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Editorial

A model for self-treatment of four sub-types of symptomatic ‘depression’ using non-prescription agents: Neuroticism (anxiety and emotional instability); malaise (fatigue and painful symptoms); demotivation (anhedonia) and seasonal affective disorder ‘SAD’

S U M M A R Y

This article will present a model for how ‘depression’ (i.e. depressive symptoms) can be divided into four self-diagnosed sub-types or causes which might then be self-treated using agents available without prescription. (Another, much rarer, cause of depressed symptoms is the classical illness of ‘melancholia’, which when severe cannot be self-treated and typically requires hospitalization.) A self-management option and alternative is now needed due to the an inappropriate emphasis of modern psychiatry on treatment of imprecise syndromal ‘disorders’ which may entail treating ‘depression’ at the cost of making the patient feel and function worse. By contrast, the basic theoretical stance of self-management is that depressed mood should be seen as a result of unpleasant symptoms – and it is the symptoms that require treatment, not the mood itself. Furthermore, drugs (or other interventions) need to be classified in terms of their potential therapeutic effects on these symptoms that may cause depressed mood. The four common causes of depressed mood considered here are the personality trait of Neuroticism; the state of malaise (fatigue, aching etc) which accompanies an illness with an activated immune system; demotivation due to lack of positive emotions (anhedonia); and the syndrome of seasonal affective disorder (SAD). Each of the four sub-types is then ‘matched’ with a first-line non-prescription agent. The ‘stabilizing’ agents such as St John’s Wort and the antihistamines chlorpheniramine and diphenhydramine are used for treatment of Neuroticism; analgesics/pain killers such as aspirin, ibuprofen, paracetamol/acetaminophen and the opiates are used to treat malaise; energizing agents such as caffeine and nicotine are used for the treatment of demotivation; and bright light used in the early morning to treat SAD. Self-treatments are intended to be used after research and experimentally, on a trial-and-error basis; with self-monitoring of beneficial and harmful effects, and a willingness to stop and switch treatments. The model of S-DTM (self-diagnosis, self-treatment and self-monitoring) is suggested as potentially applicable more widely within psychiatry and medicine.

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Introduction

‘Depressive disorder’ and ‘anti-depressant’ are categories that should be discarded

The gross imprecision of the diagnosis of ‘depression’ has become farcical in recent decades, when the supposed prevalence of ‘depression’ has risen from a fraction of a percent by about a hundred-fold to anything from ten to twenty-five percent [1,2]. Nowadays, any person suffering a persistent unpleasant emotional state may be officially diagnosable as depressed, and treated with drugs termed ‘anti-depressants’.

I have previously argued that the disease category of mood (affective) disorder called depression is neither coherent nor useful; and instead it would be preferable to regard ‘depressed mood’ as secondary to a variety of unpleasant emotional states [3]. In other words, depressed mood should be seen as caused by symptoms and emotions – for example anxiety, fatigue or lack of positive emotions (anhedonia) can all lead to depressed mood. Diagnosis and treatment of ‘depression’ should therefore be focused on the emotional states which cause depressed mood, and not upon treating a vaguely-defined – hence over-inclusive – syndrome termed ‘depressive disorder’. In principle there might be an unbounded number of causes of negative, depressed states of

unhappiness – in practice, I will focus upon four which are apparently amenable to improvement by therapeutic intervention.

I have also argued that the term ‘anti-depressant’ should not be used, since there are no drugs which have a general action to alleviate depressed mood: what the effective drugs are really doing is to alleviate the causes of depressed mood [3]. There are a variety of different drugs types which can alleviate some symptoms that may lead to depressive symptoms in some people. For example, when anxiety is causing depressed mood then any drug which reduces anxiety (including alcohol, neuroleptics/antipsychotics, benzodiazepines or selective serotonin-reuptake inhibitors – SSRIs) may all (for a while) alleviate ‘depression’. But when a person’s depressed mood is not caused by anxiety then these same drugs could be ineffective or may actually worsen the depressed mood.

I believe that a self-management option and alternative [4] is now urgently needed (at least in the UK and USA) due to the incorrect and counter-productive theoretical stance of modern psychiatry [3], the corruption of modern psychiatry by industrial and political influences [2], and the inappropriate emphasis of modern psychiatry on treatment of syndromal ‘disorders’ [3,4]. This focus on syndromes may lead modern psychiatrists to treat ‘depression’ at the cost of making the patient feel and function worse [5].

This is the rationale and justification for the following article, which represents a personal view – speculative and tentative – of

a possible future for psychopharmacology in psychiatry, specifically in relation to negative symptoms of ‘depression’ such as sadness unhappiness, lack of motivation, long-term miserable anxiety, unpleasant mood swings and the inability to feel happiness. My hope is that these ideas are sufficiently accurate and valid to be useful and applicable – but also that they will stimulate discussion and serve as a basis for a process of evolution and improvement.

By extension, this general model of self-diagnosis, self-treatment and self-monitoring (S-DTM) could potentially be extended to other areas of psychiatry and medicine in which symptoms are the focus and where effective treatments are available without prescription. Indeed, as well as being used to alleviate negative states, the model is also applicable to lifestyle/quality of life enhancement [3,5].

Imprecise diagnosis and treatment of depression

I believe that, on the one hand, the treatment of depression can be more specific and effective than at present; but on the other hand it is also correct that the psychoactive drugs are all imprecise in their effects, and in particular tend to affect different people differently. This means that psychiatric treatment (whether self-treatment or treatment by professionals) is almost inevitably a trial-and-error matter, and should be embarked-upon in an experimental spirit.

Psychiatric drugs (and also some other psychiatric interventions such as electroconvulsive therapy and perhaps bright light) tend to be non-specific in relation to traditional diagnostic syndromes [3]. Different categories of drugs such as ‘antidepressants’ and the neuroleptics/antipsychotics often have over-lapping therapeutic effects, side effects and indications – mainly because many of the most-used drugs were chemically-developed from a relatively small number of coloured dyes which were initially made into antihistamines during the 1940s then further modified over the following decades to make the neuroleptic/antipsychotics, tricyclic and SSRI antidepressants [1,6,7].

So, drug recommendations for symptomatic treatment in psychiatry are mainly about suggesting which drug to try first. There needs to be an attitude of trial-and-error; with self-monitoring of the effects of treatment, willingness to change to stop treatment or change to another treatment if the first choice has undesirable side effects or is apparently ineffective.

With these cautions in place, I see no compelling reason why people should not self-treat for psychiatric symptoms using drugs which are available ‘over the counter’ and without prescription. After all, in a country such as the UK or the USA people in their tens of millions already self-treat for headaches and back pains, constipation and diarrhoea, runny noses and blocked noses, hay fever and eczema, high cholesterol, skin infections and duodenal ulcers. And in a world where it is common to assert that anything up to half the population have significant psychiatric symptoms of some sort (e.g. depression, anxiety states, various phobias and compulsions, insomnia) then self-treatment become a practical necessity.

Furthermore, I suggest that symptomatic self-treatment for ‘depression’, when done by careful and informed people, might well be superior to the average treatment on offer from psychiatric professionals. The main constraint is the limited range of drugs available without prescription (especially, see below, in the case of demotivated depression); but this restrictive public policy may change over time or be circumvented by the increased ease of purchasing pharmacological agents without prescription.

Self-diagnosis by introspection – the ‘phenomenological’ approach

The process by which self-diagnosis may be accomplished requires some elucidation. I have previously termed the sequence

S-DTM – meaning Self-Diagnosis, self-Treatment and self-Monitoring. The aim is to introduce to self-management a helpful degree of thoroughness and formalization to make the process both safer and more effective than unstructured self-management.

The first step involves developing self-awareness of symptoms. The word ‘phenomenology’ refers to the process of introspection or inward-looking by which a person can become aware of their inner, subjective states – psychiatric symptoms are one of the body states which may be accessible to such introspection [3,8,9]. To self-diagnose by introspection requires a skill which may be unfamiliar. For example, it is possible to be anxious but unaware of the anxiety [10,11]. To become aware of anxiety as a feeling, a person needs to be able to identify their own state of mental angst, muscular tension, rapidly beating heart, sweatiness, ‘butterflies in the stomach’ and so on.

Furthermore, inner states must be identified in terms of a system of classification – because body sensations tend to be experienced as formless and undividedly ‘holistic’ unless there is a systematic classification which can describe them. Without some such analytic scheme, it may not be possible for someone to be aware of, and to express even to themselves, much more than a simple dichotomy of feeling either ‘good’ or ‘bad’. Self-treatment, however, requires that different types of ‘feeling bad’ can be distinguished and identified.

In terms of ‘depression’ – the process begins with recognition of a depressed mood, in other words a negative or unpleasant mood state which could be characterized by some kind of unhappiness. Then there is a further introspective process by which the sufferer tries to identify some inner physical, bodily state which may be the main cause of this unhappiness. The assumption is that if this causal symptom can be alleviated or eliminated then the person may become happier.

Happiness is not necessarily entailed by removing the cause of unhappiness, but it is easier and more probable that a currently-unhappy person will become happy if they are relieved of unpleasant symptoms. For example, it is hard to be happy when suffering a headache and relief of the headache may therefore cause a person to become happy who would otherwise have remained miserable.

More exactly, there is an attempt to match-up inner states against a pre-determined classification. Four body states which may cause unhappiness include emotional instability with anxiety (Neuroticism); fatigue and bodily aches and pains (malaise); lack of emotion – especially loss of the ability to anticipate future pleasures (demotivated depression); and sleepy, hungry, irritable mood specifically during the winter season (SAD).

Having identified a particular aversive body state as a probable cause of depressed mood, this symptom is then made the focus for self-treatment; and the symptom is monitored for its response to treatment. A treatment agent or mode is selected as being both safe and potentially able to alleviate the specific symptom, and a trial of this treatment is made. So, if the symptom underlying depressed mood is identified as anxiety and unstable emotions then stabilizing drug is chosen (such as St John’s Wort or chlorpheniramine – see below); and the symptom is monitored to see whether it responds to this treatment.

The self-diagnosis, -treatment and- monitoring (S-DTM) model [4] treating depressed mood pharmacologically

Self-diagnosis

1. Recognition of a depressed, unhappy, low mood.
2. Introspective self-diagnosis of the sub-type of symptomatic and emotional cause of depressed mood.

3. Matching the symptoms and emotions to one of the four sub-types of 'depression'.
4. Matching the sub-type of depression to the drug class which is most likely to alleviate those symptoms and emotions.
5. Researching the scientific literature on the effects, side effects and possible interactions of the drug class – and choose a (probably) safe first-line agent.

Self-treatment

6. Begin trial of treatment.

Self-monitoring

7. Very careful monitoring for effects and side effects for the first 4 hours after taking the agent, and continued vigilance for several days. Keep a record. (e.g. Consider self-monitoring blood pressure when using psychostimulant type drugs.)
8. If immediate problems of side effects or feeling worse after taking a drug, consider stopping immediately – or continue with vigilant self-monitoring.
9. If no benefit at all after a few days consider increasing dose or stopping and trying another agent.
10. If side effects are bad, or there is concern over dependence, or if unsure about whether or not the drug is having benefit, or if wanting to stop taking the drug; consider stopping the drug and self-monitoring the result of stopping – then consider restarting and monitor the results of restarting.
11. Go through the process for each new drug tried. Avoid interactions between the drug stopped and a new one started, and between multiple agents.

Four sub-types of self-treatable depression

I will consider four sub-type causes of depressed mood ('depressive disorder') which may be suitable for self-treatment: these are Neuroticism, Malaise, Demotivation and Seasonal Affective Disorder-SAD. I will also refer to a fifth type of depressive disorder - Melancholia - which was the original type of depression recognized for centuries, and is often too severe and debilitating to be self-treated and for which the best treatment (electroconvulsive therapy) cannot be self-administered.

This list of five sub-types is not exhaustive, and there almost certainly are other well-defined syndromes that are causes of depressed mood (or these four sub-types may fruitfully further be subdivided), and these might require different treatment, or treatments that are not available without prescription, but probably those sub-types described here are the commonest.

So, my suggestion is that sustained depressed mood (i.e. so-called depressive disorder) is 'caused' by least five more – specific sub-types. Naturally, each of these sub-types must have its own cause. Typically this cause is unknown or uncertain – and I will not consider the matter further here; because – whatever their cause may be – each sub-type has somewhat different symptoms and there are relatively specific treatments which have the potential to alleviate these symptoms.

I further suggest that there is no general purpose 'antidepressant' action of a drug. Instead of there being 'anti-depressants', in actuality there are several types of intervention which alleviate different unpleasant symptoms and emotions, and which may as a result make people feel less depressed. A drug which alleviates depression in one person may actually cause depression in another person because the effect on depression is secondary to the effect

on the symptoms or emotions. In what follows, drugs are classified according to their effect on symptoms; drug types considered here include stabilizing drugs; analgesics/pain killers and energizing drugs.

Melancholia – not self-treatable

Probably it is best to note and set-aside the 'melancholia' type of depression at this point. Melancholia is probably best described in textbooks from at least thirty years ago, before the diagnosis of depression became over-inclusive [12]. This is the classic, severe, debilitating form of 'endogenous' depression which may have psychotic features such as hallucinations, delusions, thought disorder, catatonia and psychomotor retardation.

Melancholia typically renders the sufferer incapable of work with severely-diminished or absent self-care and often suicidal tendencies. Subjectively, the mood state may be one of profound sadness, despair, emptiness, guilt, nothingness – speech and movement are slowed to near inertia, appetite may be absent, and death by starvation is a possibility.

Patients usually require admission to a hospital or similar institution for the treatment of melancholia – and they may require close supervision to prevent suicide. The episode of illness usually lasts for several months and the most effective treatment to improve symptoms is electroconvulsive therapy/electroshock therapy (ECT/ECS) [13,14].

Neuroticism

Anxiety is a normal, evolved human emotion which functions to increase alertness and avoid harm. However, anxiety is almost certainly the most frequently-experienced psychiatric symptom, and anxiety and depression are major feature of the 'neurotic' personality type characterized by emotional instability.

Neuroticism is one of the 'Big 5' personality traits, and was derived from the work of Hans Eysenck [15,16]. Neuroticism is an underlying disposition which is substantially hereditary and tends to endure throughout life. The personality type extends from high Neuroticism with extreme unpleasant mood swings at one extreme, to emotional stability at the opposite extreme. Other aspects of high Neuroticism include guilt feelings, low-self esteem, irrationality, shyness, moodiness and emotionality. Low Neuroticism personalities are described as emotionally stable, and display the opposite traits: calmness, cheerfulness, confidence.

I regard Neuroticism as more-or-less the same entity as Nutt's category of 'depression with anxiety' [17]; very similar to Neurotic Depression on the Newcastle Diagnostic Scale [12] and essentially the same entity as DSM IV dysthymic disorder [18]. Watson calls it 'negative emotionality' – the tendency to experience strong negative emotions [19].

Neuroticism is a kind of hypersensitivity to the environment, akin to feeling the hyper-vigilant state of being alone in an unfamiliar and threatening environment. The average level of Neuroticism is higher in women, and high Neuroticism may be commoner in modern mass societies [3].

Since it is a type of personality and not a disease, Neuroticism probably cannot be 'cured'. But severity of symptoms related to Neuroticism tend to wax and wane, probably in response to life stresses and also factors such as age, illness, drug usage etc. Given the ineffectiveness of psychotherapy and counseling, the psychiatric treatment of Neuroticism is essentially a matter of using drugs either to blunt exacerbations or else to promote long-term stabilization of emotions.

Because Neuroticism is a dispositional trait, emotion blunting drugs – when they work – are perceived to have caused a change

in personality – and such change in personality may be perceived either positively or negatively [20,21].

Stabilizing drugs for Neuroticism

The anxiety component of a personality high in Neuroticism can be treated using a variety of anti-anxiety agents (e.g. neuroleptics/antipsychotics, benzodiazepines, propranolol – and people may self-medicate with alcohol) but since the core problem is emotional instability then the more relevant classes of drugs seem to be those that stabilize by buffering or blunting emotions. I shall term these the class of ‘stabilizing’ drugs.

The most powerful emotion stabilizing drugs are the neuroleptics/antipsychotics; but these tend to blunt emotions to the point of blank inertia [7]. Indeed, the neuroleptic core effect is to induce Parkinsonism as a method of non-sedating behavior control – as implied by the name which means ‘nervous system-seizing’ (i.e. seizing and holding the nervous system, so it does not react) [22,23].

So assuming that people do not wish to suffer from self-inflicted Parkinson’s disease, neuroleptic/antipsychotics should be avoided and instead the most appropriate class of drugs for treating emotional instability are probably those which have serotonin-reuptake-inhibiting properties of which the class of selective serotonin-reuptake inhibitors (SSRIs) are the best-known and most widely-prescribed examples. These can buffer or blunt the strength of emotions [3] (Healy has termed them ‘serenic’ in their effect [2]) but without necessarily demotivating the individual. Indeed, the emotional stability induced by SSRIs might provide previously-Neurotic people with better focus and direction.

‘Over the counter’ versions of the SSRIs that are available without prescription include at least two of the drugs sold as ‘antihistamines’ [4]. These antihistamines were used as the basic molecules from which SSRIs drugs were manufactured [6,7,24–26]. (They were also the base molecules for the tricyclic antidepressants such as imipramine, and the earliest neuroleptics/antipsychotics such as chlorpromazine – consequently there are overlapping therapeutic effects and side effects among these drug classes [7].)

Diphenhydramine was the base molecule for synthesizing fluoxetine (‘Prozac’) which was the first SSRI to reach market [6]. Diphenhydramine is marketed as a sedative cough suppressant; and is probably an SSRI in terms of blocking reuptake of serotonin more potently than noradrenaline [24] (this is the pharmacological definition of an SSRI).

Chlorpheniramine was the base molecule for the synthesis of zimelidine; which was the first SSRI to be made but which never reached market due to its side effects [25,26]. Chlorpheniramine is sold as an anti allergy/anti-hay fever medication and is regarded as very safe; even being used in pregnancy for the treatment of nausea [27]. Chlorpheniramine blocks the reuptake of serotonin and also of noradrenaline [24], so is probably best regarded as a Serotonin and Noradrenalin Re-uptake Inhibitor (SNRI) resembling venlafaxine [26].

To support the use of these antihistamines in treating depressive symptom exacerbations due to Neuroticism there is the above strong theoretical argument plus a small literature of the beneficial effects of chlorpheniramine as an anti-anxiety drug and probably stabilizing agent (e.g. [28–30]) – evidence for the benefits of diphenhydramine is at present more theoretical and anecdotal. However, with a self-treatment approach using safe and non-prescription drugs, the evidence of effectiveness comes from personal experience – it is relatively easy to discover whether the drug ‘works for you’ since typically the benefits (and side effects) on the core symptom of emotional instability can be felt (or not felt) as soon as the drug is absorbed – i.e. within an hour or two. However, drug effects on mood are much more indirect and more

variable, and mood improvement may take days or weeks to emerge [3].

However, probably the best drug for producing emotional stabilization is the herb St John’s Wort/*Hypericum*. The evidence concerning the usage and value of this drug is conveniently gathered in an excellent Wikipedia survey [31]. According to the preponderance of randomized trials, St John’s Wort (SJW) seems to be the equal or superior of the SSRIs; in terms of equal or better therapeutic effectiveness, fewer unwanted side effects and greater drug safety. St John’s Wort has mainly been evaluated as an anxiolytic and/or ‘anti-depressant’; but my inference is that SJW is essentially an emotion stabilizing drug akin to SSRIs. SJW is available in measured doses without prescription from pharmacists and supermarkets, usually being sold as a food supplement alongside vitamins, minerals and other herbs.

In conclusion, an exacerbation of ‘depression’ due to Neuroticism may imply a first-line self-treatment with St John’s Wort, chlorpheniramine or diphenhydramine. Since Neuroticism is a personality trait, when stabilizing drugs are effective they produce a change in personality, and potentially may make the neurotic individual feel more positive than ever before – they may seem to themselves and others as if they are ‘better than well’ [20]. Alternatively, stabilizing drugs such as SSRIs in another individual, or too high a dose, might cause a ‘hardening’ of personality (making the person more indifferent to things which ought to be of concern) and this may cause a reduction in motivation and a reduced inability to enjoy life (anhedonia) [21].

Very rarely SSRIs (and other psychoactive drugs, such as neuroleptics) can provoke extreme unpleasant states of inner turmoil or suicidal feelings in predisposed individuals [2] – and this may be a feature of the chemical structure of stabilizing drugs [7].

Malaise

Malaise is a term I suggested in 2000 for a sub-type of depression which is underpinned by that state of exhaustion which is familiar as the effect (and persisting after – effect) of infectious disorders such as influenza or glandular fever [3,32]. Since this description, some of the main features of the Malaise theory of depression have been confirmed by further studies (e.g. [33–38]).

The main symptoms of malaise are fatigue, feeling physically ‘TAT’ (tired all the time – and by ‘tired’ is meant physically-exhausted rather than sleepy), a washed-out or drained sensation in the body and limbs, heaviness in the head or limbs, aching, headaches and low-grade pain or tenderness in trunk and limbs. Malaise corresponds to Kurt Schneider’s ‘vital’ symptoms of depression, which he regarded as being of primary diagnostic significance [39].

Depressed mood is the response to this state of malaise, so that malaise depression is primarily a problem of the body, and not necessarily the brain. The idea of malaise comes from a general recognition of ‘sickness behavior’ as the general behavior which is characteristic of a sick mammal (summarized in [32]). Sickness behavior is regarded as an evolved adaptation to acute infectious disease – a behavioral state that is energy-conserving, risk-minimizing and immune-enhancing to allow an all-out (but temporary) attack on invading micro-organism.

So, malaise is caused by activation of the immune system, and is associated with increased blood levels of immune chemicals called cytokines – eg interferons, interleukins, Tumor Necrosis Factors (TNFs) and dozens more types. There is considerable evidence of raised levels of cytokines in depression (e.g. [32,36,39]). But blood cytokines are typically also increased in autoimmune diseases (such as rheumatoid arthritis) and disseminated cancer – and these types of disease are also associated with ‘sickness behavior’ and malaise which can lead to depressed mood [3].

From anecdotal observation and general reading, I believe that sleep disruption is probably a common cause of malaise. Potentially there can be neurotransmitter and/or hormone changes triggered by sleep deprivation or sleep disruption. For example, malaise often follows sleepless nights, shift working or as an aspect of 'jet lag' due to crossing several time zones; or post-operative states with catabolism triggered by tissue destruction and sleep disruption; or following childbirth (with a combination of major hormonal and psychological changes, tissue damage and sleep disruption).

Since malaise is characterized by unpleasant, pain-like physical states, it follows that an appropriate treatment for malaise is with analgesic or pain killer drugs [32]. For example, painkillers often alleviate (to some extent) the aching and exhausted physical state associated with influenza or its aftermath.

Analgesics/pain killers for malaise

There is considerable anecdotal and indirect evidence to suggest that analgesics are effective in treating some types of depression. I am aware of one formal trial designed partially to test this hypothesis – which confirmed it [38].

However, the effectiveness of the traditional 'tricyclic' antidepressants (TCAs) in 'major depressive disorder' (which includes malaise symptoms in its definition) may be interpreted as being due to the drugs' analgesic properties [40]. Especially this applies to the effectiveness of amitriptyline, which has been the most widely-prescribed TCA for depression [1]; and which is also currently used in the treatment of cancer pain in terminal/palliative care, migraine etc. Furthermore, opiates (which are analgesics) have, at various times throughout history – most recently during the 1980s – been apparently successfully used in the treatment of depressive symptoms [3,32,41]. (By contrast, SSRIs probably do not have significant analgesic properties [3,40].)

When depressed mood is associated with a malaise state, there could be a trial of the various simple analgesics available without prescriptions: aspirin, ibuprofen, paracetamol/acetaminophen and the mild opiates such as codeine or dihydrocodeine. Either aspirin or ibuprofen can also be combined with paracetamol and/or an opiate. Individual responsiveness to these analgesics is variable, and so are the experienced side effects – so there may need to be a period of trial-and-error before concluding that analgesics are ineffective.

As when treating Neuroticism with stabilizing drugs; the analgesics/pain killers would be expected to have a rapid effect in alleviating malaise symptoms as soon as the drug has been absorbed – i.e. in just a few hours [3]. But because mood is not directly related to malaise symptoms, it may take days or weeks before a reduction in malaise symptoms leads to an improvement in mood. So even when malaise is alleviated with treatment, the mood may remain depressed for other reasons – perhaps due to other unpleasant emotions, or to circumstances or habit [3,9,32].

Demotivated depression

Demotivated depression is characterized by reduced positive emotions; and it is this inability or impaired ability to experience pleasure (i.e. anhedonia) that is the cause of demotivation.

Motivation is at root a product of the ability to feel current pleasure in anticipation of future situations – it is this pleasurable anticipation of future positive states of emotion which provides the immediate motivation needed for present action [3,10]. If one cannot experience pleasure, and if nothing seems likely to induce pleasure, then there will be a generalized loss of interest in life and its opportunities, and this will be experienced as a lack of vitality and drive (including reduced sexual drive).

Life usually involves a trade off between present and future pleasure and pains – normal people will often do something which

is worse in the here-and-now, if this leads to the prospect of something better in the future. Something as simple as making the effort to visit a friend is done in the expectation that the here-and-now inconvenience of walking or catching a bus will be compensated by the future pleasure of conversation. But if the thought of having a conversation with a friend does not lead to a here-and-now sense of pleasure (pleasurable anticipation), then the deterrent effect of the unpleasant aspects of walking or catching a bus will weaken the motivation to visit the friend.

Demotivated depression is therefore a concept derived from and almost identical with Nutt's 'Depression with loss of interest and energy' [17] and Watson's state of low positive emotionality [19]. Causally, demotivated depression may be an exacerbation of the personality trait of introversion (asocial, quiet, submissive, timid, avoidant) [15,16] or a sub-clinical state of early Parkinsonism [42]. Demotivation may be a consequence of taking certain types of drug – especially drugs that lead to a reduction in dopaminergic – or noradrenergic/norepinephric – activity in brain; agents such as the neuroleptic/antipsychotic drugs which block dopaminergic receptors [7,23]. The motivational system seems to involve mainly the dopaminergic neurotransmitter system, and this seems to interact with noradrenergic, serotonergic and cholinergic systems – among others [43].

It is important to recognize that demotivated depression would probably be made worse by stabilizing drugs which tend to blunt emotions – since stabilizers would blunt the positive emotions which are already deficient in demotivated depression.

Energizing drugs for demotivated depression

The suggested treatment of demotivated depression is with energizing drugs which enhance dopamine or norepinephrine actions – either directly or indirectly [44]. The classic examples of such drugs include the psychostimulants such as dexamphetamine or methylphenidate ('Ritalin'). Other energizing drugs include bupropion, monoamine-oxidase inhibitors such as phenelzine or moclobemide, amineptine, reboxetine, and the tricyclic desimipramine [17,44]. However, these drugs are only available with a prescription.

There are few energizing drugs which are available without prescription (probably due to fears of inducing addiction or dependence). The best-known and by far the most widely used energizers are caffeine and nicotine.

Caffeine [45] is found in coffee and tea and available in tablet form without prescription. It is a weak psychostimulant which increases alertness. Caffeine probably has properties as an analgesic or painkiller; and probably also has beneficial effects in preventing and perhaps treating Parkinson's disease (suggesting that caffeine acts like a dopamine agonist) [46,47].

Nicotine [48] is found in tobacco but is also available as a non-prescription drug (for example as lozenges, chewing gum, or skin patches). While nicotine works directly upon the cholinergic neurotransmitter system, it appears to have indirect effects as a 'dopaminergic' psychostimulant – it often increases energy and alertness and like caffeine (but with stronger evidence) seems to have both a preventive and therapeutic effect on Parkinson's disease [47–50].

In conclusion, the range of possibilities for self-treatment of demotivated depression with non-prescription drugs are at present both limited and somewhat speculative.

Seasonal Affective Disorder – SAD

Seasonal Affective Disorder (SAD) is winter depression or winter blues: low mood that occurs with greater frequency at more extreme latitudes (north or south) almost certainly due to the short daylight hours during winter months [51]. It is treated, not

with drugs, but with bright artificial light, usually administered in the early morning [52,53].

The typical symptoms of SAD include excessive sleeping (hypersomnia) i.e. still tired when waking in the morning and sleepy throughout the day; increased appetite with carbohydrate craving and weight gain; irritability; fatigue; reduced motivation and sociability. In other words, while sleepiness and carbohydrate craving with weight gain are somewhat distinctive to SAD; many of the symptoms of SAD overlap with the three other sub-types of depression (i.e. irritability overlaps with Neuroticism, fatigue overlaps with malaise, and reduced motivation and sociability overlap with demotivated depression).

It is therefore the seasonal pattern of depressed mood and the characteristic behaviors which are crucial for the diagnosis rather than the specific subjective symptoms being experienced. Another diagnostic factor is the rapid and profound improvement of these symptoms in response to exposure to bright early morning light – which makes the response to light treatment something of a ‘diagnostic test’ for SAD.

SAD needs to be distinguished from the increased seasonal incidence of malaise symptoms which would be expected during winter months due to the increased prevalence of infectious diseases in many parts of the world (especially upper respiratory tract infections such as colds and influenza). Indeed, discriminating SAD from malaise could be tricky, since in the first place the main symptom of malaise is physical tiredness or ‘fatigue’ while the main symptom of SAD is mental tiredness or ‘sleepiness’. In the second place there is no reason why a person should not simultaneously suffer from both malaise and SAD, therefore from ‘tiredness’ due to both fatigue and sleepiness.

Indeed, it is plausible that the circadian hormone disruptions which are plausibly associated with SAD might themselves lead to immune activation as a secondary consequence – so that SAD might precipitate malaise.

Bright light therapy for the treatment of SAD

Bright artificial light usually administered early in the early morning seems a very effective treatment for SAD [51–53] – this requires no prescription but only the purchase of a device delivering suitably bright light.

Especially-bright artificial light is needed to treat or prevent SAD because the aim is to simulate the kind of brightness that is provided by natural outdoor light. Normal indoor house lighting in a kitchen is only about 400 lux (there is even less light in bedrooms), while outdoors, even on a cloudy day, there is about 10 times greater intensity of light – 4000 lux.

Specialized ‘light boxes’ generate about 10000 lux of suitable-wavelength light at close range. This should be sufficient to cure SAD if administered for 30 minutes – however the subject must usually be sedentary and near to the light. A ‘light visor’ shines the light from much closer to the eye for about the same length of time, while allowing the subject to be mobile. ‘Dawn simulators’ are like an alarm clock that brightens up to about 400 lux over a period of about an hour – apparently these also seem to work for some people.

If a person has SAD, then bright early morning light will probably produce a marked improvement in their symptoms within just a few days and continued use of bright light therapy would probably prevent a return of SAD symptoms.

SAD is a syndrome

SAD therefore is an example of diagnosing and treating a syndrome; rather than the symptom based-management model as recommended for Neuroticism, malaise and demotivation.

Table 1

Four sub-types of ‘depression’ and first-line agents for their treatment

Sub-type	Emotions	Treatment
Neuroticism	Anxiety, unstable emotions	Stabilizing drugs
Malaise	Fatigue, pains	Analgesics/pain killers
Demotivated	Anhedonia – lack positive emotions	Energizing drugs
SAD	Winter seasonal symptoms	Bright morning light

The delineation of SAD is actually a tremendous success story of psychiatry within the past few decades. And interestingly, (although perhaps not surprisingly) this process of definition and development of treatments happened largely outside of the professional structures of modern psychiatry, presumably because bright light treatment is non-pharmacological and not patentable.

It is a significant paradox (and one which supports the need for self-management in psychiatry) that probably the most valid and most effectively-treatable of recently-defined psychiatric syndromes arose mostly outwith the ‘official’ field of heavily-funded psychiatric research.

Conclusion

The main benefits of the S-DTM approach to self-management of psychiatric symptoms using non-prescription drugs (Table 1) is that it allows people to avoid contact with modern psychiatry and to maintain control of their own therapy and tailor treatment to their own needs. The main limitations are those of limited (or inaccurate) knowledge, difficulties of introspection and self-monitoring, and the restricted range of treatments available without prescription.

One major advantage of a more specific approach to diagnosing and treating sub-types of ‘depression’ in a symptomatic fashion is that of avoid the damaging consequences of treating demotivated depression with stabilizing drugs. Under the currently prevailing standard model of depressive disorder, it is quite likely that any person with a depressed mood would first be tried-out with the stabilizing and emotion blunting SSRIs; and any observed worsening of mood would probably be blamed on the ‘disease’ of depression instead of the drug. But since demotivated people lack strong positive emotions, SSRIs would probably make them feel worse by blunting their emotions still further. (i.e. if your problem is insufficient pleasurable experience, the last thing you need is to be made even less responsive to pleasurable stimuli.)

The five sub-types of ‘depression’ (including melancholia) are not mutually exclusive. An individual might suffer from two or more of these syndromes simultaneously. Various combinations are possible. For example, a person with depressed mood due to a neurotic and unstable personality might well in addition suffer from seasonal affective disorder during the winters, or from malaise following influenza. Or a person with lack of drive due to a chronic demotivated depression might in addition experience malaise secondary to a chronic infection or autoimmune disease, or SAD. Such subjects might perhaps, rarely, go on to develop severe melancholia.

In such situations of several simultaneous diagnoses, alleviation of one type of symptom could fail to improve mood due the persistence of other types of symptom. More than one type of treatment may be required simultaneously, as in any situation characterized by multiple causal pathologies.

In terms of the treatments available without prescription, the biggest problem is that there is only a limited number of energizing drug treatments available without prescription; and the premier energizing drugs all require prescription at present. On the other hand, St Johns Wort is quite possibly the best all-round sta-

bilizing drug (better than SSRIs), bright light therapy is certainly the best treatment for SAD (short of moving to live in a latitude nearer the equator and with sunnier weather); and there is a reasonable range of effective analgesics available 'over the counter' for treating malaise – stronger opiates and stronger NSAIDs being the main categories of pain killers currently requiring a prescription.

In sum, the ability of individuals to self-manage 'depression' is already powerful, and the future looks promising. I hope the above ideas will be useful and will also stimulate debate. And, looking beyond depression, it is possible that the general S-DTM model might be more widely-applicable within psychiatry and medicine, and for enhancement of the quality of life.

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