In Review

Anxiety: An Evolutionary Approach

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Anxiety disorders are among the most common mental illnesses, with huge attendant suffering. Current treatments are not universally effective, suggesting that a deeper understanding of the causes of anxiety is needed. To understand anxiety disorders better, it is first necessary to understand the normal anxiety response. This entails considering its evolutionary function as well as the mechanisms underlying it. We argue that the function of the human anxiety response, and homologues in other species, is to prepare the individual to detect and deal with threats. We use a signal detection framework to show that the threshold for expressing the anxiety response ought to vary with the probability of threats occurring, and the individual's vulnerability to them if they do occur. These predictions are consistent with major patterns in the epidemiology of anxiety. Implications for research and treatment are discussed.

Clinical Implications

- Understanding the evolved function of the anxiety response can aid in understanding why people's propensity to become anxious varies with their life circumstances and developmental history.

- Theory predicts that the threshold for mounting an anxiety response should depend on the probability of dangerous events occurring in the current environment, and the vulnerability of the person to those events should they occur. Numerous epidemiologic findings relating to anxiety disorders can be integrated within this framework.

Limitations

- The evolutionary criteria for when a mechanism is functioning adaptively are different from the criteria for identifying when a psychiatric disorder is present.

- Evolutionary thinking has not yet been used to develop new treatment strategies for anxiety disorders, although it may aid in understanding why existing ones are effective.

Key Words: anxiety, anxiety disorders, emotions, signal detection theory, behavioural ecology, evolutionary medicine

It is not informative to study variations of behaviour unless we know beforehand the norm from which the variants depart.[1, p 109]

Anxiety disorders are among the most common mental illnesses, with huge attendant quality of life and financial costs. For example, during 12 months in 2001–2002, 4.6% of Canadians had symptoms meeting DSM-IV diagnostic criteria for an anxiety disorder,[2] and, during the course of their lifetimes, around 28.8% of Americans are estimated to be affected.[3] Although treatments for anxiety exist, these currently have limited efficacy. For example, for GAD, antidepressants, though more effective than placebo, have a number needed to treat of 5.54, meaning nearly 6 patients need to be treated with the drugs to produce 1 more clinically significant symptomatic improvement than placebo.[4] Only around 46% of GAD patients show a clinically significant response to psychological therapy.[5] The pervasiveness of anxiety disorders and their resistance to treatment suggest a need for a deeper understanding of the sources of anxiety. We argue that to make some progress understanding clinical anxiety disorders, we need to start by obtaining a better understanding of the normal anxiety response: What is it for, and why does it have the features that it does? As evolution by natural selection is the source of all complex physiological—including neural—organization in nature, this necessarily means taking an evolutionary perspective. Our concern is specifically with generalized anxiety.

Anxiety is highly comorbid with depression, but we do not have the space to explore evolutionary approaches to depression (see instead Dr Hagen's In Review paper[6]) or why their comorbidity would be so high. We also do not review the extensive evolutionary literature on specific phobias.[7]

Why Do People Become Anxious?

Behavioural biologists divide explanatory questions into 2 broad categories: those concerning ultimate evolutionary function and those concerning proximate mechanisms.[8,9] The ultimate function of a structure refers to the reasons that the structure in its current form has been retained through evolutionary time, against the many alternative forms of the same structure that are thrown up each generation through genetic mutation and recombination. For example, the ultimate function of the pigmentation in the skin of humans is to protect tissue from the damaging effects of ultraviolet light. This claim is not a just-so story, because it makes principled predictions that can be tested in contemporary humans. It predicts that people whose skin colour is at the lightest end of the normal range of variation will suffer increased rates of cancers and other ultraviolet-related health problems, such as neural tube defects in their offspring. These predictions are consistently confirmed.[10–12] Thus, given that these serious health problems affect survival and reproduction, we can state with some confidence that this account of the function of skin pigmentation is the correct one.

Mechanistic questions concern how a structure works. Human skin colour is produced by the pigment melanin, which is synthesized from the amino acid tyrosine in specialized cells called melanocytes, and transported to other epidermal cells in vesicles called melanosomes. These are all details of how pigmentation fulfills its function. Functional and mechanistic explanations can be pursued somewhat independently of one another in the first instance. We can work out what pigmentation is for without necessarily knowing all the details of how melanin is produced. However, the 2 must ultimately be integrated.[13] Functional thinking is potentially useful, even for the most mechanistically oriented researchers, as the function of a structure places constraints on how it can work and thus limits the search space of possible hypotheses researchers may want to pursue.

In psychiatric research, the focus has largely been on mechanistic explanations for symptoms of anxiety (for example, which drugs have anxiolytic or anxiogenic functions, and which brain areas and neurophysiological systems are implicated). While generally accepted that anxiety serves to prepare a person for threats,[14] so far only a few pioneers have thought in any detail about the implications of this being anxiety's function.[15,16] We believe that there may be considerable benefit for clinicians and researchers in thinking more functionally, as this will shed light on the interconnectedness of the different components of anxiety and help guide and integrate research into the brain mechanisms involved.

Are Some Cases of Anxiety Disorder Adaptive?

One question that tends to arise when considering psychiatric conditions from an evolutionary point of view is where the boundary lies between evolved function and dysfunction. A mechanism is working functionally in the evolutionary sense if it has a level of responsiveness that will, averaged across all individuals and the environments in which they live, maximize survival and reproduction. This is a very different criterion from those used to demarcate clinical boundaries in psychiatry, which are mainly based on level of suffering and quality of life. If a mechanism is producing distress or impairing quality of life, this does not necessarily mean that it is malfunctioning in the evolutionary sense. For many adaptations, such as the pain system, it is part of their design that they cause subjectively unpleasant states, and individuals' viability would be reduced if they did not do so under the appropriate circumstances. Thus, while undoubtedly some cases of anxiety disorder are pathological, in that the control mechanisms regulating the anxiety response have become dysregulated, it is also possible that some cases represent appropriate adaptive responses to the situation in which the person currently finds him or herself. As a corollary, insufficient anxiety-proneness may be a commonly occurring dysfunction. However, as it is not associated with subjective distress, presumably the people affected do not present for treatment.

Anxiety-proneness and anxious symptoms are distributed along a continuum in the human population, and symptom levels predict outcomes in a graded fashion.[17,18] Moreover, epidemiologic evidence suggests that the probability of long-term survival is lower in people with a low level of anxiety-proneness than those in the middle of the distribution.[19,20] It is not even well established that, in people with clinical levels of anxiety, the anxiety necessarily impairs biological fitness. In a recent study,[20] patients with clinically defined comorbid anxiety and depression had lower mortality than those with depression alone, despite having poorer health and more disability. All of this supports the view that clinical anxiety shares a continuum with the normal, protective anxiety response, and that locating the boundary between adaptive function and pathology is not straightforward, either philosophically or empirically. Moreover, the presence of dysfunction may not be the appropriate criterion for allocating treatment (for discussion, see Wakefield[21] and Cosmides and Tooby[22]). In view of this continuity, it is important to consider in detail the evolutionary forces that have shaped the anxiety response.

Abbreviations

ALR	anxiety-like response
DSM	Diagnostic and Statistical Manual of Mental Disorders
GAD	generalized anxiety disorder

Figure 1 The signal detection problem
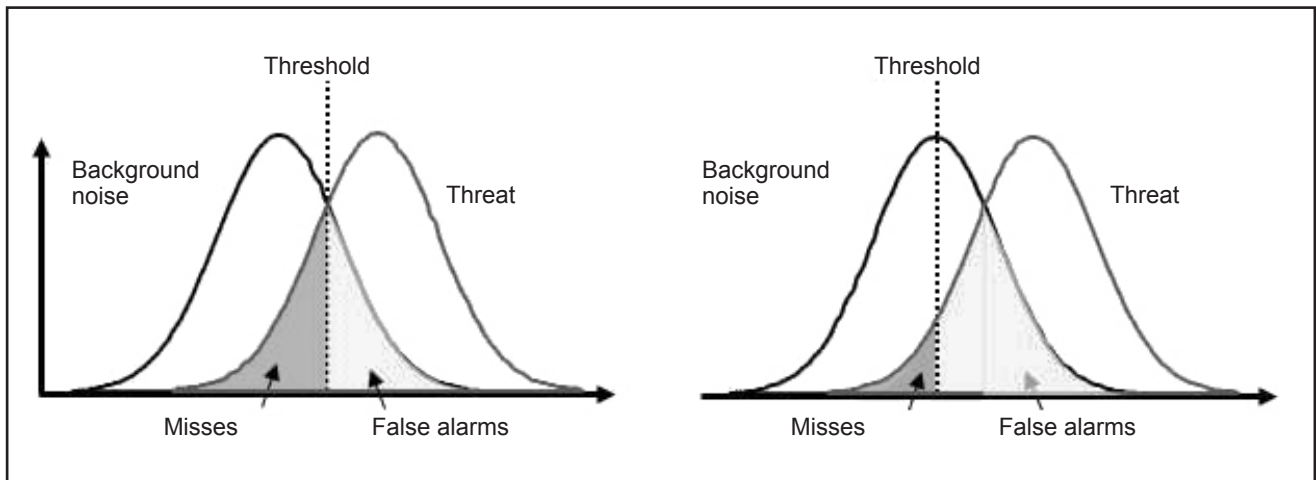
The horizontal axes represent the strength of sensory information reaching the subject, while the 2 normal distributions represent the probability of different amounts of evidence generated under the presence of a threat, and under background noise only. Crucially, the 2 distributions overlap. If the subject sets a high threshold for mobilizing a threat response (left panel), there will be a fair number of real threats missed as well as a fair number of false alarms. If the subject sets a lower threshold (right panel), the subject will miss many fewer real threats, but only at the cost of much more frequent false alarms. We argue that anxiety disorders represent unusually low personal thresholds for threat response.

Function of ALRs in Other Species

Evolutionary thinking in behavioural biology provides the researcher with a toolbox of different approaches. We will employ 2: the comparative and the optimality approaches.

Comparative Evidence

The comparative approach is based on comparing different species, or different populations of the same species, to discover the ecological correlates of a behaviour pattern, and thereby to test hypotheses about function. Psychiatrists have long employed comparative evidence from other species when thinking about mechanisms of anxiety (most psychopharmacology is done initially in animal models), but there is scope for greater use of comparative evidence in also investigating function.

Animals of many species display a suite of responses that we call the ALR in their normal behaviour. The ALR consists of increased heart rate, stress hormone secretion, restlessness, vigilance, and fear of potentially dangerous environments, and decreased feeding and exploratory behaviour. It also involves an increased tendency to interpret ambiguous stimuli as threatening.[23–25] In Belding's ground squirrels (*Spermophilus beldingi*), the components of the ALR are more strongly expressed in populations facing higher levels of predation.[26] Differences in stress hormones in high- and low-predation environments are detectable within a few weeks of the pups emerging from the nest. This suggests that the function of the ALR is to detect and deal with threats, specifically from predators in this case. The changes in physiology, cognition, and behaviour characterizing the ALR can be interpreted as ways of fulfilling this function. The cognitive changes increase the likelihood that imminent threats will be detected early, and, at the physiological level,

the ALR prepares the body for action by hyperventilating to oxygenate the blood, diverting blood to the muscles and sweating to cool the skin. Thus both the contexts in which anxious symptoms appear and the nature of these symptoms support the view that the function of anxiety is to prepare, physiologically, cognitively, and behaviourally, for detecting and dealing with threats to survival.

An Optimality Approach

Optimality approaches in behavioural biology involve building theoretical models of what behaviour we ought to expect if the structure has indeed been selected to fulfil the function we hypothesize (for an example in the context of low mood, see Nettle[27]). Model predictions can then be compared to actual data on the behaviour in question. We have already suggested that the function of anxiety is to prepare the individual for threats. How should such a threat detection system be designed? Nesse[28,29] introduced the use of signal detection theory to address this problem, an approach we extend here.

Signal detection theory is a body of mathematics concerned with the problem of how to decide whether a given event has occurred (here, the event is whether a threat is present; for example, was that rustle in the bushes a tiger or only the wind?). Ideally, sensory evidence would indicate this unambiguously, but, in practice, there is fluctuating ambient noise; although, on average, tigers make more rustling than the wind, in some instances a tiger generates less rustling than in other instances where there is no tiger. Therefore, there are overlapping distributions of cue intensity reaching the receiver from environments with tigers and from safe environments (Figure 1). The receiver must establish some criterion (called the signal detection threshold) above

which they will accept the evidence as indicating that the event is present (here, a level of rustling they will accept as sufficient to prepare for a tiger). Below this threshold, the rustling is dismissed as noise. Because the 2 distributions in Figure 1 overlap, any threshold that the receiver sets will generate some errors; it is impossible to set a threshold where the outcomes are all correct, whether detections (hits) or rejections. Specifically, there will always be some false alarms, where the receiver prepares for a tiger when it turns out to be nothing but the wind, and some misses, where the receiver does not prepare but there is a tiger.

The optimization problem concerns where to set the threshold. In general, there is a direct trade-off between the frequencies of false alarms and misses; raising the threshold will reduce the former but only at the expense of increasing the latter. The optimal threshold (λ) for the receiver to adopt can be derived mathematically,[30] and can be simplified for our purposes as follows in Equation 1:

$$\lambda = \frac{p_{nt}}{p_t} \cdot \frac{w_{fa}}{w_{miss}}$$

where p_{nt} and p_t are the probabilities of there being no threat present, and a threat present, respectively, and w_{fa} and w_{miss} are the respective costs of a false alarm and a miss (see also Haselton and Nettle[31]). Note that costs here are in terms of biological fitness. Thus, if misses usually lead to death, w_{miss} is very large. The p_{nt}/p_t term is determined by the objective chance that a threat is imminent (that is, the threat probability). We call the w_{fa}/w_{miss} term the individual's vulnerability, as it specifies how bad it would be for that individual's fitness if a real threat went undetected.

A couple of predictions follow from this equation (summarized in Figure 2). First, as the actual prevalence of threats in the environment goes up (that is, the probability increases), the optimal threshold for mounting the ALR gets lower. This means that when an optimally behaving animal is in an environment where there is objectively a lot of danger (or where it has received prior cues that dangers are frequent), it should require much less evidence of the presence of a threat to trigger the ALR than it would when in a safe environment. This means we should predict that animals will display many more anxiety symptoms in such environments. Some of the additional ALRs will be hits, owing to the greater prevalence of threats, but many will be false positives, as lowering the detection threshold necessarily entails increasing the false alarm rate. Figure 2 shows that as probability increases, the optimal threshold decreases initially at a faster than linear rate, which means that as the hit rate goes up, the false alarm rate goes up even faster. Thus, as the individual's best estimate of the amount of danger around goes up, the amount of both correct and baseless threat responses ought to increase. We have already seen that this prediction holds in the case of Belding's ground squirrels.

Figure 2 The optimal threshold for mounting a threat response, as a function of the probability of that threat being present in the environment, for individuals with 3 levels of vulnerability
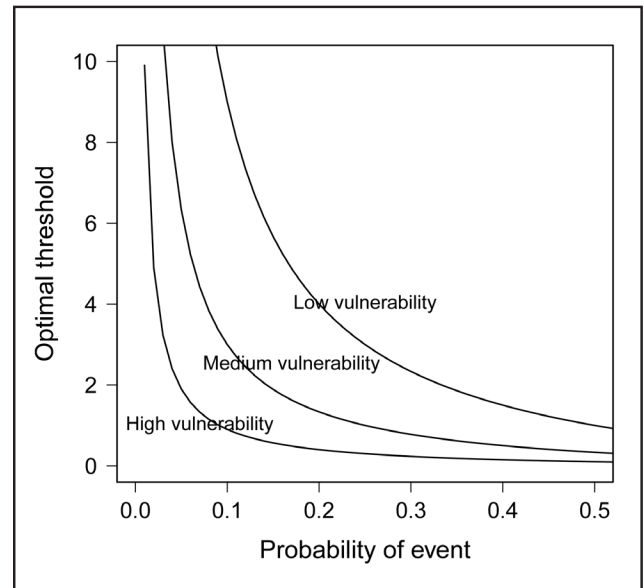
Values are generated from Equation 1, with the vulnerability term set at 0.1, 0.33, or 1.0. Increasing the probability of threatening events occurring always lowers the optimal threshold for responding, as does increasing the individual's personal vulnerability to threats should they occur.

The second prediction is that the relative costs of false alarms and misses (that is, the vulnerability) will affect the threshold. If misses are generally costly, as seems reasonable for the detection of mortal dangers ("few failures are as unforgiving as failure to avoid a predator"[32, p 619]) and the cost of a false alarm is only some unnecessary vigilance and increased heart rate, then an optimally functioning system will produce many times more false alarms than misses. This is Nesse's smoke detector principle[28,29]; when they are set correctly, smoke detectors go off when there is no fire moderately often, but never miss a real fire. Setting the threshold higher may be nicer for residents of the building, who would not have to troop outside for no reason so often, but in the longer term it would be suboptimal, as one missed fire is a catastrophe.

More specifically, though, any factor which affects the individual's ability to cope with the danger, and which thereby increases the likely cost of a miss for that individual, ought to cause that individual to lower their threshold accordingly. From Figure 2, we can see that an individual with higher vulnerability has a lower optimal threshold for any given level of probability than a less vulnerable individual. For example, this means that animals ought to have a lower threshold for mounting an anxiety response when they are lame than when they are healthy, and when they are in an environment with no cover, compared with one with cover. There is certainly evidence for such effects, both from the laboratory and the field. In the laboratory,

Table 1 Symptoms of anxiety and their function in defence against potential threats

Symptom	Functional significance
Easily startled, hypersensitive to noise	Response to threat easily evoked
Insomnia	Constant alertness
Restlessness, increased heart rate	Body is prepared for action
Preferential attention to cues related to threats	Notice threats sooner
Interpretation of ambiguous information as threatening	Reduce probability of missing possible threats
Ambiguity aversion	Avoidance of situations whose threat level is unclear

placing animals in isolation or exposed settings produces increased ALR, including cognitive changes toward interpreting ambiguous stimuli as negative.[24,33] In the field, individuals who lack cover, are far from conspecifics or at the margins of social groups, show increased vigilance.[34,35] Pregnant and nursing females are also more vigilant.[36,37] Presumably these effects are due to individual vulnerability in the event of an attack being elevated. Additionally, in reef fish *Chromis dimidiata* and *Pseudanthias squamipinnis*, individuals who have had access to a cleaner wrasse have an attenuated stress response, compared with individuals who have not.[38] The interpretation of this finding is that cleaner wrasse, by removing ectoparasites, keep the fish in better overall health. Better health means their ability to cope with subsequent threats is greater, and thus, in our terms, their vulnerability is lower. Thus it makes sense that their threshold for mounting an ALR would be higher.

In summary, we expect an ALR optimized by natural selection to exhibit the following features.

1. Its ease of evocation would not be fixed but rather modulated, depending on the individual's environment and current state.
2. It would be more readily evoked in environments where threats are more common (that is, where the probability is high).
3. It would be more readily evoked when individuals are in a condition that means they will find it difficult to cope with undetected threats (that is, where the vulnerability is high).
4. It will consist of a suite of physiological, cognitive, and behavioural changes that divert the individual's resources from other concerns, and facilitate the detection of and response to threats.
5. Even when functioning optimally, the system will produce many more false alarms than hits, and the greater the probability and vulnerability, the greater the ratio of false alarms to hits will be.

There is some support for at least the first 3 of these propositions from nonhuman examples. To what extent, though, do they help us understand anxiety in humans?

Application of the Evolutionary Approach to Anxiety in Humans

Let us assume that human anxiety is the homologue of the ALR found in many other species and that it serves the same function, to detect and prepare for threats. How does what we know from the psychiatric literature fit in with this assumption?

Implications for Symptoms of Anxiety

Many symptoms of human anxiety, although unpleasant, make functional sense in the terms we have outlined (Table 1). At the cognitive level, vigilance is increased, threatening information is given processing priority,[39] and ambiguous information is interpreted as threatening[40] because threats are judged as more likely to occur[41,42] (though see Nesse and Klaas[43]). The functional interpretation of these changes is obvious. We make a more specific claim: anxiety disorders, specially, GAD, can be equated to having a low threshold in the signal detection model. This makes sense of many GAD features, which are basically extreme forms of the normal anxiety response features outlined in Table 1. The DSM-IV specifies that the anxiety must be characterized as excessive for a period of at least 6 months.[44] The excessive in this condition is hard to operationalize exactly, but it could be taken to mean the presence of threat-related cognition with an unusually high proportion of false alarms. Such a high proportion follows directly from having a low threshold. The other GAD symptoms—for example, sleep difficulties, tension, and poor concentration—all follow straightforwardly from the person's threshold for threat detection being set so low that almost any information from the external environment becomes significant to provoke a response. We side-step the question of whether the threshold is low owing to system malfunction—pathology—or that being the best threshold for that person's current life situation. No doubt it varies from person to person. Moreover, we ought to expect there to be both traitlike (that is, temperamental) variation in where people's thresholds are, and within-individual change in thresholds according to their current life situations. Nonetheless, GAD's suite of symptoms is coherent and can be linked to having a low threshold in the signal detection model.

Implications for Epidemiology

If anxiety has the functions suggested for the ALR in our section Function of ALRs in Other Species, and if the signal detection model is a good framework for understanding its design, we can make clear predictions about the epidemiology of anxiety. These really all come down to 2 claims: as the objective probability of threats increases, so should the severity of anxious symptoms, and as the vulnerability of people to threats, should they occur, goes up, so should the severity of anxious symptoms. Many of the specific epidemiology patterns observed in the literature can be related to these 2 claims (Table 2). We find the evolutionary framework useful in that a large number of empirically derived associations presented in the literature

Table 2 Epidemiologic associations with anxiety that can be interpreted from the standpoint of the signal detection model		
Pattern	Reference	Interpretation
Low birth weight predicts higher trait anxiety later in life	Lahti et al[45]	Higher vulnerability: low birth weight individuals in poorer somatic state and have higher lifelong vulnerability to mortality and morbidity
Low socioeconomic position associated with greater anxiety	Najman et al[46] and McMillan et al[47]	Higher probability and vulnerability: low socioeconomic position characterized by more bad events, and fewer resources to cope with them when they occur
Injuries or disabilities causing loss of physical mobility associated with increased anxiety (and this seems to be independent of the association between depression and physical conditions[48])	Cano et al,[49] Suh et al,[50] Lenze et al,[51] and Bellin et al[52]	Higher vulnerability: loss of mobility reduces people's ability to deal with threatening situations
Members of discriminated-against minorities have less anxiety when living in neighbourhoods with more people of the same ethnicity	Das-Munshi et al[53]	Higher probability: probability of threats higher for minority individuals suffering discrimination when there are few others nearby
High absolute levels of anxiety in Zimbabwe, Central African Republic, and Gaza Strip	Langhaug et al,[54] Vinck and Pham,[55] Elbedour et al[56]	Higher probability: high rates of violence, unrest and disease mean probability of mortal threat is high in these environments
More anxiety among women than men	Breslau et al[57]	Higher probability and (or) vulnerability: greater level of threat and (or) vulnerability to threats among women than men
Anxiety associated with living alone or being a lone parent	Rimehaug and Wallander[58]	Higher vulnerability: threats more difficult to cope with in the absence of social support
More anxiety among women suffering domestic violence	Tolman and Rosen[59]	Higher probability: prevalence of physical danger is higher for such women

as unconnected all emerge from the general framework in a coherent way. Some of these are obvious and would have been predicted anyway, but others, such as the strong effect of loss of physical mobility on anxiety, are not intuitive until one adopts the evolutionary view.

Implications for Therapy

We believe that the framework outlined here could be useful for developing novel approaches to treatment, or at least refining existing ones. The effects of actual levels of probability and vulnerability must be mediated by a person's appraisal (conscious or otherwise) of their probability and vulnerability.[60] This means that relief of anxiety can, in principle, come in 2 ways; reducing people's objective probability and vulnerability, and changing their appraisals thereof, which may or may not be realistic (for a related discussion, see Nesse and Ellsworth[61]).

Some existing therapies can work by changing appraisals of probability–vulnerability. Cognitive therapies[62] for anxiety may address either the vulnerability or the probability term of Equation 1. Recalibrating either probability or vulnerability ought to result in raising the threshold for evocation of anxious symptoms. Exposure therapy is a long-established and often effective framework for treating specific phobias, which involves the controlled exposure of the patient to the source of the anxiety.[63] Exposure therapy may be having its effect by recalibrating the person's estimate of the probability of a specific threat occurring; if the patient can handle a dog many times, and

never experience it responding with threatening behaviour ("repeated disconfirmation" is a term used to describe the therapeutic methodology in this source"), then their best estimate of the probability term in Equation 1 for the case of dogs will be much lower. Their threshold for the evocation of anxious responses when dogs are in the environment should thus be raised, and symptomatic relief follows.

Drug treatments for anxiety disorders work by down-regulating threat detection mechanisms pharmacologically. An interesting possibility raised by our analysis is that drugs that treat nonanxiety-related symptoms that feed into perceived vulnerability, such as, for example, providing good analgesia to someone with chronic pain, may also reduce symptoms of anxiety.

The perspective outlined here draws attention to the fact that at least some people may be anxious because they are correct to be anxious. That is, their appraisals of high probability and vulnerability may be relatively realistic, and their symptoms, although unpleasant, reflect an adaptive response to this situation. Thus, on a holistic view, it is a priority to address ecological issues, such as poverty, economic insecurity, lack of social support, poor housing, and urban safety, as well as challenging people's cognition. These ecological factors are strongly associated with mental distress at both the individual and the societal level.[64] Tackling these factors will shift the population distributions of experienced probabilities of and vulnerabilities to threats, and thus ought to have population-level benefits regarding lower rates of anxiety.

If we accept that some cases of clinical anxiety may represent adaptive responses to the situations in which people find themselves, this raises an important question about the consequences of treatment. If the treatment involves reduction of actual levels of probability or vulnerability, this is fine, because reduced anxiety is appropriate in these circumstances. However, if cognitive or pharmacological treatment involves a change in a person's appraisal of the levels of probability or vulnerability without a change in the real levels, then there is a danger that this could increase the chances of a miss, with dire consequences for the person. Removing the battery from a smoke detector may seem like a good solution to frequent false alarms, until a real fire hits. The very fact that evidence is starting to accumulate that anxious people are less likely to suffer various forms of mortality should at least make us question whether there may sometimes be huge costs to inappropriately treating anxiety disorders.

Conclusions

We have outlined a simple functional framework for understanding human anxiety and homologous responses in other species. An individual's threshold for mounting a threat response ought to be sensitive to the likelihood of bad things happening in the environment (probability), and the individual's ability to cope if they do happen (vulnerability). If either increases, the individual anxiety response should be more easily triggered, and should therefore produce a greater number of false alarms. Epidemiologic patterns of anxiety symptoms can be interpreted within this framework, and existing treatments for anxiety disorders can be understood as reducing the patient's appraisal of either their probability or vulnerability.

The evolutionary perspective we have outlined does not immediately change what psychiatrists do. However, it does bring a broader biological lens to bear on a complex issue that is associated with a huge burden of human suffering. It raises the potential for integration between psychiatric research and the rich traditions of research in behavioural ecology and ethology. It may lead to fresh, more holistic public health approaches to anxiety prevention, and stimulate further research by promoting the exchange of ideas between scientists working on research topics as diverse as epidemiology, neurobiology, clinical psychology, and animal behaviour. In particular, thinking in evolutionary terms draws attention to the way high levels of anxiety may be associated not only with neural malfunction but also with genuinely dangerous or precarious life situations. That is, to understand behaviour, one must consider what is not only inside the organism but also going on in the organism's immediate environment.

Acknowledgement

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Résumé : L'anxiété : une approche évolutionnaire

Les troubles anxieux comptent parmi les maladies mentales les plus répandues, et une intense souffrance les accompagne. Les traitements actuels ne sont pas universellement efficaces, ce qui suggère qu'il faut une compréhension plus profonde des causes de l'anxiété. Pour mieux comprendre les troubles anxieux, il est d'abord nécessaire de comprendre la réponse normale à l'anxiété, ce qui veut dire examiner sa fonction évolutionnaire de même que les mécanismes qui la sous-tendent. Nous alléguons que la fonction de la réponse humaine à l'anxiété, et des homologues d'autres espèces, est de préparer la personne à détecter et à traiter les menaces. Nous utilisons un cadre de détection des signaux pour montrer que le seuil d'expression de la réponse à l'anxiété devrait varier selon la probabilité que des menaces se produisent, et selon la vulnérabilité de la personne à ces menaces si elles sont présentes. Ces prédictions concordent avec les principaux modèles de l'épidémiologie de l'anxiété. Les implications pour la recherche et le traitement sont discutées.