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Anxiety: Splitting the phenomenological atom

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ABSTRACT

Anxiety is one of the most studied constructs in psychology and one of the most prevalent features seen in clinical disorder, presented either as the defining symptom (e.g., generalised anxiety disorder) or comorbid with other disorders. But despite considerable work directed towards the elucidation of its neuropsychological bases, its adaptive value in everyday life, and its possible evolutionary roots, significant problems remain in the development of an adequate integrative model of its multidimensional nature. To address this problem, this paper argues for a closer integration of biological, cognitive, behavioural and experiential systems, including the functions of higher-level controlled systems: it is the interplay of these systems that lead to the construction of the phenomenological angst of anxiety that is represented in consciousness. A summary is provided of a recently formulated model centred around the well-known behavioural inhibition system (BIS), which takes equal account of lower-order (automatic) and higher-order (controlled) systems. This model assigns significance to two specific theoretical problems that have hindered previous attempts at model development: (a) the 'lateness' of higher-level controlled systems (which are often accompanied by conscious awareness); and (b) the different functions served by automatic-reflexive and controlled-reflective systems, especially how they relate to each other (the 'interface' problem). Targets for pinpointing primary dysfunctions in anxiety-related processes are suggested, and the implications for clinical intervention are highlighted.

1. Introduction

Anxiety is multidimensional, with causal roots in evolutionary, psychophysiological, cognitive, and behavioural mechanisms; and, phenomenology, it is fundamentally subjective: the *angst* of anxiety is constructed qualia, and this is one of its most important defining qualities. This complex nature continues to pose a significant obstacle to the development of a viable model of anxiety. Research tends to focus on only some of these features to the, relative, neglect of others, producing a failure of theoretical integration. This is understandable, although to be regretted. However, instead of being seen as a major obstacle, multidimensionality may be the key to unlocking the brain-mind construction of anxiety. This paper poses the question: are we now at a stage where a viable model of the multidimensionality of anxiety is possible? This grand question may be broken down into two sub-questions. (1) What are the theoretical problems that any such model would need to address? (2) What might the contours of a sketch of any such model? These last two questions are the focus of this paper.

The other papers in this Special Issue provide ample testimony to the multidimensional nature of anxiety research, each presenting specific theories and findings through the lens of one major research perspective. This paper aims to provide a meta-model that has the potential to incorporate these different perspectives. The editors asked contributors to focus on a number of specific questions (see Editorial), and my contribution bears principally on two of these questions:

1. What is the most appropriate level of explanation to understand human anxiety? Is it possible, or indeed desirable, to attempt to understand anxiety at all levels of explanation (e.g., evolution, DNA, brain, endophenotype, and behaviour)?
2. What is the role and importance of conscious awareness in anxiety (i.e., the subjective feelings associated with various forms of anxiety?) Are these subjective aspects causally important or impotent?

2. Preamble

The approach taken in this paper is an extension of a general model of behavioural control developed in the context of multiple level processing in personality psychology; and its extension to anxiety is straightforward given that the core of this general model is the well-established behavioural inhibition system (BIS: Gray, 1982; updated by Gray & McNaughton, 2000; and further revised by McNaughton & Corr, 2004, 2008). In particular, the model helps to account for the phenomenological aspects of anxiety (e.g., excessive worry) and its subjective representation in conscious awareness: these aspects are defining features of the state/disorder. For example, symptoms of Generalised Anxiety Disorder

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(GAD; American Psychiatric Association, 2003) include: (1) excessive worry (apprehensive expectation); and (2) difficulty controlling worry, which are accompanied by such features as problems with concentrating, disturbed sleep, and being easily fatigued – these features interfere with attention to concurrent tasks, which have a special significance in understanding the causal basis of anxiety, as discussed below. However, immediately mention is made of higher-level controlled processes, and conscious awareness in particular, some very thorny theoretical problems are encountered; namely: (a) the relationship between automatic (reflexive) and controlled (reflective) processing (the 'interface' problem); and (b) the fact that controlled processes (including, but not restricted to, the generation of conscious awareness) come too late in the causal chain of events to influence the events they represent (the 'lateness' problem) – this latter problem questions the functional significance of the subjective-emotional aspects of anxiety (its *angst*). It is here argued that these problems, and their resolution, hold crucial implications for understanding the functional (distal) significance of anxiety, its mediating (proximal) processes, and its psychological consequences.

The implications for anxiety research of these two major problems are summarised in this paper, and the proposed model of anxiety is aimed at their solution. The model itself is based on the concept of the BIS, which accounts for: (a) why certain anxiogenic stimuli are extracted for controlled processing (i.e., those that are not 'going to plan', as detected by an error mechanism); and (b) the functions of controlled processing (including conscious awareness) in anxiety in terms of the adjustment of the cybernetic weights of automatic processes – which, it is argued, are always in control of *immediate* behaviour – which, then, usually influence *future* automatically-controlled behaviour (however, this influence can break down when the 'interface' between automatic-reflexive and controlled-reflective processes is dysfunctional (this can occur for a number of reasons; see below). The general model of behavioural control upon which this specific anxiety model is based was the topic of a target paper, with peer commentaries and author's response (Corr, 2010a, 2010b) – the reader is referred to these sources for a full description of the general model.

2.1. Multi-level processing in anxiety

Anxiety entails many features that are nonconscious, including passive avoidance of potentially threatening stimuli (objects and situations), behavioural dithering/indecision, and a generally hyper-vigilance to threat; and these features can be observed, by behavioural observations, in the rat where we usually assume there is very little higher-order controlled processes and, largely, nonconscious processing (Blanchard, Blanchard, Griebel, & Nutt, 2008) – to the extent that rats engage in any form of controlled/conscious processing, we can be confident that it is of orders of magnitude below that experienced by human beings. In contrast to the relatively humble (though still highly sophisticated) rat, in human beings conscious awareness of anxiety is a crucial component of the state and its various clinical expressions – without compliants by the patient of their experience it would be very difficult to diagnose, for example, GAD – for sure, motoric and cognitive disruptions loom large but these are not sufficient to define the condition in terms of the experience of the patient.[1] How to reconcile these different levels of processing in anxiety should be a key task for the anxiety theorist; furthermore, it is possible that some crucial features of anxiety are to be understood only by reference to the *interplay* of these different levels of processing. On a positive note, the multidimensionality of anxiety may not be the problem it, at first blush, may seem; instead, it may be an important clue to its very nature – however, only once the theoretical issues presented above are adequately acknowledged and addressed. (In passing, it may be noted that multi-level models of processing are ubiquitous across psychology (Carver, Johnson, & Joormann, 2008), and have implications for how behaviour is controlled and which aspects of internal and external worlds get extracted for higher-level controlled (and sometimes) conscious processing (Corr, 2010a).)

A multiple processing perspective on anxiety poses problems for understanding its functional nature. It is widely agreed that anxiety is an evolved adaptation; but is all its different levels of processing equally functional? This question is nowhere more contentious than in the case of conscious awareness, which already has been highlighted as one of the defining features of anxiety: phenomenological angst. Despite the claim that conscious awareness has no functional significance, including that involved in the phenomenology of anxiety, and therefore need not play a central role in formal models of information processing and emotion (e.g., Matthews, 2010), there are reasons for thinking that such awareness is not merely epiphenomenal. Indeed, for some theorists, it is obvious that consciousness is functional, and to suggest otherwise leads only to even greater theoretical troubles (e.g., Morsella & Hubbard, 2010; Revelle, Wilt, & Condon, 2010). For anxiety to have functional significance, it must be shown to have survival value and fitness-enhancing functions. Space prevents further discussion of this matter here, which has been dealt with elsewhere (Corr, 2010a, 2010b). The rest of this paper highlights some of the specific functions of anxiety.

2.2. Behavioural inhibition system (BIS)

Updated versions of the Reinforcement Sensitivity Theory (RST) of personality (e.g., Corr, 2008) retain the BIS as central to understanding normal and abnormal anxiety. It is seen to work in conjunction with, at least, two other brain-behavioural systems: the *Flight-Flight-Freeze System* (FFFS; related to processing of all aversive stimuli, which includes nonreward); and the *Behavioural Approach System* (BAS; related to processing of all appetitive stimuli, which includes nonpunishment). Importantly, revised RST makes a distinction between FFFS-related fear and BIS-related anxiety (for a discussion of this distinction, see Cooper, Perkins, & Corr, 2007; Perkins, Kemp, & Corr, 2007; also see McNaughton, 2011).

In general terms, the BIS is responsible for the detection and resolution of goal-conflict, of whatever kind (e.g., between BAS-approach and FFFS-avoidance)[2], and evolved to permit an animal to withhold entrance (i.e., passive avoidance) or to enter a dangerous situation (i.e., leading to cautious 'risk assessment' behaviour in the context of simultaneous BAS-mediated behaviour), such as a foraging field where predators may be present. The BIS serves the function of resolving the evolutionarily-important conflict resulting from risk-aversion (FFFS) and risk-proneness (BAS); and it is involved in the processes that finally generate the emotion of anxiety.

The BIS entails the inhibition of prepotent, conflicting, behaviours (e.g., those related to the FFFS and BAS), the engagement of risk assessment processes, including the scanning of memory and the environment to help resolve concurrent goal-conflict. This is all experienced subjectively as worry, apprehension and the feeling that actions may lead to a bad outcome. The BIS resolves goal-con-

[1] Given the multidimensional nature of the neurophysiological processes involved in anxiety (see McNaughton & Corr, 2004, 2008), there may well exist states/disorders that, in cognitive and behavioural terms, resemble anxiety but which lack the subjective-emotional component (e.g., as seen in alexithymia).

[2] The BIS is activated by all forms of goal-conflict, including approach-approach conflict (e.g., which of two academic positions one should take?). The aversiveness of this form of conflict resides in the perceived opportunity costs of making the wrong choice.

flicts by increasing, through recursive loops, the negative valence of stimuli (held in cortical stores), via activation of the FFFS, until resolution finally occurs either in favour of approach (i.e., a return to pre-conflict behaviour) or active avoidance/escape. Theta activity is the neural signature of this BIS activity and can be identified by EEG theta coherence during emotionally-charged rumination (Andersen, Moore, Venables, & Corr, 2009).

The BIS may have a privileged role to play in integrating biological, cognitive and experiential features of anxiety; and this is in spite of the fact that BIS theory is often seen to be operating exclusively at the automatic-reflexive level of processing. However, recent expansion of the concept of the BIS has clarified its full range of applicability to anxiety (Gray & McNaughton, 2000; McNaughton & Corr, 2004, 2008). In particular, focus on the BIS may be warranted because it provides a basis not only for the behavioural functions of anxiety but also for how (certain types of) automatically processed information (i.e., prepotent behaviour) gets extracted and subjected to higher-level cognitive analysis by controlled processes (some of which enter conscious awareness, and form the content of the complaints of the anxious person). Important in this respect is the way that the BIS affords an explanation for this transition, in the form of a mismatch error signal (i.e., between expected and actual states of the world), which is detailed below.

This expanded account of BIS functioning offers a viable account of the challenges faced by evolution: how to achieve adaptive 'fast and dirty' behavioural responses, especially in basic defensive situations, as well as 'slow and clean' behavioural responses, especially in complex or novel environments (LeDoux, 2002) – however, the latter process created its own problems, namely the 'lateness' of controlled and conscious processes, and how these 'late' components interface with the neural machinery that control immediate behaviour. In order to avoid misunderstanding, it is necessary to appreciate that, to the extent that conscious awareness, and the controlled processes that underlie it, play any role, and in order for them to have causal efficacy, they must interface with the machinery that controls immediate behaviour which, as discussed below, is always controlled at a preconscious, automatic level. Put another way, behaviour is initiated and executed at a preconscious, automatic level: mind events must follow brain events (for a justification of this theoretical stance, see Corr, 2010a).

The vast range of psychological processes, computed entirely nonconsciously and without the involvement of higher-level controlled processes, does not pose a problem for our understanding of pre-existing behavioural routines (see Velmans, 1991, for a review of these processes). Likewise in clinical conditions, we see automatic modes of processing, for example as in 'blindsight' (i.e., subjective blindness but intact visual performance; Weiskrantz, 1986), and in a variety of other clinical conditions (see Frith, 2007). In the wider realm of psychology, such processes are consistent with the (re)discovery of implicit personality processes (Bargh & Williams, 2006). But anxiety phenomena are different to these forms of exclusively automatically-controlled phenomena: although there is much of an automatic nature to anxiety, conscious awareness looms large and is a defining feature – failure to explain this fact and to incorporate it into a causal model must render any resulting theory, at best, incomplete and, arguably, inadequate.

So, how should we deal with the finding that controlled processing, and especially its conscious awareness components, comes after the initiating brain-behavioural event (indeed, some 300–500 ms; this is the 'neuronal adequacy' period)? That is, on a millisecond-by-millisecond basis, the engagement of controlled processes and their representation in conscious awareness lags behind the brain's initiation and execution of the behaviour itself. (Based on the extensive work of Libet, 1982, 1985, 2004, the evidential basis for these assertions is summarised by Corr, 2010a.) In consequence, only the results of the processes are accessible to conscious awareness, not how the behaviour was initiated and executed (the production of language is an obvious example of this distinction).

2.3. Outlines of the functional model of anxiety

The model of anxiety outlined in this section, and expanded in the sections to follow, is based upon the general model of behavioural control developed by Corr (2010a,b). In turn, this is based on Gray (2004) functional theory of consciousness, which took seriously the implications of the work of Libet (1982, 1985, 2004) on the lateness of conscious awareness. Gray's (2004) model postulates three aspects of consciousness, which can be extended to anxiety phenomena.

1. It contains a model of the relatively enduring features of the external world; and the model is experienced as though it is the external world (qualia generation; e.g., colour is not part of the external world, but is a brain-mind construction from the electromagnetic energy that in the eye and brain are no more than electrochemical signals that convey information for higher-level interpretation and the construction of qualia). Qualia of the anxious person (cognitive aspects, e.g., worry and rumination; physiological aspects, e.g., digestion problems; subjective aspects, e.g., feeling of imminent danger) result from a heightened perception of threat and a risk assessment scanning of internal and external environments, readying the body for rapid (FFFS) defensive action.
2. Features that are particularly relevant to ongoing motor programs, or which depart from expectation, are monitored and emphasised. An over-active BIS triggers easily error signals, the contents of which pervade the conscious mind – these error signals are aversive in nature which colours consciously-accessible emotion by virtue of its risk assessing processes, one of which is to serve as an input into the FFFS which adds further to the aversive quality of qualia.
3. The control variables and set-points of the brain's nonconscious servomechanisms are juxtaposed, combined and modified; in this way, error can be corrected. However, in the case of anxious rumination, this process is dysfunctional for a variety of reasons; for example: (1) controlled processes are overwhelmed with material; (2) the FFFS will tend to be highly activated; and (3) this coactivation will tend to feed upon itself by generating further error-triggering signals.[3]

2.4. What-if modelling

In relation to point (3), error triggering stimuli – which according to this model are always aversive in nature - are displayed in the medium of conscious awareness, and it is here that further 'what-if' controlled processes take over – at this point, stimuli can become detached from the external world. Imagination can create its own aversive stimuli, for example, in the form of secondary appraisal. This 'what-if' virtual reality environment in which the world is modelled, especially those features that depart from

[3] This cybernetic view of the brain-mind was foreshadowed by Kenneth Craik (1943), who proposed that the nervous system is a calculating machine capable of modelling external events; and that the mind constructs a small-scale model of these external events and of its own possible actions in relation to them, so as to provide the facility to try out various alternatives to ensure that reactions to future situations are appropriately based on prior knowledge of likely outcomes. This internal model frees the organism from responding to the external world in a, real-time, trial-and-error manner (where such error can lead to death).

expectation, may be assumed to have conferred enormous evolutionary advantage, especially in social contexts; however, in the anxious person it can be the source of self-generated error-signals and thus fuel further cycles of worry, anxious rumination, etc. Typically, this rumination is self referential, adding to its salience.

2.5. Function of subjective-emotion?

At this point, it may be instructive to pause to consider the wider context of the function of emotion in terms of 'lateness' of subjective states – which, it needs to be remembered, always run hundreds of milliseconds behind the initiating brain-behavioural events (see Corr, 2010a). The idea that we feel an emotion and then act cannot be accepted within the terms of the proposed model: emotion comes too late in the causal chain of events to affect the behaviour it represents. So what function does emotion serve, if any? This is a problem identified by other researchers. For example, Baumeister, Vohs, DeWall, and Zhang (2007, p. 194) make the following observation,

"The assumption that the purpose of full-blown, conscious emotion is to cause behaviour directly appears to be widespread and indeed deeply embedded in psychological theorizing. Yet it appears to be far less true than many researchers (ourselves included) have assumed".

Therefore, (a) if emotion (e.g., anxiety) does not have a direct causation on behaviour, and (b) conscious processing has a functional role, then (c) what could be the functional role for anxious rumination? One possible answer is that anxious rumination does influence behaviour but not the behaviour it represents but rather future instances of behaviour. In normal anxiety this process works effectively, but in clinical anxiety this process becomes dysfunctional. Perhaps by knowing the processing system characteristics it might be possible to suggest the primary dysfunctions that lead to pathological anxiety. The remainder of this paper addresses this point.

3. Anxious awareness as a product of error detection

A major question to be answered is: what might be the functions of the generation of conscious awareness involving anxious rumination? The model proposes that these functions are to be found in the inhibition of inappropriate prepotent (automatically processed) behavioural routines that are not going to plan. This serves the immediate function of stopping the behaviour that is producing the error signal, and subjecting the error-triggering environment to detailed controlled analysis: in the case of anxiogenic stimuli, the result is experienced as negative rumination, worry and the feeling that something bad might happen.

At the core of this model is a 'comparator', which compares actual stimuli with expected stimuli – this function is performed by the BIS (Gray, 1982; Gray & McNaughton, 2000; McNaughton & Corr, 2004, 2008; for a summary, see Corr, 2008). When there is no discrepancy, and 'all is going to plan', the comparator is said to be in 'just checking mode' and behavioural routines run uninterrupted and stimuli are not extracted for detailed processing by higher-level cognitive processes. When a mismatch is detected, between the actual and expected states of the world, then the comparator goes into 'control mode', and the salient features of the error-triggering environment are subjected to controlled, attentional, analysis and (often) represented in the medium experienced as conscious awareness. This type of analysis is not uncommon throughout psychology; for example, as stated by Mayr (2004, p. 145),

"It has been often noted that we usually become aware of those aspects in the internal or external world that interfere or interrupt routine action – which are very same events that typically elicit executive control operations".

To illustrate these inhibitory functions, imagine you are confronted by a dangerous snake. Your FFFS-fear system would be activated and automatic brain-behavioural routines (e.g., simple fleeing reaction) triggered. This activation would be initiated long (i.e., hundreds of milliseconds) before you were aware consciously (i.e., 'see' and 'feel' qualia) of this event. Now, it would be highly adaptive for you to have the facility to 'replay' this immediate past in order to analyse its contents – this facility would be especially beneficial at times when your automatic behavioural routines did not achieve their goal (e.g., avoiding the dangerous snake in the first place). According to the model, this is one of the major functions of controlled processes.

The inhibitory function of consciousness solves one major evolutionary problem: how to ensure that automatic responses are appropriately activated; and how controlled processes are invoked only at critical junctures, when a definite choice has to be made and a cautious, risk-assessing, mode of processing is more appropriate than the prepotent response. At these critical junctures, and after fine-grained analysis afforded by controlled processing, cybernetic adjustments can be made to the automatic system, such that when the same (or similar) stimulus (e.g., snake) is encountered in the future, automatic-reflexive behaviour will be more appropriate (see below). In this way, controlled/conscious effects come to determine automatic/nonconscious effects, albeit with a time lag. Fig. 1 shows the flow of information in automatic and control modes.

It should be immediately obvious that anxious rumination may be the result of the BIS error triggering mechanism being too sensitive, flooding the medium of conscious awareness with negatively valenced stimuli. This should then have the secondary effect of impairing the efficiency of controlled processes to rectify the error and adjust the cybernetic weights of the automatic system; in consequence, the next time when the same or similar stimuli are encountered, instead of the system running smoothly without error signal activation and an appropriate response to the initial stimuli, error signals continue to be generated: error begets error; anxiety begets anxiety.

In addition, the sensitivity of the BAS and FFFS will have a significant impact upon the detection of goal-conflict. When they are sufficiently and (approximately) equally activated they will trigger goal-conflict by causing the planning and predictor system to fail to decide upon a dominant form of behaviour: this leads to BIS-related behavioural dithering, cognitive indecision, and controlled processing. Thus, there may be forms of anxiety that are not the result of an over-active BIS, but rather the simultaneous activation of the FFFS and BAS – this could be the result of a weak inhibitory link between these two systems (see arousal-decision model of Gray & Smith, 1969). The problem finally resolves itself by the whole system becoming more risk averse and when conflict continues, in consequence, behavioural control reverts to FFFS-mediated active avoidance/escape, or if the magnitude of perceived threat declines than a return to BAS-mediated behaviour. Important in this sequence of events are coping and appraisal mechanisms (both primary and secondary) which have a significant impact on predictions (including self-efficacy) about the world. Therefore, individual differences in sensitivity and activation of FFFS, BAS and BIS give rise to the personality components of this model, as do personality concepts related to self-efficacy, perceived control and appraisal of the consequences of mismatch between the expected and actual state of the world.

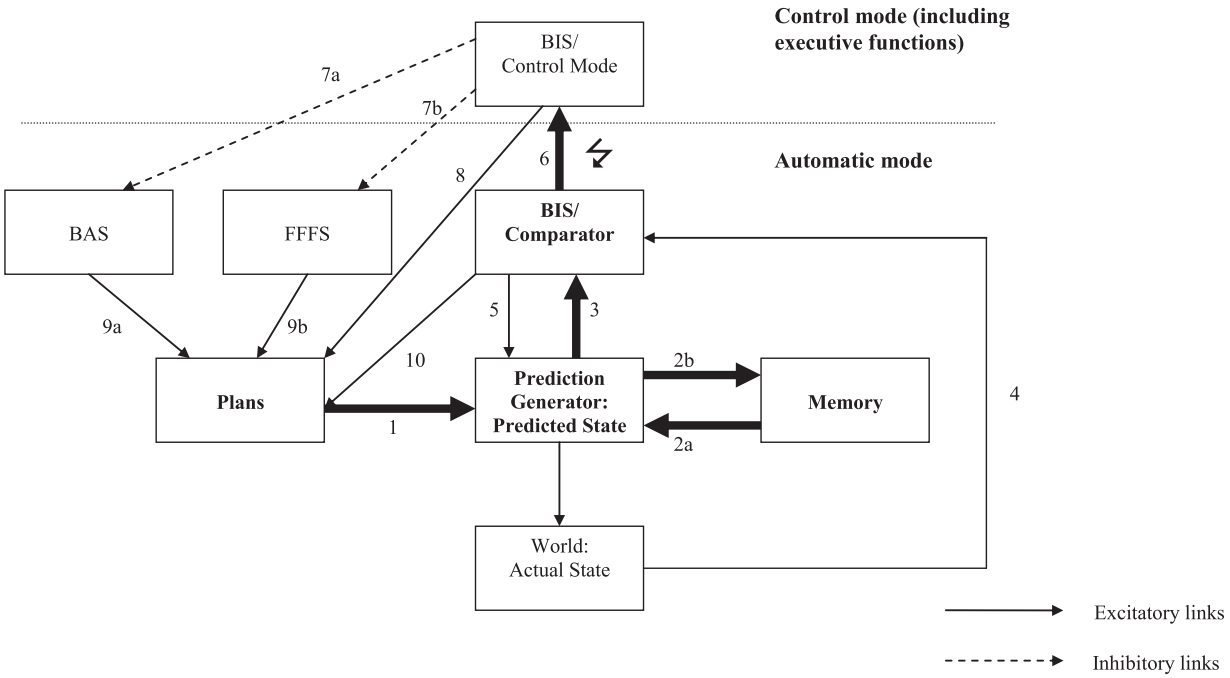

Fig. 1. Information processing diagram of the functioning of the behavioural inhibition system (BIS) in 'automatic mode' and 'control mode', containing basic approach (BAS) and avoidance (FFFS) factors that, along with the BIS, comprise three fundamental brain-behavioural systems that underlie the major dimensions of personality. The state of anxiety arises from activation of the BIS by error-generating signals which are extracted for further detailed analysis by the controlled-reflective system. Behavioural plans (Plans) lead to predictions (Prediction Generator; 1) of future states of the world, which receives input from (2a), and sends output to (2b), stored previous experience (Memory). The BIS (BIS/Comparator) receives input from the Prediction Generator (3), and then compares the response-reinforcement outcomes (World: Actual State) with predictions (4), and then one of two things happen: (a) 'everything is going to plan', and the BIS/Comparator sends input to the Prediction Generator to continue the motor program ('just checking mode'; 5); or (b) the BIS/ Generator detects a mismatch between prediction and outcome and generates an error signal (ϟ), which leads to activation of controlled processes (BIS/Control Mode; 6). Once the BIS/Control Mode is activated, there is inhibition of the behavioural approach system (BAS; 7a) and the fight–flight–freeze system (FFFS; 7b); and at this time the BIS initiates cautious behaviour and risk assessment (see text), which then informs Plans (8), which simultaneously receives input, about current states, from the BAS and FFFS (9a, b), as well as input, about the nature of the conflict, from the BIS/Comparator (10). Plans initiate appropriate behaviour and the above cycle is repeated, until behavioural resolution is achieved in the form of FFFS-mediated active avoidance/escape or BAS-mediated approach. According to this model, pathological levels of anxiety are generated by an over-active BIS, which inappropriately extracts stimuli for controlled processing – the weight of this information serves to over-whelm controlled processes and leads to a failure to adjust cybernetic weights of the behavioural control system (Fig. 2) so as to avoid error signals in the future. Bold lines highlight possible sources of dysfunction seen in anxiety states/conditions, which may include: (a) inappropriate plans (1); (b) inadequate prediction generation (3); (c) inappropriate retrieval/storage of information from/to memory (2a/2b); or (d) over-active BIS/comparator (6) – shown in bold and thick arrows. (Adapted from Corr, 2010a.)

4. The anxiety model

The flow of information between automatic and controlled processing is shown in Fig. 2. This model adopts Gray (2004) idea that actions that are organised at the automatic-reflexive level (e.g., fleeing from a predator) can, nonetheless, be affected by controlled-reflective processes. As an extension of this idea, for example, a fear state that is experienced consciously has the capacity to sensitise the whole defensive system (e.g., in a particular environment) and, thereby, affect all *subsequent* fast, automatic responses in that environment; alternatively, more specific controlled process alterations can be made to automatic behavioural routines. In other words, over the course of hundreds of milliseconds, behaviour is modified by experience: *learning* occurs. This view is endorsed by leading emotion researchers; for example, Baumeister et al. (2007) (p, 195) state, "Evidence indicates that conscious emotion is helpful for learning" and "Emotion stimulates reflection on prior events".

In order for sensitization of the whole defensive system, anxiety-related emotions seem able to provide automatic-reflexive level valence to context (e.g., foraging field *x* is potentially dangerous; or a specific person may be harmful); and this valence bias is then imposed upon incoming preconscious information (primary appraisal), such that during the initial ~300–500 ms 'neuronal adequacy' period, when behaviours are prepared and initiated, automatic routines are biased to respond ("without thinking") in specific ways to general context, as opposed to specific stimuli (e.g., what is currently in the field or what the person just said). By this route, controlled processing can have a direct causal influence on automatically-elicited behaviour, but in the *next* iterative cycle of behaviour. This form of valence bias would serve the additional function of priming the inhibition system so that inappropriate prepotent behaviour is halted faster the next time it generates an error signal: in consequence, the anxious person will be easily distracted, undecided, and have the feeling that bad things are just around the proverbial corner. They would be primed to respond in an anxious manner to even low level anxiogenic stimuli (whether real or imagined).

In relation to more specific controlled process alterations to automatic behavioural routines, anxiety-related emotions often provide the appropriate valence to controlled processing representations over the longer time frame, especially when 'what-if' simulations are computed to determine the likely future outcome of a specific action. The outcome of these simulations would be to affect the cybernetic weights of automatic behavioural routines. As shown in Fig. 1, individual differences in the sensitivity of reward (BAS), punishment (FFFS) and conflict (BIS) systems may be expected to influence the ease of generation of the emotion, thus allowing these systems to affect the contents of consciousness. In the case of anxiety, the ease of activation of the BIS may be especially important; however, if the FFFS is easily activated then this might lead, as a secondary consequence, to activation of the BIS.

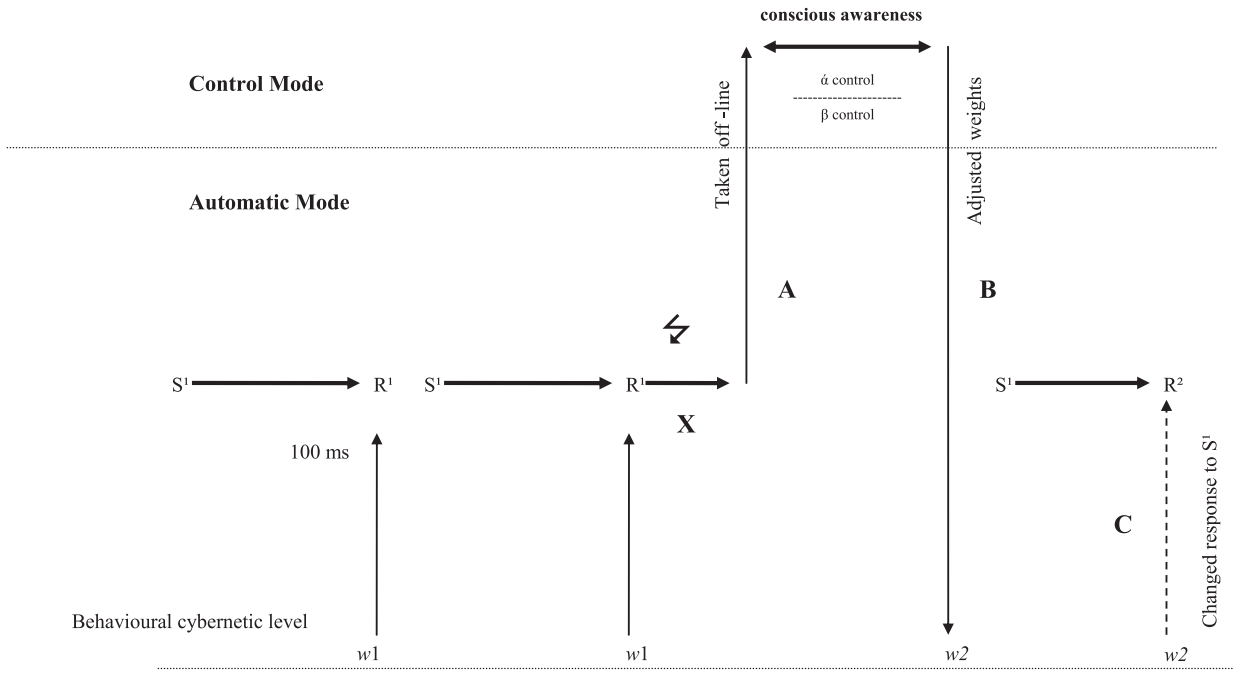

Fig. 2. Information processing diagram of the functioning of the cybernetic behavioural control system in the generation of anxious controlled processes and consciousness. When 'everything is going to plan', automatic processes proceed uninterrupted (see Fig. 1). When an error signal (↯) is detected (i.e., mismatch between expected and actual state of the world; e.g., encountering a snake in a familiar field, or a self-generated image of something unpleasant), the salient features of internal (e.g., memory) and external worlds are extracted and subjected to detailed controlled processing (Control Mode), which may result in a perceptual-based representation and display in a medium that is experienced as conscious awareness (shown as 'α control'). Control mode processing may occur in the absence of conscious awareness (shown as 'β control'). In either form of processing, extracted stimuli are subject to (varying degrees) of fine-grained analysis—all of this happens within hundreds of milliseconds (this period of time required for the generation of conscious awareness is the 'neural adequacy; period; **X**). Controlled processing can alter the cybernetic weights (e.g. w^2) of automatic processes and, thereby exert a causal influence on *future* automatically controlled responses (e.g. R^2) when the same (or similar) stimuli/worlds are encountered. High states of anxiety result from: BIS-activated error-signal generation (**A**; different reasons for this generation are given in Fig. 1); weak 'interface' between control mode processing and automatic mode processing (**B**); and, therefore, a subsequent failure appropriately to adjust the cybernetic weight that control immediate behaviour (**C**). For these reasons, iterative reactions to anxiogenic stimuli are difficult to stop, and the result is a vicious cycle of ruminative anxiety. This cycle may be stopped by therapeutic intervention designed, for example, to attenuate the salience of stimuli (primary appraisal), modify the attributions once stimuli are extracted for controlled processing (secondary appraisal), and facilitation of the 'interface' between control mode and automatic mode processing by, for example, augmentation of serotonin activity (see text). (Adapted from Corr, 2010a.)

In any case, BIS activation will activate the FFFS (see above), and for this reason fear and anxiety are often co-activated. The important question though is: which process initiated the goal-conflict in the first place? Thus, the model predicts two general forms of anxiety: primary (BIS-initiated) or secondary (FFFS-initiated). We may account for anxiety symptoms by these processes as well as the pathological running of mental 'what-if' simulations (e.g., worry) which serve to maintain and exacerbate symptoms.

Thus, controlled process analysis exerts an influence on automatic routines by *general* and *specific* influences, that is by the changing of specific automatic cybernetic weights such that when the same stimuli, that previously led to an error signal, is encountered again a different (more appropriate) reaction occurs. Pathological levels of anxiety consist in the dysfunction in these normal processes of behavioural control.

4.1. Executive control

Anxiety is unlikely to involve just the activation of the BIS, whether or not in association with the FFFS and BAS. BIS activation may be a necessary condition, but higher-level processes provide the sufficient conditions for, at least, pathological states/conditions of anxiety (for a summary of this literature, see Eysenck & Derakshan, 2011). This conclusion flows from the realisation that flexible behaviour requires a high level of coordination, involving attention, decision-making, etc. Whilst the hippocampus (and other distributed structures) of the BIS may be necessary to mediate error signals (McNaughton & Corr, 2004, 2008; see Corr, 2010a, 2010b), they function in collaboration with cortical stores of information reflecting the conflicts between *goals*. In this regard, prefrontal cortex (PFC) is important. Miller and Cohen (2001) provide a review, and an outline of a model, of how the PFC functions to achieve cognitive and behavioural coordination. They note that, in order to avoid behavioural confusion, mechanisms must have evolved that coordinate low-level sensory and motor processes according to the representation of internal goals. This theory is consistent with the cybernetic view adopted in this paper. (For a discussion of the evolution and hierarchical nature of emotion, also see McNaughton & Corr, 2009.)

More specifically, PFC is a network of neocortical areas that send/receive projections from nearly all sensory and motor systems, and many subcortical areas. The 'top-down' functions of PFC are guided by internal goal states; and these are especially important when there is a mapping between sensory input, cognition and action that are either weakly developed, relative to existing ones, or are rapidly developing. As noted by Miller and Cohen (2001), one of the most important aspects of cognitive control is the ability to select a weaker, task-relevant response, in the face of stronger competing, prepotent, but task-irrelevant, ones. PFC is seen to be important in executive control, especially the active maintenance of goals and the rules of the task.

In relation to anxiety, selective attention and behavioural inhibition may be seen to be two sides of the same coin. On the one hand, attention is the effect of biasing competition in favour of salient information (in this case, threat-related information); and inhibition is the consequence this has for the irrelevant information

(e.g., the inhibition of BAS-related behaviour). PFC-mediated control is complemented by another form of control dependent on the hippocampal system (Miller & Cohen, 2001), which is important for binding together information into a specific episode; in contrast, PFC, like other neocortical areas, is more important for extracting the regularities across episodes (corresponding to goals and task rules).

Added to the above, there is also an important role for the anterior cingulate cortex (ACC) which is active during a variety of cognitive tasks that entail mental effort, and that its involvement in these tasks may be explained by its role in the detection of conflicting response tendencies, although only when conflicting stimuli are consciously perceived (Dehaene et al., 2003). In addition, Moeller and Robinson (2010) reviewed a large literature which shows that reactivity to error feedback is related to ACC – itself part of the distributed BIS – which is also activated by subjective experience of distress and pain; and, furthermore, that patients with anxiety-related disorders show enhanced activation of ACC when stimuli are discrepant from expectations. (For further discussion of the neural loci of detection of goal-conflict, see Corr, 2010b.)

4.2. Serotonin

The importance of serotonin in anxiety-related disorders is well known, although not much is known about the signals encoded by individual serotonin neurons. However, there is evidence suggesting that this neurotransmitter plays an important role in the control of behaviour by aversive events, punishment and losses. For example, animals with lesions of the serotonin system have difficulty acquiring stimuli associations that require the inhibition of a response and difficulty inhibiting the response if the lesion is produced following training (Harrison, Everitt, & Robbins, 1999; Soubrie, 1986). In terms of the proposed model, serotonin is seen as one of the most (if not the) important neurotransmitters as it innervates the entire defensive system (including both FFFS and BIS; see McNaughton & Corr, 2008). It is noteworthy that the treatment of choice for many neurotic disorders entails enhancement of serotonin neurotransmission (e.g., Selective Serotonin Reuptake Inhibitors; SSRIs). The above findings are consistent with Carver et al. (2008), who claim that serotonin is important in the transition to/from automatic/controlled processing (this is the 'interface' problem).

4.3. Experimental evidence

There has been a paucity of experimental studies linking different features of anxiety to the *interface* of, and transition to/from, automatic and controlled processes; however, there have been many studies focusing on only one of these levels (see papers in this special issue). In support of the idea that, in high scorers on neuroticism, dysfunctions are observed between these two levels of processing, Corr (2003) reported that these individuals have impaired automatic processing (i.e., the procedural learning of the sequence of stimuli) in the presence of controlled (attentional) dual-task processing, especially when the dual-task is cognitively demanding (e.g., counting backwards). In terms of the model proposed here, impaired automatic processing of stimulus regularities may underlie the inability of high neuroticism individuals to resolve BIS-related cognitive/motoric conflicts, hence producing the rumination, threat-perception, and worry that accompanies high neuroticism and anxiety. This finding suggests that highly anxious individuals, with an hyperactive BIS, tend to experience a high level of error signals, leading to material being extracted for controlled process analysis, which then lead to an inundation of negatively valanced material competing for processing resources on ongoing tasks: this interpretation accounts for experimental dual-task effects as well as the high level of distractibility seen in clinical anxiety. This position is consistent with the association of low serotonin with (a) neuroticism (and related clinical disorders), and (b) disrupted transition to/from automatic-controlled processing (Carver et al., 2008).

The adaptive function of the BIS is to resolve goal-conflict and thus change the probability of responses to future instances of stimuli that previously led to conflict. However, at high levels of BIS-related error signal generation, controlled resources will be overwhelmed and this failure to resolve conflict itself generates further conflict: by this route, normally functioning anxiety can become pathological. In a different context, it is interesting to note that worry impairs occupational performance in, relatively, low cognitive ability people but enhances it in, relatively, high cognitive ability people (Perkins & Corr, 2005, 2006). Such findings suggest that cognitive resource capacity determines whether worry is functional or dysfunctional. In determining these effects, the functional capacity and efficiency of the PFC-related processes (e.g., working memory and executive control) must be assumed to be important.

4.4. The split anxiety atom

We have arrived at a picture of anxiety as reflecting the operation of multiple, interrelating, processes. At the lowest, automatic-reflexive, level are basic systems (e.g., FFFS) that are responsible for prepotent defensive behaviours, producing heightened reactions to aversive stimuli (real and imagined). When goal-conflict occurs, the BIS is activated, leading to behavioural inhibition, indecision, and caution and the subjective aspects of anxiety – over the course of time and repeated activation, this automatic-reflexive system can come to be primed to detect threat and goal-conflict, producing a chronic, trait-like, over-readiness to respond with prepotent defensive outputs. Once the BIS is activated by goal-conflict, salient features of the aversive environment, which have been tagged as 'not going to plan', are extracted for higher-level analysis by the controlled-reflective system, where detailed information processing occurs, often entailing the generation of conscious awareness where the immediate past ($\sim$300–500 ms) is replayed for the purpose of 'what-if' analysis: subjectively, this is experienced as worry and rumination, leading to, among other things, cognitive distractibility. At pathological levels, the controlled-reflective system is overwhelmed with the quantity of information to be processed, leading to the cognitive inability to resolve the underlying goal-conflict; in turn, this forms the basis for further goal-conflict and this pathological cycle is further fueled by activation of the FFFS by the BIS. By this route, the behavioural, cognitive, emotional and subjective qualities of anxiety can become self-propelling, with ever greater levels of anxiety being fueled by dysfunctional interface of automatic-controlled processing (i.e., controlled processes fails to modify the cybernetic weights of the automatic machinery of behaviour).

4.5. Fractionating the BIS

In comparison with Gray's (1982) original formulation of the BIS, the revised model of Gray and McNaughton (2000) (see further revisions in McNaughton & Corr, 2004, 2008) is multi-process, distributed over a number of structures. These developments point to additional sources of influence that may be studied in anxiety. It is now preferable to view the BIS as dysfunctional rather than being simply under or over-active; and this dysfunction may be seen in any of its distributed components. For example, it is quite feasible that the cognitive and motivational components of the BIS are under-active, but that the processes leading to the generation of the subjective state of anxiety, and the fuelling of general negative

emotionality via inputs to the FFFS, are functioning relatively normally, which given (a) the high level of BAS and FFFS, and (b) the inability of the BIS to resolve their conflicts, should be expected to generate considerable anxiety because the BIS is not deactivated by successful goal-conflict resolution. These processes show how under-activity in some parts of the BIS can lead to, seemingly, over-activity in other parts (although the 'over-activity', leading to anxiety, may be entirely appropriate to the goal-conflict information that it receives and fails to resolve – it is not the anxiety that is maladaptive, but the underlying processes generating it).

In terms of the neurology of revised RST (for latest version, see McNaughton & Corr, 2008), the following systems may be associated with the psychological processes outlined above. First, the detection of conflict is based in the hippocampus as the main locus; however, they may also be related detectors at all levels of the BIS, ranging from the periaqueductal gray, medial hypothalamus, amygdala, septo-hippocampal system, posterior cingulate and prefrontal dorsal stream (McNaughton & Corr, 2008). The systems relate to different forms of BIS processing: lower levels detect conflict between quick and dirty goal representations, and higher ones with slower more sophisticated ones (with the top end involving "planning"). This distinction maps onto the distinction between automatic vs. controlled processing. Secondly, in terms of attentional processing, neurotransmitter systems, principally, acetycholine and norepinephrine, are likely to be heavily involved. Thirdly, behavioural inhibition is likely to be controlled by the inferior frontal gyrus, or under very tight time constraints the presupplementary motor cortex. Fourthly, in terms of the inhibition of prepotent behaviour, inhibition involves output from the BIS to whatever motor areas provided the input that generated the conflict. The output will be to the lower levels of the motor system, leaving the activation of the goal representation itself intact but preventing its normal capture of the motor system. Given this impairment of the BIS, common stimuli are less likely to be tagged as similar, and thus these stimuli act as novel inputs further disrupting the smooth operation of attentional processing by, for example, triggering orienting reflexes. Lastly, activation of the BAS, FFFS and the BIS is likely to lead to high levels of arousal, especially emotional arousal via the amygdala, which serves only to exacerbate existing symptoms. These areas provide a clue to where dysfunctions may be observed in different expressions of anxiety and may form the targets for future research.

4.6. Implications for clinical intervention

According to the above proposals, there are a number of possible targets for interventions designed to break the pathological anxiety cycle. For example, there is evidence that some forms of anxiety are anxiolytic insensitive (Gray & McNaughton, 2000), suggesting the greater involvement of frontal processes, perhaps related to 'what-if' simulation, with less of an primary influence of lower-level BIS processes (e.g., hippocampus). In other expressions of anxiety, as seen in alexithymia, presentation of the subjective component is defective in the controlled-reflective system. Seen in this theoretical light, anxiety symptoms may be presented in different forms. In order to recommend a specific intervention, it would be necessary first to isolate the components of primary dysfunction; for example, over-active BIS; over-active mental 'what-if' stimulation; tendency for BAS and FFFS to be simultaneously activated; inadequate capacity or efficiency of controlled system to resolve underlying goal-conflict; or dysfunctional transition to/from automatic and controlled processes?

This general approach to understanding the multidimensionality of anxiety, therefore, suggests that it should be possible to fractionate 'the anxiety system' into its separate systems and processes. The challenge though would be to design tests that assess primary component dysfunction, as opposed to secondary activation of other components of the system or whole-system activation. Work along these lines should also contribute to furthering understanding of the normal process of self-regulation that maintains adaptive forms of anxiety (cf. Carver & Scheier, 1981), as well as identifying the nature of the absence of certain forms of anxiety as seen in clinical conditions such as psychopathy (Corr, 2010c).

5. Concluding remarks

This paper identified a number of theoretical problems that need to be recognised before attempting to construct a viable model of anxiety capable of integrating evolutionary, genetic, neurophysiological, neuropsychological, cognitive, behavioural and experiential factors. These two problems (i.e., 'lateness' and 'interface') were addressed from the perspective of multiple levels processing which included the functional role of conscious awareness in subjective angst. An integrative model was outlined, pointing to the different functions of anxious emotion and its cognitive and behavioural components in terms of inhibition of inappropriate prepotent behaviour, extraction of error-prone stimuli for detailed, higher-level, controlled processing, and the adjustment of the cybernetic weights of the automatic machinery in order that future behaviour can be appropriately modified. Clinical anxiety was seen as dysfunction in these otherwise normal processes of emotion and behaviour regulation. Different types of dysfunction highlighted the possibility that there exist different expressions of anxiety (e.g., some more behaviourally based, others more emotion based). Targets for isolating primary dysfunctions, and their implications for clinical intervention, were discussed.

This paper may help to resolve venerable debates in emotion research (e.g., James-Lange and Zajonc-Larazus debates) because, according to the proposed model, all emotion must be, *at the point of initiation and execution*, nonconscious; but that, higher-level, cognitive processes are also involved and important in influencing future behaviour. Control can, and is, exerted by automatic processes as well as controlled processes. In this way, the multi-level processing nature of anxiety is not the problem but the solution. Finally, this paper may have gone some way to suggesting answers to William James (1884) seminal question: 'What is an emotion?'.

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