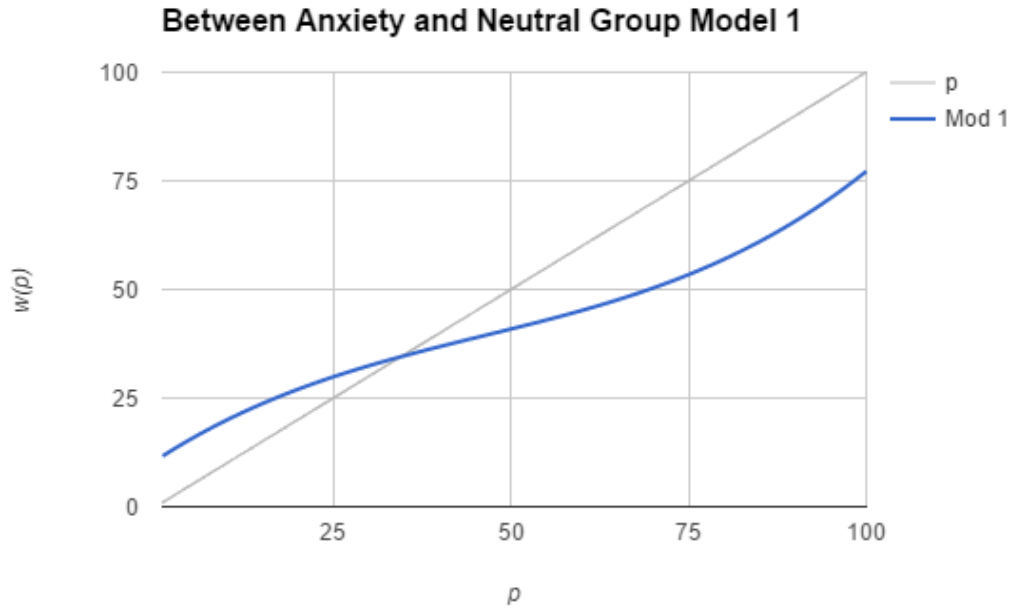


Figure 1. Graph of decision weights versus probability using fitted values of Table 12, Model 1.

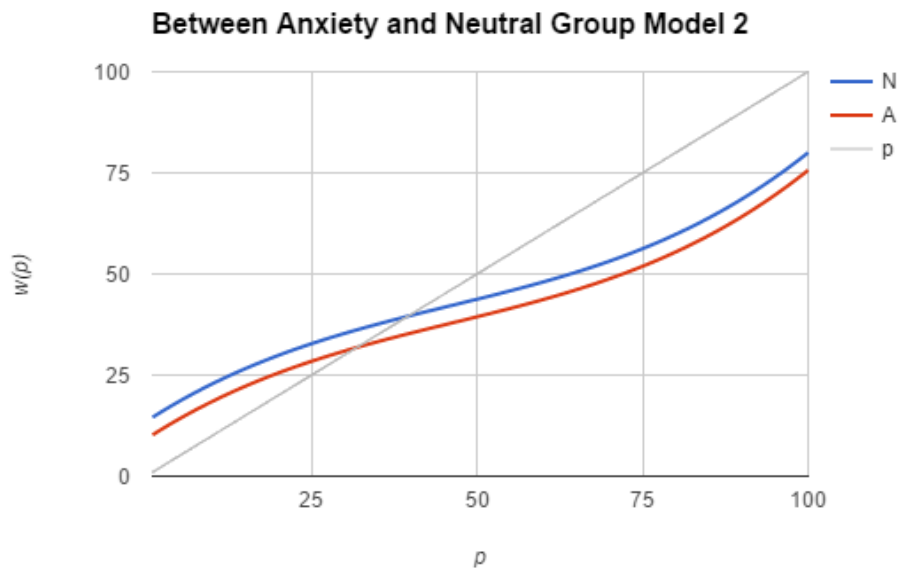


Figure 2. Graph of decision weights versus probability using fitted values of Table 12, Model 2.

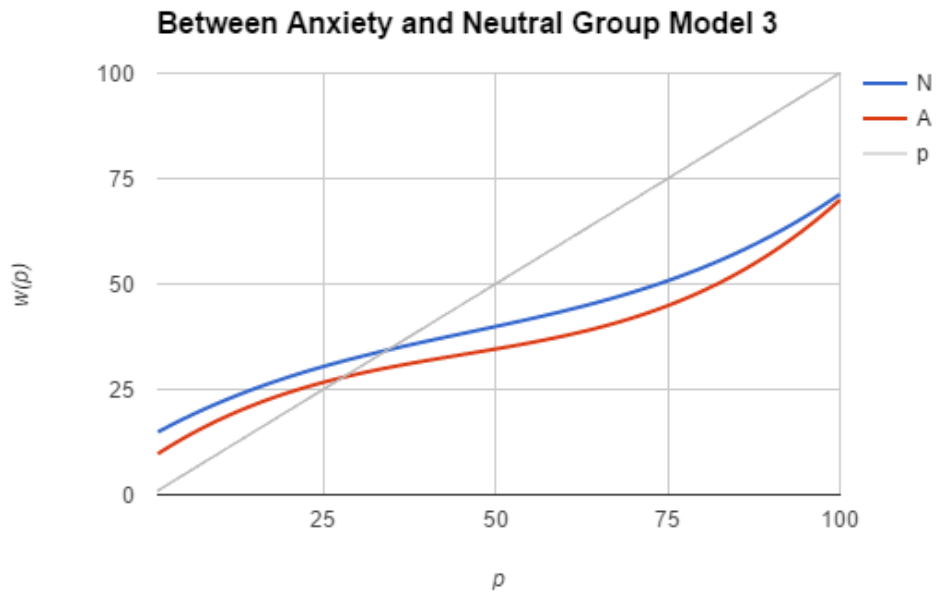


Figure 3. Graph of decision weights versus probability using fitted values of Table 12, Model 3.

Appendix E: Graphs of Between Anxiety and Reducing Group Fitted Polynomial Regression Results

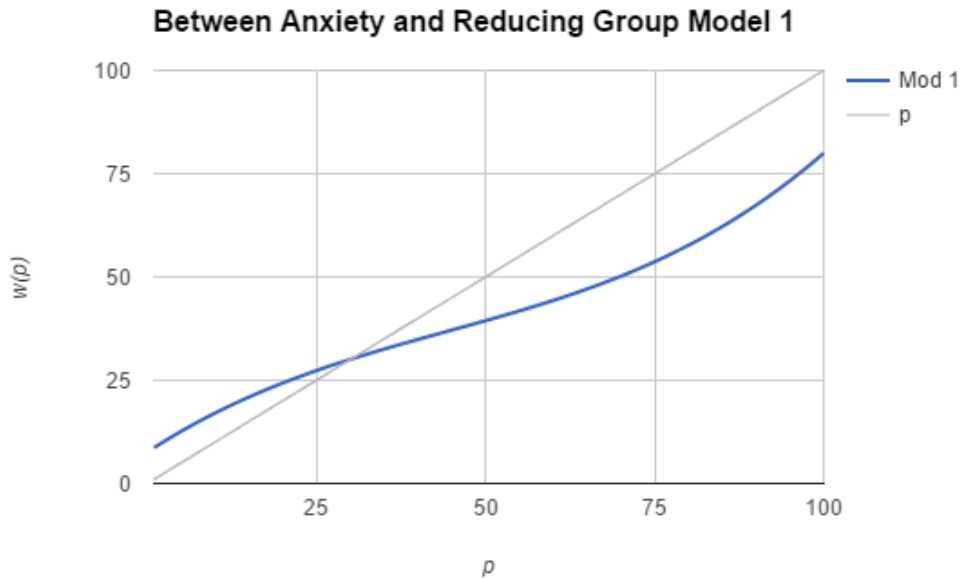


Figure 1. Graph of decision weights versus probability using fitted values of Table 13, Model 1.

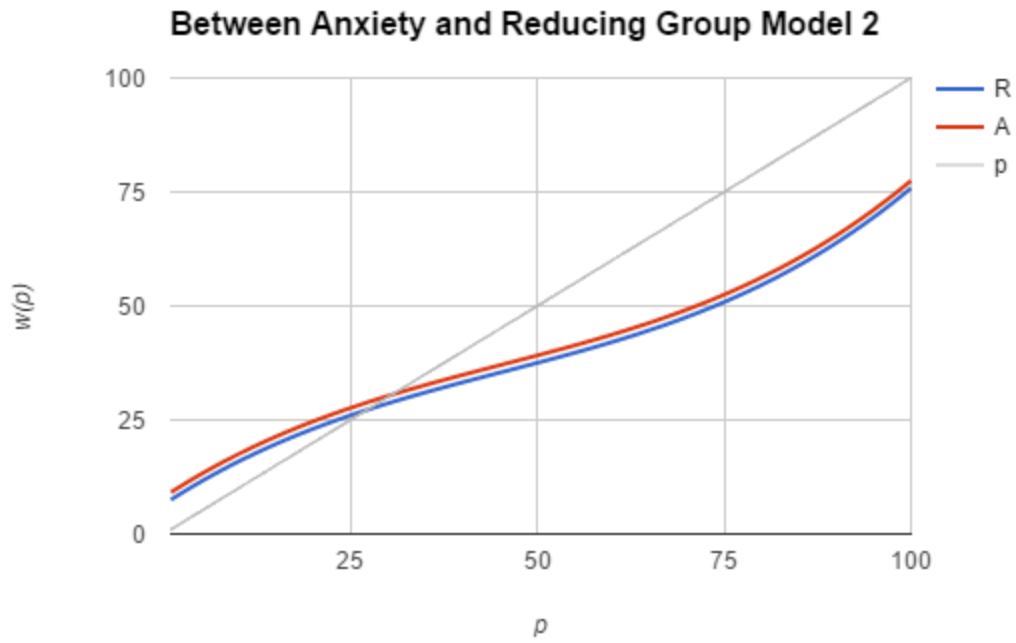


Figure 2. Graph of decision weights versus probability using fitted values of Table 13, Model 2.

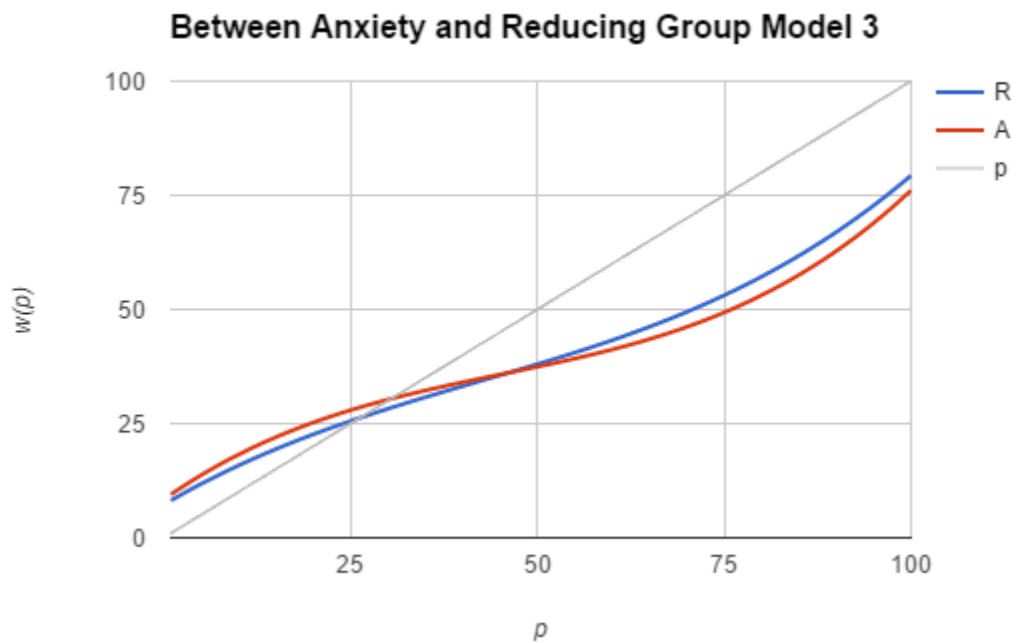


Figure 3. Graph of decision weights versus probability using fitted values of Table 13, Model 3.

Appendix F: Graphs of Between Neutral and Reducing Group Fitted Polynomial Regression Results

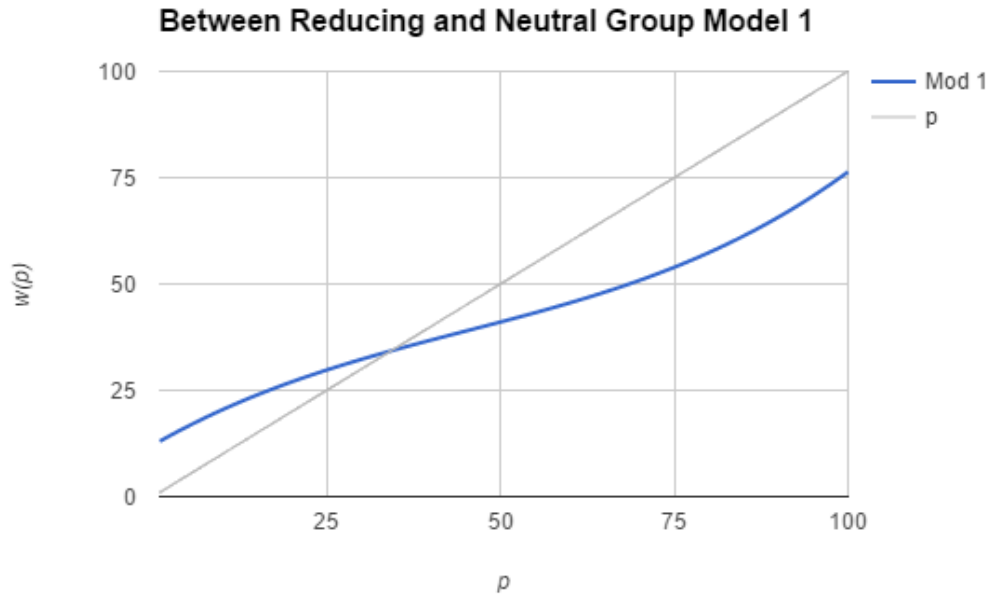


Figure 1. Graph of decision weights versus probability using fitted values of Table 14, Model 1.

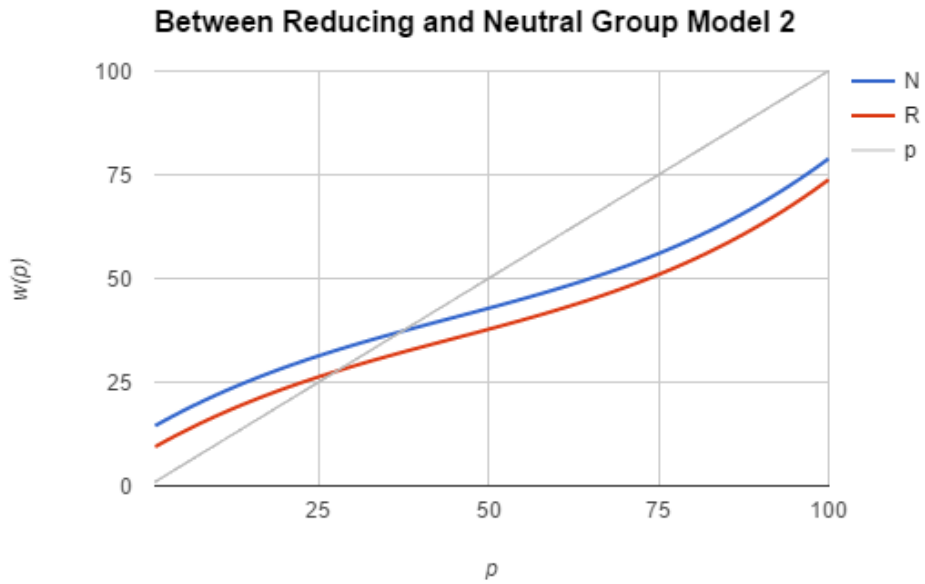


Figure 2. Graph of decision weights versus probability using fitted values of Table 14, Model 2.

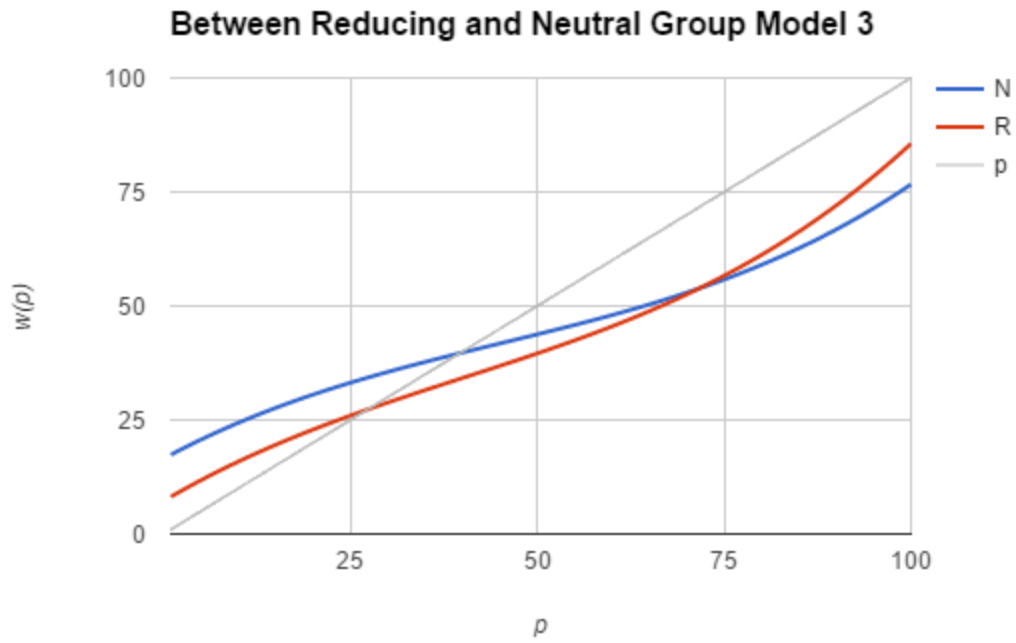


Figure 3. Graph of decision weights versus probability using fitted values of Table 14, Model 3.