

In the first chapter, Hagop Akiskal and Olavo Pinto make a case for “bipolar spectrum disorder” which includes not only patients with classic mania, but also depressed patients who also have subclinical elevations of mood, or bipolar family histories, or who have “manic” types of depressed symptoms such as racing thoughts and hypersexuality. Whereas true bipolar disorder has an incidence of 1%, bipolar spectrum disorder affects 5% of the population. According to current practice, many of these patients get diagnoses of unipolar depression or borderline personality disorder. The authors claim that bipolar spectrum disorder responds to mood stabilisers rather than antidepressants, and so it is clinically important that these patients should be recognised and properly treated.

How does this relate to our idea of affective disorders being based on the agonistic strategy set of escalation and de-escalation (Gardner & Price, 1998)? We have suggested that elevation of mood represents the inappropriate or excessive deployment of an escalating strategy, whereas depression represents the de-escalating strategy. Escalation is a strategy which increases the chances of winning in competition, but at the expense of increasing the costs. De-escalation reduces both costs and chances of winning.

The idea of a strategy set which contains the alternative strategies of escalation and de-escalation is borrowed from behavioural ecology (Krebs & Davies, 1993). The strategy set is typically accessed by ranking stress, at a time when either elevation of rank or loss of rank would be appropriate, but the environmental indicators are not sufficiently clear as to whether there should be the increase of investment necessary to bring about a rise of rank, or whether a reduction of investment in the form of depression would result in a better overall payoff. As with other strategic choices, the factors which determine the choice of strategy are different from the factors which cause the strategy set to be accessed. What seems to have evolved in this situation is a randomised choice between elevation and depression of mood. This fits with what we know of bipolar disorder. There may be elevation or depression of mood, but we cannot predict in which direction the mood change will go.

According to the behavioural ecologists, the randomisation is not entirely random, but rather each individual has a probability of choosing escalation (or de-escalation), and this probability could be genetically determined or contingent on some factor during development. For instance, the occurrence of secure attachment during early life might result in a higher probability of escalation. The same could be true of bipolar patients, who might have a genetically determined probability of developing mania rather than depression, or this probability could be influenced by developmental factors.

Having said this of bipolar disorder, what does our theory say of unipolar depression? Here we should point out an important difference noted by behavioural ecologists about the evolution of the agonistic strategy set. There are two separate situations which would lead to the evolution of de-escalating strategies (which appear counterintuitive because they involve the giving up of resources). The strategies can be either “mixed” or “contingent”. The mixed strategies are described by the classic hawk/dove game, in which a dove always de-escalates and a hawk always escalates, and each individual has their own probability of choosing hawk and dove. Hawks succeed because they win more, doves succeed because they avoid destructive fighting. The strategy set evolves to an equilibrium, called an “evolutionarily stable strategy” or ESS, in which each strategy (defined as a probability of escalating on any particular occasion) has equal fitness.

However, there is another situation in which a de-escalating strategy could evolve, and this is what the behavioural ecologists call a contingent strategy, or more informally, a “best of a bad job” strategy. Say one is fighting a rival, and suddenly he is joined by three friends: there is an indication for a change of strategy, but there is not much point in adopting an escalating strategy, and so a switch to a de-escalating strategy is likely to give the better contribution to fitness. In other words, our protagonist needs to run like hell. Or the choice of strategy may be “phenotype dependent”: say, for instance, our protagonist is competing for social success at a party, and then looks in the mirror and sees that she has a huge pimple on the end of her nose, she is likely to de-escalate and adopt a less self-assertive profile. If, for any reason, you realise you are not going to do well in the arena, it makes evolutionary sense to de-escalate and remain out of the “action”.

This “contingent de-escalation” would seem to be an appropriate model for unipolar depression. There is never any likelihood of escalation. Either environmental adversity or one’s own deficiencies are so overwhelming that the possibility of a choice of strategy does not arise. De-escalation is the only answer.

Therefore most of the unipolar depressed patients one sees are adopting de-escalation as a “best of a bad job” strategy. Some, of course, will be operating a mixed strategy, but for some reason (chance) they have not yet plumped for the escalating option. They may have a very low inherited (or environmentally determined) “probability of escalation”. Some people may even have a probability of zero, and it would be difficult to distinguish these people from “best of a bad job” strategists.

I think our model has some contribution to make to understanding affective disorders:

1. It draws attention to two types of “stressful” situation which may cause the agonistic strategy set to be accessed. One type is “bad news” to which the only logical response is de-escalation, and this we predict to be the type of stress which elicits unipolar depression. However, another type of stress is likely to elicit bipolar disorder. This is the ambiguous situation, in which it is not possible to predict whether it would be better to invest more or to cut one’s losses. There may be advantage in either escalation or de-escalation, and only natural selection will decide whether the right strategy was chosen.
2. It draws attention to the ratio of episodes of mania to episodes of depression which may be genetically determined or affected by developmental factors. This ratio may turn out to be more heritable than the occurrence of bipolar disorder.
3. It emphasises the possible importance of randomisation in choice of strategy. In whatever way the ratio of mania to depression may be determined, on any one occasion the choice of strategy is likely to be randomised.
4. By postulating different mechanisms for unipolar and bipolar disorder, it supports the idea that they may respond to different treatments.

Mixed states

The next two chapters concern mixed states, in which the features of depression and mania occur at the same time. The authors make the case that this category might include agitated depression, which they suggest responds better to neuroleptics or mood stabilisers than to antidepressants. They suggest that mixed states may result when depression occurs in a person of manic temperament, or when mania occurs in a person of depressive temperament.

In the case of mixed states, too, our model may be helpful. We have suggested that escalation and de-escalation may occur at any of Paul MacLean's three brain levels, and that in the case of mood disorders we are seeing escalation and de-escalation at the lowest level of the forebrain, what MacLean called the reptilian brain, anatomically situated in the basal ganglia. But it is possible that a depressed person, de-escalating in the reptilian brain, has chosen to escalate at the level of the paleomammalian brain situated in the limbic system. This would lace the depressive state with the escalating emotions, particularly irritability and anger; there might also be an energy contribution from limbic escalation, giving rise to agitation and/or racing thoughts.

Likewise, a manic patient escalating at the reptilian level might choose to de-escalate at the limbic level, and this would add depressed emotion to the manic mood, resulting in tearfulness, shame and guilt.

It might be profitable for researchers into mixed states to bear these possibilities in mind, in case they might account for a proportion of their cases.

Psychotherapy

A chapter on psychosocial interventions reports the high rate of non-compliance with treatment, which is due in wealthy patients to side effects such as tremor and weight gain, and in poorer patients to denial of illness. Even though bipolar disorder is more respectable than schizophrenia, it still carries a stigma. The best way to ensure compliance is to develop a good therapeutic relationship. Four manual-based techniques are outlined.

They report two longitudinal prospective studies which found that patients returning to high expressed emotion (EE) homes were "five times more likely to relapse over a 9- to 12-month than those whose families exhibited low levels of EE." Since EE is a construct consisting of criticism, hostility and overintrusiveness on the part of family members, it does not seem surprising that patients subjected to this hostility might find themselves accessing their agonistic strategy sets more often than was healthy.

Conclusion

Not only was Hagop Akiskal editor of this volume, and first author of the first chapter, he was also co-author of three other chapters. This represents a major achievement on his part. He is doing here for bipolar disorder what he previously did for dysthymic disorder - putting it on the map. His idea of "soft" bipolar disorder (or bipolar spectrum disorder) without clinical attacks of mania is an improvement on the numerical classification. And if these patients turn out to respond selectively to mood stabilisers, a lot of patients now labelled as anxious, obsessional, phobic, addicted or borderline personality disorder are going to benefit. There is a strong case for a prospective randomised trial, and this volume makes the case clearly.

Gardner, R. Jr. & Price, J.S. (1999) Sociophysiology and depression. In: The Interactional Nature of Depression: Advances in Interpersonal Approaches (eds. T. Joiner and J.C.Coyne). Washington, DC: APA Books. Pp. 247-268.

- Krebs, J.R. & Davies, N.B. (1993) An Introduction to Behavioural Ecology, 3rd edition. Oxford: Blackwell Scientific Publications.

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