The problem with explaining symptoms: The origin of biases in causal processing

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Abstract

Understanding causation is complex, especially where it involves 'the person'. Advances in physiological, psycho-social understanding, and associated health interventions have nevertheless been made despite this complexity but often in the face of weak comprehension of the actual causal framework of any particular disorder. One of the problems highlighted by CauseHealth and the European Society for Person Centered Healthcare is that our tools (e.g., the Scientific Method, Evidence Based Medicine, Random Control Trials) are disappointingly weak in comparison with the model of understanding to which we aspire. Research from experimental psychology highlights a further constraint to our understanding; all animals have evolved neural mechanisms that solve natural causation in a manner that is similar to the scientific method. Our behaviour and thinking suffers from the same weaknesses as our methods. We will discuss experiments that have been conducted to test causal perceptions and mental representations. We address 1) a primary route for how causes are extracted from experience the 2) single cause bias and 3) representational complexity. These experiments have implications for both patient and practitioner as well as how they interact. Not only is the world more complex than we generally tend to acknowledge but we have evolved to think more simply than we might wish.

"Am I gluten intolerant? Allergic to dogs? Is my rash related to the coarseness of the fibers of the new clothes I am wearing or is it a precursor to cancer?" The answers to these causal questions require a person to analyse associations gathered from their experience. And, the person contacting their doctor must acknowledge the reality of the modern patient-doctor relationship; which is that the diagnostic process is a complex almost intractable causal problem (Mumford & Anjum, 2011).

In reality then, diagnosis is often more trial and error than true causal identification. The patient brings their thoughts, feelings and memories to the examining room. Some of this data will be helpful for making a diagnosis, but these communications may also hinder or even have their own causal role in the manifestation of the syndrome. The modern doctor understands the inherent limitations of the 10 minute diagnostic meeting because of the sheer complexity of the causal relations that underlie many disorders. The scale of the patient's task is made all the more acute by public health messages; bluntly put, that many fatal conditions are preventable if only the patient had noticed the symptoms earlier "early diagnoses saves lives and reduces treatment costs" (NHS, UK).

It is in this context that psychological research has been making advances in the understanding of the biases that constrain our thinking and behaviour in relation to cause and effect (e.g., Matute et al., 2011; Msetfi et al., 2015). When the practitioner queries the patient about factors possibly responsible for the new rash that has appeared, the diagnostic task requires

that the doctor not only recall memories from previous patient's rashes and their possible causes, but also requires that the patient provide an accurate recall of their current state and of any previous relevant associations. The branch of psychology responsible for this work studies how people think and reason about causes (e.g., Oaksford & Chater, 1998) but also the psychology of learning about associative connections which underlie such thinking about causation (e.g., Murphy et al., 2005).

This latter work recognises the limitations in perception, memory, and the processing biases that affect our remembered associations. Of course sometimes a single causal factor with a known diagnostic trail is the appropriate model for understanding a particular patient's symptom. However, the person-centered approach to medicine eschews a simple reliance on grouped data or evidence based research to diagnose a patient. Rather, this approach recognizes the unique set of causal factors that might affect an individual and the psychological limits of a human searching for causal understanding in this complex environment. Here we attempt to inform person -centred health professionals on the types of factors that have been found to impact a person's understanding of their symptoms and how this might colour the information provided to their practitioner.

Associative Learning

The experimental study of causal learning in humans differentiates two classes of causal problem. The first is how people acquire associations from

passively observing relations (Predictive learning; see Key terms Figure 1; e.g., Vallee-Tourangeau et al., 1998c), which is distinguished from tasks that emphasize the advantage of active instrumental involvement (Instrumental learning; see Key terms; Murphy et al., 2005; Vallee-Tourangeau & Murphy, 1999). A further elaboration relies on considering the mental processes that underlie learning, modelled on biologically plausible networks found in neural tissue and using simple machine learning algorithms (e.g., Baker et al., 1996). This field treats causal learning as an association problem.

The study of associative learning examines how relationships among the events of human experience are connected. The assumption is that these connections form the building blocks of our causal understanding (e.g., Murphy et al., 2009; Shanks, 1987). Researchers studying these processes made a link between how animals acquire predictive associations (e.g., between stimuli and food) and how humans learned to associate and remember word pairs (e.g., Dickinson, 1984; Murphy & Baker, 2004; Rescorla, 1988). Studies of human association learning have discovered that the associations we learn seem to be related to how events co-vary. Suppose we are interested in knowing whether we have an allergy to cats. Our initial suspicions will not be based on a blood test, but rather our experience. However, we do not simply remember the time that being near a cat made us sneeze, but rather the allergy suspicion is produced by an association, which is based on four types of experience that we tend to aggregate (illustrated in the contingency table of Figure 2). Association learning reflects, sensitivity to

the contingency between events. This sensitivity to contingency bears some resemblance to the experimental method and random control trials and how they are used in science to understand causal effects. Participants in our experiments are presented with multiple instances of events and asked to make a judgement of the strength of the relation based on conditional outcomes.

For example, we tested whether people could learn the causal relation between a virus and its ability to cause a particular disease (Vallee-Tourangeau et al., 1998b~~a~~, c~~b~~). Naïve participants with no particular medical experience were presented with data and asked to make judgements about the possible causal relations. The data were presented as sets of fictitious patient records in which each record presented a single patient and the medical data for the presence of a virus (the cause) was related to a disease (the effect). Each set of patient records contained a different virus-disease relationship or contingency (see Figure 1 and 2 for calculation of contingency). Participants were extremely accurate at judging relationships; independent of the number of times the virus and disease were paired, people's judgements reflected the overall contingency. This result was in contrast to earlier work that suggested that people might base their beliefs on how often the virus and disease co-occurred (see Smedslund, 1963 and Vallee-Tourangeau et al., 1998a for a discussion of this debate). People are extremely sensitive to contingencies. The fact that these results mirror findings from

other animals (Baker et al., 1996) suggests that brains have evolved to learn contingencies as a tool for solving causal problems.

In spite of this evidence, there **are** systematic biases in sensitivity to contingency, either produced by i) biasing the information or by ii) increasing the complexity of the task. For example, when information is biased, people sometimes perceive a casual relation where objectively none really exists. This is called the illusory correlation effect (see key terms Figure 1). Illusory correlations suggest that sometimes our ability to perceive a relation is biased because of our biased experience (e.g., the False Consensus Effect, Marks & Miller, 1987; our Facebook contacts are likely to share our beliefs and bias our perceptions of the wider public). In the real-world, this can mean that patients can have very strong beliefs about the cause of their illness, or, indeed, the efficacy of an in-effective treatment (i.e. alternative remedies Matute et al., 2011). These simple causal illusions are well known. However, illusions can also emerge due to the way the we process unbiased information (e.g., Murphy et al., 2011).

As in the situation of a person trying to track down the cause of an allergic reaction, real life often involves multi-cause scenarios. The presence of multiple predictors increases the cognitive load and so might be expected to make tasks more difficult and to compromise adequate processing of all relevant information (e.g., Cavus & Msetfi, 2016). These complex situations, sometimes referred to as selective association tasks, are an example of the interactive effect of multiple causal cues (see Selective Associations, Figure 1).

Importantly for medical professionals, there is evidence that people differ in how well they can learn about these multiple cause relations, thereby introducing a further route for variability into the diagnostic process.

How people learn about multiple causes?

So far, we have assumed that events and causes are simply extracted from our experience, but even simple effects can the consequence of multiple causes which themselves are combinations of predictive elements (for instance they have multiple modalities, a rash (the effect) has multiple components painful, is itchy, is red etc., but might also be caused by multiple factors, stress, clothing, itching) and have conditional dependencies (configurations). To take a simple example, a patient's cough, may be a symptom of a virus or an upper respiratory tract infection, whooping cough, or lung cancer. The combined symptoms of whooping cough are very similar to a cold (coughing, sneezing, runny nose, low fever) though the particular pattern differs. This requires that the patient configure the relevant information in order to make a judgement on whether they should seek medical advice.

This is a problem requiring the use of cues, such as symptoms, to predict the likelihood of a given outcome, in this case, a disease. Where the relationship between symptoms and disease are complex, two different approaches to representing the relationship might be possible; an elemental or a configural representation. In an elemental representation, each cue or symptom will enter into an independent association with the outcome or

disease (Atkinson & Estes, 1962; Rescorla & Wagner, 1972). In computational terms, the multiple cues may contribute algebraically to produce the overall judgement of predictive relation. In effect, the more symptoms present the more likely the particular disease.

A simple example of this relationship is summation and has been studied in numerous associative learning tasks (e.g., Collins & Shanks, 2006; Rescorla, 1997; Soto, Vogel, Castillo, & Wagner, 2009). For example, if a single symptom, A is a reliable predictor of a particular disease, and a second symptom B is also a reliable predictor of that same disease, our expectation of the disease will be elevated in the combined presence of A and B relative to either A or B alone.

In contrast, if cues or symptoms are represented configurally, multiple symptoms are represented simultaneously, such that the configuration of symptoms forms a unique profile, which alone predicts the disease (e.g., Pearce, 1987). Following a configural representation, summation is not necessarily observed; where a unique symptom profile is seen to predict a disease, the relationship between symptoms is not linear (e.g., Vallee-Tourangeau et al., 1998b~~a~~).

Neither of these theoretical approaches is 'correct', however, elemental representations may often oversimplify the relationship between symptoms and disease, as conditional or interactive relationships between symptoms are not captured in the representation. There are, for example, relatively simple

discrimination problems that cannot be solved with a purely elemental representation. For instance, a drug may have a beneficial effect, but there is a limit to the relation between drug dosage and efficacy, and in fact doubling the dose might result in death. Another common example is the negative patterning (also referred to as the XOR) problem (e.g., Grand & Honey, 2008). This problem captures the idea that sometimes two independently useful factors may cancel each other out; drug A and B are both effective but in compound they interact to produce a negative effect. This is a scenario where elemental representation proves to be error prone and configural representation may be recruited. Following a configural representation, the problem is resolved easily as the co-occurrence of A and B is treated as a unique configuration.

While configural representations may be important for accurately representing some complex relationships people can and will use both types of representation (Williams, Sagness, & McPhee, 1994). However, research has identified factors relating either to the information being learned or the person that can reduce the ability to recruit a configural representation. For example, people may be less likely to recruit a configural representation when the co-occurring relevant symptoms have an unequal salience or an unequal relative validity history (Byrom & Murphy, Under review). Further, individual difference affects these tendencies. Cultural background affects whether people are more likely to invoke configural or elemental thinking about causes (Msetfi, Kornbrot, Matute, & Murphy, 2015). In addition, we

have shown that cognitive factors associated with stress reduce an individual's ability to recruit a configural representation and consider a unique relationship between a set of symptoms and associate disease (Byrom & Murphy, 2014; Byrom & Murphy, 2016). Instead, under stress, individuals are more likely to represent the relationship between symptoms and disease elementally. Thus, when a patient makes a judgement on whether to seek medical advice, or recounts the story of their symptom experience to their medical practitioner, it is important that the practitioner has clarity on the person/patient factors that will enter into this complex causal story.

Studying differences in configural and elemental predictive reasoning

In order to study differences in the representation of complex causal discriminations, we asked our participants to complete a biconditional discrimination. Like the XOR problem, the solution of the biconditional discrimination requires a configural representation. The predictive cues (e.g., virus) are combined in such a way that no individual cue predicts the outcome (e.g., disease), but rather the outcomes are always predicted by the co-occurrence of causes (i.e., AX+, AY-, BX-, BY+). When A and B are more salient than X and Y, that is, they catch the participants' attention more readily, participants find it harder to solve the discrimination, than when the salience of all the stimuli is roughly equivalent. Similarly, if A and B have previously been more reliable predictors of the outcome than X and Y,

participants again find the biconditional discrimination harder to solve (Byrom & Murphy, Under review). This suggests that highly salient cues and cues that have previously been reliable predictors of outcome can overshadow subtler cues, reducing our ability to recognise how these cues interact and configure. The recognition that salience contributes to the diagnostic problem means that where the patient experiences some symptoms more intensely than others, they will find it harder to represent the unique, configural, symptom profile, and reasoning may be biased towards a linear representation between symptoms and disease even though the salient features may not be as causally relevant.

Using these tasks, we have also shown that key individual differences in attentional scope influence configural representation. Importantly, narrow attentional scope is associated with stress, emotional arousal and mental health issues (Basso, Schefft, Ris, & Dember, 1996; Fredrickson & Branigan, 2005; Gasper & Clore, 2002; Rowe, Hirsh, & Anderson, 2007). We found that people who presented with a narrow attentional scope, as well as typical people trained to adopt a narrow attentional scope, are slower to arrive at configural solutions to complex causal problems (Byrom & Murphy, 2014; Byrom & Murphy, 2016). They find it harder to discriminate between configurations containing overlapping cues that predict different outcomes (Byrom & Murphy, 2014). This suggests, firstly, that our ability to engage configural representations may decline under situations of stress, and,

secondly, that some people will fail to notice important co-occurring symptoms.

Causal control and Instrumental learning

The majority of the situations we have already discussed relate to the observation or prediction of passively experienced cause. However, key to health care, is a person's experience of their own causal control; that is their perception of the extent to which their own actions will produce an effect. There of numerous example of this, including adherence to medication to alleviate symptoms, as well as patients' false beliefs in completely ineffective and expensive 'quackery' (Matute et al., 2011). As with tendencies toward elemental and configural processing, both situation and person factors affect these causal belief tendencies. Here, we describe an example of causal control in terms of the alternative causes of effects that people consider, and how the most frequently diagnosed mental health disorder, depression, affects causal learning although mood more generally is expected to have an effect on causal thinking

With its origins in the animal research literature on predictive (Classical) and Instrumental conditioning (see key terms; Baker et al., 1996), it became clear that learning could provide animals with important new conditional relations that allow them to interact with their environments in better and more complex ways. For example, a rat could be taught to extract food from a tray on the basis of the presence of visual and auditory stimuli or the time of day, and can use such statistical regularity to meet their biological

needs. However, it was also evident that particular causal relations induced behavioural symptoms in animals resembling those found with people suffering from depression (Seligman & Maier, 1967).

This finding inspired research with humans, which initially involved studying the effects of fluctuation in mood on the ability to judge the strength of simple instrumental causal relationships; that is the extent to which people thought that their actions were the cause of effects. Over a series of experiments, Alloy and Abramson (1979) exposed healthy students and those with mild depression symptoms to a contingency relationship between a simple action and an outcome stimulus (it was a button press and a light bulb), and asked them to judge their control over the effect. In both of the situations tested, participants had no control at all; the probability with which the light came on following the button press was the same as the probability of the light when the action did not occur.

Findings showed that when the outcome rate was frequent, healthy people assumed that their actions had a moderate degree of causal control but when with infrequent outcomes, the causal control illusion did not occur. Much follow up research supports the replicability and validity of these findings (Msetfi, Murphy, & Kornbrot, 2012; Msetfi, Murphy, Simpson, & Kornbrot, 2005), Thus, healthy people are more likely to believe that they have control over frequently occurring effects, which randomly coincide with their actions, and maintain this strong belief in spite of considerable experience with the random relationship. This finding can explain why

people tend to maintain unhealthy behaviours, or ineffective treatment options.

If this is the case, why is it then that when people are recommended a treatment option that is known to 'work' (i.e., strong causal relationship between action and effect), they often do not adhere? This is obviously a complex issue but at least some part of it relates to the perception that no causal relationship exists. Thus, research on conditions in which people do not perceive their causal control is informative. Mildly depressed participants did not tend to exhibit the causal illusion (e.g., Byrom, Msetfi, & Murphy, 2015; Msetfi, Wade, & Murphy, 2013) and under specific conditions can produce a learned 'causal helplessness' that may reflect their mood state.

As we have suggested, causal learning involves multiple causal factors and a key aspect in the process of learning about one's own causal control is learning what else might be causally relevant. So if a causal event occurs frequently and 'I' am not in control of the occurrence of that event, then something else must be. Learning theory assumes that the possible causes of outcomes – one's own actions versus non-self causes- compete in the learning experience in terms of acquiring causal strength. This leads to an important prediction that, if people with depression are so adept at perceiving lack of causal control, then the source of this difference may be in the way that external causes are processed.

We tested these predictions initially and found that depressed people's causal models were impervious to manipulations in exposure to non-self (i.e.,

context; Msetfi et al., 2005, 2007), which could be regarded as a means of manipulating the strength of the context- outcome relationship. We have also shown ways of experimentally blocking healthy people's ability to integrate context in their causal control model (Cavus & Msetfi, 2016). Manipulations of cognitive capacity and attention to causes other than self during the causal learning process, induce a healthy causal illusion (Msetfi, Cavus, & Brosnan, 2016).

Any intervention which de-emphasises external causes as a cause of effects will consequentially boost people's own feelings of causal control. Thus, counter-intuitively, when health care providers make contextual causes of effects salient to the patient, i.e., in the case of treatment adherence - the importance of support groups, the clinic, the pamphlets, any non-self controllers, this may detrimentally impact a person's own feelings of efficacy and result in the 'wrong' behaviour. Obviously, we are not recommending that non-self causes are discarded! We are suggesting that healthcare practice may need to fold in an understanding of perceptions of cause.

Summary

We provide a summary of the some of the experimental evidence relevant for an understanding of the psychology of causal thinking, in particular as it is captured by associative learning theory. This work suggests that our perceptions of cause are governed by principles that are well understood in the lab. We would like to argue that these principles will also

be useful in the clinic both from the perspective of the patient understanding their medical symptoms but also from the perspective of the diagnostician. There is considerable evidence that humans struggle to perform complex causal thinking. The way we learn is likely to contribute to biases in how we think causes have their effects on our bodies which ultimately is what medicine is trying to understand.

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Figure Captions

Figure 1: Box with key terms

Figure 2. A 2 x 2 contingency table describing the relation between two binary events a virus and the presence of symptoms for illness. The four events that describe the relation between the two events are labelled A, B, C and D.

Figure 1.

Predictive learning: Perception provides evidence for the effect of causes. Experiments with animals studying the role that predictive learning plays for behaviour is known as Pavlovian conditioning (Rescorla, 1988). In these experiments a stimulus is presented in a predictive relation with an outcome of biological significance (i.e., food or water) and animals behave towards a predictive cue once they learn that it is a reliable predictor of the outcome. Medical diagnoses has many of the same features in that predictive cues are used to anticipate their effects.

Instrumental learning: Active involvement provides a different form of learning experience instead of observing the relation, the agent can test the effectiveness of cue. Instrumental learning involves learning the strength of the association between particular responses (R) and particular outcomes (O).

Contingency: Refers to the strength of the dependent relation between the occurrence of any two things. The relation can be understood by considering an example from medical reasoning in which the virulence of a virus can be evaluated. The relation between a virus and illness is imperfect but the strength of the relation can be determined by considering the evidence for the relation. How often does someone with the virus become ill (Cell A), how often does someone with virus not have the disease (cell B), how often does someone without virus have the same disease and how often do neither occur (Cell D). We can calculate the quantitative strength of the contingent relation using various metrics (see Allan, 1980). One measure relates the frequencies with which the four types of event that relate the two variable A, B C and D in Table 1. $\Delta P = [A/(A+B) - C/(C+D)]$.

Causal Relation: In addition to the statistical relation captured by contingency, events and outcomes also have **causal** dependencies (as opposed to correlations) if they are necessary and sufficient for the outcome

to occur but in normal life we do not usually have access to the evidence to make such judgements but rely on statistical evidence to infer causal relations. Philosophers such as Hume suggested that causation might involve causal forces but that we do not usually use a perception of the force to make our inferences, but rather we infer causation on the basis of basic principles (Priority, Covariation, Temporal Contiguity and Similarity). In the example presented in Table 1, we have a causal model of understanding that suggests that Viruses can cause symptoms, but we might also understand this relation first by seeing the symptoms and then measuring the patient with a test for the virus.

Illusory Correlation: In some situations no statistical or causal relation exists in the real world, but people persist in believing that one does. An example of an illusory correlation is seen in the sports star who believes that they have lucky socks , but is also present in the stereotypical beliefs we have about minority groups (Murphy et al., 2011). In medical diagnosis the illusory correlation is found in beliefs about some forms of alternative medicine that persist in the face of evidence that there is no effectiveness.

Selective Associations and Blocking: Selective learning refers to situations in which multiple cues are present for a particular outcome, but in which learning is selective to particular cues. More salience cues are learnt about at the expense of less salient cues (Byrom & Murphy, in press) and bias learning. Similarly prior knowledge interferes with learning, a phenonmenon called Blocking (Kamin, 1969).

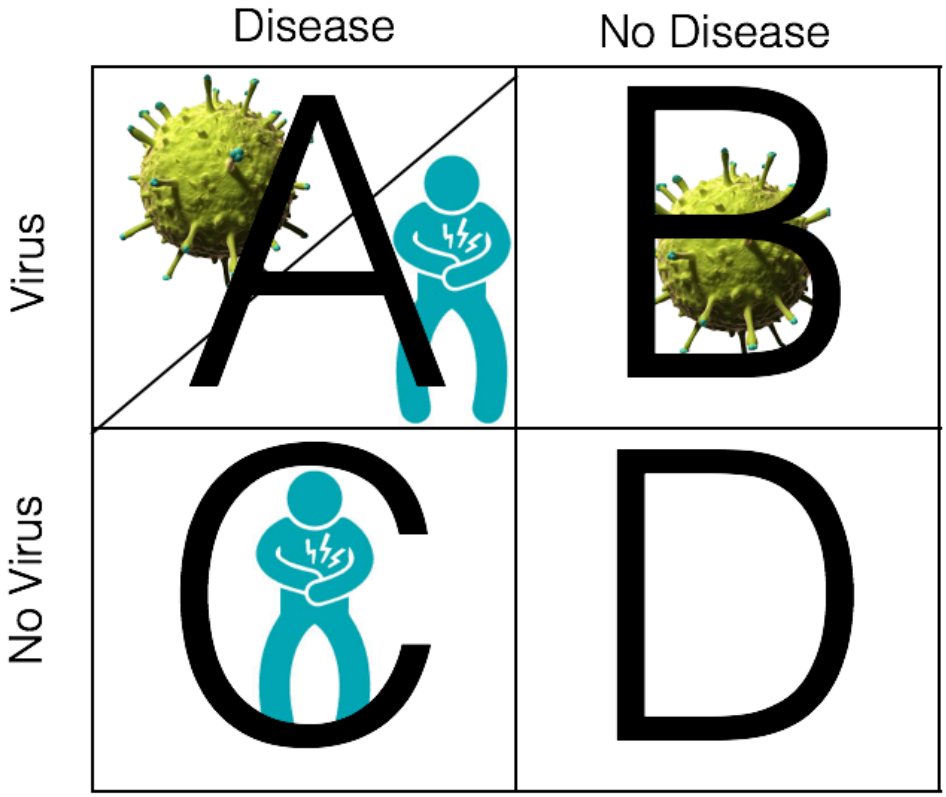

Table 1.2X2 Contingency Table describing the relation between two binary events. In this case a virus ( ) and the presence of symptoms for illness ( ).