appg for Prescribed Drug Dependence

UKCP UK Council for Psychotherapy

the british psychological society promoting excellence in psychology

nsun National Survivor User Network

THE NATIONAL COUNSELLING SOCIETY professional standards authority accredited register

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Guidance for Psychological Therapists

Enabling conversations with clients taking or withdrawing from prescribed psychiatric drugs

December 2019

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Contents

A welcome guidance

BACP has been proud to be part of this important project to produce much-needed guidance for our members.

The increase in the prescription of psychiatric drugs means many of our members are working with clients who are taking or withdrawing from them, and this can have an impact on their work.

We know from a recent survey of practising therapists that the majority feel ill-equipped to deal with these issues in a therapeutic setting.

This work will provide our members with up-to-date evidence and relevant guidance to help clients deal with the issues around taking or withdrawing from such drugs and understand the impact on clients and therapy.

We fully support the guidance and recommend it as a resource for our members and training providers.

Hadyn Williams

Chief Executive Officer, BACP

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The BPS fully endorses this guidance and is proud to have produced this in collaboration with our partner organisations.

We believe the official recognition of the increasing numbers of people being prescribed psychiatric drugs, and the difficulties withdrawing from them, is a positive step in helping both patients and psychological therapists.

Our members have consistently told us that they need guidance, information and training to help them work more confidently with clients either taking or withdrawing from prescribed drugs.

The evidence reviewed in this guidance provides an up-to-date summary of the main effects, adverse consequences and possible withdrawal reactions for each of the main classes of psychiatric drug.

We strongly recommend this guidance as a resource for our members.

Sarb Bajwa

Chief Executive, BPS

the british psychological society
promoting excellence in psychology

We are absolutely delighted to endorse this guidance document, which will be an invaluable resource to countless therapists both now and for years to come. It's commonplace for UKCP members to be working with individuals taking psychiatric medication, yet many don't feel properly equipped to discuss this in therapy. This guidance not only provides therapists with deeper knowledge of these medications, but will enable them to discuss confidently issues that are often central to the emotional distress that people they are working with are experiencing. The importance of this cannot be underestimated. It constitutes yet another important step in improving the care for the alarming number of people currently being prescribed psychiatric medication.

Professor Sarah Niblock

Chief Executive, UKCP

UKCP
UK Council for Psychotherapy

As a network led by people who experience long-term mental distress, during the creation of this guidance NSUN helped to provide the perspective of people directly affected by prescribed drug dependence. This guidance is needed more than ever, given the widescale prescribing of psychiatric drugs continues to increase.

It is vitally important to support the prevention of drug harms and dependency by offering practitioners crucial information about withdrawal management, the monitoring of symptoms, while supporting regular medication review and the limiting of unnecessary long term use. Above all people should have a choice about whether to take medication or not and have information and access to a range of alternatives.

NSUN welcomes the guidance and hopes it will help to reduce unnecessary long term use of psychiatric medication, while taking us closer to developing and using non-drug alternatives.

Sarah Yiannoullou

Managing Director, NSUN

nsun National Survivor User Network

The National Counselling Society fully support this guidance, and commend the authors and organisatons involved in its compostion. The increase in psychiatric drug prescriptions should be viewed through a critical lens. The research laid out in this guidance clearly indicates an urgent need for more education on the impact of such prescribing, as well as critical evaluation of the paradigm by which it is enabled. As medication is being used in a wide variety of mental health settings, our members will have experienced the impact of these drugs upon clients and therapy. In many cases, therapists may be unaware how medication, and psychaitric drug withdrawal itself, can subtly yet significantly impact the therapeutic process. We would ideally like to see all therapists and allied professionals develop greater awareness of the potential impact of prescribed drug dependency. We strongly encourage our members and training providers to familiarise themselves with this guidance.

Vicky Parkinson

Chief Executive Officer, NCS

THE NATIONAL COUNSELLING SOCIETY

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I am delighted that the APPG for Prescribed Drug Dependence has brought together the leading therapy organisations and relevant experts to produce this guidance, which will help to tackle the problem of prescribed drug dependence highlighted by Public Health England's recent report. It is clear that many people end up taking unnecessary and potentially harmful psychoactive drugs for years, and that there has been inadequate recognition of the problem and very little support for those wishing to withdraw. This guidance, along with the other recommendations from PHE such as a national helpline, are therefore part of an overdue response to this important public health issue.

Sir Oliver Letwin MP

Chair, APPG for Prescribed Drug Dependence, October 2018–November 2019

appg for Prescribed Drug Dependence

Acknowledgements

Organisational roles

The All-Party Parliamentary Group for Prescribed Drug Dependence (APPG for PDD) has facilitated the creation of this guidance by bringing together key professional bodies representing psychological therapists in the UK with relevant subject matter experts.

The British Association for Counselling and Psychotherapy (BACP), British Psychological Society (BPS) and United Kingdom Council for Psychotherapy (UKCP) have collectively funded and steered the creation of the guidance in conjunction with members of the APPG for Prescribed Drug Dependence Secretariat (all members of the Council for Evidence-based Psychiatry (CEP)), and the National Survivor User Network (NSUN). The professional bodies, including in addition the National Counselling Society (NCS), endorse the guidance and will promote it to their members and relevant training organisations.

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The guidance has been developed in conjunction with and reviewed by experts by experience (clients, carers, therapists and campaigners).

The APPG for PDD will distribute the guidance and host a website for this purpose, seeking to make it as widely available as possible both in the UK and, where appropriate, internationally.

The following were officers of the APPG for PDD until November 2019:

Chair: **Sir Oliver Letwin MP** (Ind)
Co-chair: **Sir Norman Lamb** (Lib Dem)
Co-chair: **Luciana Berger MP** (Lib Dem)
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1. Introduction

Dr James Davies, Professor Rosemary Rizq & Dr Anne Guy

In September 2017 the All-Party Parliamentary Group for Prescribed Drug Dependence (APPG for PDD) met senior representatives from Public Health England (PHE) to present data and research (including work undertaken by the British Medical Association, 2016[1]) revealing the mounting social and individual problems associated with prescribed drug dependency and withdrawal. As a result, Steve Brine MP, then Under Secretary of State for Public Health and Primary Care, commissioned PHE to undertake the largest review to date into prescribed drug dependency and withdrawal. This comprehensive review has now been published and has called for the following:

- A 24-hour national helpline and associated website to provide advice and support for those adversely affected by prescribed drug dependency and withdrawal.
- Updated clinical guidance and improved doctor training.
- Provision for better patient-information about drug risks and benefits, as well as alternatives such as therapy and social prescribing.
- Further research into the nature and severity of withdrawal and its successful treatment.
- Appropriate support from the NHS for patients, including dedicated support services.[2]

In May 2019 the Royal College of Psychiatrists issued a new position statement on antidepressant withdrawal,[3] following new research[4,5] and campaigning by people who have been harmed by psychiatric drugs, (also known as the prescribed-harm community), the Council for Evidence-based Psychiatry (CEP) and the APPG for PDD. The Royal College's statement acknowledged that antidepressant withdrawal is more widespread than previously thought and can be more severe and protracted than our current clinical guidelines acknowledge.[6] Joining the campaigners, the Royal College also called for NICE to update its guidelines to better reflect the evidence base.

In October 2019, NICE heeded calls by CEP, the APPG for PDD and the RCPsych to remove its previous advice that antidepressant withdrawal is usually mild, self-limiting and resolving over 1-week, and acknowledge that, while many people may experience only mild withdrawal, there is 'substantial variation' in people's experience 'with symptoms lasting much longer (sometimes months or more) and being more severe for some patients'.[7]

While these changes largely relate to antidepressants (and, in the case of PHE, benzodiazepines, Z-drugs, GABA-ergic medicines and opioid pain medications), they demonstrate that thinking around psychiatric drug withdrawal has shifted considerably since early 2018. Today, in the UK, it is now widely acknowledged that we previously underestimated the incidence, severity and duration of withdrawal effects and the extent to which those people affected need support. Relevant organisations are therefore now considering how best to support people who have suffered harm. PHE has recommended a helpline, better training for doctors on appropriate withdrawal management, and more support for GPs; recommendations now supported by the Royal College of General Practitioners, the Royal College of Psychiatrists , the British Medical Association and all the organisations involved in either the creation and/or endorsement of this current document (i.e. the APPG for PDD, CEP, the British Association for Counselling and Psychotherapy (BACP), United Kingdom Council for Psychotherapy (UKCP), the British Psychological Society (BPS), and the National Counselling Society (NCS)).[8]

In endorsing this document these organisations are taking their share of responsibility for addressing the withdrawal problem, by equipping psychological therapists with the information and guidance necessary to help them better inform and support clients who are either taking or withdrawing from psychiatric drugs.

1.1 What are the aims of this guidance?

Psychiatric drugs such as antidepressants and antipsychotics are more prescribed today than at any other time in our profession's history. Around a quarter of the UK adult population was prescribed a psychiatric drug last year, with around 16% being prescribed antidepressants (2016–17) (DHSC 2018).[9] The steep rise in prescriptions (which have broadly doubled in the last 20 years[10]) means that most therapists now work with clients who have either taken or are taking psychiatric drugs. These drugs will produce effects that may or may not be experienced as positive by the individual in question. These drugs can also produce adverse effects, while many clients will struggle to reduce or withdraw from them. To date, the lack of summarised evidence, information and training for therapists who work with such clients, constitutes a growing problem for therapists whatever their modality or setting in which they work.

This lack of knowledge and training is reflected in data gathered from a 2018 survey of approximately 1,200 practising therapists – all members of BPS, UKCP or BACP. While 96.7% of the therapists reported that they currently work with at least one client who is taking a psychiatric drug (e.g. an antidepressant, anxiolytic or antipsychotic), only 7.3% reported that their training equipped them 'very well' in responding to questions about withdrawing from or taking psychiatric drugs. Additionally, 42.5% of therapists reported feeling a lack of confidence in knowing where to find appropriate information (or ethical or professional guidance) on how to work in the most therapeutic way with people taking or withdrawing from psychiatric drugs. This lack of support, training and information may well explain why 93.1% of the therapists surveyed reported they would find it either 'useful' or 'very useful' to have professional guidance to help them work more competently and confidently with such clients.

It is therefore now essential for the therapeutic professions to respond jointly to this growing need for clear guidance about how best to work with and support clients either taking or withdrawing from psychiatric drugs. This guidance seeks to provide such support in two distinct ways:

Firstly, it aims to support therapists in deepening their knowledge and reflection on working with the said client group. The evidence reviewed in this guidance means that therapists will now have access to an up-to-date summary of the main effects, adverse consequences and possible withdrawal reactions for each of the main classes of psychiatric drug. Using this evidence base, the guidance aims to empower therapists to talk about prescribed drugs with their clients (and where appropriate with prescribers) as well as to identify and work with the impact that psychiatric drugs may exert on the process of therapy itself.

Secondly, it invites therapists to familiarise themselves with core issues relating to the role of psychiatric drugs in therapy. Many therapists prefer to avoid discussing the client's relationship to prescribed psychiatric drugs, assuming any consideration or discussion of this relationship is best left to prescribers. This preference may be rooted in feelings of anxiety about navigating alternative views on psychiatric drugs, not having sufficient knowledge to engage other professionals, or feeling uncertain about managing the boundaries of one's professional competence or role. Indeed, while this guidance agrees that *it is not the role of the therapist to tell a client either to take, continue to take or withdraw from psychiatric drugs, nor to decide when, if or what drugs need to be withdrawn*, this guidance actively encourages therapists to support clients in whatever decisions they reach with their prescribers. It also encourages them to engage with the views and perspectives of other professionals whilst at the same time honouring the distinctive and important contributions therapists can make in supporting a client through withdrawal from psychiatric

drugs. Finally, it is also important to note that this guidance does not aim to disrupt or comment on the NICE guidelines as used by medical doctors, which, for example, recommend drugs for many conditions in addition to psychological therapies. However, it is also important to note that NICE's recommendations are continually being updated in the light of new debates, disputes, interests and evidence. For example, at the time of writing the guideline on depression (CG90) is undergoing an additional period of consultation in response to criticisms from a coalition of stakeholders, which includes many therapy organisations.

1.2 Who is this guidance for?

This guidance aims to be relevant to a wide variety of theoretical models as well as professional and personal positions held by members of the main accrediting bodies. These are: BPS, BACP and UKCP and NCS. It is clear that therapists' professional training as well as their personal and therapeutic experience means there are likely to be significant differences in how they think about and work with the various issues relating to prescribed drugs. Many therapists will be highly critical of their use whilst others may believe prescribing privileges should be extended to therapists.[11] It is also important to note that, in addition to holding different views, therapists also operate within a variety of settings. These may include NHS primary care, secondary care and specialist services; statutory or third sector services; private practice and other private sector services and agencies. Different professional settings will shape therapists' decision-making as well as the opportunities available to them for working collaboratively with other healthcare professionals.

Given this rich diversity of professional backgrounds, trainings and settings, this guidance does not aim to be prescriptive nor attempt to offer a set of therapeutic 'competences' or 'guidelines'. Rather, by using the available evidence base, therapists of all persuasions will be invited to consider a number of key questions and concerns relevant to their therapeutic work with clients who are either taking or withdrawing from prescribed psychiatric drugs.

Therapists will also be invited to reflect on their own professional background and training, their personal and practice-based experiences as well as their relationship to and understanding of the 'medical model' and its associated interventions.

While this guidance is therefore written for therapists, much that is included may be of professional interest to those working in allied helping professions (e.g. nursing; occupational therapy; social work; and those in relevant caring and medical roles). It is therefore hoped that allied professions might be able to make use of some or all of these materials in ways that will serve their clients' interests while at the same time best honouring their own professional and ethical values.

1.3 The medical model and the emerging crisis

Whatever an individual's view regarding the best model with which to understand and respond to emotional and mental distress, it is clear that since the mid-2000s there has been growing professional and public criticism of the utility and validity of the 'biomedical' model and associated interventions – a model in which distress has been assumed by some to be rooted in an underlying disease mechanism or organic pathology. It is important to note, however, that such criticism has been advanced not only by non-medical professionals. Indeed, many of its proponents stem from the medical and psychiatric community itself, where today there is a diversity of views regarding the utility and validity of this model. In short, the lines of debate cut through all mental health disciplines, and so can

no longer be framed in disciplinary polarised ways. Furthermore, such debates now resonate beyond the disciplines themselves, in ever larger sections of the academic, political, media and service-user communities, and similarly stem from increasing concern that our mental health services are not just failing due to lack of investment, but owing to peoples' emotional and behavioural difficulties being over, unduly and unhelpfully medicalised. It has been argued that over-medicalisation has led, in turn, to the consequent over-prescribing of psycho-pharmaceuticals,[12,13] rising mental health stigma,[14] the proliferation of unnecessary and harmful long-term prescribing, and the crowding out of effective alternatives that people both need and want.[15,16] These arguments have dovetailed with others that pertain to the medical model, such as the value or otherwise of psychiatric diagnosis more broadly[17–21]; the role of conflicts of interest between the pharmaceutical industry, prescribers and drug-researchers[22–24]; the lack of biomarkers for 'mental disorders' or evidence for the chemical imbalance theory of mental distress [25,15]; the evidence that antidepressants may yield no clinically significant benefits over placebos for most people despite ever-rising prescriptions[30–33]; the expanding knowledge of withdrawal problems[34,4,5], and the growing understanding that long-term use of psychiatric drugs is often associated with poor outcomes and increased harms[3]. These concerns, criticisms and areas of debate have been articulated, advanced and engaged with not only by psychologists, academics and therapists, but also by many psychiatrists who have seen in the psychiatric perspectives and treatments once championed in the 1990s, many promises left unrealised.

Each individual involved in the composition of this guidance will have a particular view on these separate debates and criticisms, as will its readers. As no guidance can ever be written in a vacuum, and as many contributors have been involved in some of the above debates, it is important to be explicit about how these criticisms may have informed the content of this guidance.

The first obvious influence is that this guidance departs from the increasingly contested belief, both within psychiatry and beyond, that psychiatric drugs 'cure' mental 'illnesses' that are rooted in brain pathologies. Rather, it takes the view that psychiatric drugs, like all other psychoactive substances, alter states of mind in ways that may or may not be experienced as helpful by the individual in question. Also, like many other psychoactive substances, psychiatric drugs can cause side, adverse and withdrawal effects that can complicate a person's recovery, certainly if not acknowledged as such.

The second influence concerns the language used in this guidance. Medical terms such as 'illness', 'disorder', 'pathology' and 'dysfunction' do not merely describe the suffering they depict but shape how it is understood, managed and perceived. Medical language imports meanings that may not always accord with how many psychological therapists frame distress. A common view in the psychological community is that medical language broadly assumes what it should rather demonstrate: that the suffering it describes is in fact medical 'illness', 'disorder' or 'pathology'. Rather than seeing suffering as an illness, many therapists would understand it as a rational reaction to hurt, trauma or impairment. In many cases it may be a call for change or an instance of what may be termed 'social suffering' – namely, a non-pathological, distressing, yet understandable human response to harmful social, political, relational and environmental conditions (past or present).

Given that medical language carries meanings that extend well beyond the way in which many therapists understand psychological distress, including meanings that assume, rather than demonstrate the biological causes of mental distress, this guidance will avoid medical terminology where possible. Instead, it will adopt non-medical descriptors, such as those recommended by the British Psychological Society.[36] There are occasions, however, where the meaning of alternative words is not clear and so some language has been retained for the sake of simplicity and readability, but this should not be taken as an acceptance of its full medical implications. Quotation marks have been used in some places to denote a disputed term.

1.4 Glossary

In this guidance the terms below will be preferred and used for the following reasons:

- **Therapists** – this term is used to denote the range of different psychological therapists represented by the bodies endorsing this guidance (e.g. counsellors, clinical and counselling psychologists, psychotherapists, psychoanalysts). This term is used simply for the matter of convenience, and its usage in no way intends to minimise or overlook the differences that may exist between different therapeutic professionals and modalities.
- **Psychiatric drugs** – this term is used throughout to refer to all prescribed psycho-pharmaceuticals including antidepressants, antipsychotics, stimulants, tranquilisers, anxiolytics etc. regardless of who has prescribed them.
- **Client:** this term is used throughout this guidance to refer to anybody meeting a therapist for therapy.
- **Dependence** – this term is used throughout this guidance to denote physical dependence on a drug. This is not to deny the relevance of meanings and beliefs and the psychological effects of taking and stopping psychiatric drugs, but only to specify that the research covered in the evidence sections of this guidance predominately relates to dependence in its physical form.

Also, this guidance draws the distinctions between the following terms:

- **'Dependence' rather than 'addiction'** – this distinction is drawn for two reasons: the term 'addiction' is generally associated with dependence on non-prescribed substances (such as illicit drugs and alcohol). As such it may be read, rightly or wrongly as having negative connotations. In contrast, 'dependence' largely avoids those connotations, which is why the prescribed-harm community, in general, prefers the term, as it better captures the experience of becoming dependent by following a prescriber's recommendations. Secondly, and following Public Health England's preferred language, dependence refers to an adaptation to the repeated exposure to a drug. This is usually characterised by tolerance and withdrawal, (though tolerance may not occur with some drugs).[2]
- **Psychiatric 'drugs' rather than psychiatric 'medication'** – this distinction is drawn since the term 'medication' is defined as a substance that is used to cure or treat a disease or medical condition, or to alleviate symptoms of an illness.[37] As it is contestable as to whether psychiatric drugs either 'cure' or 'treat' a 'disease' or a 'medical' condition or 'illness', the term 'drugs' is preferred, in particular as the definition for drugs (i.e. 'a substance which has a physiological effect'[38]), better captures the evidence-base for how psychopharmaceuticals work.
- **Drug-Centred vs Disease-Centred model of drug action** – this distinction is drawn to clarify how psychiatric drugs work: the disease model assumes psychiatric drugs work by reversing or partially reversing an underlying 'disease', while the drug-centred model asserts that psychiatric drugs work by producing physiological and psychological effects, as all psychoactive substances do, which may or may not be experienced as beneficial. This guidance prefers the drug-centred over the disease-centred model, as it better captures the evidence-base as to how psychopharmaceuticals work. This is further expanded on in section 2.

1.5 Scope

This guidance relates to psychiatric drugs that have been prescribed in the course of clinical practice. It does not tackle illicit or recreational drug use (nor prescribed painkiller/opioid use) and any associated problems. Naturally, such hard and fast distinctions may belie clinical complexity, given some clients may present with multiple prescribed and non-prescribed dependencies.

A further area beyond scope is first, the impact of other physical health conditions on both drug and talking therapy, and conversely, the impact of prescribed psychiatric drugs on physical health (beyond those problems associated with dependence and withdrawal). As important as these areas are, the number of variables involved renders making any general statements unfeasible, beyond recommending that such adverse reactions must always be discussed with the prescriber.

Finally, systemic, child and family therapies, as well as social prescribing are not discussed in this guidance, although we clearly recognise the vital contribution they make in this area. The parameters of a project must be drawn somewhere, and ours reflect pragmatic constraints rather than any implied grading of the relative importance of the topics omitted.

1.6 How to use the guidance

It is suggested that the sections giving general information should where possible be read in full, whilst readers might selectively read information relating to specific classes of drugs in sections 4 and 5, depending on which they are most likely to encounter, or on an as-needed basis.

This guidance aims to empower and support conversations often already taking place between therapists and their clients. Therapists will need to decide for themselves whether, and to what extent, they wish to use this guidance in the context of their therapeutic work. These decisions will depend on their theoretical modality, practice setting and the individual needs of the client. The client's agency, as always, should be supported and respected at all times. Clients should be encouraged to discuss withdrawal from prescribed psychiatric drugs with a knowledgeable prescriber who can give medical advice, oversee and manage any withdrawal process appropriately. While this guidance advocates the importance of informed client choice based on full information about potential benefits and risks, *it does not advocate therapists telling their clients to take, not take, stay on or withdraw from psychiatric drugs. These matters should be left to the prescriber and client to decide.*

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2. Introduction for therapists on how psychiatric drugs work

Professor Joanna Moncrieff & Dr Tom Stockmann

2.1 The place of prescribed drugs in Mental Health Services

Drugs have been the mainstay of psychiatric treatment since the 1950s. Nowadays, most people who receive specialist psychiatric services are prescribed one sort of psychiatric drug, and often several. General practitioners prescribe such drugs to millions of other people.

Before the 1950s, drugs, especially sedatives, were used extensively in psychiatric hospitals and prescribed to outpatients. However, they received little attention because they were generally regarded simply as a means of chemical restraint.[1,2] However, during the 1950s and 1960s new ranges of drugs were introduced into psychiatry. Views about how they worked gradually transformed: they came to be seen not simply as inducing useful but crude states of sedation and passivity, like the older style drugs, but as acting to reverse underlying psychiatric diseases.

The naming of psychiatric drugs reflects this assumption; so 'antipsychotics' are thought to act on the biological abnormality that produces symptoms of psychosis or 'schizophrenia', 'antidepressants' are thought to reverse the basis of depressive symptoms, 'mood stabilisers' are thought to help rectify the process that gives rise to abnormal fluctuations of mood and 'anxiolytics' are thought to address the biological mechanism that creates anxiety.

2.2 How do psychiatric drugs work?

The assumption that the major types of drug used in psychiatry work by reversing or partially reversing the underlying disease process may be termed the 'disease-centred' model of drug action. There is little evidence to support this model, however. An alternative 'drug-centred' model states that psychiatric drugs produce a global state characterised by a range of physiological and psychological alterations, which are superimposed on, and interact with, the 'symptoms' of mental 'disorders' in ways that may or may not be perceived as beneficial.

2.2.1 The disease-centred model

The disease-centred model has been imported from general medicine, where most modern drugs are correctly understood in this way. Although most medical treatments do not reverse the original disease process, they generally act on the physiological processes that produce symptoms, or on specific physiological targets to reduce symptoms via an identified mechanism. Thus, chemotherapeutic agents counteract the abnormal cell division that occurs in cancer, while analgesics act on the physiological processes that produce pain. Some anti-hypertensives (medications for high blood pressure) relax blood vessels by acting on specific receptors to lower blood pressure, even though the cause of the hypertension may be unknown.

The disease-centred model of drug action is closely related to theories that mental health conditions are caused by abnormalities in particular brain chemicals, or a 'chemical imbalance'. Chemicals that facilitate or inhibit the transmission of nervous impulses with the brain are called 'neurotransmitters'. Following the observation that psychiatric drugs act on neurotransmitter systems, it started to be proposed that an abnormality in these systems might be the cause of psychiatric disorders. The best-known example of this way of thinking is the dopamine hypothesis of 'schizophrenia', which followed from the discovery that early antipsychotics reduce dopamine activity (dopamine is a brain-based neurotransmitter). The idea that depression is caused by a deficiency of the neurotransmitters serotonin or noradrenalin is another example sometimes referred to as the 'monoamine theory' of depression (serotonin and noradrenalin are both classified as monoamine-type brain chemicals or neurotransmitters).

Some researchers still support the dopamine hypothesis of 'schizophrenia'[3], and some antipsychotic drugs certainly affect dopamine transmission, although others have only weak effects on dopamine. However, evidence that there are dopamine abnormalities in people with psychosis or 'schizophrenia' prior to starting drug treatment is inconsistent.[4,5] Few people now accept the idea that depression is caused by a serotonin or noradrenaline abnormality, and again the evidence is highly inconsistent.[6]

In fact, despite decades of intensive research on all sorts of aspects of biological science including various neurotransmitters, genetics and neural networks, definitive causes of mental health difficulties have not been established. Recently, the former head of the US National Institute of Mental Health admitted that $20 billion of funding for investigating the neuroscience and genetics of mental disorders, had produced no benefit to people suffering from mental health difficulties.[7]

There is also little evidence that drugs that are meant to have specific effects in certain conditions, according to the disease-centred model, are better than other sorts of drugs.[8,9] For example, numerous drugs that are not considered to be antidepressants have been shown to have equivalent effects to antidepressants in people with depression, including anti-anxiety drugs like diazepam (Valium), stimulants and antipsychotics. Antipsychotics are not clearly distinguished from other sedatives in studies of people diagnosed with psychosis or schizophrenia[10], and lithium is not superior to other drugs in studies in people diagnosed with acute bipolar or manic states.[11] In addition, two studies that attempted to distinguish between the effects of lithium and antipsychotics in people with different diagnoses (bipolar or affective disorder versus non-affective psychosis or schizophrenia) failed to do so.[12,13]

Even if the mechanisms of mental health difficulties could be identified, however, we would still be uncertain whether or not psychiatric drugs impact symptoms by affecting those mechanisms. This is because to draw this conclusion, we somehow need to discount the effect of the general mental and behavioural alterations that these drugs are known to cause in anyone, regardless of whether or not they have an identified neurochemical abnormality.

The drug-centred model of drug action suggests it is these alterations that are significant.

2.2.2 The drug-centred model

This model highlights that psychiatric drugs can be considered to be 'psychoactive' drugs in the sense that they are substances that cross the blood/brain barrier and affect brain functioning. By doing so they produce an altered global state characterised by a range of physiological, psychological and behavioural changes. There is no essential distinction, according to this view, between drugs used for psychiatric treatment and recreational psychoactive drugs like alcohol and cocaine. All psychoactive drugs produce altered physical and mental states that can influence the way people think, feel and act, with different sorts of substances having different sorts of effects. The effects of recreational drugs are experienced as desirable by at least some people, but some drugs produce mental and physical changes that are generally experienced as unpleasant (e.g. antipsychotics and lithium). The drug-centred

model suggests that it is these psychoactive properties that explain the changes seen when drugs are given to people with mental health difficulties. Drugs like benzodiazepines and alcohol, for example, reduce arousal and induce a usually pleasant state of calmness and relaxation. This state may be experienced as a relief for someone who is intensely anxious or agitated. But taking a drug like this does not return the individual to 'normal', or to their 'pre-symptom' state. The drug-induced state is superimposed on the 'symptoms' and is found to be preferable, either by the sufferer themselves, or by others.

In psychiatry, an accepted example of a drug-centred treatment is the recognised benefits of alcohol in social anxiety (also referred to as social phobia). Alcohol can help people with social anxiety because a state of mild intoxication is associated with a lessening of social inhibitions. Rather than reversing an underlying biochemical imbalance, alcohol works because it substitutes the alcohol-induced behavioural and emotional state, with its characteristic lessening of inhibitions, for the previous anxious state.

The brain reacts to the presence of a drug in various ways, and often adapts to the drug in ways that counteract the drug's effects. Therefore, the effects that a drug has when it is first taken may wear off and increasing doses may be required to sustain the initial effects. Sometimes this is referred to as 'tolerance'. Biological adaptations to the presence of a drug are also responsible for withdrawal symptoms. When a drug that has been taken for some time is stopped, the body's adaptations are no longer opposed by the presence of the drug and can give rise to unpleasant and debilitating sensations and experiences.

Whereas the disease-centred model assumes that psychiatric drugs help to restore normal brain functioning, the drug-centred model stresses that taking a drug creates an abnormal biological state. Some effects associated with this altered state may be perceived as worthwhile in certain situations. Often however, by distorting normal bodily function, drugs have an adverse impact. They may therefore do more harm than good, particularly in the long term.

Much of the material in this section is a condensed and updated version of material contained in *A Straight Talking Introduction to Psychiatric Drugs* by Joanna Moncrieff, published by PCCS Books, and used with the publisher's kind permission.

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3. Implications for therapeutic practice

Professor Rosemary Rizq, with Professor Tim Bond, Dr Anne Guy, Dr David Murphy, Paul Sams, Professor Marcantonio Spada & Georgina Whitney

Having introduced the broader context and the different ways of viewing prescribed psychiatric drugs in sections 1 and 2 of this guidance, this section considers the implications for therapeutic practice. It invites therapists to reflect on their own position in relation to the biomedical paradigm and to consider a number of key issues in relation to prescription psychiatric drugs. Practice-related guidance includes suggestions for working with clients at different stages of their prescribed drug journey: those who are considering taking prescribed drugs; those who are considering withdrawing; and those who are already withdrawing and who may be experiencing withdrawal reactions. For ease of reference, brief summaries of the evidence presented in sections 4, 5 and 6 are included.

3.1 The biomedical paradigm and its relationship to different therapeutic modalities

From the outset, it is important to acknowledge some of the tensions that exist between the biomedical model currently dominating healthcare and the psychological paradigm adopted by therapists working with those in emotional distress.

As stated in the introduction, the growing medicalisation of distress in society reflects the widespread assumption that mental illness exists in the same way as physical illness and can be diagnosed and treated like flu or a virus. The idea that psychological distress may be understood as the symptom of an underlying disease process or organic abnormality, to be treated with prescribed psychiatric drugs, reflects a disease-centred model of practice in line with the biomedical approach to science, policy and practice currently dominating mental health services.

Of course, the prevalence of the biomedical approach does not exclude mental health services advocating better access to psychological therapies, particularly where therapy is assumed to be complementary to the use of psychiatric drugs. However, the majority of psychological therapists hold a framework for understanding emotional pain that conflicts with the prevailing disease-centred model of practice. Therapists from all professional backgrounds draw from paradigms that predominantly emphasise the psychological and psychosocial aspects of experience thought to underpin mental suffering, rather than on models that emphasise notions of deficit, symptomatology and medicalisation. Indeed, there has been a growing professional movement in the psychotherapeutic field away from a disease-centred model of practice and the use of prescribed psychiatric drugs. For example, there have been recent attempts to offer alternative ways of understanding mental distress (e.g. the *Power Threat Meaning Framework*[1]). The BPS[2], too, has stated that *'clients and the general public are negatively affected by the continued and continuous medicalisation of their natural and normal responses to their experiences; responses…which do not reflect illnesses so much as normal individual variation…This misses the relational context of problems and the undeniable social causation of many such problems'* (p2). Underpinning these and other critiques lies the call for a 'paradigm shift' within mainstream psychiatry[3] that takes account of the complex interplay of social, cultural, economic and psychological forces that are thought to result in much mental distress today.

However, the continuing cultural dominance of the biomedical approach means that it is likely to shape the attitudes, beliefs and values of therapists from all psychotherapeutic backgrounds and to pervade their practice in both explicit and implicit ways. Whilst Elkins (2009)[4] suggests that the 'medical model' in the psychological therapies is essentially an analogy: *'a descriptive schema borrowed from the practice of medicine and superimposed on the practice of psychotherapy'* (pp67–71), it is clear that different psychotherapeutic disciplines will understand, take up and respond to it in different ways. For example, some have argued that the field of applied psychology is significantly permeated by a biomedical perspective[5], whilst others prefer to adopt a critical position in relation to notions of 'pathology', 'illness' and 'disorder'.[6] Within the different theoretical traditions too there is considerable variation in philosophical stance and attitude, reflective of different tensions and discourses within the field. Although therapists principally draw from psychological paradigms that differ from the biomedical approach, it is clear some psychotherapeutic frameworks actively recruit the 'medical model' by analogy, borrowing language and classificatory systems that give rise to an apparent alignment in practice.

In the face of these and other complex debates and professional differences, therapists using this guidance will need to consider carefully the degree to which they think a biomedical perspective currently influences their practice. Clearly there will be considerable differences here, depending on each therapist's professional background, professional training, work context and personal preference. For example, there will be some therapists whose work setting privileges a biomedical framework, requiring them to use the language of psychiatric classification and to incorporate standardised assessments and manualised 'clinical' techniques into their therapeutic work. By contrast, others may work in settings that allow them to reject the language of medicalisation and symptomatology entirely and to focus instead on the therapeutic relationship and client self-determination. There are many possible configurations here and many possible variations in the extent to which therapists feel they can or must adhere to a biomedical perspective. For this reason, it is important for therapists to reflect on the personal and professional ways in which they relate to and engage with the 'medical model', as this is likely to influence significantly, if implicitly, their attitude towards people who are taking prescribed drugs, prescribers and the drugs themselves.

How do the main modalities relate to the medical model?

The authority of the medical model means that many therapists consider issues of prescribed psychiatric drugs to be the exclusive remit of doctors, psychiatrists and neurologists. However, the specialist training of therapists means they all subscribe to a conceptual system of mental distress that is primarily psychological rather than biomedical. They are therefore well placed to help their clients in ways that are additional to and distinctive from medication.

The next section offers a brief summary of how the three main therapeutic modalities traditionally position themselves in relation to the biomedical model of practice. It is clear that such a summary cannot be exhaustive, nor can it do justice to the variations that exist within and between theoretical orientations. Rather, it aims to offer a starting-point of reference for therapists who wish to locate their practice on the continuum discussed above.

3.1.1 Humanistic models of training and practice, including person-centred, experiential, existential and Gestalt approaches are concerned with notions of subjective experience, personal meaning and the development of potential, with therapy seen as inherently relational. The client's potential for actualisation, uniqueness, autonomy and authenticity contrasts with the medical model's focus on 'illness', 'disorder' 'psychopathology' and its use of standardised assessments, 'objective' outcome measures and the specificity of 'clinical' techniques. Psychological distress is considered to be the result of thwarted actualisation due to sub-optimal

social/environmental conditions. Humanistic therapists seek to develop a therapeutic relationship characterised by authenticity and transparency rather than by hidden agendas or 'expert' positions, instead emphasising emotional engagement, collaborative work, responsibility for the self and the client's freedom to self-direct. The fundamental call for humanistic therapists to 'be with' rather than 'do to' the client means they do not direct or actively encourage clients to make changes in their lives. Instead, they prefer to support clients in taking responsibility for themselves through a spirit of collaborative, empathic enquiry, exploration and acceptance.

3.1.2 Psychodynamic models of training and practice

range from long-term psychoanalytic and relational psychotherapy through to shorter term models such as brief, psychodynamically oriented counselling and Dynamic Interpersonal Therapy (DIT). Whilst there are important differences between the various psychoanalytic schools of thought, all approaches emphasise the centrality of unconscious mechanisms and processes in relationships and tend to focus on the emergence of transferential and countertransferential material within therapy. The client is seen as 'divided', and therapeutic work aims to bring unconscious material to the surface, allowing it to be experienced safely and to become available for thought and processing with the therapist. Psychodynamic therapists, like humanistic therapists, tend to reject the 'expert' position characteristic of biomedical approaches, although some schools of psychoanalytic thought adhere to diagnostic classifications that are closely linked with medical psychiatry. Most therapists prefer to adopt a 'neutral' stance that allows the client to project on to the therapist feelings and fantasies deriving from his or her early relationships. The traditional injunction to keep the therapeutic space free for transferential work means that psychodynamic therapists may vary in their willingness to offer direction or advice to clients, and they are likely to consider carefully the unconscious implications of the therapist's contact with prescribers or other people involved in the client's care.

3.1.3 Cognitive-behavioural models of training and practice

cover a number of approaches including Beckian cognitive-behavioural therapy (CBT), dialectical behaviour therapy (DBT) and rational-emotive behaviour therapy (REBT) as well as those that would be considered under the heading of 'third wave' approaches such as mindfulness-based CBT (MBCBT), compassion-focused therapy (CFT) and acceptance and commitment therapy (ACT). These are all structured, focused approaches emphasising the use of specific techniques and strategies to promote measurable change. Whilst there is some debate about the extent to which its proponents align themselves with the 'expert' position characteristic of the biomedical model, the therapist stance endorsed by most CBT practitioners is collaborative, seeking to develop a helpful therapeutic alliance, a shared formulation and therapy goals. Psychoeducation and self-monitoring may be used to help clients identify unhelpful patterns of thought, behaviour and action that are seen as maintaining their current psychological difficulties. This may be followed by therapeutic work aimed at addressing underlying issues such as the impact of trauma. CBT is a common approach within public sector services, where therapists routinely work within a multi-disciplinary team.

In concluding this section, it can be seen that the different perspectives outlined above all carry very different assumptions about the nature of emotional suffering. The biomedical paradigm sees much of mental distress as an unproductive 'disorder' or 'symptom' that is best removed with the help of prescription psychiatric drugs. Within the humanistic and psychodynamic traditions, however, suffering is conceptualised as having potential value and purpose rather than something that is merely 'pathological' or otherwise useless. Therapists from these traditions tend to see emotional distress as a signal that there is something wrong in the individual's life: suffering represents an opportunity for change and transformation if it can be explored and managed productively. By contrast, in approaches such as cognitive-behavioural therapy, the main focus is on removing symptoms of distress by altering patterns of cognition, emotion and behaviour that are understood to maintain emotional suffering.

3.2 Key issues for therapists to consider when working with clients who are taking or withdrawing from prescribed psychiatric drugs

A general principle emerging from the evidence base in this guidance is that there is little to support a 'disease-centred' model of drug action. Prescription psychiatric drugs act on the brain to alter mood and consciousness. In general, they control reactions to emotional distress by numbing, tranquilising or sedating a person, thereby producing subjective states that may or may not be experienced as helpful to the individual. Where psychiatric drugs produce effects experienced as helpful, they are best thought of as a temporary tool or coping mechanism that can be a helpful precursor to psychological change.

In the instances where prescribed psychiatric drugs produce short-term relief, they do not change the underlying causes of psychological distress and may do some harm in the long term. It should also be remembered, however, that psychiatric drugs can be prescribed for physical conditions such as migraine. As we will see from the evidence presented in sections 4–7, all prescription psychiatric drugs come with withdrawal costs to some people. What follows is a brief summary of that evidence.

Evidence box A: Summary of adverse effects and withdrawal reactions to broad classes of prescribed psychiatric drugs
(For full details, including references, see sections 4 and 5)

Benzodiazepines (e.g. Diazepam) have sedative properties and are generally prescribed for anxiety and sleep disturbance. They carry a significant risk of dependence if used for more than a month and for this reason should be prescribed for no longer than that. Adverse effects include drowsiness and impaired cognitive ability and, at higher doses, slurring of speech, loss of balance and confusion. Withdrawal effects are often severe and generally include an acute period over two weeks to two months with symptoms such as anxiety, agitation, insomnia and muscle stiffness. There can also be tingling, numbness, electric shock-type feelings, hallucinations, delusions and nightmares. Some people will experience longer-term withdrawal symptoms lasting a year or more.

Antidepressants come in two main classes: Tricyclic antidepressants which are sedating, resulting in slowed reaction time, drowsiness and emotional indifference. In high doses they can also cause heart arrhythmias; SSRIs/SNRIs can cause nausea, drowsiness, but also sometimes insomnia. They usually have sedative effects and appear to numb emotions but may sometimes cause anxiety and agitation. There is also some evidence that SSRIs may increase suicidal impulses and possibly also violent behaviour in children and young people. Withdrawal effects can include nausea, dizziness, anxiety, depression, 'brain zaps', insomnia, hallucinations, vivid dreams, agitation and confusion. These symptoms typically last a few weeks but may continue for up to a year and occasionally for several years.

Stimulants (e.g. Ritalin) are generally prescribed for behavioural problems in children (and now often adults). They increase arousal and improve attention in the short-term, but suppress interest, spontaneity and emotional responsiveness. Insomnia is common. An important adverse effect for children is growth suppression. There may be rebound effects on withdrawal as well as tearfulness, irritability and emotional lability (rapid often exaggerated changes in mood).

Mood stabilisers (e.g. Lithium) are most commonly prescribed for those who have been given a diagnosis of bipolar disorder. All have a sedative effect, suppressing physical activity and reducing or flattening emotional responses. There is decreased ability to learn new information, prolonged reaction times, poor memory, loss of interest and reduced spontaneous action. Weight gain is common. Withdrawal from Lithium does not result in the physical withdrawal symptoms typical of other drugs but can cause a relapse of mania if undertaken too quickly.

Anti-psychotics (e.g. Olanzepine) all produce a sedative effect, dampening or restricting emotional reactions and making it difficult to take the initiative. There are a number of adverse neurological and metabolic adverse effects, including muscle stiffness, tremors, slowness in movement and thought, and akathisia (restlessness). Weight gain, increased risk of diabetes and cardiovascular disease are also common, and long-term use leads to shortened life span. Suicidality and sexual dysfunction are common adverse effects. Tardive dyskinesia or involuntary movements of the face, tongue, arms and legs is common, and may become evident, or be exacerbated, after withdrawal, reduction or switching medication. Withdrawal effects typically start within four days and may include symptoms such as nausea, headache, tremor, insomnia, decreased concentration, anxiety, irritability, agitation, aggression and depression. Rebound psychosis may also occur.

For ease of reference, the following table summarises the main adverse effects and withdrawal reactions for each class of prescribed psychiatric drug. For fuller lists and a review of the evidence (including references), please see sections 4 and 5.

Evidence box B: Summary of psychiatric drug effects by class.

Drug class	Effects that may be perceived as adverse	Possible withdrawal reactions
Benzodiazepines and Z-drugs E.g. Temazepam, Diazepam, Pregabalin, Gabapentin, Zoplicone	■ Sedative ■ Significant risk of dependence ■ Drowsiness and impaired cognitive ability	■ Sweating, nausea, dizziness, abdominal cramps ■ Anxiety, agitation, insomnia, muscle stiffness ■ Tingling, electric shock type feelings. Risk of epilepsy ■ Panic attacks, poor memory ■ Hallucinations, delusions ■ Nightmares
Antidepressants E.g. Fluoxetine, Paroxetine	■ Sedative ■ SSRIs/SNRIs: nausea, drowsiness, insomnia ■ Sexual dysfunction ■ Anxiety and agitation ■ Emotional blunting ■ Suicidality	■ Anxiety ■ Nausea, dizziness, insomnia. ■ Mood changes ■ Hallucinations ■ Vivid dreams ■ Confusion
Stimulants E.g. Ritalin	■ Insomnia ■ Growth suppression in children	■ Rebound effects, including tearfulness, irritability, emotional lability
Mood stabilisers E.g. Lithium Tegretol	■ Sedative ■ Drowsiness, tremor, lethargy, decreased ability to learn new information, prolonged reaction time, poor memory, reduced spontaneity ■ Weight gain ■ Reduced emotional responses ■ Toxic state: levels have to be regularly monitored	■ No physical withdrawal effects ■ Relapse or rebound of mania
Anti-psychotics E.g. Chlorpromazine, Haloperidol, Olanzapine, Risperidone	■ Sedative ■ Dampened emotional responses and motivation ■ Dizziness, sexual dysfunction, weight gain ■ Cardiovascular effects ■ Akathisia and extra-pyramidal effects ■ Tardive dyskinesia ■ Anticholinergic effects: dry mouth, blurred vision, constipation ■ Restlessness ■ Suicidality	■ Nausea, headache, tremor ■ Sleep disturbance, irritability, aggression, depression. ■ Possibility of 'supersensitivity', psychosis, particularly when withdrawing from clozapine ■ Rebound psychosis.

For fuller lists of possible drug effects and withdrawal reactions, please refer to sections 4 and 5.

3.2.1 Potential effects of taking prescribed psychiatric drugs on therapeutic work

The evidence detailed in section 4 suggests that research aimed at demonstrating the superiority of a combination of psychiatric drugs and psychotherapy over either intervention alone is not conclusive. Indeed, given the predominantly sedative effects of many prescribed psychiatric drugs, it is not unrealistic to suggest they can significantly and unhelpfully affect therapeutic work.[7] Therapists may find that prescribed psychiatric drugs act in ways that limit their emotional access to clients and the problems for which they are seeking help. Clients may feel 'out of reach' or emotionally cut off and their difficulties may seem vague or difficult to define. In addition, prescription psychiatric drugs have the potential to significantly alter the way clients think, feel and behave.

Effects on thinking may include: loss of memories; poor recall; poor concentration; confusion; losing track of ideas; difficulties in making links; difficulties in structuring thought; problems staying focused; and an inability to retain insights over time.

Effects on feeling may include: emotional withdrawal; being uninvolved, distanced or 'not really there'; inability to reconnect with feelings relating to past experiences; suppressed anger, sadness or fear; and a lack of emotional congruence.

Effects on behaviour may include: passivity with the therapist; passivity outside therapy sessions; uncooperativeness or over-compliance; denial of responsibility; absences due to lateness, cancellations or missed appointments; apparently poor motivation; repetitive speech or behaviour; and disengagement from work or social activities.

These effects will vary according to the particular drug, its dosage and the period of time over which it has been taken as well as the individual taking it. A picture will build up over time of how and in what way the client's life has been affected and shaped by taking prescription psychiatric drugs, bearing in mind that no-one is likely to display all of the above signs.

Given the above evidence for a range of effects and adverse reactions to taking or withdrawing from prescription psychiatric drugs, therapists may wish to consider a number of key issues when working with those who are currently taking, have previously taken or have now been advised to take these drugs. The following section invites therapists to consider questions relating to **reflexivity, evidence, context and ethics.**

3.2.2 Reflexivity: where am I in this?

Therapists will need to consider their personal position in relation to the medical model, reflecting on their own beliefs, values and attitudes towards prescribed psychiatric drugs together with any relevant personal or professional experiences that might have contributed to their stance. A complicating factor is that the widespread use of prescription drugs means it is possible, even likely, that therapists themselves will have been prescribed psychiatric drugs at some point in their lives. They may also have witnessed family members, partners or friends taking psychiatric drugs. Where this is the case, they may also wish critically to reflect on their own and others' experiences of such drugs and to consider how and to what extent this might impact on their therapeutic stance.

Question box 1: What do I feel about prescribed psychiatric drugs?

- What do I understand by the term 'medical model'?
- How does the medical model 'sit' with my preferred therapeutic modality?
- What position do I take up in relation to the medical model? Where do I locate myself?
- How does my professional training and clinical experience influence the way I understand and work with issues relating to taking or withdrawing from prescribed psychiatric drugs?
- Do I have any experience of taking prescribed psychiatric drugs myself? Am I aware of any family members or friends who have taken prescribed drugs?

- If so, what do I think and/or feel about these drugs, based on my own knowledge and experience?
- How might this facilitate or hinder a discussion with the client?
- Do I need to reflect on any of these issues in my own personal therapy? Do I need to discuss in supervision?

3.2.3 Evidence: what do I know?

Therapists will find it helpful to develop a basic understanding of the evidence relating to the possible effects of the main classes of psychiatric drugs, together with their withdrawal effects. This includes being aware of general information about tapering, such as the need to avoid any sudden cessation of psychiatric drugs when withdrawing. They may also find it useful to understand the likely impact of prescribed drugs on therapeutic work and how some withdrawal reactions can be mistaken for relapse back into psychological distress.

Question box 2: What evidence do I need to consider?

- Which drugs do I most commonly hear about from my clients?
- Am I familiar with the main classes of psychiatric drugs and what they are used for? (See section 4).
- Am I familiar with their common effects and withdrawal symptoms? (See sections 4 and 5).
- What do I know about the evidence for the impact of prescribed psychiatric drugs on therapy? (See section 4 per drug, and 3.2.1).
- Do I understand the importance of slow withdrawal or tapering strategies? (See section 5.4).
- What knowledge and skills do I need to best support my client?

3.2.4 Context: what are the key influences on my work and me?

Other issues will need to be considered in the light of each therapist's theoretical framework, work setting and personal and professional judgment.

It is important that therapists use the evidence base included in this guidance to develop their therapeutic understanding and skills in the light of their particular modality and professional context, as well as the particular needs of the client. As an example, therapists working within public sector services such as the NHS are likely to be expected to liaise where appropriate with prescribers, other mental health professionals and in some cases with partners, carers and relatives as well. Therapists working in independent practice may have less opportunity for such collaboration. Different theoretical models will also take diverse perspectives on the likely impact of collaboration on the therapeutic relationship. In these and other situations, therapists may wish to draw on the evidence-base to tailor their support of clients in ways that are appropriate to the particular model and context within which they are working.

Question box 3: What contextual issues do I need to consider?

- How does my preferred theoretical framework enable me to think about the role and function of prescribed drugs in my client's life?
- Given my preferred therapeutic model, what position do I take up in relation to working with other health professionals if requested by the client?
- Should I consider liaising with the client's prescriber? Given my current workplace, what are the possible channels of communication with other people involved in the care of my client?
- How does my preferred framework influence whether I signpost information where this is in the best interests of the client?

- Might it be helpful to find out more about multidisciplinary models of work in cases of prescribed psychiatric drug withdrawal? (See section 6.2)
- What is the likely impact of contact or collaboration with others on the therapeutic relationship?
- Should I consider signposting the client to further relevant information or evidence about their drugs?
- Should I consider referring the client to specialist agencies or other forms of support?
- What is the likely impact of such a referral on the therapeutic relationship?

3.2.5 Ethics: what are the principles that might apply to this issue?

Finally, working with issues of prescribed drug dependence raises legal and ethical questions relating to the importance of therapists working within the boundaries of their professional competence and role. It may be useful here to clearly distinguish between medical *advice* and medical *information*. Whilst it is clear that psychological therapists are neither trained to issue medical diagnoses nor to prescribe medical or pharmacological treatment, they may frequently be asked by clients for medical *information*. Discussing facts, scientific evidence or information where appropriate with clients differs substantially from offering a diagnosis, prescribing drugs or advising withdrawal. It is important to be clear about this distinction with clients.

Let us consider the difference between offering information to clients (sometimes called psycho-education) and giving them advice. As therapists, we may prefer to talk with clients about the common features of – and helpful reactions to – a panic attack rather than telling them what they should or should not do. The former is a common therapeutic strategy that enables therapists to help clients think about and understand the range of options available. It allows the client to decide what they feel is best or most helpful for them.

The latter places the therapist in the position of 'expert' and may risk undermining the client's autonomy and decision-making capacity. In the same way, the therapist who offers general information about the effects of psychiatric drugs is not offering any specific advice about 'what to do' but is rather providing information on the ethical basis of 'informed consent'. Clients can then decide for themselves how best to proceed.

Clearly, this process is not always straightforward, and will be dependent on a number of factors:

- The skill of the therapist in engaging the client in ways that support them to make informed decisions i.e. decisions based on understanding the benefits and risks of any proposed psychiatric drug treatment.
- The capacity of the client to engage in decision-making processes, which will vary according to their personal circumstances, history of psychological problems and current level of distress.
- The tendency of clients to favour interventions that claim immediate relief for their emotional distress, rather than longer-term interventions whose future outcome may appear less certain. This bias arguably skews the entire informed consent process, no matter how conscientiously implemented.
- Additional processes and care will be required where a client lacks the mental ability to make informed decisions about what is best for them.

It remains the case that there is currently no specific legal or ethical guidance on how therapists should respond to issues relating to taking or withdrawing from prescribed psychiatric drugs. This means that general ethical principles provided by all the main professional accrediting bodies will remain an important touchstone for their therapeutic practice and therapists may need to consider which principles are likely to be particularly relevant when working with those who are taking or withdrawing from prescribed psychiatric drugs.

For example, BACP's *Ethical Framework* (2018)[8] covers the following areas:

- Working on the basis of informed consent. Helping the client to understand the potential impact of their prescribed psychiatric drugs on the therapeutic process can be seen to be part of the therapist's responsibility to ensure the client's informed consent within therapy. This should be clearly distinguished from the prescriber's responsibility to inform the client about the physiological and psychological effects of their prescribed drugs. However, it may be helpful for therapists to support this process where appropriate, for example by directing clients to relevant sources of information.
- Respecting the client's best interests. This includes supporting the client to take action, or where necessary, for therapists to consider doing so themselves, in order to prevent significant harm to the client or others.
- Keeping knowledge and skills up to date. This may include therapists referring to the evidence-base included in this guidance and supplementing their competence in the areas proposed.
- Demonstrating accountability and candour. This includes being open and honest with clients about the potential problems or risks associated with dependence on or withdrawal from prescribed psychiatric drugs.
- Working respectfully with colleagues. Whilst it is important that therapists do not undermine a client's relationship with other colleagues or prescribers, they may need to be prepared to support clients where they have had unhelpful experiences or advice.

Although the ethical frameworks of the UKCP, BPS and NCS also endorse these ethical principles, therapists will need to reflect on and apply them to their own particular therapeutic practice and professional work setting, as well as taking any associated organisational policies into account.

All psychological therapists should be aware that working with issues of prescribed drug dependence is becoming an increasingly contested and fast-moving field of practice. The rapid growth of scientific knowledge can make it difficult for professional guidance, including medical guidelines, to keep pace with the speed of change, leading to the potential for significant differences of opinion between those who are caring for the client. Where therapists disagree with a prescriber's medical advice to the client (e.g. which they believe to rest on erroneous or out-dated medical information), they may, with client consent where possible, consider contacting the prescriber to raise their concerns. However, differences in professional expertise, as well as variations in how a patient presents can also lead to well-founded disagreements within or across teams and disciplinary divides and practice settings. Therapists will need to be mindful of the need to communicate thoughtfully, sensitively and courteously with other professionals whilst prioritising the best interests of the client at all times (see Note in 4.3 below).

Question box 4: What ethical and legal issues do I need to consider?

- Am I aware of the distinction between medical *advice* and medical *information*?
- How might I ensure that my client does not interpret any information giving as advice?
- Am I aware of the relevant principles and ethics of professional practice recommended within my current professional accrediting body? E.g.:
  - working on the basis of the client's informed consent
  - respecting the client's best interests
  - taking steps to keep my knowledge and skills up to date
  - demonstrating accountability and candour
  - respecting the client's autonomy and self-determination.

3.3 Practice-related guidance for therapists

It is not possible for this guidance to address all the possible implications of taking or withdrawing from prescribed psychiatric drugs, for all therapeutic practice, in all contexts. Rather, the intention is to promote critical thinking and awareness of the impact of prescribed psychiatric drugs, and for therapists to extend their competence by considering issues particular to their own clients and practice settings.

This part of the guidance is divided into three main sections for ease of reference. Each section addresses issues that are relevant to the client's drug 'journey', i.e. where the client is in relation to taking prescribed psychiatric drugs. The sections are as follows: a) clients who are considering a prescription for psychiatric drugs; b) clients who are already taking prescribed psychiatric drugs; c) clients who are considering withdrawing from their prescribed drugs; and, d) clients who are currently withdrawing from prescribed drugs and who may be experiencing withdrawal effects.

In each section, a number of key information areas are highlighted alongside links to relevant sections in the guidance that provide further material, resources and/or evidence for therapists to consult. Implications for the client and for therapy are also discussed. At the end of each section there are a number of practice-related questions for therapists to consider. These are designed to help therapists think critically about their therapeutic work and its particular context, and are not necessarily to be asked of clients. Given the considerable differences within and between theoretical frameworks, these questions are intentionally broad, aiming to help therapists reflect on their personal knowledge, skills and experience in working with clients who have issues relating to taking or withdrawing from prescribed psychiatric drugs.

Note 1: Working with prescribers and family members or carers

Throughout the guidance, therapists are encouraged to consider if, when and how it might be appropriate to contact prescribers. It is clear that there can be no hard and fast rules here about the best course of action where therapists are concerned about a client's use of or withdrawal from prescribed psychiatric drugs, and in many cases therapists may decide against such contact. The decision to get in touch with a prescriber will inevitably be a function of multiple, overlapping factors: whether contact is at the request of and in the best interests of the client; whether the client has consented to the therapist making contact; the therapist's preferred therapeutic model and rationale for communicating – or not – with the prescriber concerned; the work context in which therapy is taking place; and the therapist's own confidence in and previous experience of initiating contact with prescribers and other medical professionals.

Where contact with the prescriber is considered appropriate and where the client has given consent, a short email to request a discussion or meeting can be helpful, followed up where necessary by a telephone call or message. For therapists working in public sector services like the NHS, such communications are usually straightforward, particularly where therapists are working side-by-side with prescribers. Where it proves difficult to contact prescribers, it may be necessary for the therapist to discuss their concerns with colleagues and/or supervisors. Where appropriate, they may wish to consider bringing their concerns to a multidisciplinary team meeting for discussion (details of models for supporting withdrawal in multidisciplinary teams can be found in 6.2). In other settings such as independent practice, communication with prescribers is frequently more complex and will be dependent on therapists obtaining the GP or prescriber contact details. Where possible, therapists can email or write to request a conversation or meeting, indicating their professional qualifications and role together with their reasons for being concerned about the client. Following initial contact, therapists may need to be prepared to maintain communication particularly where the client is withdrawing from prescribed drugs.

Therapists are also encouraged to consider whether it might be appropriate, with the client's consent,

to be in contact with carers or family members such as partners or other relatives. In the case of some older adults, or those with learning disabilities or communication difficulties, carers, partners and families are likely to be involved in supporting the client. In some work settings, particularly within public sector services, collaboration with family members and carers is seen as a relatively straightforward element in therapeutic work. In other settings, such as independent practice, there is less opportunity or need for contact and collaboration. Therapists will need to consider carefully, from the perspective of their preferred therapeutic framework and practice setting, the range of issues and implications associated with contacting and working with carers and/or family members.

Note 2: Working with the beliefs clients hold about prescribed psychiatric drugs

Therapists may also wish to explore the beliefs clients hold about taking prescription psychiatric drugs, as well as the psychological 'message' that a pharmacological intervention inevitably carries. For example, some people believe, or have been told, that depression, anxiety and other psychological problems are caused by biochemical changes to the brain, while others believe there are genetic factors underlying their emotional distress. In these cases, prescription psychiatric drugs carry a strong psychological message for the individual that they are 'ill' and require medical 'treatment' in order to manage. Indeed, where clients believe that they are 'weak' or have failed to live up to social expectations and norms, it may be preferable for them to treat what they believe to be the physical or biochemical causes of their distress rather than to explore painful life experiences or interpersonal dynamics that may be contributing to the problem. Clients may also believe that it is not good for them to experience strong feelings of distress, and that prescribed psychiatric drugs will quickly and effortlessly get rid of feelings of sadness or anger. In these and many other situations, therapists will need to take into account the beliefs and meanings held by the client, exploring any unrealistic or over-optimistic expectations about prescribed psychiatric drugs that prevent the client from accepting an alternative view of their difficulties, which would in turn enable therapy to be of more benefit.

The relationship that clients have with their prescribed psychiatric drugs becomes more complex where drugs are taken as a consequence of being detained under the Mental Health Act or being treated under a Community Treatment Order (CTO). In these circumstances, therapists will need to be alert to the way in which pharmacological treatments are likely to impact on the therapeutic relationship, working with clients to support them within the limitations imposed by legal frameworks. Difficulties are also likely to arise where clients rely on prescription psychiatric drugs to demonstrate eligibility for benefits such as Employment and Support Allowance (ESA). In these situations, therapists will need to explore sensitively and with care the extent to which anxiety about any possible loss of benefits underpins the client's understanding of the causes of their emotional distress and drives any decision about withdrawing from prescribed psychiatric drugs. Therapists should also bear in mind the extensive debates in the field about the overprescribing of psychiatric drugs in marginalised groups, including those from black and ethnic minority backgrounds. For further information about this, it may be helpful to consult the British Psychological Society's (2017) *Understanding Psychosis* document.

3.3.1 Working with clients who are considering a prescription for psychiatric drugs

Useful information for therapists to know:

- Main effects of the proposed psychiatric drug (section 4).
- The potential risks of drug dependence (sections 4 and 5).
- The likely impact of the proposed prescribed drug on therapy (section 4 by drug and 3.2.1 above).

a) Implications for the client

- Based on the principle of informed consent, therapists may wish to enquire whether the client's prescriber has discussed with them the possible effects of or potential for dependence on the proposed psychiatric drug. If not, they can encourage the client to discuss this further with their prescriber. Therapists may also need to ensure the client is aware of the potential impact of taking the proposed drug on the process and progress of therapeutic work.
- Therapists should be aware of the implications of prescribed psychiatric drugs for working with particular groups of clients. For example, older adults who have diminished physical or cognitive capacities may be at increased risk of falling whilst taking prescribed drugs. Clients who are pregnant or planning a pregnancy may incur risks to the unborn child. In these and other cases, therapists are well-placed to encourage the client, where appropriate, to discuss the potential impact of prescribed psychiatric drugs with their prescriber in order to ensure they are making an informed choice about the use of such drugs.

b) Implications for therapy

- Therapists will need to explore sensitively and with care the client's perception of his or her psychological problems. It is important to judge whether the client wishes, or is ready, to talk about any issues associated with their planned use of prescribed psychiatric medication.
- Therapists should consider whether and to what extent the client's planned use of prescription psychiatric drugs is likely to affect therapy. Where possible, it is helpful to address issues around psychiatric drug use at an early point in the therapeutic relationship in order to better assess its likely impact on successful therapeutic work.
- Where clients directly ask therapists for advice concerning prescribed drugs, therapists will need to ensure they do not offer any personalised suggestions about the advisability or otherwise of taking prescription psychiatric drugs. They should not be drawn into discussions about the type, dosage or frequency of any drugs that the client's prescriber has recommended, and should always refer the client back to their prescriber for medical advice, remaining alert to any reluctance on the part of the client to question their prescriber. Acting on the principle of informed consent, therapists may wish to explore any concerns the client may have about their prescribed drugs and where appropriate direct them to relevant available sources of information (e.g. BNF) or evidence in a sensitive and non-leading manner (see 3.2.5).

c) Practice-related questions for therapists to consider

Question box 5

- What does the client think and feel about taking prescribed psychiatric drugs?
- Why might the client wish, or feel they need, to accept (or not) a prescription?
- What is the likely impact of the proposed drug on the client's ability to engage in psychological therapy?
- Is the client directly requesting advice about drugs? If so, can I support their agency in relation to the prescription? Do they need more information?
- How can I best support the client's choice either to start their prescribed drugs or to revisit the GP/prescriber to consider alternatives to drugs?
- Is therapy appropriate for the client at this time, or is referral to another service required?

3.3.2 Working with clients who have already started taking prescribed psychiatric drugs

Useful information for therapists to know:

- Main effects of client's prescribed psychiatric drug (see section 4).
- Impact of prescribed drugs on therapy (section 4 by drug and 3.2.1 above).
- Risks of abrupt discontinuation, reduction or switching prescribed drugs.

a) Implications for the client

- Clients may experience a range of effects whilst taking their prescribed psychiatric drugs. If therapists are familiar with some of the adverse effects of the main classes of psychiatric drugs (e.g. benzodiazepines and antidepressants) they may be able to help the client identify if and when they might be experiencing them.
- Taking prescribed psychiatric drugs may have significant implications for the client's partner, family, carers or other people involved in their care. This may be of particular relevance for older adults and those with learning disabilities or communication problems. Therapists will need to consider carefully, from the perspective of their work setting and preferred therapeutic framework, the range of issues associated with contacting and working with carers and/or family members (see Note 1, 3.3).

b) Implications for therapy

- Therapists will need to explore sensitively and with care the extent to which the client wishes, or is ready, to talk about any issues associated with their use of prescribed psychiatric drugs. In some cases, therapists may decide that it is not in the client's best interests to start therapy and instead may choose to refer the client to alternative sources of help and support. However, given the lack of currently available services, therapists should remain cautious about assuming other professionals are better able to offer emotional or psychological care. Depending on the type and level of prescription psychiatric drugs taken by the client, therapists are generally well-placed to offer support, though it may be necessary to adjust therapeutic expectations of what kind of work will be possible.
- Clients may make a 'late reveal' in therapy that they are or have been taking prescribed drugs for some time but have not previously been able or willing to discuss this. In some cases where the client's prescription drug use is known but has not been discussed, the therapist may make a decision to raise it as an issue where previously it had not been part of the work, if in the interest of the therapy.

c) Practice-related questions for therapists to consider

Question box 6

- If the client's prescribed drug use was not raised at the start of therapy, why has it been raised now?
- Why might the issue of prescribed drugs be significant within the therapy at this particular time?
- What are the implications of taking prescribed drugs for the progress of therapy?
- What is the client's relationship with their prescriber?
- How can I support the client to contact their GP, psychiatrist or other prescriber? Might it be helpful for me to do so?
- Does the client want me to contact any family members, carers or others who may be involved in the client's care? What are the implications of this for the therapeutic relationship?

3.3.3 Working with clients who are considering withdrawing from prescribed psychiatric drugs

This guidance aims to empower and support conversations often already taking place between therapists and their clients. Therapists will need to decide for themselves whether, and to what extent, they wish to use this guidance in the context of their therapeutic work. These decisions will depend on their theoretical modality, practice setting and the individual needs of the client. The client's agency, as always, should be supported and respected at all times. Clients should be encouraged to discuss withdrawal from prescribed psychiatric drugs with a knowledgeable prescriber who can give medical advice, oversee and manage any withdrawal process appropriately. While this guidance advocates the importance of informed client-choice based on full information about potential benefits and risks,

it does not advocate therapists telling their clients to take, not take, stay on or withdraw from psychiatric drugs. These matters should be left to the prescriber and client to decide.

During the course of therapeutic work, clients may consider withdrawing from their psychiatric drugs and either moving to therapy alone or ending all interventions if they are feeling better. In these cases, it will be helpful if therapists are aware of the following:

- The process and possible experiences of withdrawing from prescribed psychiatric drugs.
- Awareness of the importance of planning for withdrawal: preparation, timing, knowledge and support.
- Understanding the likelihood of withdrawal effects.
- Understanding the potential impact of withdrawal on the client's family and other social networks.
- Understanding the importance of the client having informed medical support and supervision during withdrawal.
- Key definitions about relapse and withdrawal.

Box C: Evidence summary – useful information for therapists to know

Although there is a lack of formal research into the effectiveness of therapeutic strategies aimed at supporting withdrawal, the theoretical, experiential and anecdotal evidence from those working in this field nonetheless offers useful suggestions. What follows is a summary of the 'combined wisdom' from these sources (for full details, including references, see section 6).

There are five relevant factors that have been found to be helpful in supporting withdrawal. These are:

1. Access to accurate information about withdrawal.
2. The involvement of a knowledgeable prescriber to devise, help monitor and manage, a tapering programme that is tolerable and agreeable to the client.
3. Access to client-centred, non-authoritarian support.
4. Access to information about and help with engaging with useful coping strategies and/or supportive lifestyle changes.
5. Awareness of the need to suspend customary assumptions about sources of distress and their associated interventions (i.e. emotional processing or analysis) for the duration of withdrawal.

The 'combined wisdom' approach

The combined wisdom of those therapists who have worked in depth with this client group describes three stages of support:

Stage 1: Preparation before withdrawal is started

Preparation is essential to successful withdrawal. Understanding the withdrawal process, alongside a stance of non-judgmental acceptance, allows the therapist to engage the client in a discussion about the advantages and disadvantages of withdrawal. Ten areas to consider reviewing with the client are:

- Exploring whether a client feels ready to begin the withdrawal process.
- Exploring who is going to provide medical support, and their relationship with their prescriber.
- Signposting and discussing relevant information on withdrawal (*see list of examples below).
- Discussing the possibility and general nature of withdrawal effects so clients know what to look for.
- Clarifying the high-level definitions of relapse, rebound, recurrence and withdrawal and how they might be mistaken (e.g. adverse withdrawal reactions that result from reducing or discontinuing a drug might be mistaken as 'relapse', a term which refers to the gradual return of the original issue, at the same intensity, for which the drug was initially taken – see 5.4.2).
- Addressing any potential fears about the withdrawal process.
- Identifying possible ways the attempt might be inadvertently sabotaged.
- Identifying potential support networks.
- Discussing the idea of the client using a diary or log to keep track of drug reductions and experiences.
- Discussing the availability of extra sessions or other contact if needed in between scheduled meetings, being clear about the limits of what can be provided.

* Examples of information about withdrawal that may be shared with the client if appropriate (see 5.4.1 for fuller information):

- Withdrawal from prescribed psychiatric drugs should be carefully planned and carried out under the supervision of an informed prescriber.
- Withdrawal should never be sudden or abrupt; people's experience can vary significantly, with some experiencing no withdrawal reactions whilst others can experience severe and protracted withdrawal.
- Schedules should be flexible and the reduction rate based on the individual's withdrawal reactions, intensity of reactions, their ability to cope and whether there is sufficient support available. Drugs may need to be tapered very slowly over months or beyond.
- Where reactions to withdrawal are severe, it is sometimes possible for a client to get a liquid prescription from the GP/prescriber. This helps to ensure accuracy in making small reductions to the prescribed drug.

Further details about the 'combined wisdom' approach, including references, can be found in section 6.

a) Implications for the client

- Clients may not have considered the possibility of withdrawal reactions, nor of the need to prepare for withdrawing from their prescribed psychiatric drugs. Indeed, where clients are planning to finish therapy and subsequently to withdraw from their prescribed drugs because they feel better, they may not have considered how a therapist could support them in the withdrawal.
- Withdrawing from prescribed psychiatric drugs may have significant implications for the client's partner, family, carers or other people involved in their care. This may be of particular relevance for older adults and those with learning disabilities or communication problems.

b) Implications for therapy

In addition to reviewing the information outlined in box C:

- Therapists should be aware that withdrawing from prescribed drugs requires planning and preparation and may take some time. The process of withdrawal itself can take months or years, not days or weeks. A rushed or unplanned withdrawal process is less likely to succeed.
- While it is beyond the professional remit of psychological therapists to give direct, personalised withdrawal or tapering advice, therapists may wish to consider in advance their position on giving or signposting relevant information to clients. This may be particularly important if the relationship between client and prescriber is problematic or has broken down.
- Adopting a stance of non-judgmental acceptance allows the therapist to engage the client in a discussion about the advantages and disadvantages of withdrawal.
- Where relevant, therapists will need to consider carefully, from the perspective of their work setting and preferred therapeutic framework, the range of issues associated with contacting and working with carers and/or family members (see 3.3, note 1).

c) Practice-related questions for therapists to consider

Question box 7

- If the client wishes to withdraw from their prescribed drugs, why now? What has precipitated their decision?
- Has the client discussed their decision to withdraw with his or her prescriber?
- What is the client's relationship with their prescriber?
- Does the client have a plan for withdrawal?
- How can I support the client to contact their GP, psychiatrist or other prescriber? Might it be helpful for me to do so?
- Does the client want me to contact any family members, carers or others who may be involved in the client's care? What are the implications of this for the therapeutic relationship?

3.3.4 Working with clients who are currently withdrawing from prescribed psychiatric drugs

Clients may already have started to withdraw from their prescribed drugs before starting work with a therapist. Some may not wish to tell a therapist that they are doing so. In these cases, it may be useful for the therapist to consider the following, in addition to the information given in section 3.3.3:

a) Implications for the client

- If a client has chosen to start withdrawing without talking to a prescriber or researching how to taper, any information given may come as a surprise. Discussing the helpfulness of informed medical support and supervision during withdrawal will need to be done in such a way as to not undermine the client's agency.
- Clients may have a range of experiences when withdrawing from prescribed psychiatric drugs (summarised in section 3.2, full information in section 5). Withdrawal reactions such as anxiety, agitation or insomnia, especially those that continue past the acute stages are commonly

Evidence box D: Summary – useful information for therapists to know

The 'combined wisdom' approach
As introduced in 3.3.3 the combined wisdom of those therapists who have worked in depth with this client group describes three stages of support. The second and third stages are as follows:

Stage 2: During withdrawal – support
Therapists are likely to have more regular contact with a client than a prescriber. They are therefore in a strong position to offer the client ongoing support for the withdrawal process. Possible areas for therapeutic work may include:

- Helping clients to identify withdrawal reaction and offering reassurance that they will pass. It is important to assume that any reactions that emerge during the transition are due to withdrawal unless proven otherwise.
- Encouraging clients to make sense of their experiences and to accept them as normal to the withdrawal process. For example, clients may experience intense anxiety and fluctuating levels of physical and mental pain.
- Helping clients to manage withdrawal reactions that can come and go. This is sometimes referred to as 'waves' and 'windows', where the 'waves' of reaction slowly decrease in intensity, interspersed with 'windows' of reduced or very limited reactions. Some clients may only experience 'waves' within 'waves'.
- Helping clients to identify supportive practices which enable them to manage and tolerate withdrawal experiences while they last. These may include coping strategies such as: acceptance; maintaining a non-resisting attitude to withdrawal experiences, or breathing exercises. (For the full range of potential coping tools, see 6.1.1.1).

Stage 3: After withdrawal is complete

- At the end of withdrawal, therapists may find it useful to review the client's experience and to determine with them what further therapeutic needs they have. In addition:
- If the client has experienced any cognitive problems as a part of their withdrawal experience it may take a while for confidence in decision making to rebuild (including the ability to say 'no' to others).
- If the clients' withdrawal was experienced as traumatic this might need to be considered in any further therapeutic work.
- Both client and therapist should be aware that post-withdrawal reactions can occur for some time after stopping taking prescribed psychiatric drugs.

Working in multidisciplinary teams

- There are examples in the theoretical and research literature of psychiatrist-led models to support withdrawal that may be of interest for further reading if a therapist has an opportunity to suggest this in a multidisciplinary team setting (see 6.2).

Further details about withdrawing from prescribed psychiatric drugs, including references, can be found in sections 5 and 6.

assumed by clients and their prescribers to signal a return of the client's psychological problems and to require further medication. In such cases, therapists will need to work with clients to help them understand their experiences of withdrawal as physiological rather than psychological in origin, and to agree what is realistic therapeutically during the process.

- If a client experiences protracted or severe withdrawal reactions they will naturally need to adjust their expectations of the withdrawal process and how long it might take. They may also need to consider more fully what support is available to them from family and friends, or from a continued relationship with a therapist.
- Withdrawing from prescribed psychiatric drugs may have significant implications for the client's partner, family, carers or other people involved in his or her care.
- Where clients have been sedated and inactive due to long periods of drug use, they may need to find new and more satisfying ways of occupying themselves.

b) Implications for therapy

In addition to those elements described in Box D, and always dependent on the therapist's theoretical framework, therapists should also consider the following elements that may form part of therapeutic work over the withdrawal period:

- If there was no opportunity to work with the client to prepare for withdrawal, therapists should consider the list of 10 areas listed in Box C to see if any would be still helpful to address.
- If clients experience 'waves and windows' during the withdrawal process (see Box D) where reactions fluctuate over time, therapists can help monitor the course of these episodes if they happen, providing the client with support and information and tailoring therapeutic work appropriately.
- Whilst it is clear from Box D that any reactions that emerge during the transition should be treated as a result of withdrawal unless proven otherwise, it is possible that new emotions may also emerge. The therapist may need to

consider these feelings carefully together with the client, deciding whether they are further material for therapeutic work and if so, when they might be best addressed.

- Where the client is unable to process emotional material due to high levels of anxiety or physical/mental pain or discomfort, it will be necessary for the therapist to revisit any previously agreed therapeutic aims in order to provide support, guidance and reassurance.
- The therapist will need to anticipate, discuss and work through potential problems, feelings or setbacks with the client. It is important to maintain an accepting and non-judgmental therapeutic stance, identifying risks in the event of the client becoming emotionally unsafe.
- If there are concerns about prolonged or adverse reactions during withdrawal, therapists should consider discussing with the client the advantages and disadvantages of seeking advice from the prescriber and/or other mental health professional.
- Therapists will need to encourage the client's sense of responsibility and autonomy whilst remaining clear about the support they are able to provide.
- As for 3.3.3, therapists will need to consider carefully, from the perspective of their work setting and preferred therapeutic framework, the range of issues that arise when asked to contact and work with carers and/or family members (see 3.3, note 1).

c) Practice-related questions for therapists to consider

Question box 8

- Am I aware of the 'combined wisdom' approach in relation to withdrawal strategies? (Boxes C & D).
- Does the client want me to contact his or her GP, psychiatrist or other prescriber?
- It may not be possible to distinguish between withdrawal symptoms and any re-emergence of the client's presenting psychological problem. Can I tolerate my own and the client's uncertainty about this?
- Is the client aware of the potential impact of withdrawal from drugs on existing relationships such as family, partners and colleagues?
- What might I need to do or change in my therapeutic practice to accommodate the client's withdrawal reaction distress?
- What additional relevant tools or strategies might be helpful, and how might these impact the therapeutic relationship? Which do I know enough about to provide information on, and which would I need to simply signpost for the client?
- Do I need to consider additional therapeutic support for the client? Where would this come from?

This guidance aims to empower and support conversations often already taking place between therapists and their clients. Therapists will need to decide for themselves whether, and to what extent, they wish to use this guidance in the context of their therapeutic work. These decisions will depend on their theoretical modality, practice setting and the individual needs of the client. The client's agency, as always, should be supported and respected at all times. Clients should be encouraged to discuss withdrawal from prescribed psychiatric drugs with a knowledgeable prescriber who can give medical advice, oversee and manage any withdrawal process appropriately. While this guidance advocates the importance of informed client choice based on full information about potential benefits and risks, it does not advocate therapists telling their clients to take, not take, stay on or withdraw from psychiatric drugs. These matters should be left to the prescriber and client to decide.

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4. What psychiatric drugs do by class

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4.1 Interpreting the evidence on psychiatric drugs

The most robust evidence for the use of psychiatric drugs is generally agreed to come from randomised controlled trials that compare a particular drug or intervention with a standard or 'control' condition, such as a placebo. Randomisation is important because it allows the effects of the intervention being tested to be distinguished from the effects of other things, such as the natural history of the condition and general factors that might produce improvement like seeing a specialist. To further reduce the risk of bias, the investigators and participants may be 'blinded', or made unaware of who receives the drug and who the control treatment or placebo.

Combinations of the results of several different trials of the same treatment, called meta-analyses, are also regarded as providing high quality evidence. However, a meta-analysis is only as good or as poor as the trials it combines. A meta-analysis of poorly conducted trials summates their deficiencies or biases and so the result may be more misleading than the original studies.

Randomised controlled trials were developed to test the outcomes of interventions for physical medical conditions. Translating them into the area of mental health is not straightforward and there are various difficulties with interpreting the results.

4.1.1 The validity of measurements

Emotional states and behaviours are properties of living human beings and cannot be described and quantified in the same way that we measure the properties of physical objects. Therefore, the meaning and validity of measurements of mental symptoms is not clear-cut.

4.1.2 Ignoring drug-induced alterations

Since most research is premised on the disease-centred model of drug action, the general alterations that drugs produce on physical and mental functioning are often ignored and interpreted as changes in the underlying 'disorder'. Yet these alterations may change people's experience and behaviour without affecting the underlying problem.

4.1.3 'Publication bias'

Studies that find positive effects of drugs are more likely to be published than studies that find they have no benefits or cause harm.[8] In addition, published reports of studies often emphasise the measures that show the drug in the best light.[8] Measures that show no benefit or that indicate harmful effects may not be published or may be concealed in the small print of the article.

Some pharmaceutical companies have been shown to withhold data that do not show their drug in a favourable light.[9] But doctors, researchers and editors have also played a part in focusing on research that highlights the positive and plays down the negative effects of drugs. There are extensive financial relationships between these groups, that have been shown to bias the undertaking, interpretation and reporting of research.

4.1.4 Unblinding

The use of a placebo is meant to prevent participants and researchers from knowing whether they are getting the real drug or not. This is why studies using a placebo are referred to as 'double blind'. However, it is often quite easy for people in trials to tell whether they are taking the

drug or the placebo, due to the mental and physical alterations drugs produce independently of any effect they might have on the underlying disorder. The chance that people will detect whether they are taking a drug or a placebo is heightened because people who take part in trials are given detailed information about the 'side' effects of the drug being tested.

What this suggests is that many trials that are supposed to be double blind are not. Many of the participants and some of the professionals involved are likely to be able to work out who is taking the real drug and who is on the placebo. Trials in which people are asked to guess what they are taking show that in most cases people can detect the nature of the pill they have been given.[10] If people taking part in trials believe that drugs are likely to help them, they may have a heightened expectation of improvement if they suspect they are taking the real drug. Conversely, they may have lowered expectations if they believe they are on the placebo. Any differences in the outcome of treatment may be due to these different expectations, rather than the effects of the drug.

4.1.5 Drug withdrawal effects in trials

Most trials of long-term treatment, and many trials of short-term treatment too, involve people who are already taking the drug that is being tested, or something similar. The people who are randomised to placebo are then taken off their existing treatment and may therefore be vulnerable to adverse effects related to the withdrawal of the prior treatment.[11] This is especially problematic because the withdrawal and transfer to placebo is usually done abruptly. Therefore, many studies, particularly those assessing long-term treatment, may assess the effects of withdrawing from prescribed drugs rather than the impact of starting on it in the first place.

This array of potential problems suggests that care must be taken when interpreting research on psychiatric drugs, and the clinical guidelines based upon them.

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4.2 Antidepressants

4.2.1 History

During the 1950s, certain drugs were tried out on people who were depressed, which started to be called antidepressants. One group of these drugs, which are similar in structure to some of the early antipsychotics, is known as the tricyclic antidepressants. Another group is the monoamine oxidase inhibitors or MAOIs. These were the main types of antidepressant used until the late 1980s. Prozac was launched in 1988 and was the first of a series of new antidepressants introduced during the 1990s called the 'selective serotonin re-uptake inhibitors' (SSRIs). These were joined by a variety of other sorts of drugs also branded as antidepressants (including venlafaxine, duloxetine and mirtazapine).

From the beginning of the 1990s, industry advertising campaigns and professional publicity increased the prescribing of these drugs substantially. Antidepressants are now by far the most commonly prescribed class of psychiatric drug, and their use continues to rise. In 2016 in England, over 65 million prescriptions were issued for antidepressants, a 6% increase on the previous year and over 500% increase since 1992.[1]

Antidepressants are regarded as useful treatments for depression and a range of other conditions and their use is recommended in various situations by official guidance.

4.2.2 Common short-term uses

Antidepressants are recommended for what is judged to be moderate or severe depression, and for less severe depression that is not helped by psychological interventions[1]. SSRIs are usually the first choice.

People diagnosed with depression who recover after being prescribed an antidepressant for the first time, are generally advised to continue the drug for at least six months.[2] Antidepressants are usually prescribed for a longer period if the patient has suffered several episodes, the symptoms have not disappeared entirely, the person has a long-term physical health condition, or has ongoing life stressors. In such cases, antidepressants are recommended to be continued for a minimum of two years[2], but increasingly people end up taking these drugs for multiple years and beyond.

They are also prescribed for individuals who have received a diagnosis of a variety of other mental health difficulties, including anxiety, obsessive compulsive disorder, panic disorder, phobias, bulimia and post-traumatic stress disorder. Tricyclic antidepressants are sometimes also used to treat chronic pain, particularly pain of a neurological origin, or insomnia, usually at lower doses than those recommended for depression.

4.2.3 Theories of action

The traditional view of antidepressant action, based on a disease-centred model (outlined in section 2), suggests that antidepressants help correct a chemical imbalance presumed to be present in depression. They are said to increase the availability of certain neurotransmitters that are thought to be deficient in depression. Older drugs, like the tricyclic antidepressants and the MAOIs are thought to act by increasing the availability of the neurotransmitter noradrenalin. The SSRIs are still generally believed to improve depression by correcting a deficiency of serotonin.

Although the idea that depression is caused by a chemical imbalance has entered the public consciousness, the 'monoamine' theory of depression is not supported by evidence or expert opinion.[3,4] Studies of serotonin receptors, for example, show contradictory findings, with some showing that receptor numbers are reduced in people with depression, compared to people without, some showing no difference, and some showing they are increased. Studies that aim to induce a lowering of serotonin levels through dietary means do not show any association with the onset of depression in people with no history of depression, although some studies show a deterioration of mood in people with a

previous history of depression who have been treated with SSRI antidepressants. Evidence on noradrenaline is also contradictory.[5] In addition, numerous randomised trials have shown that drugs that are not thought of as antidepressants, and have actions on other neurotransmitter systems, including benzodiazepines, opiates, stimulants and antipsychotics, are as effective as recognised antidepressants in people with depression.[5] Leading psychopharmacologists have concluded that direct evidence for the monoamine hypothesis is lacking.[6,7] Indeed, the whole 'chemical imbalance' theory of depression is now dismissed as overly simplistic by academic psychiatry.[8] Some official sources continue to suggest that antidepressants work by increasing 'levels of chemicals in the brain' that are linked with depression[8], but others, such as the Royal College of Psychiatrists public information leaflet, no longer mention reduced serotonin as a potential cause of depression.[9]

The drug-centred model as outlined in section 2, suggests that antidepressants produce mental and physical alterations, which interact with the symptoms of depression. These may potentially account for certain differences between antidepressants and placebo in randomised trials. For example, the sedation produced by older antidepressants may be experienced as helpful by some people with anxiety and insomnia, while the emotional numbness induced by some antidepressants may reduce the intensity of negative feelings for some. The mental and physical alterations may also reveal to people participating in randomised trials that they are taking an active drug, increasing the placebo effect.

4.2.4 Drug effects

There has been little effort to characterise how antidepressants alter normal physical and mental functioning.

Antidepressants come from many different chemical classes, and therefore can be expected to vary in the effects they produce. Tricyclic antidepressants, for example, appear to be pharmacologically similar to some of the older type of antipsychotics. They are strongly sedating drugs. They increase sleep and cause drowsiness during the day. Studies with healthy volunteers show that taking tricyclic antidepressants makes people slower in their reactions and impairs intellectual abilities such as attention and memory. Taking them is usually an unpleasant experience for volunteers (it is associated with 'dysphoria' in volunteer studies).[14,15]

SSRIs have more subtle effects in volunteer studies apart from their effects on the gut (most of the body's serotonin is present in the gut). They commonly cause nausea and sometimes diarrhoea and vomiting. SSRIs also commonly produce mild drowsiness but can also cause insomnia. They can induce a state of emotional numbing or restriction.[13] In addition, they can cause lethargy, reduced libido and sexual impairment. They also occasionally produce an unpleasant state of agitation and tension, especially in young people.[14,16] These effects can be difficult to recognise.

4.2.5 Evidence of efficacy

4.2.5.1 Short-term use in depression

Antidepressants are one of the standard recommended treatments for depression and many people regard them as useful. Their use is based on evidence from hundreds of placebo-controlled trials, which show that antidepressants are slightly better than a placebo in terms of scores on a depression rating scale, the principle outcome measure of these trials. Studies are inconsistent, however, and differences are small, especially when unpublished trials are included.

The small difference between antidepressants and placebo raises questions about whether the effects are, indeed, worthwhile. For example, in an analysis, which combined the results of several American trials of SSRIs and other new drugs, the difference between the drugs and the placebo was less than two points on the commonly used Hamilton Rating Scale for Depression (HRSD).[17] Other meta-analyses, including the largest ever conducted, published in 2018, report similar small

differences between antidepressants and placebo.[18] The HRSD usually has 17 items and scores up to 54 points. When a difference of around two points is compared to ratings on a commonly used global measure of people's overall condition, the Clinical Global Impressions Scale[19], it does not register as showing any difference at all. Indeed, a difference of eight points on the HRSD would be required to register as a 'mild' level of improvement on the Clinical Global Impressions Scale, a difference that is way above that found in any combined analysis of placebo controlled antidepressant trials.[20] An analysis of trials conducted by the (then) National Institute of Clinical Excellence[21] also found that the difference in depression scores between people randomised to antidepressants and people randomised to placebo was so small that it was, in the words of the Institute's report, 'unlikely to be of clinical significance'.[21]

Although depression rating scales scores are the principle outcome measures of placebo-controlled trials, results are often presented in terms of the proportion of people who show a 'response' to the antidepressant compared with the proportion that respond to the placebo. The largest antidepressant meta-analysis reported, for example, that people randomised to take antidepressants were one and a half to two times more likely to show a 'response' than people allocated to placebo.[18] There is no objective marker of 'response', however. It is simply defined, quite arbitrarily, as a certain level of reduction in depression measurement scale scores. When scores are categorised in this way, however, the difference between the groups can be inflated, so that small absolute differences in scores become quite large differences in response rates.[22] Therefore the depression scores are the most reliable measure of the outcome of these trials.

The small difference between antidepressants and placebo that is indicated by depression scale scores may not even be a genuine difference in actual levels of depression, however, but may be an artefact of research designs or a consequence of the mental alterations produced by antidepressants. Publication bias, not accounting for withdrawal effects from previous treatment and various statistical issues may have artificially inflated differences between antidepressants and placebos in randomised trials and meta-analyses of these trials.[23]

Additionally, antidepressants may produce alterations that reduce depression-related symptoms without actually acting on depression itself. Depression often involves insomnia or sleeping difficulties and sometimes involves anxiety and agitation. Any drug with sedative properties will improve this aspect of the problem. The HRSD, for example, contains three items on sleep alone and these items can score up to six points. So, any difference between drugs and placebo may reflect the sedative qualities of some commonly used antidepressants (tricyclic antidepressants and mirtazapine, for example).

Any drug that alters our consciousness may also obscure or suppress depressive feelings. SSRIs appear to dull or numb emotions, which could reduce the intensity of depressive feelings.[16,24] Tricyclic antidepressants may also promote a state of emotional indifference, given their affinity with antipsychotic drugs that are known to have this property. All these effects may reduce scores on depression rating scales.

These and other alterations also mean that people involved in antidepressant trials are sometimes able to detect whether they are taking the active drug or the placebo. This may produce an unequal, amplified placebo response in people who are taking antidepressants in randomised trials. If people can improve by taking an inert placebo, what is known as the ordinary placebo effect, then people who take a drug that has noticeable effects may have an amplified placebo response. Conversely, people who take the placebo may realise this because they do not experience any of the 'side' effects they have been told to expect. Such people may do worse than they might do if they had not been enrolled in a trial in the first place. As such, the difference between antidepressants and placebo detected in clinical trials may be a result of 'amplified' placebo effects.[25]

The idea that antidepressants may be working through inducing 'amplified' placebo effects

is supported by the finding that other drugs with noticeable effects, including stimulants, benzodiazepines, opiates, and antipsychotics have been found to have equal effects to standard antidepressants in randomised studies in people with depression.[8]

In summary, antidepressants are only marginally better than placebo in randomised trials in people diagnosed with depression. Some evidence suggests the differences are unlikely to translate into meaningful clinical benefit. Moreover, there is no current evidence that strongly supports the idea that antidepressants produce their effects by acting on the underlying biological mechanism of depression.[26] Although much research has been conducted to look for the underlying mechanisms of depression, no such mechanism has been confirmed, and there remains little evidence that serotonin or other neurochemical abnormalities are associated with depression, or account for antidepressant action. Moreover, there are other convincing explanations of how antidepressants affect people with depression.

4.2.5.2 Antidepressants in severe depression

It is commonly stated that antidepressants are most effective in severe cases of depression. Overall, the evidence around this is contradictory. A NICE review claimed antidepressants have their most marked benefits in people with more severe depression, but the data actually found the greatest effects compared with placebo in people whose depression was in the middle range of severity, rather than in those with the most severe depression.[21] A recent meta-analysis that specifically examined this issue found that the severity of depression was not correlated with drug-placebo differences.[27]

4.2.5.3 Long-term use for relapse prevention in depression

There are several studies that show that if you take people whose depression has improved while they are taking antidepressants, and you randomise some of them to have their antidepressant stopped and substituted with a placebo, then the people transferred to placebo will have more 'relapses' of depressive symptoms.[28] Based on these studies, people who have had a single episode of depression are recommended to continue taking antidepressants for at least six months. People who have had recurrent episodes are recommended to take antidepressants on a longer-term basis.

However, the interpretation of these studies has been challenged particularly because the people transferred onto placebo are liable to experience withdrawal effects provoked by stopping antidepressants (see section 4.1.5).[29–31] These effects include anxiety and mood changes and may be mistaken for a relapse of the original problem.[32]

In addition, people who experience withdrawal effects may realise that they have been swapped onto the placebo and this may make them anxious and vulnerable. The next time they experience problems they may lapse into a state of depression because they have come to believe that they need the drug to remain well and because they realise that they have been taken off it. This situation is likely, because the participants of these maintenance treatment trials are a selected group who have made a good initial response to treatment.[33] They may already be persuaded of the benefits of drug treatment, or, at least, they are likely to be nervous about having it withdrawn.

However, non-randomised observational studies provide no evidence that antidepressants improve long-term outcomes of depression. In fact, some studies indicate that long-term antidepressant use is associated with increased relapse rates,[34] and worse long-term outcomes,[35,36] compared to people who do not use antidepressants.

One such recent non-randomised study analysed the association of antidepressant use from the age of 20 and depressive symptoms over the course of the succeeding 30 years. Involving 159 people, it found that those who used antidepressants were more likely to have more severe symptoms during follow-up. However, it is likely that all these studies reflect the fact that people who take antidepressants generally have more severe

problems initially than those who decide not to take them, which may account for their worse outcomes. Some studies have taken indicators of initial severity into account in the statistical analysis to some degree, but it is difficult to exclude this problem altogether.[37]

4.2.5.4 Use in anxiety disorders

A recent meta-analysis of studies of the treatment of anxiety showed that SSRI and SNRI antidepressants were superior to placebo in reducing scores on anxiety rating scales, but again the effect was modest. The difference in improvement between people taking the drug and those taking placebo was between two and three points on the Hamilton Anxiety Rating Scale, which has a maximum score of 56 points.[38] Another meta-analysis of 12 trials of the SSRI drug paroxetine found that, on average, people randomised to take paroxetine improved by 2.3 points more than people randomised to placebo.[39] Studies comparing SSRI antidepressants with benzodiazepines for anxiety symptoms find that benzodiazepines have larger effects.[40]

SSRIs and other antidepressants, particularly clomipramine, one of the old tricyclic antidepressants, are commonly prescribed to people who are diagnosed with obsessive compulsive disorder (OCD). They improve symptoms more than a placebo by around 3.2 points on a 40-point OCD measurement scale.[41] Behaviour therapy has larger effects than medication, but most studies of therapy include people who are also on prescribed drugs.[42]

4.2.6 Common adverse effects

Tricyclic antidepressants can slow down the conduction of electrical impulses in the heart and in high doses may cause dangerous irregularities of the heartbeat known as arrhythmias. Overdosing on these drugs is dangerous and often fatal. They also cause postural hypotension (a drop in blood pressure on standing up), which can lead to falls, and they increase the risk of seizures. They tend to have 'anticholinergic effects' including dry mouth, constipation, difficulty passing urine and blurred vision. At higher doses they may cause confusion. They also cause weight gain and sexual dysfunction including impotence, loss of libido and delayed orgasm.

The effects of SSRIs and SNRIs are similar, although SNRIs may produce more noticeable effects. Both types of drug commonly affect gut activity and cause nausea, vomiting, diarrhoea, constipation, and abdominal pain. They are also associated with sexual dysfunction, especially delayed orgasm. There are mounting anecdotal reports that the sexual dysfunction associated with SSRIs can occasionally persist after the drugs are discontinued, sometimes for months or years.[43]

Both SSRIs and SNRIs can cause lethargy and SNRIs may cause drowsiness. The state of emotional numbing or detachment they produce can be experienced as unpleasant and debilitating[44] and is associated with sexual dysfunction.[45] They can also produce a state of anxiety and agitation, especially in younger people,[46,47] which can also be extremely unpleasant and may be predictive of increased suicidal impulses (see below).

4.2.7 Other adverse effects

Some SSRIs, particularly paroxetine, have been linked with birth defects,[48] and as a class these drugs can thin the blood and produce bleeding disorders.[49]

4.2.8 SSRIs and suicide

Several meta-analyses of antidepressant studies in children and adolescents show increased rates of suicidal behaviour associated with use of SSRIs.[50–53] Some meta-analyses of trials in adults indicate small increases in suicide attempts or self-harm in people on SSRIs compared with placebo.[54,55] but others do not.[56–58] A recent re-analysis of one of these negative studies revealed a significant increase in suicidal ideation and behaviour using a different statistical approach.[59] However, where they have been compared with other types of antidepressants, SSRIs have not been found to be any worse in terms of increasing suicidal ideation and behaviour.[60,54] A recent meta-analysis based on data from original trial reports (which can provide more transparent data than official publications) found increased rates of suicidal thoughts and behaviour in children and

young people taking antidepressants compared to those taking placebo, but there was no difference in adults. This analysis also found an increase in reports of aggressive behaviour among young people taking antidepressants compared to those on placebo.[61] This confirms evidence from case reports of violent incidents, including legal reports and data from drug-monitoring agencies.[62] It appears that these behaviours may be related to the state of agitation that SSRIs and related antidepressants can occasionally produce, which, for reasons that are not understood, seem to be more common among young people.[47]

It is difficult to evaluate the conflicting evidence and claims about the relationship between antidepressants and suicide and violence because these situations are rare. On balance, the majority of evidence suggests that antidepressants can increase suicidal impulses and possibly also violent behaviour in children and young people. The evidence in adults is less conclusive.

4.2.9 Conclusion

Although antidepressants have been claimed to work by reversing underlying neurochemical abnormalities, no consistent abnormalities have been demonstrated in depression, and there is little evidence that antidepressants work in this way. Antidepressants show a minimal degree of superiority over placebo in short-term clinical trials (usually eight weeks) of depression. The small difference could be explained by drug-induced effects of antidepressants, such as sedation and emotional blunting, boosting improvements on depression measurement scales, as well as methodological factors in trial design, analysis and publication, which can artificially inflate drug-placebo differences. Finally, the findings of the many short-term trials do not enlighten us about the effects of long-term treatment. Despite the fact that many people end up taking antidepressants for months and years, there is little robust research on the benefits and harms of long-term treatment.

Some psychoactive effects of antidepressants may be experienced or perceived as useful for some people diagnosed with depression. Such effects vary in strength and character depending on chemical class and composition of the particular antidepressant. For example, tricyclic drugs are strongly sedating, which might be experienced as useful for insomnia, or to reduce anxiety and agitation. SSRIs, whilst exerting weaker and more subtle effects, can induce a state of emotional numbing or restriction, which may reduce the intensity of people's feelings. However, the fact that drug-placebo differences are so small, and easily accounted for by non-pharmacological factors, suggests that antidepressant-induced alterations may not be clinically useful. Moreover, emotional restriction and other drug-induced mental alterations may complicate successful engagement in psychotherapy.

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4.3 Benzodiazepines and related drugs

4.3.1 History

Benzodiazepine is the chemical name for a group of drugs discovered in the 1960s, otherwise known as (minor) tranquilisers. The individual drugs are often more familiar by their trade names. One of the most commonly used benzodiazepines is diazepam, whose trade name is Valium. They also include chlordiazepoxide (librium), lorazepam and temazepam.

From the 1960s onwards, benzodiazepines were widely prescribed to people with sleeping difficulties and people with anxiety and 'neurotic' disorders, especially women, often for long periods of time. In the 1980s it became apparent that many people who take benzodiazepines for more than a few weeks become physically dependent on them and experience significant withdrawal symptoms when they stop. Recommendations were then made that they should not be prescribed routinely other than for short periods.

Starting in the late 1980s, the Z-drugs (zopiclone, zolpidem and zaleplon) were introduced. These are chemically different from benzodiazepines but have similar effects and are now widely prescribed for insomnia. The drugs pregabalin and gabapentin also bear some similarities to benzodiazepines in terms of their pharmacological actions. In psychiatry, they are prescribed for anxiety. They are also used for epilepsy and nerve pain. In 2013, a UK study reported that pregabalin and gabapentin prescribing had increased by 350% and 150% respectively in just five years[1]. Withdrawal reactions have been described following discontinuation, which are similar to benzodiazepine withdrawal reactions.[2,3]

4.3.2 Theories of action

Benzodiazepines act by enhancing the activity of the brain chemical known as gamma aminobutyric acid (GABA). GABA has an inhibitory effect and benzodiazepines increase this. Therefore, they lower the activity of the brain, causing sedation and relaxation at lower doses, progressing to sleep and then coma and death at very high doses. Z-drugs also work by stimulating the GABA system.

In most situations benzodiazepines are regarded as non-specific treatments. In other words, they are thought to work according to a drug-centred model by producing an artificial drug-induced sedative state, rather than reversing an underlying disease. Since it is well known that they induce similar effects in everyone, regardless of whether or not they suffer from a psychiatric problem, it is difficult to deny the impact of their drug-induced effects. An exception to this is the case of anxiety. It has been suggested that anxiety is caused by abnormalities of GABA activity, which can be specifically reversed by the action of benzodiazepines on the GABA system. However, there is limited evidence of this.[4]

4.3.3 Drug effects

Benzodiazepines and similar drugs have sedative properties, similar in nature to alcohol. They cause a sensation of relaxation, which is both mental and physical, and they are recognised muscle relaxants. Like alcohol they may occasionally lead to disinhibited or aggressive behaviour, although there is little robust evidence in clinical or help-seeking populations.[4a] The alterations they produce are usually experienced as pleasurable, and they are used for recreational purposes, especially by those who prefer sedative drugs or 'downers'.

4.3.4 Evidence of efficacy

Short-term studies of benzodiazepines show that they reduce anxiety more than a placebo and are slightly more effective than other common drugs treatments for anxiety such as SSRIs.[5] However, studies generally only last a few weeks, so it is not certain whether this effect persists, since the body adapts to counteract their effects. This is the mechanism of dependence. The body's arousal mechanisms are stepped up to counteract the effects of the drugs, leading to the need for greater doses to produce the same effects and causing unpleasant withdrawal symptoms when they are stopped.

Randomised controlled trials of benzodiazepines for insomnia show that they increase duration of sleep by around an hour on average, but do not improve the time it takes to get to sleep (sleep latency).[6] In contrast, a recent meta-analysis of Z-drugs found that sleep latency was reduced by an average of 22 minutes compared to placebo, a difference which the authors concluded may not be clinically meaningful, and there was no evidence of improvement of sleep duration, although there was insufficient evidence on this particular outcome.[7]

4.3.5 Common uses

Benzodiazepines are recommended for the short-term treatment of anxiety and Z-drugs for the short-term treatment of insomnia. Benzodiazepines are also prescribed for the treatment of alcohol withdrawal and are frequently prescribed to people with severe psychiatric problems because of their sedative properties. As such, they are prescribed extensively to psychiatric inpatients with various diagnoses.

Within psychiatric hospitals, benzodiazepines are commonly used in emergency situations to sedate people who are behaving in a disturbed or aggressive way. Studies show that benzodiazepines are effective and comparable to other sedative agents (such as antipsychotics) in this situation.[8] However, evidence about whether they can reduce disturbed behaviour over a long period is lacking.

Benzodiazepines and Z-drugs have modest effects in insomnia and so they might be useful, temporarily, in someone who is having trouble sleeping. However, this effect will wear off, and if they are taken for more than a few weeks, withdrawing from them will itself produce sleeping difficulties. It is a similar situation with anxiety. Benzodiazepines can have remarkable effects in reducing anxiety initially, but these effects are likely to decline with time. When the drugs are stopped, anxiety will be induced by the process of withdrawal. For this reason, it is recommended that benzodiazepines be reserved for short-term use only.[9]

Despite benzodiazepines being generally recommended for short-term use only, many people appear to be prescribed benzodiazepines over long periods. Recent research estimates that the current number of people taking benzodiazepines long-term (beyond one year) in England is over 266,000.[10]

4.3.6 Common adverse effects

Like all sedative drugs, benzodiazepines impair people's ability to perform simple physical and mental tasks like driving and mental arithmetic. As with alcohol, people are often unaware of their impairment and rate themselves as functioning better than they are. It may only be after they withdraw from the drugs that they realise how impaired they were.[11] Other effects that derive from the ability of benzodiazepines to suppress nervous activity include confusion, slurring of speech and loss of balance and usually only occur at higher doses, or if some other factor (like a physical illness of some sort) is present. These effects are more likely to occur in elderly people, and when they do, elderly people can have falls and suffer other accidents because of being over-sedated.

At very high doses, such as when they are taken in an overdose, benzodiazepines can, like other sedative drugs, suppress the respiratory system and cause death.

There has been some concern that benzodiazepines may occasionally lead to disinhibited behaviour and aggression. This mainly seems to occur when high doses are used in people with a prior history of behavioural problems and in people who are more vulnerable to this, like children, the elderly and people with learning disability.[12]

Pregabalin and gabapentin also suppress the activity of the central nervous system, and their use can result in drowsiness, sedation, and reduced breathing. These risks are raised by higher doses, such as might be taken in an overdose, or when they are used in combination with other drugs that depress the nervous system. Like benzodiazepines, this can lead to respiratory failure and death in extreme cases. They are also associated with weight gain, which is not generally thought to occur with benzodiazepines.

Benzodiazepines are well-recognised recreational drugs, often used alongside other illicit substances like opiates. The agents with the shortest half-life are the most susceptible to abuse, and some, like temazepam, have been added to the schedule for controlled drugs. There have also been calls to make pregabalin and gabapentin-controlled substances, due to their propensity to become drugs of recreational or illicit use.[13] Both are reported to produce a 'high' in those taking them. The abuse potential may be higher with pregabalin, which is absorbed faster and is more potent than gabapentin.[14,15] However, gabapentin can also produce euphoria.[16]

4.3.7 Long-term harm

A few studies have looked at whether long-term use of benzodiazepines affects the structure of the brain. Two of these studies found a reduction in the amount of brain matter after long-term use of benzodiazepines, similar to findings with antipsychotics.[17,18] However, two other studies found no effects.[19,20]

Some studies have reported an increased incidence of dementia in people taking benzodiazepines compared to those who are not taking them.[21,22] However, since people with dementia can often present initially with increased anxiety or depression, for which they may be prescribed benzodiazepines, these studies do not necessarily indicate a causal relationship. However, a meta-analysis found that the risk of dementia was raised in people who had taken benzodiazepines in the past as well as those taking them currently or recently.[23] It also found that the risk of dementia was higher in people who took higher doses of benzodiazepines compared to those who took lower doses, and a correlation between increased risk and dose is generally regarded as a likely indicator of causation. A more recent study, however, suggested that the association might be accounted for by other drugs being taken alongside the benzodiazepines.[24] Despite remaining uncertainties, the evidence reinforces current recommendations that the drugs should be reserved for short-term use where possible.[9]

Benzodiazepines are definitely to be avoided during the last part of pregnancy, as they can cause neurological toxicity in the newborn infant.[25]

Together with drowsiness and confusion caused by their sedative properties, the most pressing concern regarding benzodiazepine use is dependence. The occurrence of withdrawal syndromes after stopping benzodiazepines and Z-drugs is well established and also reported in relation to gabapentin and pregabalin (see section 5 for further information).

4.3.8 Conclusion

Benzodiazepines are effective in reducing feelings of anxiety and have a modest effect in insomnia in the short-term. The main concern with their use is the significant risk of tolerance and dependence, and the associated difficulties that people can experience when trying to withdraw from the drugs.

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4.4 Antipsychotics

4.4.1 History

The types of drugs that are now commonly called antipsychotics were previously referred to as neuroleptics or as major tranquillisers.

The first drugs of this sort were introduced in the 1950s and 1960s. At that time, psychiatrists viewed them following a drug-centred model as substances that happened to have the ability to suppress thoughts and emotions without simply putting people to sleep in the way the older sedatives did. The mental restriction the drugs produced was noted to be part of a general state of physical and mental inhibition that at extremes resembled Parkinson's disease. Early psychiatrists regarded this state of neurological suppression as useful but as potentially damaging to the brain. Over time, these drugs have come to be regarded as treatments that target an underlying brain abnormality, particularly through their effects on the neurotransmitter, dopamine. Parallel to this view, they have come to be called 'antipsychotics'.[1]

The first of these drugs are now sometimes referred to as 'first generation' or 'typical' antipsychotics. From the 1990s a new range of these drugs was introduced, known as 'atypical' or 'second generation' antipsychotics. The second-generation antipsychotics were claimed to be more effective and less prone to side effects than the older drugs, but this is now known not to be the case. In fact, the distinction between the two classes is regarded now as unhelpful. Both classes contain a diverse range of individual substances with varying pharmacological profiles and range of effects.

4.4.2 Common uses of antipsychotics

Antipsychotic medication is a mainstay of treatment for people diagnosed with psychosis and schizophrenia. They are used to treat acute episodes of psychotic disturbance. People who experience a first episode of psychosis in the UK are often cared for by specialist early intervention in psychosis teams. After taking antipsychotic drugs for a further one to two years after recovery from the acute episode, they may be supported to stop. People who have more than one episode are recommended to stay on these drugs long-term for relapse prevention.

As well as being used for treating those with a diagnosis of psychosis or schizophrenia, antipsychotic drugs are also used in a range of other situations, particularly to calm and subdue people who are agitated or aggressive. Therefore, they are also prescribed to people who are diagnosed with mania, personality disorder, dementia, learning difficulties, autism and anxiety. They are also prescribed for depression and insomnia. Some antipsychotics are considered to be 'mood stabilisers' and prescribed for long-term treatment of people with bipolar disorder (see section 4.7 on 'mood stabilisers').

4.4.3 Theories of action

Antipsychotic drugs had been in use for at least a decade before it was discovered that some of them strongly counteract the effects of the brain chemical called dopamine. This finding led to the 'dopamine hypothesis' that suggested that 'schizophrenia' was a result of abnormally increased dopamine activity. In this view, antipsychotics are thought to reverse the chemical imbalance causing the symptoms of 'schizophrenia' or psychosis.

The dopamine hypothesis has developed over time, and now incorporates ideas about other causal factors including genetics, environmental stress and other neurotransmitter abnormalities, but amongst this complexity, the general assumption remains that dopamine dysfunction is part of the causal pathway to psychosis. The disease-centred view of antipsychotics is that they work by correcting, or partially correcting, this underlying abnormality by lowering dopamine activity.

Although some experts still adhere to the dopamine hypothesis[2], the majority of evidence

that has been collected over the last 50 years has not confirmed any differences in indicators of dopamine activity between people with a diagnosis of psychosis or schizophrenia and people without.[3] The few studies that show differences include very few people who have not already been treated with antipsychotics (which modify dopamine activity in themselves) and have not controlled for the other factors that are associated with increased dopamine activity, such as stress and arousal.[3,4]

4.4.4 Drug effects

Antipsychotic drugs vary in their pharmacology and profile of effects, but they all produce a state of global physical and mental inhibition or restriction. Many older antipsychotics act predominantly by blocking dopamine receptors, which produces a global neurological state resembling Parkinson's disease, a condition caused by degeneration of the dopamine producing cells. Its symptoms reflect a reduction of the activity of the dopamine system, which consist of reduced movement and slowed mental processes. However, all antipsychotics affect other neurotransmitter systems to some degree, and some, such as clozapine, have relatively weak actions on the dopamine system and a wide array of actions on other systems that are likely to be relevant to the mental and behavioural alterations they produce.

All antipsychotics appear to dampen down emotional responses. Associated with this, people find it difficult to motivate themselves to do things, or to take the initiative to act. Two Israeli doctors who took an injection of haloperidol for experimental purposes described how they were unable to read, use the telephone or perform household tasks of their own will, but could do so if instructed to by somebody else.[5]

Animal and volunteer studies show that individuals taking antipsychotics perform less well on tests of learning, memory, attention, reaction times and other tests of cognitive abilities.[6–9] Psychotic symptoms can also impair cognitive function, so antipsychotics may actually improve functioning in people who are symptomatic. However, there is some evidence that long-term use of antipsychotics may impair some aspects of cognitive performance in people who have recovered from their psychosis.[10]

In contrast to the disease-centred view that antipsychotics work by correcting an underlying dopamine abnormality, the drug-centred model suggests that the 'antipsychotic' effect is achieved by this state of neurological restriction that antipsychotics induce. This artificial state of suppression may reduce the intensity of 'abnormal' thoughts and experiences such as delusions and hallucinations and render them less distressing and intrusive. In this way, antipsychotics can be useful for the symptoms of acute psychosis or what are known as the 'positive symptoms' of 'schizophrenia'. However, there is no evidence that antipsychotics are selective for 'abnormal' thoughts or psychotic symptoms, and evidence from volunteer studies[8,9] and accounts by people who have taken these drugs for a variety of problems[4] suggest that they affect a wide range of mental processes.

4.4.5 Evidence of efficacy

4.4.5.1 Short-term use in psychosis

Although there is no evidence that antipsychotics treat or target the condition known as schizophrenia or psychosis, placebo-controlled randomised trials show that antipsychotics reduce the general disturbance in people who have an acute psychotic episode or exacerbation, improve their global condition and reduce abnormal experiences like delusions and hallucinations more than placebo.[11,12] However, a significant proportion of people does not improve substantially with antipsychotic treatment and have persistent symptoms despite treatment.

Evidence about whether antipsychotics are superior to other sorts of sedative drugs is more equivocal. Two trials suggested they were superior to barbiturates, but studies comparing antipsychotics to opium and benzodiazepines have not clearly differentiated the different types of drugs.[13–15]

The question as to whether people with psychosis can recover without the use of antipsychotics

received interest several decades ago but has been neglected more recently. A study in the 1970s compared people who entered the Soteria project, a small homely unit in California designed to care for people with psychotic disturbance or a diagnosis of schizophrenia, whilst avoiding the use of antipsychotics if possible, to similar people treated with antipsychotics at a conventional hospital. Thirty percent of people randomised to Soteria avoided the use of antipsychotics, but both groups did equally well.[16] A more recent study in Finland of people with a first psychotic episode also found that 43% of people could be successfully managed without antipsychotics.[17] So a reasonable proportion of people with an episode of psychosis may recover without the need for antipsychotics, but more research is needed in this area.

4.4.5.2 Long-term use for relapse prevention

The evidence base for the long-term prescription of antipsychotics to people diagnosed with schizophrenia or other psychotic conditions consists of many randomised and non-randomised studies showing that people on placebo or no treatment relapse more commonly than those who take continuous antipsychotic treatment. These studies have important limitations, however.[18–20]

First, the studies are too short to provide useful information about the benefits and risks of long-term antipsychotic treatment, with most lasting less than six months. Second, the randomised controlled trials all involve people who are already taking antipsychotics, often for many years before the study begins. The people who are randomised to placebo, therefore, have their previous drug treatment discontinued, usually abruptly over a few days, and replaced by placebo. They are therefore liable to the adverse effects associated with discontinuing antipsychotics. Withdrawal effects include agitation and insomnia, which may be mistaken for relapse, especially in trials that use broad definitions of relapse. There is some evidence, moreover, that antipsychotic withdrawal may precipitate a relapse of the underlying disorder that would not otherwise have occurred at that point, or that withdrawal itself can produce a psychotic state.[21] Therefore, the outcome of people in the placebo group in randomised trials of long-term treatment likely reflects the effects of antipsychotic discontinuation rather than the benefits of initiating preventive treatment.

Finally, most studies of long-term antipsychotic treatment have not investigated outcomes other than relapse, such as people's overall ability to function, their ability to work, to have relationships and to enjoy their lives.

4.4.5.3 Recent evidence on long-term antipsychotic use

Recent naturalistic, non-randomised follow-up studies suggest that long-term antipsychotic use may be associated with poorer outcomes. For example, studies in the USA, Finland and Denmark found that people who took antipsychotics on a continuous basis did less well in terms of 'symptom' levels and general functioning, than people who did not take antipsychotics or took them only occasionally after 10–20 years of follow-up.[22–24] However, these studies were not randomised and those patients able to stop their antipsychotic drugs may have had a milder condition than those who continued. The results are consistent, though, with the findings of a long-term follow-up of participants from a Dutch randomised trial.[25]

This study randomised people who had recovered from a first episode of psychosis to routine 'maintenance' treatment with antipsychotics, or to have their antipsychotics reduced in a flexible manner and stopped if possible. After the first follow-up at 18 months, twice as many people had experienced a relapse in the discontinuation group as in the maintenance group, although relapse was defined broadly as an increase in a single 'symptom' of psychosis, and rates of hospitalisation were not different. Only 20% of the discontinuation group had stopped antipsychotics at this point. Seven years later, 42% of the discontinuation group and 24% of the maintenance treatment had stopped antipsychotics or were taking only very low doses. By this point, there was no longer a difference in relapse rates, and levels of psychotic symptoms were similar in both groups. However,

people in the discontinuation group were more than twice as likely to have recovered from a functional point of view (40% vs 18%).

Since this was a randomised trial, differences between groups cannot be attributed to differences in the severity of their underlying condition. Therefore, the results provide some evidence that long-term antipsychotic use impairs some people's ability to function, which may be expected given their known inhibitory effects. It also suggests that attempting a gradual and supported reduction of antipsychotics may lead to people doing better in the long-term.

The results of a 10-year follow-up of people who took part in a placebo-controlled trial of quetiapine have also been reported recently.[26] This trial was reported as showing that people who were originally randomised to placebo had poorer outcomes than those who were randomised to quetiapine at 10 years. However, people who were defined as showing a 'poor' outcome included people with a mild increase in symptoms, and also included people whose symptoms were measured only after the original trial, and not at the 10-year follow-up. In fact, the 'symptom' scales and measures of functioning indicated no difference between people who were originally randomised to quetiapine and those originally randomised to placebo at the 10-year follow-up, which is not surprising, since the original trial was only a few months' long for the majority of participants.[27]

Long-term antipsychotic treatment may be helpful to reduce the intensity of ongoing psychotic symptoms or to prevent recurrence in some people. The balance of benefits and harms still needs to be elucidated, however, especially given the serious physical complications that antipsychotics can produce. A randomised controlled trial to evaluate a strategy of gradual reduction and discontinuation of antipsychotics compared with maintenance treatment in people with recurrent psychosis or a diagnosis of schizophrenia is currently under-way in the United Kingdom to provide more evidence in this area.[28]

4.4.6 Evidence for use in other disorders

There are studies that show that some antipsychotics are more effective than placebo for people diagnosed with depression, but as described in the section on antidepressants, almost any drug with noticeable effects has been found to have 'antidepressant' effects in one study or another, strongly suggesting that the effect is in fact an amplified placebo effect.

By reducing physical movement and arousal, antipsychotics may theoretically be useful in people who are hyperactive, agitated or aggressive. Trials have been conducted of the use of antipsychotics for the treatment of short-term aggressive behaviour, which show their effects are comparable with those of other types of sedative.[29–31] Trials of longer-term treatment for challenging behaviour in people with learning disability and dementia have found little or no benefit.[32,33] With regards to the diagnosis of personality disorder, only the category of 'borderline' or 'emotionally unstable' personality disorder has been frequently studied.[34] No evidence has been found for a positive effect of antipsychotics on the core features of the diagnosis itself, but NICE guidelines suggest short-term use of antipsychotics can be considered for crisis symptoms, such as impulsivity and aggression.[35]

Antipsychotics have been found to provide no net benefit for the core symptoms of autism in both children and adults.[36] NICE found 'moderate to low' quality short-term evidence for a range of behaviours including irritability and parent-defined challenging behaviours, but strong evidence of adverse effects.[37] Problems with the evidence included inconsistent results and risks of bias, such as unclear blinding procedures. The NICE guidance suggests considering antipsychotics for managing severely challenging behaviour in autism if other interventions are not possible or effective. Another meta-analysis judged the evidence for the use of antipsychotics for irritability and aggression in autism to be of better quality, but also noted the risk of adverse effects.[38]

There is a lack of evidence for the use of antipsychotics in insomnia[39], and limited evidence for anxiety. According to a recent meta-analysis one antipsychotic (quetiapine) may have modest benefits compared to placebo in reducing anxiety symptoms, but it also has significant adverse effects and it is not clear that the benefits can compensate for these.[40] The results were also inconsistent, and all the individual trials were funded by the manufacturer. Other antipsychotics have been trialled for anxiety, with negative results.[41]

Overall, since antipsychotics are associated with serious adverse effects (see below), the balance of benefit to harm is not likely to be positive in less serious mental health difficulties, especially with long-term use.

4.4.7 Adverse effects

Antipsychotics frequently produce a variety of bodily alterations that can be harmful, including metabolic disturbance and neurological effects. Less commonly, they are associated with dangerous and sometimes life-threatening complications.

Extra-pyramidal effects: This is the term used to describe symptoms produced by the effects of antipsychotics on a part of the brain involved in bodily movement called the extra-pyramidal system. They include Parkinson's disease-type symptoms of muscle stiffness, tremor and slowness of both movement and thought. Sometimes a 'dystonic' reaction can occur, when the muscles uncontrollably spasm. Most often this occurs shortly after starting the drug, but it can occur after longer periods of treatment too. Acute dystonia, which most often affects the head and neck muscles can be frightening and painful, and potentially fatal, if it is severe and not treated quickly. Another 'extra-pyramidal side effect is akathisia, which is a state of intense restlessness, causing people to feel compelled to move about, together with a feeling of psychic tension or anxiety. Although this is classified as an extra-pyramidal side effect, the exact mechanism behind it is unknown.

Metabolic abnormalities: Antipsychotics frequently cause people to gain weight.[42] They cause a noticeable increase in appetite and craving for carbohydrate-rich foods and decrease movement and energy use. Antipsychotics are also linked to disruptions of the body's normal metabolic processes that can lead to diabetes and raised cholesterol. These may, in turn, lead to increased rates of cardiovascular disease (including heart attacks and strokes).[43]

Structural brain changes: Recent studies in both animals and people have revealed that long-term antipsychotic treatment is associated with reduced brain weight and volume.[44, 45]

Tardive dyskinesia: This is a neurological condition involving involuntary movements, usually of the face. Several studies suggest that intellectual or cognitive deterioration also forms part of the syndrome[46,47]. Recent studies find that tardive dyskinesia affects approximately 4%–5% of people per year who take antipsychotics[48,90] (although this may be lower in ordinary psychiatric practice in the UK, possibly due to use of lower doses). It occurs more frequently in the elderly. It can be permanent, persisting after the drugs are stopped.

Neuroleptic malignant syndrome: This is an uncommon and dangerous reaction that occurs in around 0.5% of people newly started on antipsychotics. The exact mechanism is not known. It consists of a sudden reaction in which people have a high temperature, muscular rigidity and there is a risk of death.

Effects on the heart: All antipsychotics can cause a defect in the ability of the heart muscle to conduct electrical impulses. In particular, the drugs can cause prolongation of part of the heart's cycle of activity and they can cause irregular heartbeats or arrhythmias. Rarely, these effects can lead to sudden death, which is more common with higher doses.[50]

Hormonal abnormalities: Dopamine inhibits the production of the hormone prolactin. Therefore, reducing dopamine activity leads to an increase in prolactin levels. This is the hormone that stimulates

production of breast milk, and high levels can result in breast growth in men, lactation, infertility, impotence, reduced sex drive, and the bone-wasting condition, osteoporosis. This effect is more common with some individual antipsychotics, but sexual dysfunction is a common side effect of all, or most, antipsychotics.

Increased mortality: Evidence on whether long-term use of antipsychotics increases the risk of premature death is inconsistent. It is well known that people with a diagnosis of schizophrenia or another severe mental illness die earlier than the general population, partly due to lifestyle factors such as high rates of smoking and lack of exercise. Some studies suggest that antipsychotic drugs play a role, after taking account of these lifestyle factors.[51,52] However, other studies have reported reduced mortality among people who use antipsychotics compared to those who do not.[53,54] Being on more than one sort of antipsychotics is associated with a particularly high risk of early death.[52]

Other adverse effects: Many antipsychotics block the activity of the transmitter acetylcholine and produce what are called 'anticholinergic effects'. These include symptoms such as dry mouth, blurred vision and constipation. Many of the drugs cause postural hypotension, a drop in blood pressure on standing, due to effects on a type of noradrenalin receptor. Many antipsychotics can cause epileptic fits, particularly at higher doses, especially clozapine.

Clozapine can also cause a dangerous drop in the white blood cells that provide the body's immunity from infection. This can be dangerous and can lead to death from common infections. Everyone who takes clozapine requires regular monitoring of his or her blood cells to detect this effect early when it occurs.

4.4.8 Conclusion

Antipsychotics are powerful drugs. Most of them produce a state in which people's physical actions and mental processes are slowed up and restricted. These effects may be useful in suppressing certain mental experiences like delusions and hallucinations and in controlling disruptive behaviour, especially in the short term. The evidence on the benefits and harms of long-term drug treatment for people with a diagnosis of psychosis or schizophrenia is more difficult to interpret. Over the long term, the drugs are probably beneficial for some, but not necessarily for everyone with these conditions, and they are undoubtedly associated with severe adverse effects.

For an individual, the decision about whether to take antipsychotic drugs, or whether to stop taking them once they are started, depends on a fine balance between many considerations. For someone suffering unpleasant symptoms such as abusive hallucinations, they may have useful effects. People testify that antipsychotics help suppress distressing psychotic symptoms, but they also highlight how these benefits come at a price. Many of those taking these drugs experience the mental slowing and emotional restriction produced by antipsychotics as unpleasant, and their use can lead to a variety of physical complications. The harms related to antipsychotic drugs are particularly likely to outweigh any benefits they might produce in people with less severe mental health difficulties.

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4.5 Lithium and other drugs referred to as mood stabilisers

4.5.1 History

Drugs labelled as 'mood stabilisers' are most commonly used by people diagnosed with bipolar disorder. Bipolar disorder replaced the term manic depression to describe a pattern of behaviour that had been recognised for a long time, consisting of episodes of extreme arousal, hyperactivity and elation, known as mania, often followed by episodes of severe depression. Although the idea of a 'mood stabiliser' implies specific effects on the underlying biological basis of mood variability, in fact nothing like this has ever been demonstrated for any of the drugs referred to as mood stabilisers. The term 'mood stabiliser' merely refers to drugs that have been licensed for, or are commonly used in, the treatment of people diagnosed with bipolar disorder or manic depression. The first drug that was regarded as a specific treatment for manic depression was lithium. Lithium is an alkali metal, with sedating effects that are closely linked to neurological toxicity. The concept of a 'mood stabiliser' appeared in the 1990s at about the time that an old epilepsy drug, sodium valproate, started being marketed for the treatment of manic depression in a new preparation known as Depakote.[1] Other epilepsy drugs, such as carbamazepine and lamotrigine have also been marketed for this use. The implication that they stabilise mood has allowed these drugs to be prescribed to a wide proportion of 'psychiatric patients' who exhibit emotional turmoil from time to time. Since the invention of the concept of the mood stabiliser, such signs of emotion can be interpreted psychiatrically as a pathological or abnormal instability of mood and used as the justification for prescription of 'mood-stabilising' drugs. Hence, a large proportion of people who attend psychiatric services are now prescribed one of these drugs. However, there is little evidence that any of these drugs normalise emotional responses or stabilise mood.

Currently the group of drugs recommended for long-term treatment of manic depression or bipolar disorder includes lithium, sodium valproate other anti-epileptics such as carbamazepine and the newer drug lamotrigine and several antipsychotics (e.g. olanzapine, quetiapine and ariprazole). The antipsychotics that are officially referred to as 'mood stabilisers' are the ones that have been tested and marketed for this indication, but most antipsychotics are commonly used for the treatment of people diagnosed with bipolar disorder or acute mania.

4.5.2 Common uses

The commonest use of lithium and other 'mood stabilisers' is for the long-term treatment of people diagnosed with bipolar disorder. Guidelines suggest that they should be prescribed on a long-term basis to reduce the risk of relapse into a further episode of either mania or depression. An episode of acute mania is usually treated with various sedative agents including drugs referred to as mood stabilisers, like sodium valproate, but also benzodiazepines and all sorts of antipsychotics.

Over the last few decades, the idea that there are less severe forms of bipolar disorder has been popularised and the concept of the disorder has become malleable. Concepts such as 'bipolar 2 disorder', which is said to consist of recurrent depression with mild periods of mania, and 'bipolar personality' have been created, but are not universally accepted. It has been claimed that up to 20% of the population may suffer from some sort of 'bipolar spectrum' disorder.[2] Alongside these changing notions of the condition, there have been increasing rates of prescription of drugs referred to as 'mood stabilisers', particularly newer or atypical antipsychotics[3], some of which have been heavily marketed for this indication.[4]

4.5.3 Theories of action

Lithium is chemically similar to sodium, which is involved in many biological processes. Researchers have proposed various theories of the mechanisms for lithium's supposed anti-bipolar action. These

include correcting 'abnormal' sodium and calcium levels within cells, correcting 'abnormal' sodium dependent processes, effects on dopamine and serotonin pathways, and neuroprotective effects.[5] However, it remains widely acknowledged that there is no clear evidence as to the mechanism of action of lithium. This is also the case for the other drugs referred to as mood stabilisers.[6]

Although there is no clear biochemical theory, such as the dopamine hypothesis of 'schizophrenia', that helps to rationalise a disease-centred view of the action of these drugs, they appear not to be regarded simply as sedatives. If this were the case, the risk of toxic effects, especially with lithium, would be difficult to justify. Instead lithium and the other drugs are regarded as having specific, although yet unidentified, actions on a presumed biological basis of abnormal mood or manic depression.

From a drug-centred perspective, all drugs currently designated as 'mood stabilisers' have sedative effects and hence they are likely to reduce arousal and the emotions associated with heightened arousal like elation and irritability. The main research that has been conducted into their effects on people with relevant diagnoses, and that is used to justify the term 'mood stabiliser', concerns whether they suppress signs of mania and prevent relapse in people diagnosed with classical manic depression, now known as 'bipolar 1 disorder'. The only tests that have been done to look at how these drugs affect the variability of mood in healthy volunteers were done with lithium and found that lithium did not reduce normal fluctuations of mood.[7,8]

4.5.4 Drug effects

Lithium is a metal that can have dangerous effects on the nervous system, the gut and the kidneys at relatively low doses. Mild symptoms of toxicity include neurological symptoms such as tremor and lethargy. Progressive toxicity results in diarrhoea and vomiting, incontinence, drowsiness, disorientation, abnormal jerking movements, loss of balance (ataxia) and slurred speech (dysarthria), finally giving rise to convulsions, coma and death.

The effects deemed therapeutic are on a continuum with the manifestations of the toxic state. Thus, before the signs of full-blown toxicity start, lithium causes suppression of nervous conduction leading to sedation and impairment of cognitive functions.[9]

These effects are clearly demonstrated in volunteer studies.[8,10] After two to three weeks on lithium volunteers show decreased ability to learn new information, prolonged reaction times, poor memory, loss of interest and reduced spontaneous activity. Therefore, it is not surprising that people with mania and other forms of over-arousal are subdued when given lithium. The trouble is, the doses required to achieve a potentially useful sedative effect are close to those that cause a dangerous toxic state. Hence patients on lithium must have their blood lithium levels monitored on a regular basis.

Other drugs now referred to as 'mood stabilisers' all suppress nervous activity in different ways. They can all cause drowsiness at normal therapeutic doses and, like lithium, the anticonvulsant drugs cause signs of nervous toxicity such as slurred speech (dysarthria) and loss of balance (ataxia), usually at higher doses.

4.5.5 Evidence for their efficacy

4.5.5.1 Treatment of acute mania

Lithium reduces the symptoms of acute mania better than a placebo, but there is little evidence that it is better than other sorts of drugs with sedative effects. In fact, two studies of drug treatment for people with acute mania found that lithium was inferior to antipsychotics, probably due to the limitations caused by its toxicity.[11,12] In contrast, a Japanese study found lithium to be superior. However, doses of lithium were four times those of the antipsychotic used and patients were less severely ill, and therefore probably did not require the same level of sedation as patients in the other studies.[13]

Two studies have examined whether people with a diagnosis of mania do better with lithium compared to people with a diagnosis of another sort of acute psychosis, such as acute

schizophrenia. Both studies compared lithium with an antipsychotic and found that diagnosis did not predict which drug treatment people responded to. In other words, people with mania responded just as well to the antipsychotic drug as they did to lithium and people diagnosed with acute schizophrenia responded just as well to lithium.[14,15]

There has been little research into the effects of benzodiazepines in mania, even though they are widely used in this condition. Since they are sedative drugs, and mania is a condition of increased arousal, benzodiazepines would be a logical intervention and target for research. Some small studies that compared a benzodiazepine called clonazepam with lithium reported that the clonazepam was superior, but these were never followed up.[16,17] Whether this means that the results did not fulfil their early promise or whether the drug company that conducted them decided to aim the drug at a different market is uncertain.

Symptoms of acute mania are also improved by sodium valproate and the antipsychotic olanzapine, both of which have strongly sedating actions.[18]

4.5.5.2 Long-term use

Recommendations for long-term treatment of people diagnosed with 'manic depression' or 'bipolar disorder' are based on placebo-controlled trials, some of which show that people taking a mood stabiliser relapse less frequently than people taking placebo. However, these trials are mostly discontinuation studies. In other words, people who are already taking drug treatment are randomised either to continue to take it or to have it substituted with a placebo. Therefore, people who take placebo are, in most cases, people who have just had their previous prescribed drugs withdrawn.

There is good evidence that discontinuing lithium can induce a relapse in someone diagnosed with 'manic depression' or 'bipolar disorder', especially a relapse of mania. Several studies indicate that the likelihood of having a relapse after stopping long-term lithium is higher than it is before lithium is started.[19,20] The early studies of lithium of lithium maintenance, which were conducted in the 1970s, mostly involved people who were taking lithium prior to the study.

A few further studies have been carried out since 1990. Although not reported in all studies, where it was, a proportion of patients were reported to have been on lithium prior to entering the study. One of these studies found no difference between lithium, sodium valproate and placebo.[21] One found a difference between lithium and placebo, but it was clinically small.[22] Another reported a more substantial difference, but it appears that a large proportion of patients may have been taking lithium prior to the study (up to 69%, although the published paper does not make this clear) and the pattern of early relapses in the lithium group strongly suggests a discontinuation-related effect.[23]

The most recent study is a large trial comparing quetiapine, lithium and placebo.[24] Patients were stabilised on quetiapine prior to randomisation and may have been on long-term drug treatment prior to this. Again, the pattern of relapses suggests a discontinuation effect. Almost half the patients randomised to placebo (48%) experienced a relapse of 'any mood event' during an average of four months follow-up, versus 26.4% of the lithium-treated patients and 23.6% of those on quetiapine (during an average of six months). Relapse rates in both groups are much higher than the natural history of manic depression recorded for patients treated before the introduction of modern drug treatment in the early 20th century. Historical studies show relapse rates of around 50% over a period of two and a half to three years in the late 19th and first half of the 20th century.[25] Another problem with this study is that 54 patients who did not show adequate lithium blood levels were excluded from the population that were included in the final analysis. We know that non-compliance is associated with poorer outcomes regardless of the effects of the treatment[26], so this is also likely to have inflated the outcomes of the lithium group.

Another recent study found no significant difference in terms of rates of relapse between lithium, fluoxetine and placebo for the long-term treatment of people diagnosed with 'bipolar 2

disorder'. Time to first relapse was significantly longer with fluoxetine compared to the two other treatments, but there was no difference between lithium and placebo.[27]

Despite the mixed results and methodological issues, reviews and meta-analyses continue to recommend that lithium should be considered as the 'first line' treatment for 'bipolar disorder'.[28]

The evidence is just as poor for other 'mood stabilisers', if not worse. Despite the widespread use of sodium valproate and similar preparations, the only long-term study that compared it with placebo and lithium found no difference between any of the treatments on any of the major outcome measures.[21] Lamotrigine, a relatively new 'mood stabiliser', was found to be better than placebo for preventing depressive but not manic episodes in two trials sponsored by the manufacturer.[22,23] However, since lamotrigine is a drug with noticeable sedative drugs, there is likely to be a substantial 'amplified placebo effect' in people with a diagnosis of depression. The one placebo-controlled trial of olanzapine for the prevention of future episodes of manic depression found a lower rate of relapse (mostly of mania) in people treated with olanzapine compared to those randomised to placebo.[29] Results indicate a probable discontinuation effect, however, since the majority of the relapses in the placebo group occurred in the first three weeks of the study and all had occurred by three months. Quetiapine performed slightly better than lithium and was statistically significantly superior to placebo in the large, industry-sponsored study described above, but again a discontinuation effect is likely.[24]

4.5.6 Common adverse effects

Lithium is highly toxic to the nervous system, the digestive system and the kidneys. This means that blood levels that are only slightly higher than the levels usually associated with current doses can cause an acute toxic state. This can be fatal if lithium is not stopped immediately. This toxic state can occur if an overdose of lithium is taken, but it also occurs if blood levels increase because of dehydration or interactions with other drugs.

The toxic state can also sometimes occur at what would normally be regarded as safe blood levels of lithium.[30] Before the full-blown toxic state develops, lithium's effects on the kidneys result in extreme thirst and excessive urination. Its effects on the nervous system commonly result in a hand tremor as well as reduced reaction times, slow thinking and reduced creativity.[31] Lithium also frequently causes weight gain. In a small proportion of patients, long-term lithium treatment may result in irreversible kidney damage.[32] Lithium also frequently results in under-activity of the thyroid gland. Up to 20% of women on long-term treatment develop this complication and require treatment with thyroid hormones.[33] It is usually reversible on stopping lithium. Lithium can also affect the parathyroid gland, which affects calcium levels and bone health.

As explained above, withdrawal of lithium in someone with a diagnosis of bipolar 1 or manic depression increases the risk of a relapse, especially a relapse of mania. The mechanism for this is unclear, but it is as if removing the neurological suppression produced by lithium causes the nervous system of a susceptible person to go into over-drive, precipitating a rebound manic episode.

Sodium valproate can cause nausea, lethargy and sedation, hair loss, weight gain and polycystic ovaries, a condition associated with reduced fertility. It is also known to produce a high rate of foetal abnormalities if it is taken early in pregnancy and should not be prescribed to women of childbearing age. Valproate has dangerous but rare complications including liver failure, pancreatitis, and blood disorders.

Carbamazepine can cause a rash, nausea, sedation and signs of neurotoxicity such as loss of balance (ataxia) and double vision (diplopia). Rarely it can also cause serious blood disorders, such as aplastic anaemia and agranulocytosis, by suppressing the production of blood cells in the bone marrow. Very rarely it causes a drug-induced reaction known as 'hypersensitivity syndrome', a dangerous condition that can lead to failure of internal organs, especially the liver, and has a death rate of 8%. It can also cause a serious skin reaction (toxic epidermal necrolysis).

Lamotrigine also causes neurological symptoms such as loss of balance (ataxia) and double vision (diplopia). It can cause a serious hypersensitivity reaction and may impair liver function. It has also been associated with blood disorders.

4.5.7 Conclusion

Sedative drugs of various sorts help to reduce the manifestations of 'acute mania'. Despite mania being self-limiting and eventually subsiding naturally, while it lasts it can be overwhelming and difficult to control. Therefore, the short-term use of sedative drugs, including antipsychotics, benzodiazepines, some drugs that are referred to as 'mood stabilisers', may be helpful while the disturbance runs its course. Although lithium is recommended for this purpose, its toxicity means that other options are safer.

Based on current evidence, it is unclear whether any drug reduces the risk of having a further episode of 'bipolar disorder' because of the strong possibility that trials of preventive treatment reflect the effects of withdrawing from previous treatment. From a drug-centred perspective, it is plausible that sedative drugs might suppress the occurrence of mania, since it is a state of increased arousal. However, it is also possible that the body's adaptations to the long-term use of a drug will counteract any suppressant effect the drug might initially exert. It is less clear how the use of sedative, neurological suppressants like lithium, antipsychotic and anti-epileptic drugs would prevent the occurrence of 'depression'.

For people diagnosed with bipolar disorder, the potentially disabling and sometimes dangerous effects of the various drugs commonly on offer need to be weighed with a possible reduction in the risk of relapse. Mania can have harmful consequences and some people may feel that even the hope of protection may compensate for all the adverse effects of long-term drug treatment. Some may prefer to find other ways to try and exert some control over their experiences. For example, some people manage to identify the early warning signs of mania and use sedative drugs and lifestyle measures such as avoiding stress and taking time off work, to try and avert an impending relapse.

Other people may simply prefer to live with the risk of recurrence and seek intervention for an 'episode' if and when they need it.

With regards to people who do not have symptoms of 'classical' 'bipolar disorder', there is no clear evidence to support the use of a so-called mood stabiliser. No drugs have been shown to 'normalise' or smooth out moods. All drugs described as mood stabilisers are sedative drugs, which suppress mental and physical activity and may reduce people's emotional responses to their environment, in a similar way to antipsychotics, many of which are now regarded as mood stabilisers. For most people the adverse effects of these drugs would be likely to outweigh any benefits in terms of managing emotions that they may obtain from the alterations the drugs produce.

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4.6 Stimulants

4.6.1 History

Stimulants are a group of drugs that are still referred to by the type of effect they induce, rather than the condition for which they are prescribed. They are controlled drugs and some, such as amphetamines and cocaine, are commonly used recreationally. Stimulants are today mainly prescribed for what is referred to as attention deficit hyperactivity disorder (or ADHD) – namely, a set of behavioural problems deemed to occur in children and increasingly in adults. The stimulant methylphenidate (Ritalin) is most commonly prescribed, but various forms of amphetamine, including dexamfetamine and lisdexamfetamine, are also used, and a drug called atomoxetine is also used, which was originally claimed to be different from stimulants, but shows a stimulant-like profile of effects.

4.6.2 Common uses

In current guidelines stimulants are recommended as the first intervention for a diagnosis of severe ADHD in children, or if psychological therapy is judged to have been ineffective in less severe cases. In adults, they are the first line recommended intervention.[1]

4.6.3 Theories of action

Stimulants increase the availability and activity of excitatory neurotransmitters, such as dopamine and noradrenaline, within the brain, but they have effects on a wide range of neurotransmitters and biochemical systems.

Traditionally, stimulants are said to work by correcting a shortage or malfunction of these neurotransmitters, with most research attention focusing on dopamine. However, there is no consistent evidence of a specific chemical abnormality in the brains of people diagnosed with ADHD, and no evidence that stimulants work by reversing it.

There is a simple alternative explanation for how stimulants work in ADHD. The main physiological effect of stimulant drugs is to increase arousal. At high doses this results in increased activity and it can cause obsessive-compulsive behaviours and abnormal movements such as tics and grimaces. At lower doses the main manifestation of increased arousal is an increased ability to concentrate, and a feeling of calm. This is familiar to people who smoke cigarettes, since nicotine is a mild stimulant drug. Therefore, stimulants would be expected to improve attention and reduce hyperactivity in the relatively low doses at which they are prescribed.

This drug-centred model of how stimulants work suggests that the effects of stimulants in people diagnosed with ADHD are the same as those that are observed in people with no such diagnosis. This is confirmed by research that showed that giving methylphenidate (Ritalin) to both healthy volunteers and people diagnosed with ADHD led to similar increases of brain dopamine in the two groups and the same improvements in concentration and attention.[2] These results are consistent with the effects that are observed in animals.[3] This demonstrates that there is no need to construct a disease-centred account for the action of stimulants. A drug-centred model, in which low-level stimulant-induced alterations improve concentration and attention on a single task, can account for their effects in people diagnosed with ADHD.

Animal studies also show that stimulants inhibit spontaneous exploratory behaviour, reduce an animal's interest in its environment and reduce its social interactions with other animals. Instead, the animal shows repetitive, over-focused, pointless behaviours such as pacing, scratching, excessive grooming, gnawing and staring at small objects. They also develop tics and other involuntary abnormal movements.[4] In children too, it is recognised that stimulants can suppress interest, spontaneity and emotional responsiveness.[5] Therefore it seems stimulants increase the ability of a person or an animal to focus on a single task by reducing their interaction with the rest of the environment.

Adults typically enjoy the effects of stimulant drugs, hence their use as recreational drugs. Children, however, generally dislike the experience of being on stimulants.[6,7] However, children may also see the benefits of taking stimulants from the point of view of their behaviour or school performance.[8]

When stimulants are used recreationally, people often need to increase the dose to keep getting the same desired effect. This shows that stimulants, like other psychoactive drugs, induce 'tolerance'. In other words, the body adapts to counteract their effects, so if you use them continuously, you must increase the dose to get the same effects. Tolerance to stimulants prescribed for ADHD has been demonstrated in animals[9] and documented in children[10], although the fact that children are naturally maturing during treatment may obscure tolerance effects. If tolerance occurs, it suggests that any beneficial effects that are experienced in the early days of stimulant treatment would gradually be lost.

4.6.4 Evidence of efficacy

Studies in children and adults find that stimulants reduce the symptoms of ADHD more than a placebo, as measured by various rating scales. This is not surprising, given the alterations they are known to cause in humans and animals regardless of whether or not they have a diagnosis of ADHD. The effects are not large, however. One study of methylphenidate (Ritalin) in adults found differences of between four and five points on a 56-point rating scale, for example[11], and another found a difference of between three and six points on a 54-point rating scale score.[12] A meta-analysis of trials of methylphenidate in children found that the drug was more effective than placebo at a level that was just above that judged to be a minimally relevant difference.[13]

In addition, few studies provide data on long-term outcomes and controlled trials do not show evidence of beneficial effects on school achievement in children, or employment or other aspects of general functioning in adults. One of the few trials that looked at these sorts of outcomes, a placebo-controlled trial of atomoxetine in adults diagnosed with ADHD, found no difference in work productivity between people randomised to take the active drug and people randomised to placebo (the main outcome of the study), and no difference in driving behaviour either.[14]

Moreover, many of the studies in children and adults have been conducted by a group of researchers at Harvard University who were revealed to have received millions of dollars from the pharmaceutical industry in consulting fees and other payments.[15] Studies conducted by this group show consistently larger effects than other studies.[16]

Two large randomised studies have been conducted that explored the long-term outcomes of stimulant treatment and psychotherapy for ADHD – one in children and one in adults.

In the first study, children were randomly allocated to four different types of treatment: intensive behavioural therapy, an intensive 'medication management' regime with frequent medical reviews, a combination of behavioural therapy and 'medication management,' and routine community care, in which children often received prescribed stimulants.[17]

The first set of results, based on data from the first 14 months of the study, showed that all groups displayed a substantial decline in the severity of their symptoms. The 'medication management' group fared better than the group that had behaviour therapy on the core symptoms of inattention, as rated by parents and teachers, and hyperactivity as rated by parents only. The study showed no differences between the groups for the other factors that were evaluated, including social skills, parent–child relations, academic achievement and aggression.

However, ratings by the only blinded rater, a classroom observer, showed no difference between the treatment groups for attention or hyperactivity.[18] In addition, around 60% of the routine community treatment group were also prescribed stimulants and this group fared the same as the behavioural therapy group. Hence it may have been something about the intensity of

the contact involved in the intensive 'medication management' group that improved symptoms apart from, or as well as, their prescribed drug treatment.

At the three-year follow-up, there was no difference between the original groups in terms of any outcome measures.[19] This study is important because it is the only randomised study that has followed-up children with ADHD for more than a year. Its results are difficult to interpret and not decisive, but they suggest that stimulants, coupled with assertive monitoring, may improve teacher or parent ratings of children's attention and activity levels in the short to medium term, but long-term benefits are not established.

The study in adults was conducted in Germany and participants were randomised to one of four treatment conditions: to take methylphenidate with routine care, to take methylphenidate in combination with a cognitive-behavioural group psychotherapy programme or to take placebo in combination with routine care or the group psychotherapy programme. The first follow-up was conducted at three months, and subsequently at six months, a year, and two and a half years after the trial commenced. Methylphenidate performed better than placebo at all follow-up points in this study, but differences were small. At three months the difference in symptom scores was 1.7 points on a 36 point scale, at one year it was 2.2 points[20] and at two and a half years' follow-up the difference was 1.4 points.[21] Although these differences were statistically significant, there is no research that has established what sort of differences in symptom scales might translate into meaningful or observable improvement in ADHD, as there is for the diagnosis of depression. In other words, we cannot be sure of the clinical significance of the findings, but the differences detected appear to be modest. There was no difference in symptom scores between those who were randomised to the group psychotherapy programme and those who received routine care at any follow-up point in this study.

4.6.5 Common adverse effects

Stimulant drugs increase the activity of the heart, raising the heart rate and increasing blood pressure.[20] There is considerable debate as to whether these effects translate into serious consequences such as an increased risk of heart attacks, cardiac arrhythmias (irregularities of heart rhythm which can lead to death) or stroke. The sorts of changes to heart rate and blood pressure that are observed with stimulant treatment have been shown to lead to more serious cardiac effects in other contexts.[22] Some studies of adults who are prescribed stimulants for ADHD show an increased incidence of arrhythmias, transient ischaemic attacks and sudden death[23,24], but others have shown no detrimental cardiovascular effects.[25] A recent meta-analysis found increased rates of sudden death due to a cardiac arrhythmia with all drugs prescribed for ADHD and with methylphenidate specifically, but no increased risk of myocardial infarction, stroke or all-cause death.[26] Overall, the data suggest that prescribed stimulants cause a slight increased risk of serious cardiac events, particularly arrhythmias and sudden death. Recreational use of stimulants is well known to lead to cardiac complications in some cases, but doses taken are usually considerably larger than those that are prescribed.[27]

In some cases, stimulants induce a depressive picture, with lethargy, withdrawal, and loss of emotional responsiveness, sometimes referred to as a 'zombie' effect.[28] In others they may cause agitation and anxiety. Insomnia is very common. Rarely, stimulants can induce a psychotic episode.

A recent study found that being prescribed stimulants for an ADHD diagnosis increased the risk of developing Parkinson's disease or a similar brain condition by more than eight times.[29] The association between taking stimulants and Parkinson's disease is well established among people who take them recreationally[30], so it is plausible that prescribed use will have some effect too.

An important adverse effect of stimulants in children is growth suppression. The three-year follow-up of the MTA study showed that children

who had taken stimulants on a continuous basis were 2.3cm smaller than a non-ADHD comparison group and 4.2cm shorter than those children in the study who had not used stimulants.[31]

Although not all studies show negative effects on growth, another recent study looking at growth rates over five years confirmed the MTA findings and showed that higher doses of stimulants had a stronger retarding effect on growth than lower doses.[32] The exact mechanism whereby stimulants suppress growth is not yet known. It may be related to the fact that they reduce appetite, but they are also known to have an impact on several hormones that may be involved in growth including growth hormone, prolactin and thyroid hormones.

4.6.6 Conclusion

Stimulant drugs have generalised effects that may help to reduce symptoms such as inattention and hyperactivity in children and adults diagnosed with ADHD. Trials reveal consistent, but relatively modest, benefits on symptom levels compared to placebo, but no trials have established beneficial effects on other outcomes such as school or work performance or achievement.

Stimulants are associated with psychiatric problems – commonly anxiety and insomnia. They may be associated with an increased risk of serious conditions such as heart attacks and Parkinson's disease. The desire for short-term symptom reduction must be balanced against these potential adverse effects, as well as the evidence suggesting that the beneficial effects on attention are achieved by suppressing the person's ability to interact with their wider environment in a playful or creative manner.

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4.7 Combined psychotherapeutic and psychopharmacological intervention in depression

A combination of prescribed drugs and psychotherapy is often regarded as a superior intervention to the use of these drugs or therapy alone, particularly with depression. The current NICE guidance[1] for depression includes recommendations for combined treatment, especially for more severe symptoms and for when previous treatments or various types have been ineffective.

On the other hand, it has long been recognised that the effects produced by taking psychoactive drugs, whether prescribed or illicit, may interfere with the learning and personal development that is an integral part of therapy. For example, if someone is taking benzodiazepines that dampen anxiety, then they may not be able to learn other techniques to manage the anxiety. Any drug that dampens emotions or sensitivity may interfere with efforts to control and manage emotional reactions in non-pharmacological ways.

The idea that combined intervention is more effective is based on several assumptions. Antidepressant drugs are assumed to target the biological causes of depression, whilst psychotherapy separately targets perpetuating psychological factors, with the two interventions leading to a cumulative therapeutic effect.[2] However, this is problematic for several reasons.

As outlined in 4.2, there is no convincing evidence for any biological abnormalities underlying depression, which are effectively targeted by antidepressant drugs. In other words, current evidence does not support the idea that antidepressants improve or correct a specific biological component to depression (a disease-centred model), which could act in parallel with psychotherapy.

In addition, the evidence for the actual effectiveness of antidepressants in depression is beset by multiple flaws (see section 4.2.5). Meta-analyses have reported that antidepressants may have slightly more effect than placebo in the short-term reduction of depression symptom scale scores, but it is not clear that such an effect is clinically relevant and could provide an additional benefit to psychotherapy, or that it is a specifically pharmacological effect as opposed to an amplified placebo effect.

NICE suggest that antidepressants can enable more effective therapy through effects such as improved sleep, motivation and cognitive ability.[2] Antidepressants do produce psychological and behavioural changes, as described in Section 1.2. Some antidepressants have sedative effects that may improve sleep, but there is no evidence that any antidepressant increases motivation or cognitive ability more than a placebo. From what we know of the alterations antidepressants produce, it is not clear that they would aid psychotherapy, and they may even be counterproductive.

The sedation produced by some antidepressants, for example, may be useful in terms of increasing sleep and reducing anxiety, but may hamper therapy by impairing clarity of thinking and cognitive function during the day. The emotional restriction associated with SSRIs may, in theory, numb intense hopelessness and feelings of depression, which may help people to engage in therapy, but may also prevent people from learning how to manage their emotions in other ways.

Questions remain about the psychological effects of taking antidepressants and how they may impact on therapy. Many people understand antidepressants to work by reversing the underlying biological causes of depression, because, despite the lack of evidence for this position, this is what they have been told. Therefore, taking antidepressants can signal the idea that depression is a biological condition, over which the individual has little control. This position is logically inconsistent with the aims of therapy to enable people to gain more control over their feelings and behaviour and this is explored in section 3.

Multiple individual studies have looked at whether combined antidepressants and psychological intervention is superior to antidepressants or therapy alone. Overall, the results are contradictory. For example, when comparing a psychological intervention alone to a combined intervention, some studies found the combined intervention to be more effective,[3,4] others found no difference,[5–7] and others found the psychological intervention alone more effective.[8,9] Similarly, when comparing a combined intervention to the drugs alone, some studies found a combination to be more effective,[10–12] whilst others did not.[13–15]

As a result of this confusing overall picture, several meta-analyses of randomised studies have been performed, with many reporting an advantage, of varying degrees, for the use of a combination of antidepressants and psychotherapy over either drugs or psychotherapy alone.[16–19] However, judging by two recent meta-analyses, the quality of the individual studies included in these studies varies greatly, leading to questions about the reliability of their results.

For example, one 2009 meta-analysis combined studies that compared antidepressants alone to a combination of antidepressants and psychotherapy for depression.[18] It included 25 randomised trials and found that combination was better than antidepressants alone in the short term, in term of changes in depression symptom scores. However, the effect size was small, and whilst statistically significant, possibly not clinically relevant. There was insufficient data to look at longer-term outcomes. The number of studies included was limited, and the individual trials were also fairly small. The average number of patients per study was 81, and 15 studies contained fewer than 50 patients. The trials varied in their target population, with 16 looking at adults in general with depression, and others focusing on more specific groups, such as bereaved older people, and people with other physical or psychiatric conditions in addition to depression.

Many of the studies contained crucial flaws. Understandably, no study could blind participants to their treatment allocation, but only 18 reported blinding of assessors. In addition, only 16 studies conducted intention to treat analysis, in other words including the outcomes of all people who entered the trial. As the dropout rates varied significantly between combined and individual treatment groups, this may have impacted on results.

Another 2009 meta-analysis combined 19 studies comparing combination treatment to psychological treatment alone.[19] This also found a small difference in favour of combined treatment between the two groups in the short term, similar in magnitude to the other meta-analyses, which the authors admitted may be too small to be clinically relevant. Some limited follow up data were available, with no difference found between the two groups after three to six and 12 months.

The study populations also varied in this study. All looked at people with depression, with 14 studies looking at adults in general, and five at specific populations (adults with HIV, multiple sclerosis, chronic depression; older adults). The authors found that the difference between combined treatment and psychological treatment was much smaller when only data from the general adult samples were looked at. Only a minority of studies (11) reported blinding of assessors and a limited number of studies employed an intention to treat method. As the dropout rates were highly variable, and as high as 55% in one paper, this may have distorted results too.

Overall, evidence that a combination of antidepressants and psychotherapy is superior to either intervention given alone is not conclusive. The assumptions behind this research, for example, that antidepressants are effective, and that antidepressants and psychotherapy provide distinctive, additive mechanisms against depression have not been proven.

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4.8 Conclusion: Understanding psychiatric medication

In general, the orthodox 'disease-centred' view is that psychiatric medications reverse or partially reverse an underlying biological 'abnormality'. The existing evidence for this has been questioned. It is suggested that, in the absence of clear evidence for targeted actions by psychiatric medications on specific pathologies, an alternative, 'drug-centred' model is more valid and useful.

A drug-centred model views drugs as producing characteristic altered states, which vary according to the pharmacological properties of the drug concerned. These effects can alter, suppress or obscure the manifestations of mental health difficulties, and may be experienced as useful for some people with these problems. Within this model, there still remains a role for the careful and judicious prescribing of certain psychiatric drugs in some situations involving mental distress and disturbance.

However, the drug-centred model does entail a different relationship between person and prescriber. Rather than centring the discussion on what intervention is deemed appropriate for a specific diagnosis, people using services and their networks can ask and debate with their doctor about what sort of drug-induced effects might or might not be useful in their specific situation. They can explore by themselves and with others what the benefits of a drug-induced state would be and what negative consequences are likely to flow from that state.

People who are already taking psychiatric drugs might want to reflect on what drug-induced effects they are experiencing, and how these effects might be affecting their lives. They will want to balance any positive effects they feel they obtain against the negative or unpleasant effects and the evidence for long-term harm. People who want to stop their medication, either because they are stable, or because they feel it has not helped, will need information about the nature of the drug they are on before they can decide the best method for coming off it.

The drug-centred model makes the service user the expert in their own drug management. It is up to them to decide whether they find certain effects useful or not (unless drugs are being prescribed against the patient's wishes for purposes of social control). The model highlights how taking psychiatric drugs is always a delicate balancing act between benefits and harm. The useful effects that drugs have are part and parcel of a drug-induced state, a state of intoxication that is not the same as the ordinary state of the body and mind. Taking psychoactive drugs is likely to impair and suppress aspects of our mental and emotional functioning to a greater or lesser degree. If so, the question is whether that impairment is preferable to the distress that is being experienced.

Although many people are advised to take psychiatric drugs for long periods after their problems have subsided, the evidence for the benefits of long-term treatment is limited and harmful effects accumulate with long-term use. Therefore, deciding to stop taking prescribed drugs long-term can be a logical decision in many situations, and it is important that professionals provide support to people to do this as safely as possible.

Much of the material in this section is a condensed and updated version of material contained in *A Straight Talking Introduction to Psychiatric Drugs* by Joanna Moncrieff, published by PCCS Books, and used with the publisher's kind permission.

5. What do we know about withdrawal?

Professor John Read & Dr James Davies, with Luke Montagu & Professor Marcantonio Spada

5.1 A general introduction to dependence and withdrawal

Drugs are foreign substances from the point of view of the body, and therefore the body tries to counteract their effects. This is sometimes referred to as the body 'adapting' to the presence of a drug. The adaptation can occur in a variety of ways. In the presence of a drug that reduces the activity of a neurotransmitter, like dopamine, noradrenalin or serotonin, the body may increase the number of receptors for the particular neurotransmitter, or existing receptors may become more sensitive. Such adaptations can mean that, over time, higher and higher doses are required to achieve the same drug effect, as is seen for example with benzodiazepines and alcohol. When this occurs, the individual is said to have developed 'tolerance' to the effects of the drug.

If the drug is stopped then the body's adaptations are suddenly unopposed by the presence of the drug, and they give rise to withdrawal symptoms. For example, people taking dopamine receptor-blocking drugs such as antipsychotics will manufacture more dopamine receptors in their brains, and those receptors that already exist will change and become more sensitive to dopamine. This is the body's way of trying to increase the activity of dopamine despite the presence of a chemical that tends to reduce its activity. When the drug is stopped, these extra dopamine receptors will still be present, and may increase the activity of dopamine above normal levels until they reduce back down to normal numbers.

Usually the body's adaptations disappear gradually when the drug is no longer present, and the withdrawal reactions subside. However, we know very little about the body's response to long-term drug consumption and how the body reacts to the withdrawal of such consumption. It is possible that the adaptations sometimes persist or that it can take a long time for them to revert to normal. Even relatively shorter-term treatment can result in adaptations and resulting symptoms persisting a long time.

For example, we know that the abnormal movements of tardive dyskinesia, a potential effect of long-term antipsychotic use, often get worse when antipsychotics are reduced or stopped. This is likely to be mediated by the increased numbers and activity of dopamine receptors. Sometimes the movements improve with time as the body readjusts to the fact the drug has been stopped. However, sometimes they are permanent, implying that the adaptation of the body's dopamine system to the presence of antipsychotics can be irreversible. Something similar may be occurring after discontinuation of benzodiazepines or antidepressants, when, in some cases, withdrawal reactions last for long periods.

Most psychiatric drugs affect a range of different brain chemicals or neurotransmitters, and withdrawal effects can reflect the drug's impact on any of these chemical systems. Withdrawal reactions themselves may be mild and annoying, they may be unpleasant and sometimes they are unbearable. In addition, withdrawal from sedative drugs often causes agitation and insomnia, which can easily be mistaken for early signs of relapse. When drugs have been taken for a long time, such as several years, it is possible that the body will take a considerable time to readjust and withdrawal reactions may go on for some time.

Often the worst withdrawal effects are experienced at the end of the withdrawal process, when dosage has been reduced to almost zero. Repeated attempts at withdrawal may result in what is known as the 'kindling' effect where neuronal hypersensitivity results in the progressive

worsening of withdrawal symptoms at each subsequent withdrawal episode. It is reported that this can also happen when drugs are reinstated. Therapists should also be aware that withdrawal reactions are not limited solely to during or immediately after the withdrawal process but may last over a period from six to 18 months, and in some cases, several years.

Withdrawing from psychiatric drugs has some similarities to giving up recreational drugs. For example, people may become dependent on psychiatric drugs in both physical and psychological senses as they do with drugs of misuse. One clear difference is that recreational drugs have effects that are pleasurable, and so people crave the drug when they stop taking it. In contrast, people do not usually crave the effects of drugs such as antipsychotics or antidepressants, since these do not cause euphoria. People can, however, have strong beliefs about what such drugs do for them and may become anxious if they withdraw from them. As anxiety can occur as a withdrawal reaction it may not be possible to tell such feelings apart until withdrawal is complete. The danger is that any anxiety may be mistaken for a relapse of the original condition – see 5.4.2 for further discussion of this.

In general, if a drug is stopped suddenly, the withdrawal reactions will be more intense, but they may last for a shorter period (especially if the drug has only been taken for a short period; with longer-term use the body's response is more unpredictable). If a drug is gradually reduced, the withdrawal reactions will usually be less intense, and may not even be noticed by some people. The way the body reacts is not entirely predictable, however, and therefore withdrawal reactions can still be severe in cases where the drug is gradually reduced and when taken short-term.

Different drugs differ in their ability to cause withdrawal reactions. 'Short-acting' drugs that act quickly and are rapidly eliminated from the body cause more intense withdrawal reactions than 'long-acting' drugs that stay around in the body for longer. The rate of elimination of a drug is measured in what is called its 'half-life'. A half-life means the time it takes for the concentration in the body to decline by half. Drugs with a short half-life are eliminated rapidly, while drugs with a long half-life remain in the body for longer.

Heroin, for example, has a short half-life and causes more reactions after stopping than methadone, which has a long half-life. This is the principle behind the practice of prescribing methadone to people who are dependent on heroin to help them withdraw. The antidepressant fluoxetine is another example of a drug with a long half-life. It is eliminated from the body slowly over a period of weeks. Therefore, its effects generally wear off more gradually following cessation than those of drugs with a shorter half-life. In contrast, drugs such as the benzodiazepine lorazepam, antidepressant paroxetine and antipsychotic clozapine are eliminated rapidly from the body – they have short half-lives. That is why you must take repeated doses of them every day to get an even amount of the drug in the body over a daily period. These drugs cause more intense withdrawal reactions when stopped.

When embarking upon any account of how therapists may best support clients either withdrawing or considering withdrawing from psychiatric drugs, we encounter a number of immediate obstacles:

- In the first place, beyond the considerable work undertaken on benzodiazepine withdrawal, it is widely acknowledged that research into antipsychotic and antidepressant withdrawal is comparatively limited. One of the major outcomes of the recent antidepressant withdrawal debates[1] was that numerous admissions made by mainstream psychiatry showed serious gaps in our understanding of withdrawal. It also showed that established thinking on withdrawal (captured by NICE guidelines) is in significant need of revision (a revision which, as we mentioned in the introduction to this guidance, has now taken place).
- While the prescribing professions still have much work to do in deepening our understanding of withdrawal, research into how therapists may best support clients either in or considering withdrawal is even more sparse.

What is therefore offered in this section is largely derived from the combined experience of those who have worked directly with people withdrawing from psychiatric drugs. Although more research on such practices is still needed, experiential knowledge of successful withdrawal management has become sufficiently comprehensive to merit providing a picture of what we know, to date, works best.

This section will first summarise broadly what is known about withdrawal from each class of psychiatric drug, paying especial attention to the incidence, severity and duration of withdrawal. Finally, some background information regarding the medical management of the withdrawal process is provided alongside the definition of key terms.

5.2 Evidence on the likelihood, range of possible experiences, duration and severity of withdrawal per drug class

5.2.1 Antidepressants

Although further and better research is clearly needed in this area, it is safe to say, from the best available studies to date, that at least half of people who try to stop, or reduce, their antidepressants will experience withdrawal effects, while about half of those people describe the effects as 'severe', and the duration varies enormously.[1]

5.2.1.1 Withdrawal reactions

Table 1: Withdrawal effects of antidepressants

Flu-like symptoms	Headaches
Nausea	Insomnia
Dizziness	Anxiety
'Brain zaps'	Irritability
Emotional blunting	Diarrhoea
Sexual dysfunction	Fatigue
Sweating	Twitching
Vivid dreams	Heart palpitations
Muscle stiffness	Sensory
Hallucinations	hypersensitivity
Imbalance	Confusion
Agitation	Inability to cry

Shorter half-life antidepressants (such as venlafaxine, paroxetine, duloxetine and imipramine) are expelled from the body more rapidly (also see section 5.1). Antidepressants that are eliminated more slowly (e.g. fluoxetine) allow the body time to re-adapt to being without the drug and hence the withdrawal reactions are usually (but not always) less severe.

5.2.1.2 Incidence – How many people experience withdrawal reactions?

A recent systematic review of the literature on withdrawal from all types of antidepressants, but predominantly covering selective serotonin reuptake inhibitors (SSRIs), revealed 17 studies that contained data on withdrawal incidence – namely, on how many people taking antidepressants will experience withdrawal[1].

Seventeen different studies were reviewed (these ranged from small, industry funded drug trials to large independent online surveys of people who take antidepressants). These produced incidence rates from five percent to 97%. Of these 17 studies, three were excluded on methodological grounds.* The remaining 14 studies were methodologically diverse (comprising six RCTs, five naturalistic studies and three surveys) and produced incidence rates ranging from 27% to 86%. When grouping the three types of study together, the weighted average for each group was:

- The three surveys – 57.1% (1790/3137),
- The five naturalistic studies – 52.5% (127/242)
- The six RCTs – 50.7% (341/673)

* Two excluded studies, which reported low incidence rates (12%), were simply 'chart reviews' of medical notes (Coupland et al., 1996; Himei & Okamura, 2006) which are notoriously weak owing to their reliance on practitioners being aware of, and recording, withdrawal reactions. A further excluded study, which reported very high incidence rates (97%), comprised 693 people who were all involved in a withdrawal programme using tapering strips (and were answering a question about their previous attempts to come off) (Groot & Van Os, 2018). This sample was unrepresentative because people who have not experienced withdrawal reactions are unlikely to enter a tapered withdrawal programme. (See 5.4.1 for information about tapering).

As getting similar findings from different methodologies is typically seen to strengthen confidence in an overall, combined estimate, the most recent evidence suggests that at least half of people suffer withdrawal reactions when trying to come off antidepressants (median 55%).

5.2.1.3 Treatment duration – does the length of time spent on an antidepressant affect withdrawal?

When comparing the studies, there was no obvious relationship between incidence of withdrawal reactions and duration of treatment, but, as noted above, the information on treatment duration was incomplete. There were some useful data, however, within some of the studies. Two studies found no significant difference in the treatment duration of those who did and did not experience withdrawal reactions,[2,3] demonstrating that withdrawal reactions do not only occur in people who had been on the drugs for long periods of time. Both an international online survey[4] and an even larger NZ survey[5,6] found that those who had been on the drugs for more than three years were significantly more likely to report withdrawal effects, but these findings could partly be explained by a larger number of withdrawal attempts. Most participants in all four of these studies had been on antidepressants for months or years, so the studies were not able to assess whether there is a plateau, within the first few weeks of treatment, beyond which the probability of withdrawal reactions does not increase for most people.

5.2.1.4 Self-reported addiction

Another approach to the question of the incidence of withdrawal reactions is to ask how many people report becoming 'addicted' to or dependent on antidepressants. Traditional studies ignore this somewhat taboo topic. We do have important data, however, on how many recipients experience antidepressants as 'addictive'.

Three studies have provided percentages, which range from 27% to 37%. Of 192 people taking antidepressants in the Netherlands, 30% described their drugs as addictive. The two large online surveys found that 27% of 1,521[5] and 37% of 943[4] also described their antidepressants as addictive. The weighted average of these three studies is 30.8%. While it is difficult to extrapolate these findings to the wider population of those taking antidepressants, it is nevertheless important to note in these studies that nearly a third of those taking antidepressants, when asked, report being addicted to the drugs, according to their definition of the term.

5.2.1.5 Severity of withdrawal based on surveys

Unfortunately, questions as to the severity of withdrawal have not been sufficiently addressed in randomised trials. Therefore, the preponderance of data we have on withdrawal severity is derived from direct-to-consumer surveys.[1] As it is difficult to extrapolate from surveys to the general antidepressant population (e.g. people who experience withdrawal may be more likely to respond to surveys) population level estimates are hard to make. Nonetheless, the survey data are important as they indicate that for a proportion of those taking antidepressants withdrawal can be severe.

For example, in a recent New Zealand survey, 46% of the 750 who experienced withdrawal effects reported those effects to be 'severe' rather than 'mild' or 'moderate',[5,6] which was very similar to the 43% finding in the international sample.[4] Furthermore, a recent Dutch study found that of 671 people who had experienced some degree of withdrawal effects, 51% reported the most extreme of six levels of withdrawal. Additionally, a recent international survey of 605 people, all self-identifying on antidepressant withdrawal websites as experiencing withdrawal, asked participants to rate on a scale of 0–10 how severely withdrawal had impacted upon their life. The average rating was 8.4 with 41% indicating the highest level of severity on the scale.[7]

The percentages selecting the most extreme level of severity on offer in each of these four studies ranged from 41% to 51%, with a weighted average of 45.3%. So regardless of the scale used, nearly half of all people surveyed in these studies who experienced withdrawal effects ticked the most extreme level of severity on the scale they were presented with.

5.2.1.6 Difficulty, and duration, of withdrawal

In a recent UK survey, 245 responded to the question 'How easy did you find it to come off your medication?'

- 20% ticked 'very easy';
- 51% ticked 'fairly easy'; and
- 29% ticked 'not easy at all'.[8]

Of the 247 who responded to 'How long did it take you to come off your medication?'

- the majority (68%) did so within three months;
- but 21% took between three to six months;
- 6% took between six and 12 months; and
- 5% took more than a year.[8]

5.2.1.7 Duration of withdrawal reactions

A recent systematic review of antidepressant withdrawal identified 10 relevant studies that had gathered data on the duration of withdrawal reactions.[1] While this review could not provide firm conclusions about the average duration of withdrawal reactions (because of the variety of methodologies and ways duration was reported), it did conclude that there is far more variability in duration than previously believed.E Nine of the 10 studies found that a significant number of people experience withdrawal reactions beyond a week, while seven of the 10 studies showed that it is not uncommon for people to experience withdrawal for several months.

This review's findings were consistent with other reviews. For instance, a 2015 review of quantitative studies and case reports noted that in only four out of 18 case reports (22%) did withdrawal reactions spontaneously remit within two weeks, and in two cases withdrawal effects were ongoing a year after discontinuation. It concluded that withdrawal reactions 'typically last a few weeks' but noted that 'many variations are possible, including…longer persistence of disturbances'.[9] A more recent review, of research just on withdrawal from serotonin-norepinephrine reuptake inhibitors (SNRIs), concluded that 'Symptoms typically ensued within a few days from discontinuation and lasted a few weeks, also with gradual tapering. Late onset and/or a longer persistence of disturbances occurred as well' and recommended that 'Clinicians need to add SNRIs to the list of drugs potentially inducing withdrawal symptoms upon discontinuation'.[9]

Even longer durations have been reported by two real life samples of people experiencing difficulties with withdrawal. For instance, a recent international survey of people self-identifying as experiencing withdrawal found that when 605 people who had experienced withdrawal were asked 'How long have you experienced withdrawal symptoms?' 87% responded at least two months, 59% at least one year, and 16% more than three years.[7] Additionally, a recent content analysis of a population likely to have experienced withdrawal difficulties assessed the content of 137 online posts about AD withdrawal in the real world. The mean duration of withdrawal reactions was 90.5 weeks for the 97 taking SSRIs and 50.8 weeks for the 40 taking SNRIs. Although neither of the above two study samples are representative of all those taking antidepressants, they nevertheless indicate that it is not as rare as sometimes thought for withdrawal reactions to last more than a year.[1]

5.2.1.8 Qualitative studies on antidepressant withdrawal

Qualitative studies are consistent with and serve to bring to life the findings of the recent review of quantitative research.[1] Illustrative examples of personal testimony regarding the severity and duration of withdrawal effects follow:

"I am currently trying to wean myself off of Venlafaxine, which honestly is the most awful thing I have ever done. I have horrible dizzy spells and nausea whenever I lower my dose.

"It took me almost two years to get off Paroxetine and the side effects were horrendous. I even had to quit my job because I felt sick all the time. Even now that I am off of it, I still feel electric shocks in my brain."[10]

"It took me two months of hell to come off the antidepressants. Was massively harder than I expected.

"I forgot to take my Citalopram for two days and

woke up one morning with severe dizziness. It was so extreme that I fell over when I tried to get out of bed, and I threw up."[6]

"The withdrawal effects if I forget to take my pill are severe shakes, suicidal thoughts, a feeling of too much caffeine in my brain, electric shocks, hallucinations, insane mood swings. … kinda stuck on them now coz I'm too scared to come off it."[11]

"While there is no doubt I am better on this medication, the adverse effects have been devastating – when I have tried to withdraw – with 'head zaps', agitation, insomnia and mood changes. This means that I do not have the option of managing the depression any other way because I have a problem coming off this medication.

The difficulty of getting off has been a tough road and taken me years of trying and is something that doctors could be more knowledgeable of and supportive with."[12]

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5.2.2 Benzodiazepines and Z-drugs

The incidence of withdrawal range in different studies from 20% to 100%. Rather than report all the studies that draw these estimates, it can safely be concluded that these drugs are highly addictive and that dependence and withdrawal reactions are common. For this reason, the British National Formulary (2012)[1] recommends that uninterrupted usage, for both benzodiazepines and Z-drugs, does not exceed four weeks, because the drugs so quickly lead to tolerance and to physical and potentially psychological dependence. However, it is now clear that there are substantial numbers of people taking them for longer than two years (see 4.3.5).

Benzodiazepines are a drug-class that includes sedatives and anxiolytics:

- Sedatives (otherwise known as hypnotics or sleeping pills), such as flurazepam, temazepam, nitrazepam and loprazolam, tend to be short acting.
- Anxiolytics (also known as tranquillisers or anti-anxiety drugs), such as diazepam, alprazolam, chlordiazepoxide, oxazepam and lorazepam, are longer-acting.

'Z-drugs' are non-benzodiazepine sedatives/ hypnotics. The Z-drugs available in the UK are zaleplon, zolpidem, and zopiclone.

Both benzodiazepines and Z-drugs boost the effect of a substance in the brain called Gabba Amino Butyric Acid (GABA), which is thought to have a

calming effect. Because the Z-drugs are short-acting, it was hoped they may avoid or minimise dependence and withdrawal. However, there seems to be no robust evidence that they are significantly less addictive, or less often lead to withdrawal reactions, than short-acting benzodiazepines.

5.2.3.1 Withdrawal reactions

Someone who uses benzodiazepines for more than a few (two to four) weeks is likely to experience withdrawal reactions when they stop them. The reactions include anxiety, agitation, insomnia and muscle stiffness. Since benzodiazepines suppress nervous activity, stopping them increases the activity of the nervous system. Withdrawal can therefore induce unusual and usually unpleasant sensory experiences such as tingling and numbness, electric shock-like feelings and occasionally delusions and hallucinations.

Withdrawal reactions usually start between six and 48 hours of stopping, or after reducing the dose of a benzodiazepine, but can start later for longer-acting drugs, such as anxiolytics.

The most common withdrawal effects of these drugs include:

Table 2: Most common withdrawal effects of benzodiazepines and Z- drugs	
Sweating	Irritability
Nausea	Agitation
Dizziness	Muscle stiffness
Headaches	Twitching
Insomnia	Heart palpitations
Anxiety	Sensory hypersensitivity

But many other reactions may be experienced, including:

Table 2.1: Other withdrawal effects of benzodiazepines and Z-drugs	
Panic attacks	Restlessness
Weight loss	Abdominal cramps
Depression	Poor memory and concentration
Agoraphobia	Burning sensations in the skin

Table 2.1: Other withdrawal effects of benzodiazepines and Z-drugs	
Flu-like symptoms	Sore tongue and metallic taste
Blurred vision	Tinnitus (ringing in the ears)
Nightmares	Tingling in the hands and feet
Lethargy	Hallucinations and delusions

Sudden cessation of benzodiazepines and Z-drugs increases the probability of these withdrawal reactions and may also cause grand mal seizures, hallucinations, and suicidality.[1-7]

5.2.3.2 Severity of withdrawal

The severity of these reactions increases with:

- longer usage
- higher dosage
- the use of multiple benzodiazepines
- oral rather than injected use
- shorter half-life benzodiazepines (such as lorazepam or temazepam) because these are expelled from the body more rapidly. Drugs that are eliminated more slowly allow the body time to re-adapt to being without the drug and hence the withdrawal reactions are usually (but not always) less severe
- an abrupt cessation, and so it is recommended that benzodiazepines are withdrawn slowly.

5.2.3.3 Incidence – How many people experience withdrawal reactions?

Although initially marketed as a non-addictive alternative to barbiturates, benzodiazepines have long been recognised as highly addictive. Estimates of how many people experience withdrawal effects are determined by how long they have been on the drugs, how quickly they withdrew from them, and the definition or measure used to assess the withdrawal effects. Approximately 40% of people will become addicted within six weeks of taking them.[8] Some research finds that everyone who has been on benzodiazepines for at least six months and then tries to stop the drugs quickly will experience some withdrawal reactions, and for 40% the reactions will be moderate or severe.[3]

5.2.3.4 Duration

Estimates of how long withdrawal reactions last vary greatly, and are largely determined by duration of treatment, dosage and drug type. Almost all people stopping or reducing benzodiazepines will experience an 'acute' phase of withdrawal, which typically lasts for two weeks to two months. A minority will experience protracted (or 'post-acute') withdrawal phases, for a year or more,[9,1,10,11] with anecdotal reports of five to 10 years.

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5.2.4 Antipsychotics

The most recent survey found that of 105 people who tried to come off antipsychotics, 65 (62%) experienced unwanted withdrawal effects 'across the full-range of physical, emotional, cognitive, and functional domains'[1]

Drugs that were developed in the 1950s to treat people diagnosed with schizophrenia were initially described as 'major tranquillisers', acknowledging their powerful sedating effects. They have since become known as 'antipsychotics' or 'neuroleptics'. They are now often used on other groups besides those diagnosed with schizophrenia, including prisoners; children with learning and other difficulties, and people in care homes for the elderly. The first antipsychotics included chlorpromazine, haloperidol, pimozide and trifluoperazine. These 'first generation' antipsychotics had a disturbing adverse effects' profile (including tardive dyskinesia – a usually irreversible movement disorder). A second generation of antipsychotics, sometimes referred to as 'atypical' were developed, in the 1990s.[2] These include: amisulpride, aripiprazole, clozapine, olanzapine, quetiapine and risperidone.

We were introduced to the concept of withdrawal effects after stopping antipsychotics in section 4.4.5, in relation to understanding efficacy studies. As is the case for other central nervous system drugs, such as benzodiazepines and alcohol, the brain can develop a tolerance to antipsychotics.[3] Antipsychotics, however, are clearly not addictive, if one's definition of addiction involves a craving for the drugs. In fact, because of the unpleasant adverse effects many people try hard to stop taking antipsychotics soon after commencing them,[4,5] or have to be forced to take them against their will via the Mental Health Act, often with involuntary, long-acting injections.[6] About half of people prescribed antipsychotics for 'schizophrenia' are 'noncompliant'.[5] In one large sample, 74% tried at least once to discontinue the antipsychotics within 18 months of starting treatment.[7]

The adverse effects that lead to people trying to come off these drugs include[8-11]:

Table 3: The adverse effects that lead to people trying to come off these drugs

Sedation	Cardiovascular effects (arrhythmia & sudden cardiac death)
Dizziness	Akathisia (extreme restlessness)
Sexual dysfunction	Metabolic effects (obesity, glucose intolerance, high cholesterol and diabetes)

In an international survey of 832 people taking antipsychotics, twice as many (395) cited 'unpleasant side effects' than 'felt better and didn't need it' (195) as their main reason for wanting to stop their antipsychotics.[12]

Despite not being addictive in the strict sense of that word, there are two types of withdrawal syndrome that can make it very difficult to reduce, or come off, these drugs. The first type has much in common with the withdrawal effects of the other central nervous system drugs discussed in this guidance, such as benzodiazepines. The second type is somewhat more specific to psychosis and/or antipsychotics.

5.2.4.1 Classic withdrawal reactions

A recent review[3] found that antipsychotics share a range of 'classic symptoms of withdrawal' with all central nervous system drugs. These reactions, which usually emerge within four days of stopping, include:

Table 4: Classic withdrawal reactions associated with antipsychotic drugs

Nausea	Irritability
Tremor	Aggression
Anxiety	Depression
Agitation	Sleep disturbances
Headache	Decreased concentration

The reviewers suggest that these reactions usually last 'up to six weeks' and 'may last more than six weeks and become a post-withdrawal disorder' but the review provides no data to support these suggestions.

There are, in fact, relatively few studies of the frequency or duration of classic withdrawal reactions following discontinuation of antipsychotics. The largest direct-to-user, international survey, of 832 people prescribed/taking antipsychotic medication, found that 65% reported withdrawal effects when trying to stop or reduce, and that half of those people (51%) described those withdrawal effects as 'severe'.[12] Reported withdrawal was strongly correlated with duration of treatment ($p < .001$).

5.2.4.2 Antipsychotic induced psychosis and tardive dyskinesia

As described in section 4.4.3, antipsychotics blockade, to a varying degree, the dopamine system and other neurotransmitter systems (along with many other effects on the brain and body).[13] This led to the notion that 'schizophrenia' is 'caused' by an overactive dopamine system, a hypothesis that was never proved and that is now largely abandoned. The brain tries to compensate for the blockade.

As early as 1974 Dr Solomon Snyder, Professor of Psychiatry and Pharmacology at John Hopkins University warned that:

> *Something within the neurons recognises this sudden absence of neurotransmitter molecules at their appropriate receptor site and one way or another transmits a message back to the dopamine neurons saying something like the following: 'We don't have enough dopamine. Please send us some more!' Whereupon the dopamine neuron in question proceeds to fire at a more rapid rate.*[14]

It has since been established that the brain's attempted compensation also includes an increase in the number, and sensitivity, of dopamine receptor cells,[3] a process that is not unique to antipsychotics. When an antipsychotic, and the dopamine blockade, are removed, or reduced, the brain is effectively overwhelmed with dopamine, partly because of the abnormal drug-induced sensitivity and number of dopamine receptor cells. This process is likely to apply to the other neurochemical systems that antipsychotics influence. These effects may result in a withdrawal psychosis, which is often mistaken for a return of the 'schizophrenia' that the drugs were intended to treat. This in turn often leads to a reinstatement of the drugs that have, paradoxically, caused the neurotransmitter abnormalities.[15,2,14]

The first cases of dopamine 'Supersensitivity Psychosis' [SP] were reported 40 years ago.[16] A 2006 reviewer of the available evidence concluded:

> *There is evidence to suggest that the process of discontinuation of some antipsychotic drugs may precipitate the new onset or relapse of psychotic symptoms. Whereas psychotic deterioration following withdrawal of antipsychotic drugs has traditionally been taken as evidence of the chronicity of the underlying condition, this evidence suggests that some recurrent episodes of psychosis may be iatrogenic [caused by medical treatment]. Clinicians may therefore want to re-evaluate the benefits of long-term treatment in some patients.*[15] [Definition added]

There have been two recent, comprehensive reviews of the research literature on what now tends to be called 'antipsychotic-induced Dopamine Supersensitivity Psychosis' or 'Supersensitivity Psychosis' [SP] for short.[3,17] One of the reviewers has designed criteria for two SP-based withdrawal syndromes, differentiated primarily by duration.

5.2.4.3 Rebound psychosis

One set of criteria for 'Rebound Psychosis', or 'Withdrawal Psychosis', are new psychosis reactions occurring, or old psychotic reactions recurring at above pre-treatment levels, after antipsychotic discontinuation, reduction, switching or in between dose intervals, usually (but not always) after about three months continuous exposure to the drug (the time necessary for increased dopamine receptor density to occur), and causing distress or impairment in functioning.[3] These reactions usually appear within roughly four days of stopping oral antipsychotics but can take several weeks to emerge after cessation of long-acting injections. Rebound psychosis seems to be rather rare and the evidence most clearly supports it in relation to withdrawal from clozapine. British psychiatrist Joanna Moncrieff prefers the term 'Rapid Onset Psychosis' because it is neutral about the underlying mechanisms that, she suggests, are unclear.[15]

5.2.4.4 Persistent Postwithdrawal Supersensitivity Psychosis (PPSP)

Some researchers think that if Rebound Psychosis lasts longer than six weeks it should be reclassified as 'Persistent Postwithdrawal Supersensitivity Psychosis',[3] but this area is hard to research and there are a range of opinions on the topic. If PPSP does exist it is one of two long-lasting Postwithdrawal Disorders caused by antipsychotics. The other is the movement disorder Tardive Dyskinesia that is discussed later.

5.2.4.5 How many people experience Rebound Psychosis and PPSP and for how long?

Few studies have addressed the incidence or duration of withdrawal induced psychosis. The 2006 review mentioned earlier had reported mostly only case studies, including nine people with no previous history of psychosis, whose new psychosis (typically hallucinations or delusions) usually responded to reintroduction of the antipsychotic.[15] It was possible, however, to estimate that 20–25% of people withdrawing from a specific antipsychotic, clozapine, experienced Supersensitivity Psychosis (SP) or, as the reviewer prefers to call it 'Rapid Onset Psychosis'.

An early study estimated that between 22% and 43% of 224 outpatients diagnosed with schizophrenia had SP. Two recent studies of atypical antipsychotics have reported SP incidence rates of 65%[18] and 72%.[19] All three studies, however, included cases that occurred due to tolerance (see section 1.8) while the person was still taking the antipsychotics. In the latter study, 42% of the cases were identified as 'Rebound Psychosis', which means that overall 30% of the sample, not all of whom had tried to stop their antipsychotics, had experienced withdrawal-induced psychosis. Another study found SP in 26% of people while changing from one antipsychotic to another.[20] This is, however, a very difficult issue to research because of the fluctuating nature of the underlying psychosis.

In a recent international survey of people taking antipsychotics, 'new or increased psychosis' was the second most frequently reported 'other side effect' (after 'akathisia/restlessness'). Thirteen reported new reactions and six reported the exacerbation of previous reactions. It was not known, however, how many of the instances of new or exacerbated psychosis reactions followed withdrawal.[12]

There is some evidence that antipsychotics with shorter half-lives (e.g. clozapine, metroclopromide, sulpiride and amisulpiride) are more likely to provoke SP.[15,3]

5.2.4.6 Tardive Dyskinesia

Tardive Dyskinesia (TD), also mentioned briefly in section 4.4.7, is a disabling, often irreversible, antipsychotic-induced neurological disorder involving uncontrollable movements of the face, tongue, arms and legs. It is also associated with cognitive impairment.[21] It is likely, but not proven, to be the result of the over activity of the dopamine system caused by changes such as increased receptor numbers and sensitivity caused by antipsychotics. Some researchers consider TD to be either a component or predictor of SP.[3,18,19] The average prevalence of TD in people taking antipsychotics is about 30%,[22,23,2] rising to 57% after 15 years of treatment with first generation antipsychotics.[22] The prevalence was thought to be lower for second generation, 'atypical', antipsychotics, but the difference has found to be slight or non-existent,[22,2] or the consequence of second generation antipsychotics being prescribed at lower dosages. It is listed here as a withdrawal effect because the reactions of TD are often masked by the withdrawal of antipsychotics. When the drugs are stopped, it is thought that dopamine activity increases due to the increased sensitivity of the dopamine system produced by long-term antipsychotic treatment. Increased dopamine activity can produce abnormal movements. Thus, the overt physical reactions (but not necessarily the cognitive reactions) of TD are often either seen for the first time, or are exacerbated, after discontinuation, reduction or switching of antipsychotics.[22,2] People over 50 are three to five times more likely than younger people to develop TD.[22]

5.2.4.7 Withdrawing slowly, with support

A recent study exploring the personal accounts of individuals discontinuing antipsychotic drugs identified that 'weaving a safety net to safeguard well-being' was a pivotal process in drug reduction. This involved taking precautionary steps prior to reducing drugs taken to establish interpersonal alliances with family, friends, support groups and mental health professionals that can be activated should problems arise.[23]

In another study, 55% of 105 people who attempted discontinuation of APs described successfully stopping all APs for varying lengths of time, half reported no current use, and half described having some form of professional, family, friend, and/or service user or peer support for their attempt. Having support was associated with less relapse.[1] Furthermore, withdrawing gradually across more than one month was positively associated with successful withdrawal.[24] There will, of course, be large variability in how long people need to take to withdraw.

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5.2.5 Lithium and other 'mood stabilisers'

The relatively small amount of research conducted suggests that reducing, or withdrawing from, Lithium does not seem to cause the physical reactions caused by coming off other psychiatric drugs. Several studies, however, show that stopping Lithium can cause a relapse of mania, and that the probability of having such a relapse when withdrawing after long-term use is higher than before Lithium was started.[1–4]

Lithium is a toxic alkali metal, similar to sodium and potassium. It is prescribed primarily for people who experience relatively extreme emotional highs and lows, who often receive the diagnostic label 'Manic Depression' or, more recently, 'Bipolar Disorder'. The dose considered to be therapeutic is so close to the dose that causes a hazardous toxic state (which can be fatal if the Lithium is not stopped immediately) that levels of lithium in the blood have to be carefully monitored.[2]

The mental health charity Mind advises that:

> *There do not appear to be physical withdrawal symptoms with lithium. However, if you come off lithium too quickly you are very likely to have a rebound manic or psychotic episode and become quite ill, so you need to be cautious, reduce gradually – over at least one month, and much longer if you have been taking it for years. If relapse occurs, it happens in the first few months after withdrawal and then tails off.*[5]
> (Mind, 2018).

Some studies have reported increased suicidality following withdrawal from Lithium, especially if abrupt.[6,7]

Other drugs, sometimes described as 'mood stabilisers', include the three anticonvulsants carbamazepine (Tegretol), lamotrigine (Lamictal) and valproate (Depakote, Epilim). Little research has been conducted into the withdrawal reactions for people taking these drugs who do not have seizure disorders. A case series of six people coming off lamotrigine found distressing psychiatric reactions, especially anxiety and irritability.[8] A study of 90 people who withdrew from carbamazepine found that 26 (29%) reported withdrawal reactions within four days of withdrawal. Reactions, which alleviated within one week, included insomnia, dysphoria, hallucination, hand fremitus (vibratory sensation), and headaches.[9]

For the withdrawal effects of asenapine (Sycrest), an antipsychotic which is sometimes used as a mood stabiliser, see the section on antipsychotics.

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5.2.6 Stimulants prescribed for ADHD

The effects of withdrawing from stimulant drugs like cocaine and amphetamines that are taken for recreational purposes are well-documented. Even after taking stimulants for a day or two, people taking them typically experience a period characterised by reduced energy, depression, irritability, hunger and excessive sleeping, which can last for a couple of days. When someone has taken stimulants continuously for a long period, they may initially have insomnia, and feel anxious, sad and agitated, and they can experience chills and intense cravings for the drug.

After this, the person withdrawing will likely begin feeling both mental and physical exhaustion, start to sleep excessively, although they may still experience periods of insomnia, and become more depressed. They may continue to feel anxious and irritable and stop feeling pleasure, they may become less sensitive to stimuli such as touch and sound, be socially withdrawn and have vivid dreams.

The depression can be intense, and it may be accompanied by suicidal thoughts. The symptoms can persist for between a few days and several weeks or even months.[1]

Withdrawal from prescribed stimulants is less commonly described. Studies that have explored the consequences of withdrawal have focused only on whether or not it is associated with a relapse of the symptoms of ADHD, without considering the possible physiological and psychological effects of the withdrawal itself.[2,3] However, it has long been recognised that use of prescribed stimulants by people with ADHD is associated with the phenomenon known as 'rebound'.

This occurs when the effects of a dose of a stimulant wear off, usually towards the evening, and consists of a worsening of the symptoms of ADHD beyond their original level before treatment was started. Children, in which 'rebound' has mainly been noted, become highly excitable and distractible. Since low- dose stimulants reduce activity and increase focused attention, these rebound effects are a predictable response to the wearing off of the direct effects of the drug.

Rebound is also characterised by the onset of some new symptoms including tearfulness, irritability and emotional lability, which are not usually part of ADHD.[4–6] These rebound effects suggest that stimulants restrict or dampen emotional responses at the doses used in clinical practice. It has also been shown to manifest in the worsening of driving performance in adults who had taken a dose of a stimulant several hours earlier compared to those who took a placebo.[7]

The existence of rebound suggests the presence of the drug has modified the brain in some way, which in itself consists of a form of withdrawal syndrome. The rebound phenomenon also illustrates how quickly the body adapts to the presence of a drug and how rapidly withdrawal symptoms can occur after the effects of a drug have worn off.

A few case reports document a withdrawal syndrome following the complete discontinuation of prescribed stimulants in children which, as in adults, includes depression and malaise. New episodes of migraine and psychosis have also been reported.[8-10] However, there is no research that could confirm how common or severe this withdrawal syndrome is, and how long it might last when it occurs.

As with research on the long-term effects of other psychiatric drugs, the probable existence of a withdrawal syndrome following discontinuation of stimulant treatment is likely to confound attempts to assess relapse or recurrence of ADHD symptoms after medication is stopped.

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5.2.7 Polypharmacy

Polypharmacy, the prescribing of more than one drug at the same time, has increasingly become the norm in psychiatry.[1] By the 1990s 80% of people receiving psychiatric intervention were on more than one drug.[2] A particularly common combination is antidepressants and benzodiazepines.[3] A 2009 study found that up to one third of psychiatric outpatients were on three or more psychiatric drugs.[4]

Despite its commonality, little research has explored the role that this multiple prescribing has on the frequency, severity or duration of withdrawal effects, or has studied how polypharmacy affects the process of coming off the various combinations of drugs.

In the large New Zealand online survey[5] people who were taking, or had taken, more than one antidepressant reported a higher incidence of withdrawal effects (68.3%) than those who had taken just one antidepressant (e.g. Fluoxetine – 35.5%), with the exception of Paroxetine (75.9%) and Venlafaxine (70.4%).

In the large international online survey[6] 55.4% of those who had taken only antidepressants reported withdrawal effects, compared to 65.9% of those who had taken both antidepressants and antipsychotics. The figures for reported addiction were 36.8% and 47.7% respectively.

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5.3 Overall impacts of withdrawal on individuals

In addition to understanding the objective effects of withdrawal, it is also necessary to understand the subjective impact that withdrawal can have on the lives of individuals. It is important to be particularly mindful of how debilitating – physically, psychologically and relationally – withdrawal, in some cases, can be.

A recent survey of 319 people using services in England, all self-identifying as experiencing varying degrees of antidepressant withdrawal, showed that half reported being incapacitated in some way by the experience, with their capacity to perform basic daily tasks being impaired.[1] A full (27), reporting more extreme withdrawal reactions, indicated that the experience had 'ruined their lives' or had led them to 'lose everything'. Many individuals also reported that their reactions had a significant impact on their ability to work. Decision-making, memory, concentration and communication skills were also affected, to varying degrees, leading some participants to take time off or struggle through in a 'brain-fog' or a 'zombie-like' state.[1] As a result, many participants experienced some level of financial loss, while many experienced a significant lowering of their confidence and self-esteem.

Withdrawal can have far-reaching consequences, extending beyond those personally impacted to affect families, friends and associates. In the same survey, some individuals reported that withdrawal undermined their ability to support and take sufficient care of others, including their children. They also reported that their ability to engage socially was significantly impaired, leading to increased isolation.

A lack of understanding by family members about withdrawal reactions can also place further strain on relationships. When withdrawal is perceived as an 'over-reaction' this can lead to a breakdown of mutual trust and understanding. Alternatively, a decrease in self-care can also lead to heightened dependency on others, again compounding relational strain.

At its most extreme, then, withdrawal can lead to family break-ups, job losses and unemployment, reliance on state benefits, bankruptcy and even suicide.[2] While this study cannot be said to represent all those taking antidepressants, or even all those who experience withdrawal, it nevertheless indicates that for some, withdrawal can be a highly destructive experience, adversely impacting families and beyond.

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5.4 The withdrawal process and terminology

Before outlining some key strategies therapists can use to support clients through withdrawal in section 6, it will be useful to first provide some background information regarding the medical management of the withdrawal process.

5.4.1 Some background on tapering

Tapering is defined here as the slow reduction over time of a prescribed drug. It should be managed by a knowledgeable prescriber. There are various successful recommended protocols for tapering. All agree that people should never stop taking psychiatric drugs abruptly or use a rushed tapering schedule.

Schedules should be flexible and the reduction rate based on the individual's withdrawal reactions, intensity of reactions, their ability to cope and whether there is sufficient support available.[1] A knowledgeable prescriber will be mindful of such factors when supporting a person through tapering. People's experience can vary significantly, with some experiencing no withdrawal reactions whilst others can experience severe and protracted withdrawal.

Whilst it is beyond the remit of the psychological therapist to give specific tapering advice, there are some helpful rules of thumb and practical considerations with which it is useful to become familiar, especially with respect to those who do experience severe and protracted withdrawal:

- Schedules: there are multiple online resources that anyone can consult for information on tapering. One such resource distils wide experiential knowledge shared by individuals with lived experience of tapering and withdrawal into clear information about tapering schedules. For example, it states that:

  '...most people most of the time have the least-disruptive, least-disabling, and most successful outcomes by reducing their psychiatric drugs at a rate between 5–10% per month, recalculated each month based on the most recent, previous month's dose.'[2]

Recent research in the Lancet Psychiatry also supports the vital need for long tapering for some people.[3]

- Given the need to taper slowly, two years to complete withdrawal is not exceptional.[4]
- Tapering strips can help facilitate a successful withdrawal. These strips comprise a roll of small pouches that each contain a daily dose of antidepressant. Each strip contains 28 pouches, with the dose in each pouch getting successively lower over a 28-day period. In a recent study of 895 individuals wishing to discontinue their antidepressants, 71% were able to withdraw successfully with the use of one to three strips.[5] These are not currently available on the NHS but can be ordered by a prescriber from the Netherlands. See the resources section for links for further information.
- Some prescribed psychiatric drugs are available in liquid form, which can make reducing dosage easier.
- It is helpful to also be aware that some psychiatric drugs, such as antidepressants, may interact with other prescribed medical drugs. It would clearly be part of the role of the prescriber to decide if any readjustment is needed if a client decides to withdraw.[6]

5.4.2 Clarifying the language of withdrawal

In order to support clients who have decided to withdraw from psychiatric drugs, it is important to become familiar with the language used to describe withdrawal. Some of the key terms are:

'Withdrawal', 'withdrawal reaction' or 'symptom' or 'discontinuation syndrome'

All these terms refer to the various adverse reactions that result from reducing or discontinuing a drug. While the first three terms are non-contentious, 'discontinuation syndrome' is controversial. Its current meaning was first defined at the 'Discontinuation Consensus Panel' funded

by Eli Lilly in 1996[7] and has been criticised for obscuring and minimising withdrawal (perhaps for commercial reasons). [8] It is advised that it be replaced with one of the less problematic terms such as 'withdrawal reaction' or 'withdrawal symptom' or just simply 'withdrawal'.

'Relapse'

This term refers to the gradual return of the original issue, at the same intensity, for which the drug was initially taken.[9,6]

'Rebound'

This refers to one's pre-drug problems returning with greater intensity after the drug is withdrawn and is directly linked to withdrawal from the drug.[10]

'Recurrence'

This term is used to denote a new episode of distress (as opposed to the return of the original 'episode'). This new 'episode', following withdrawal, may be induced by the withdrawal itself.[10]

'Persistent postwithdrawal disorder'

This refers to the return of the original symptoms at greater intensity and/or additional symptoms related to a supposed new emerging 'disorder', which have persisted for at least six weeks after drug withdrawal.[6,10] This term is controversial, however, given that it can be used to ascribe wrongly the responsibility for an adverse withdrawal reaction to an unspecified, unidentified 'disorder' within the individual – thus medicalising a drug-induced reaction.

'Tolerance withdrawal'

Withdrawal reactions can be experienced at any stage during the prescription course and not just during tapering or after discontinuation. For instance, withdrawal reactions can be experienced when there is a marked decrease in the drug's effect (which may lead to higher drug doses being prescribed to maintain a said effect). This experience is termed 'tolerance withdrawal' – an experience that, if not properly acknowledged, is susceptible to being either denied or misdiagnosed (e.g. as failure to respond to treatment).

'Inter-dose withdrawal'

Clients who take their antidepressants or other drugs only sporadically, can experience what is known as 'inter-dose withdrawal'. This refers to withdrawal reactions that are caused by the drug's effects wearing off before the next scheduled dose is taken. Inter-dose withdrawal is more likely to be encountered with benzodiazepines or drugs with a short half-life (see 5.1).

In some cases, withdrawal reactions resulting from tolerance or inter-dose withdrawal can be as disabling as those experienced during and after tapering, and so should not be overlooked as reasons why a client may start displaying debilitating reactions.[1]

5.4.3 How withdrawal can be misinterpreted or misdiagnosed

When these different types of experience are either overlooked or confused, withdrawal can be misunderstood or misdiagnosed, with detrimental effects for the client.

In 2007, and with respect to antidepressants, Haddad and Anderson[11] provided an instructive list of the various ways in which withdrawal can be misdiagnosed:

i. **as relapse** (i.e. the original problem returning) with drugs being reinstated as a consequence. For example, as antidepressants are now widely prescribed for anxiety-related problems, and as increased anxiety is a common withdrawal reaction, ignorance of withdrawal reactions could have led, in the past, to relapse being overestimated when antidepressants were withdrawn.[12] This could still be leading, in the present, to genuine withdrawal being misread as relapse with drugs being reinstated.[13]
ii. **as failure to respond to treatment** (e.g. patients not taking prescribed drugs as directed, leading to withdrawal reactions which are then mistaken for the condition worsening, leading to dose increase or drug switching).
iii. **as a new mental health 'condition'** such as 'bipolar I or II' (e.g. with 'manic' of 'hypomanic' withdrawal reactions being misdiagnosed as the early onset of 'bipolar').

iv. **as side effects of a new drug** e.g. withdrawal reactions can also be experienced when 'switching' between antidepressants. If this is not correctly recognised, such reactions are liable to being misdiagnosed as side effects of the new drug to which the person has now switched.[11]

v. **as new physiological conditions** such as 'functional/somatic system disorders' or 'medically unexplained symptoms'.[14]

While we do not currently possess any clear evidence as to how common the misdiagnosis of withdrawal by doctors may be, we do know from anecdotal reports and qualitative survey data that it may be more common than traditionally supposed.

For this reason, some general rules of thumb have been devised to help safeguard against, or identify, such misdiagnosis:

- When did the experience arise? One prevailing view has been that it is possible to distinguish antidepressant withdrawal from relapse as the former usually commences within a few days of stopping the drugs and resolves quickly if the drug is reinstated, whereas relapse is uncommon in the first weeks after stopping treatment.[12,15] While this view on timing makes intuitive sense, it has limitations as many withdrawal variations are possible, including late onset of withdrawal and/or longer persistence of disturbances.[16] Also, the evidence is unclear as to whether relapse is uncommon in the first weeks after stopping treatment.
- Are emotional and physical reactions occurring at the same time? e.g. if unattributed feelings of anxiety or depression are present alongside physical reactions this increases the likelihood of their being related to withdrawal.[17,15]
- Is there any evidence of other medical problems? If physical reactions cannot be attributed to other identifiable medical problems they may well indicate withdrawal.[18]
- How does the experience 'feel'? many people say that withdrawal related reactions feel qualitatively different to the client's original presenting issue, with some describing withdrawal reactions as having a 'chemical' feel.[18]
- Fuller lists of commonly experienced withdrawal reactions can be found online, a good example being that given by the Withdrawal Project[2] (see resources section).

Guidance on how a psychological therapist might ethically consider using this information to assist both prescriber and client can be found in section 3. As mentioned previously, tapering should ideally be performed under the supervision of a knowledgeable medical professional although the current reality is sometimes the right support is not offered leaving people to withdraw on their own or with the support of online information and communities.[19]

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6. The role of the therapist in assisting withdrawal from psychiatric drugs – what do we know about what is helpful?

Dr Anne Guy, with Dr James Davies, Daniel C. Kolubinski, Luke Montagu & Baylissa Frederick

Currently in the UK there are no national dedicated services working with dependency and withdrawal issues. The services that do exist cover less than three percent of the national population (see 3.2 for further information about these). However, psychological therapists are already working with a proportion of those who are likely to be dependent on such drugs and who have no access to other services. For example, a 2018 survey of BPS, BACP and UKCP members asked what percentage of their clients were taking prescribed psychiatric drugs – it showed that:

- 27% said between 25-50%
- 23% said between 50-75%
- 31% said more than 75%.[1]

Given that all therapists are likely to have already found themselves in the position of working with a client in withdrawal, therapists may provide vital support by acquiring some basic additional knowledge. They do not need to be 'specialists' in order to be helpful. Education and awareness of the issues raised in this guidance will allow therapists to consider whether, and how, to begin integrating issues of prescribed drug dependence within their practice.

This guidance aims to empower and support conversations often already taking place between therapists and their clients. Therapists will need to decide for themselves whether, and to what extent, they wish to use this guidance in the context of their therapeutic work. These decisions will depend on their theoretical modality, practice setting and the individual needs of the client. The client's agency, as always, should be supported and respected at all times. Clients should be encouraged to discuss withdrawal from prescribed psychiatric drugs with a knowledgeable prescriber who can give medical advice, oversee and manage any withdrawal process appropriately. While this guidance advocates the importance of informed client choice based on full information about potential benefits and risks, it does not advocate therapists telling their clients to take, not take, stay on or withdraw from psychiatric drugs. These matters should be left to the prescriber and client to decide.

Therapists are often in the advantageous position of having a pre-existing therapeutic relationship with an individual. Based on this relationship it is possible that therapists can respond to prescribed drug issues, including withdrawal, in an integrated process.[2] There are two distinct aspects to the role that psychological therapists can play:

The first is that of helping the client understand and manage any causes and effects of emotional distress that led them to be prescribed psychiatric drugs in the first place.* The second is to support the client during drug withdrawal if this becomes necessary, when, dependent upon the clients' experience, *the first role might need to be largely placed on hold*.

* It is important to note, however, that some people are prescribed such drugs for physical conditions.

6.1 The combined wisdom approach

Although there is a lack of formal research into the effectiveness of therapeutic strategies aimed at supporting withdrawal, the theoretical, experiential and anecdotal evidence from those working in this field nonetheless offers useful suggestions. What follows in this section is a summary of the combined wisdom from these sources.[2–8]

There are five relevant factors that have been found to be helpful in supporting people to successfully withdraw:

- **access to accurate information** about withdrawal and an opportunity to discuss it and find answers to any questions before withdrawal starts
- **the involvement of a knowledgeable prescriber** to devise, help monitor and manage, a tapering programme that is tolerable and agreeable to the client
- **access to client-centred, non-authoritarian support** that empowers client choice and enables understanding of withdrawal experiences
- access to **information about and help in engaging with useful coping strategies** and/or supportive lifestyle changes
- **awareness of the need to suspend customary assumptions** about the source of distress and associated interventions (i.e. emotional processing or analysis) for the duration of withdrawal. This obliges both client and therapist to judge carefully when to resume conventional therapeutic work, ideally after any adverse withdrawal effects have abated.

Stages of support

The combined wisdom approach comprises three stages. First, the therapist helps the client prepare for the onset of withdrawal. Second, the therapist offers support during withdrawal. And finally, the therapist helps the client to adjust to a new 'normal' once withdrawal has ended. Each of these stages will now be considered in turn.

6.1.1 Stage 1: Before withdrawal is started – preparation

Preparation is essential to any successful withdrawal, and therapists may need to consider with the client whether they are ready to take their first reduction. Understanding the withdrawal process, alongside adopting a stance of non-judgmental acceptance, may assist the therapist in engaging the client in a discussion about the advantages and disadvantages of withdrawal. It also opens up a space where the client's motivations and goals can be discussed.

Before withdrawal begins, 10 areas to consider reviewing with the client are:

1. exploring whether a client feels physically and emotionally ready to begin the withdrawal process;
2. exploring who is going to provide medical support, and their relationship with their GP or other prescriber;
3. signposting and discussing relevant information on withdrawal (e.g. the desirability of slow tapering: see 5.4.1 and online resources at the end of this section);
4. discussing the possibility and general nature of withdrawal effects so clients know what to look for;
5. clarifying the high-level definitions of relapse, rebound, recurrence and withdrawal and how they might be mistaken (see 5.4.2 for the difference between these terms);
6. addressing any potential fears about the withdrawal process, including understanding what happened during previous attempts or concerns about living without psychiatric drugs;
7. identifying possible ways the attempt might be inadvertently sabotaged, either by the client or others;
8. identifying potential support networks. Are friends, family or others prepared to assist if withdrawal becomes either severe or protracted?
9. discussing the idea of the client using a diary or log to keep track of drug reductions and experiences (see the resources in Appendix A for examples of these);

10. discussing the availability of extra sessions or other contact if needed in between scheduled meetings, being clear about the limits of what can be provided.[2]

It may be useful here again to clearly distinguish between medical advice and medical information. Whilst it is clear that psychological therapists are neither trained to issue medical diagnoses nor to prescribe medical or pharmacological treatment, they may frequently be asked by clients for medical information. Discussing facts, scientific evidence or information where appropriate with clients differs substantially from offering a diagnosis, prescribing drugs or advising withdrawal. It is important to be clear about this distinction with clients (see 3.2.5 for further discussion on this).

6.1.2 Stage 2: During withdrawal – support

Therapists are likely to have more regular contact with a client than a prescriber – they are therefore in a strong position to offer the client ongoing support for the withdrawal process.[3,8] During withdrawal itself, practitioners have identified a number of useful ways of supporting clients:

- Helping clients to identify withdrawal reaction and offering reassurance that they will pass.[6,7] It is important to assume that any reactions that emerge during the transition are due to withdrawal unless proven otherwise.[3,7]
- Encouraging the client to proceed at whatever pace is right for them, while continuing to draw on relevant information to support the client's decision making.
- Suspending any attempt to understand deeper psychological material during periods when withdrawal reactions are strong, shifting instead to providing support.
- Helping clients to identify supportive practices, which enable them to manage and tolerate withdrawal experiences while they last. These may include coping strategies – see the list of 'coping mechanisms' below.
- Continuing to provide a warm and attentive therapeutic relationship, and, if consistent with your way of working, facilitate open communication between the individual, family members, the prescriber and other health professionals.

Frederick[7] states that as clients may experience intense anxiety and fluctuating levels of physical and mental pain during withdrawal, they should be encouraged to make sense of their experiences, as well as to accept them as normal to the process. Reactions can also come and go, and this is sometimes referred to as 'waves' and 'windows', where the 'waves' of reaction slowly decrease in intensity and are interspersed with 'windows' of no or reduced reactions. Some clients may only experience 'waves' within 'waves'.

It is also important to help manage expectations while advocating the use of self-care tools and techniques (see below). It is helpful for therapists to also be aware that 'emotional anaesthesia' – the inability to feel pleasure or pain – is a common withdrawal effect. If the client therefore feels distant from their emotions, any therapeutic work may need to take account of this, focusing on helping with withdrawal experiences rather than attempting to process deeper emotional material. Equally, as clients reduce their drugs, feelings can come back in sudden and very powerful ways; feelings that the client may be learning to cope with for the first time without drugs.[2]

6.1.2.1 Coping tools for use during withdrawal

The experience of those working with withdrawal supports the use of a range of coping tools. As withdrawal can sometimes be severe, it might be challenging for a client to learn new coping strategies during the withdrawal itself. For this reason, therapists might consider supporting their clients in selecting coping strategies that are both realistic and appropriate to clients' needs and current capacities.[7,8] Such client strategies may include:

a. **Acceptance/non-resistance:** maintaining a non-resisting attitude is one of the most important requirements for managing withdrawal. It involves clients staying with painful experiences as they become aware of them without struggling or attempting to stop them.

b. **Mindfulness*:** this encompasses a variety of practices that help clients get in closer touch with the present moment including their thoughts, feelings and physical sensations, importantly without judgment or resistance.

c. **Positive self-support and self-talk*:** this is a technique often used in CBT to help the client influence their mood by developing self-awareness of how they think about themselves, their present and future and where thought patterns start to become unhelpfully negative.

d. **Breathing exercises*:** such as diaphragmatic breathing can be generally helpful to clients when anxious or panicked.

e. **Emotional freedom technique (EFT)*:** this is an acupressure technique often described as 'psychological acupuncture' and involves tapping particular meridian points on the face, body and hands.

f. **Exercise:** (if tolerated and appropriate to the client's level of fitness and capacity – it can trigger a 'wave' of reactions in some).

g. **Faith:** where there is an existing faith or practice this can prove helpful for some people – for example, some report using prayer as a way of achieving a more tranquil and hopeful state.

h. **Grounding†:** this is a term used to describe a strong feeling of connection between mind and body, including a sense of being fully present. There are various exercises that can promote that sense including some mindfulness exercises.

i. **Healthy distractions**

j. **Hobbies:** coping with an intense withdrawal can leave some clients with a sense that all normal life has been lost, in some cases, irrevocably. For many clients it is helpful, when possible, to resume elements of a more balanced life, appropriate to their capacity and circumstances.

k. **Meditation*:** for those with less intense withdrawal reactions formal methods of meditation can be helpful to experience periods of respite.

l. **Self-compassion work*:** sometimes linked with mindfulness, this includes the idea of moving past self-criticism into self-kindness.

m. **Sleep:** it is important that clients take reasonable steps to maximise the probability of achieving satisfactory levels of sleep and rest.

n. **Keeping a diary*:** this can be used to track changes in experiences such as sleep and mood as reductions in dosages are made. It could also include goal setting for the next day if found to be helpful.

o. **Visualisation*:** this involves clients focusing on an image of what they want and visualising it as if it were already there.

p. **De-catastrophising*:** clients learning to recognise when they are thinking about worst case scenarios, while also working to bring attention back to what is actually happening.

Once a client has made a number of small reductions successfully and has learned what works for them in coping with any reactions that arise, some clients might choose to withdraw from counselling until they are completely off the drugs and can resume or review therapeutic work again if needed.[2]

6.1.3 Stage 3: After withdrawal is complete

At the end of withdrawal, therapists may find it useful to review the client's experience and to determine with them what further therapeutic needs they have. It may be helpful to remember the following points:

- If the client has experienced any cognitive problems as a part of their withdrawal experience it may take a while for confidence in decision making to rebuild (including the ability to say 'no' to others).
- Ensure the clients' aims and assessment of progress are realistic given their experience of withdrawal.
- If the clients' withdrawal was experienced as traumatic this might need to be considered in any further therapeutic work.[9]
- Post-withdrawal reactions can occur for some time after stopping prescribed psychiatric drugs.

* Some introductory sources of information for these can be found in the resources section in appendix A. Interested clients or therapists will be able to find further information on any of the above tools for themselves and the list is by no means exhaustive – it is intended to give an idea of the range of activities that might be of use.

6.2 Psychiatrist led multidisciplinary models

There are examples in the theoretical and research literature of psychiatrist/ prescriber-led models to support withdrawal that may be of interest for further reading if a therapist has an opportunity to suggest this in a multidisciplinary team setting. They are most notably:

6.2.1 Breggin's 'person-centred collaborative approach' to psychiatric care

This model was developed by the US psychiatrist Peter Breggin. It is a model for prescribers working with patients in psychiatric outpatient settings in the US, and is based on the core principles of working within an empathic relationship, communicating information openly and honestly and fostering empowerment and respect for the client's viewpoint, wishes and needs.[3]

Whilst it can be used in any circumstance in which a person might need more support than can be provided in a one-to-one relationship (with a prescriber or therapist), it is suggested that this approach might be of particular use when working with 'vulnerable' clients, such as:

- Adults who are dependent on others such as their parents or state authorities
- Adults who are seriously disabled, emotionally or cognitively
- Adults receiving routine psychiatric drugs including the elderly
- Any individual whose judgment or ability to take care of themselves is seriously impaired.[3]

6.2.2 Fava and Belaise's (2018) three-module approach6

This model for psychotherapeutic management of antidepressant withdrawal was developed in Italy by the psychiatrists, Gatti Fava and Guidi Belaise. It also advocates collaborative team working (e.g. comprising a psychiatrist trained in psychotherapy, a physician and clinical psychologists) to support the client's withdrawal from, in this case, antidepressants. They used CBT as their core therapeutic modality and, as with the common wisdom model already described, focused on different tasks in preparation for, during and after withdrawal.

6.3 How are UK therapists already working with withdrawal?

Some UK psychological therapists are already directly involved in supporting people in withdrawal from prescribed psychiatric drugs either through working in one of the very few dedicated services (which together cover just three percent of the population[8]) or as individual therapists working independently.

6.3.1 In dedicated services

Those working in dedicated services receive additional training about withdrawal from prescribed psychiatric drugs, including:

- How to help people prepare to withdraw
- How to engage and achieve the support of the persons' prescriber
- How to support people during withdrawal including offering relevant information, signposting helpful coping strategies and supporting gradual tapering (although plans should always be overseen by a prescriber)
- How to judge what kinds of therapeutic intervention are helpful at each stage of withdrawal.

First, it is helpful to recognise that under the umbrella of those dependent on prescribed psychiatric drugs there are different groups of patients. Broadly speaking there are those:

a. who are currently unaware they might be dependent and therefore need to be contacted proactively, and
b. those that know they are dependent and need support to withdraw through reactive services they can self-refer to.

The four existing dedicated services in the UK tend to be primarily aligned with one of these two groups:

a. Proactive services

The two small multidisciplinary services which currently cater for patients in the first group are the:

- Prescribed Medication Support Service (PMSS) covering six counties in North Wales, and the
- Bridge 'Addiction to Medicines' Programme based in Bradford.

The PMSS

- works alongside local GPs and pharmacists to identify patients taking painkillers or benzodiazepines who are in need of a drug review for a variety of reasons e.g. prescribing is beyond current guidelines, newly pregnant women. Patients can self-refer but not many do.
- Patients are invited in for a holistic assessment of their needs with one of a small number of Prescribed Medication Therapists (a nurse/ counsellor hybrid role).
- a plan is developed, usually including a personalised drug reducing regime, which is then signed off by the GP.
- other appropriate support is drawn from a range of services, including a traditional primary care counselling service.

The above model has been recommended, by the Welsh Government Petitions Committee,[10] as one possible model upon which to base the national distribution of similar services.

The Bridge in Bradford operates on a similar basis, and again focuses on painkillers, benzodiazepines and Z-drugs.[8]

People who are taking antidepressants and antipsychotics, and who are prescribed beyond guidelines, are not currently proactively contacted by either of these services.

b. Reactive services

The other two dedicated services offer support to people within their vicinity who contact them directly for help. They are:

- the Bristol and District Tranquiliser Project (BTP) and

- REST (Mind in Camden), recently taken over by a large substance misuse service provider.*

Both these services are staffed by a small number of counsellors trained in supporting withdrawal. Given that many people who contact these services report having had poor experiences with their doctors, meetings are offered in non-medical settings. However, it remains important that prescribers are involved in the withdrawal process. Those using services take responsibility for contacting their GP and getting their support for an agreed tapering plan. If the person is a local resident, the service might offer group or one-to-one counselling, with peer-to-peer support offered outside of meetings.

The above dedicated prescribed drug dependence services rely on psychological therapists who have some additional knowledge of withdrawal, but only two services work directly with doctors. The reactive services offer training to local GP surgeries on a request basis, but the people using the service remain responsible for establishing contact with their prescriber. This mirrors the situation generally for psychological therapists who either work in a multi-disciplinary team, or independently of doctors, either in an agency or alone.

6.3.2 In independent practice

A few therapists working independently with prescribed drug dependency and withdrawal have acquired substantial experience through working with this specific client group. They have considerable knowledge of the available literature, to which they may even have contributed via practice-based research. This knowledge is reflected in the 'combined wisdom' approach outlined in 6.1.

* It is important to note that whilst there is excellent work being done in substance misuse teams who are often working with people dependent on a mixture of prescribed and non-prescribed drugs, the majority of people who are only dependent on prescribed drugs understandably do not identify themselves as 'substance misusers' – they have taken drugs as prescribed by their doctors and so attending a service focused on substance misuse is regarded by them as inappropriate.

6.4 Conclusion

Throughout this section it has been emphasised that it is not the role of the therapist to decide when drugs should be withdrawn, how this may be best achieved or what tapering protocols should be deployed. However, this does not mean that therapists cannot have a critical role to play in supporting the client during withdrawal. By using this guidance, therapists will be better informed about some of the possible variables impacting a client's potential withdrawal experience. They may also be in a better position to communicate with other practitioners where appropriate (if the client does not wish to do so themselves), and to suggest that the client consults their prescriber in cases where adverse drug reactions arise before, during or after withdrawal.

Finally, if the therapist holds any particular concerns regarding how the prescriber may be understanding and managing an individual's withdrawal, it may be advisable (again, if the client does not wish to do so themselves and with their consent) to communicate these formally to the prescriber. The ethical therapist, while practising within their own sphere of professional competence, will always be thinking about the importance of the relationship their client has with their prescriber, assessing any ways in which that relationship can be supported in service of the client's needs and wants. This has been covered in more detail together with ethical considerations, such as the importance of 'informed choice', in 3.2.5.

This section reflects the current state of knowledge on what is helpful for psychological therapists to consider when working with clients withdrawing from, or preparing to withdraw from, psychiatric drugs. As withdrawal becomes better recognised throughout the mental health professions, it is hoped that appropriate and directed research will further add to this knowledge.

This guidance aims to empower and support conversations often already taking place between therapists and their clients. Therapists will need to decide for themselves whether, and to what extent, they wish to use this guidance in the context of their therapeutic work. These decisions will depend on their theoretical modality, practice setting and the individual needs of the client. The client's agency, as always, should be supported and respected at all times. Clients should be encouraged to discuss withdrawal from prescribed psychiatric drugs with a knowledgeable prescriber who can give medical advice, oversee and manage any withdrawal process appropriately. While this guidance advocates the importance of informed client choice based on full information about potential benefits and risks, it does not advocate therapists telling their clients to take, not take, stay on or withdraw from psychiatric drugs. These matters should be left to the prescriber and client to decide.

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7. Patient voices – examples from real life

Dr Anne Guy (Ed.)

The stories that follow have been offered by volunteers with the intention of helping therapists understand some possible experiences some people may have when taking or withdrawing from psychiatric drugs. These experiences are not presented as being representative, they are rather offered as examples that may illuminate some of the complexities involved. The people here are described as 'patients' as their stories are primarily about the impact of the drugs they were prescribed. Suggestions for further reading are provided at the end of the section.

Sarah's story

I took an SSRI antidepressant for 17 years. The reasons I ended up staying on the drug for that long are threefold:

a. I was lied to and told I had a chemical imbalance in my brain, so, until I investigated and challenged this 'diagnosis', I believed I needed the drug.
b. Whenever I tried to stop taking it and went into withdrawal, I was told that the drug was not addictive so my symptoms were an indication of the extent of my illness.
c. The only place to get advice on tapering was from the internet. This was sporadic and inaccurate and so my tapering efforts constantly failed.

The withdrawal symptoms I experienced were, in the early days: nausea, vertigo, IBS, weight loss, muscle tension, brain zaps, palpitations and insomnia. Each time I tried to come off the drug by tapering more and more slowly, those withdrawal effects got stronger as key bodily systems were affected by the absence of the drug.

As time went on my nervous system became more and more hyper vigilant as I felt unsafe, finding danger everywhere. I developed a number of phobic reactions to external and internal stimuli – e.g. a hot flush would be followed by a wave of fear. Each attempt at withdrawing sent me into a state of shock, in effect, to the point where I developed a movement disorder and symptoms of trauma. When I finally completely stopped the drug, it took four years for the majority of the symptoms to subside.

Peter's story

I had a decade of mixing and matching anti-psychotic, antidepressant and mood stabilising medications from my late teens to late twenties. During my early twenties the consultant psychiatrist I saw regularly had prescribed Largactil [editor's note: an antipsychotic], he then withdrew it in favour of another medication when I said it wasn't effective.

Firstly, I would say that the advice around withdrawing was sparse and effects that I might encounter never discussed. What ensued was a couple of weeks that I can only describe as 'scary' that saw me become extremely paranoid, have visual hallucinations and physical sensations.

My paranoia was based around the fact I was relaying information back to my consultant and on one occasion to an on-call duty psychiatrist that my wife had called because she was so worried. The information I relayed was dismissed as me 'lying' 'exaggerating' and 'making it up'.

I was explaining that in my peripheral vision I could see a dark figure and it seemed to be following me everywhere I was going, during this time I was experiencing repeated and extreme panic attacks. I was also getting repeated sensations in my brain, from temple to temple that I can only describe as electric shocks, these were extremely frightening, and I was convinced I was going to die.

My trust in the doctor and his profession was shaken at a time that I was very unwell, this eventually led to me taking a non-medication approach to my mental health, something that has proved successful as I look back on a decade of wellness but something my consultant did not support.

Molly's story

I was under the care of a psychiatrist in the community mental health team. I was taking a combination of Mirtazapine [editor's note: an antidepressant] and Trazodone [editor's note: an antidepressant which is also a sedative] with Zopiclone [editor's note: a 'Z'-drug, similar to a benzodiazepine, induces sleep] when I became tired of the side effects while the psychiatrist was on holiday and chose to stop taking the first two.

Within a couple of days, I had started to become increasingly 'up' and became hypomanic nudging into mania with symptoms of psychosis two days later. I became convinced my psychiatrist and the mental health team were conspiring to have me sectioned and managed to persuade my psychiatrist to discharge me although I was exhibiting pressured speech.

My therapist, who was separate from the mental health team but was funded by the CCG, was someone I confided in and who tried to get support from my psychiatrist by calling him directly. When we discussed it afterwards, he said he found the lack of support difficult to handle as he watched me spiraling out of control.

In the end I ran away to Paris and 'snapped out' of the episode after putting my safety at risk several times. The therapist had to manage the repercussions in terms of the impact on my mental health but also rebuilding my trust in medication, which I was cautious about taking with a fear of withdrawal if I ever had to stop.

Angela's story

In 2015 I was advised by my GP to try 10mg of Nortriptyline [editor's note: a tricyclic antidepressant] to see if it helped reduce the frequency of my migraines. It didn't help so after three months I wanted to wean off and asked my GP for it in liquid form so that I could do it gradually.

My GP refused saying the liquid was only licensed for elderly patients and suggested I cut the 10mg tablet in half for a week, then into quarters for a week, then a quarter every other day for a week and stop. I did this regime however when I stopped my nervous system went into chaos.

I felt extremely anxious, depressed, angry and irrational and couldn't eat or sleep. I went back to my GP and asked whether this was a reaction to stopping the medicine, but he said not on the low dose you were on! He suggested that I was having a relapse into an anxious state as I have had a history of anxiety due to PTSD although never to this extent before!

He gave me 14 days prescription of 3.75mg Zopiclone sleeping tablet without warning about how addictive they were if used for more than a few nights at a time! After two weeks I hadn't improved and was given another 14 days prescription of Zopiclone. By week four I had reached tolerance and needed to double the dose to sleep. The following day I had a bad reaction to the drug and my body became numb all over and I was having continuous, uncontrollable body jerking and finally I collapsed, and the paramedics were called.

It was only then that I googled Zopiclone and read the many articles warning about the high risk of dependency and the department of health's warning to all GPs that Zopiclone and other Benzos should not be taken for more than two weeks! To cut a long story short, I received no help or sympathy from my GP and had to plan my own escape from the hell I found myself in. The only help I could find was the Bristol drug project helpline and the one in Camden. I used the Ashton manual and tried reducing 10% of Zopiclone but the withdrawal symptoms were so bad that I couldn't get out of bed. The helplines and the Ashton manual recommended swapping to Valium, which has a longer half-life, compared with Zopiclone's very short half-life and would be easier to wean off. I made the swap and started weaning off at 10% every two weeks but still had horrendous withdrawal symptoms, I became housebound and couldn't work or drive for five months due to shaking most of the time. It was the worst time of my life and has taken me a couple of years to get my life back on track.

Majid's story

I have been a service user and carer for over 15 years. Initially my diagnoses was depression and anxiety and I was treated with venlafaxine [editor's note: an SNRI antidepressant].

Over the years my illness was then changed to personality disorder with severe depression. Over 10 years I have never seen the same consultant twice, therefore, no one knew how I was doing. The side effects made me deteriorate with little sleep. I would be mentally exhausted and sleep on benches in the park or on settees when visiting family. I was soon banned going to houses due to me not looking after myself and sleeping everywhere.

The medications made me put on weight and I was outgrowing my clothes. I can remember that I found walking up three steps a struggle and would be out of breath. I would often feel dizzy and faint, (and thought this was normal). Yet I was told by the team to carry on taking the medicines.

I took venlafaxine for 10 years and tried to come off them twice but was advised to stay on them by a locum psychiatrist and a support worker. The medicine in the long-run made me more alert and I would often be awake till 5am in the morning, which ruined my relationship with my family.

When I eventually saw a psychologist, she told me that I needed 'tweaking up' and would attend my psychiatric appointment. The problem was identified that I would say I am fine (because we were told in group therapy to be positive and I had no insight into my illness – 10 years made it a normality).

My psychologist argued my case, but the psychiatrist was saying that I was not taking my medication! (Not true and this was the first time he saw me but looked at history notes) that is why I was behaving manic. After another appointment with the psychiatrist and psychologist he changed my tablets to mirtazapine. I had no tapering of the other drug just a straight swap and had to endure sweating sleeplessness, panic attacks, anxiety attacks, paranoia and I could not trust anyone in my family.

I was later told the side effects of venlafaxine had made me manic. I am now on a different medication but still have issues with people and have flashbacks of how I was. I am more relaxed with this medication but would like to come off them so I can concentrate and do more things, because the medications make me tired and forgetful. Since I am calm, the family is calm and not alert.

Medicines are dangerous if not checked upon and can make you do stupid things which you have no control over. Venlafaxine was making me suicidal. Thank Allah I am off them and in more control of my life. Due to this I have lost my benefits and had housing eviction letters because I was not filling in the forms on time (poor concentration/sleep).

I am now diagnosed with general anxiety and depression. Not personality disorder or Bipolar which they were thinking of labelling me. It's amazing what medicines can do to you.

Suggestions for further reading

i. International survey into withdrawal from psychiatric drugs[1]

In Sep 2017 the All-Party Parliamentary Group for Prescribed Drug Dependence, in conjunction with researchers at the University of Roehampton, undertook one of the largest direct-to-consumer international surveys of its kind into withdrawal from psychiatric drugs (antidepressants, antipsychotics and benzodiazepines). There were approximately 1,700 respondents, 319 of whom were taking antidepressants living in the UK. This report summarises both the quantitative and qualitative data on those in the UK taking antidepressants (319) who reported their withdrawal experience.

ii. Petition submissions – Scotland & Wales

One hundred and fifty-eight personal accounts of people impacted by prescribed drug dependence and withdrawal (specifically for antidepressants and benzodiazepines) were submitted in response to two petitions lodged with parliamentary Petitions Committees in Scotland[2] and Wales[3] in 2017. These submissions have also been analysed and summarised in a report.[4]

References

1. Davies, J., Pauli, R. & Montagu, L. (2018). *Antidepressant withdrawal: A survey of patients' experience* (an APPG for PDD Report).
2. Scottish Petition PE01651 http://www.parliament.scot/GettingInvolved/Petitions/PE01651.
3. Welsh Petition reference P-05-784 http://www.senedd.assembly.wales/ieIssueDetails.aspx?IId=19952&Opt=3.
4. Guy, A., Brown, M. & Lewis, S. (2018). *The patient voice: An analysis of personal accounts of prescribed drug dependence and withdrawal submitted to petitions in Scotland and Wales* (an APPG for PDD Report).

Appendix A – Resources

Withdrawal Support Sites

The Ashton Manual
Research information and protocol for the treatment of withdrawal
benzo.org.uk/manual/

Battle Against Tranquilisers
Support for withdrawal from benzodiazepines, tranquillisers and sleeping pills, and drugs with similar effects
www.bataid.org

Benzo Buddies
Online support for benzodiazepine withdrawal
benzobuddies.org

Benzo.org
Articles, information, expert medical documents, news stories and personal accounts on benzodiazepine withdrawal
benzo.org.uk

Bloom in Wellness
Free info or membership at nominal monthly fee for benzodiazepine and anti-depressant withdrawal
baylissa.com

British Psychological Society
Understanding psychosis and schizophrenia
https://www.bps.org.uk/what-psychology/understanding-psychosis-and-schizophrenia

Coming Off Psych Drugs: A Meeting of Minds
A film by Daniel Mackler
bit.ly/1UcVqNh

The Council for Evidence Based Psychiatry
Providing evidence of the potentially harmful effects of psychiatric drugs to the people and institutions in the UK that can make a difference
cepuk.org

Icarus Project and Freedom Centre
Harm reduction guide to coming off psychiatric drugs
willhall.net/files/ComingOffPsychDrugsHarmReductGuide2Edonline.pdf

Lehmann, P. (Ed), (2004). *Coming off psychiatric drugs: Successful withdrawal from neuroleptics, antidepressants, lithium, carbamazepine and tranquilizers.* Berlin: Peter Lehmann Publishing.

Mad in America
Learning about psychiatric withdrawal
madinamerica.com/2017/11/learning-psychiatric-drug-withdrawal/

Mind
Coming off of psychiatric drugs
mind.org.uk/media/4727659/coming-off-psychiatric-drugs-2016-pdf.pdf

NHS
Information on coming off of antidepressants
nhs.uk/conditions/antidepressants/dosage/

Nice Guidelines
British National Formulary – Guidance, advice and information for health, public health and social care professionals.
cks.nice.org.uk/benzodiazepine-and-z-drug-withdrawalbnf.nice.org.uk/treatment-summary/antidepressant-drugs.html

Recovery Road
Antidepressant and benzodiazepine withdrawal support
http://www.recovery-road.org

Royal College of Psychiatrists
Information on antidepressants
rcpsych.ac.uk/healthinformation/treatmentsandwellbeing/antidepressants.aspx

RxISK.org
This is owned and operated by Data Based Medicine Americas Ltd. (DBM), based in Toronto, Canada. It is run by a group of high-profile medical experts with international reputations in early drug-side-effect detection and risk mitigation, pharmacovigilance, and patient-centered care.

Guide on stopping antidepressants: https://rxisk.org/guide-stopping-antidepressants/
https://rxisk.org

Surviving Antidepressants
Online forum providing peer support for tapering and withdrawal syndrome
survivingantidepressants.org/

The Withdrawal Project
Support for tapering off psychiatric medication withdrawal.theinnercompass.org

Tapering strips

For general information about tapering strips and how then can be legally ordered from the UK see: http://www.taperingstrip.org

A petition to make such strips available in the UK http://www.change.org/p/provide-tapering-strips-to-help-people-withdraw-from-antidepressant-and-antipsychotic-drugs

Dedicated services currently offering support in the UK

Bristol & District Tranquiliser Project
Support for withdrawal from tranquilisers and antidepressants
btpinfo.org.uk

The Bridge Project, Bradford: New Directions
'Addiction to Medicines' service (painkillers, benzodiazepines and Z-drugs)
https://thebridgeproject.org.uk

The Prescribed Medication Support Service (PMSS) – North Wales
https://www.nhsdirect.wales.nhs.uk/localservices/ViewLocalService.aspx?id=2556&s=Health

Shared Decision Making

NHS (2012). *Liberating the NHS: No decision about me, without me – Government Response.* Available online: https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/216980/Liberating-the-NHS-No-decision-about-me-without-me-Government-response.pdf

Coping tools for use during withdrawal – introductory links

The Withdrawal Project
Coping Mechanisms a-z, available online: https://withdrawal.theinnercompass.org/page/coping-techniques-z

Mindfulness

Baer, R. A. (Ed)(2014). *Mindfulness based treatment approaches.* https://www.sciencedirect.com/book/9780124160316/mindfulness-based-treatment-approaches#book-description

Anthony, Wen & Howard, Matthew & Garland, Eric & McGovern, Tricia & Lazar, Michael. (2017). Mindfulness treatment for substance misuse: A systematic review and meta-analysis. *Journal of Substance Abuse Treatment,* 75. 10.1016/j.jsat.2017.01.008.

Mind (2013). *Mindfulness exercises and tips.* Available online: https://www.mind.org.uk/information-support/drugs-and-treatments/mindfulness/mindfulness-exercises-tips/

NHS (2018). *Guide to mindfulness.* Available online: https://www.nhs.uk/conditions/stress-anxiety-depression/mindfulness/

Positive self-support and self-talk

Battles, M. (2019). *Self talk determines your success.* Available online: https://www.lifehack.org/504756/self-talk-determines-your-success-15-tips

Sound Mind (2017). *Positive self talk.* Available online: https://www.sound-mind.org/positive-self-talk.html

Scott, E. (2019). *Reduce stress and improve your life with positive self talk.* Available online: https://www.verywellmind.com/how-to-use-positive-self-talk-for-stress-relief-3144816

Breathing exercises

Breathing techniques
https://withdrawal.theinnercompass.org/coping/breathing

Boyes, A. (2016). Breathing techniques for anxiety. *Psychology Today.* Available online: https://www.psychologytoday.com/us/blog/in-practice/201607/breathing-techniques-anxiety

Human Givens Institute (2012). *7–11 breathing: How does deep breathing make you feel more relaxed?* Available online: https://www.hgi.org.uk/resources/delve-our-extensive-library/resources-and-techniques/7-11-breathing-how-does-deep

Emotional freedom technique (EFT) aka "tapping"

It's suggested that this is ideally learned from an EFT practitioner, although there are some You Tube videos from its founder, Gary Craig, which describe its use e.g. EFT Intro
https://www.youtube.com/watch?v=5r4kVp1yf5E

Grounding

Get self help (2018). *Grounding techniques for coping with flashbacks and distress.* Available online: https://www.getselfhelp.co.uk/flashbacks.htm

Taibbi, R. (2019). Upset? 10 grounding techniques first-aid for when you feel stressed, angry, overwhelmed. *Psychology Today.* Available online: https://www.psychologytoday.com/gb/blog/fixing-families/201905/upset-10-grounding-techniques

Meditation

Villines, Z. (2017). What is the best type of meditation? *Medical News Today*
Available online:
https://www.medicalnewstoday.com/articles/320392.php

Inner Compass, Guided Meditation Resources, Available online: https://withdrawal.theinnercompass.org/coping/guided-meditation

Headspace.com, What is Meditation? Available online:
https://www.headspace.com/meditation-101/what-is-meditation

Self-compassion work

Good Therapy, Self-compassion, Available online: https://www.goodtherapy.org/learn-about-therapy/issues/self-compassion

Neff, K. & Germer, C. (2018). *The mindful self-compassion workbook.* Guilford Press, New York.

Keeping a diary

Mind (2013). *'Self-care for anxiety' encourages keeping a diary.* Available online: https://www.mind.org.uk/information-support/types-of-mental-health-problems/anxiety-and-panic-attacks/self-care-for-anxiety/

Scott, E. (2019). *A 'how to' guide on keeping a diary.* Available online:
https://www.verywellmind.com/journaling-a-great-tool-for-coping-with-anxiety-3144672

Bipolar UK, Mood Diary (a template for tracking medications and feelings). Available online:
https://www.bipolaruk.org/FAQs/mood-diary

Visualisation

Okhai, F. (2003). *The power of deep relaxation and guided imagery.* Human Givens Institute, Available online:
https://www.hgi.org.uk/resources/delve-our-extensive-library/case-histories/power-deep-relaxation-and-guided-imagery

De-catastrophising

Beck, A.T. (1985). *Anxiety disorders & phobias.* NY: Harper & Row

Blair, L. (2017). *De-catastrophising.* Available online: https://www.theguardian.com/lifeandstyle/2017/dec/29/stop-catastrophising-expert-guide-psychologist

Drug classes and their uses – summary table

Drug class	Subtypes	Examples	Common uses
Antidepressants	■ SSRIs	■ Sertraline ■ Fluoxetine ■ Citalopram ■ Paroxetine	Depression, anxiety disorders, obsessive compulsive disorder, post-traumatic stress disorder, bulimia nervosa
	■ SNRIs	■ Venlafaxine ■ Duloxetine	Depression, anxiety disorders
	■ Other	■ Mirtazapine	Depression
	■ Tricyclic antidepressants	■ Amitriptyline ■ Lofepramine	Depression, chronic pain (amitriptyline)
Antipsychotics	■ First generation antipsychotics	■ Chlorpromazine ■ Haloperidol ■ Zuclopenthixol	Psychotic disorders, acute mania, sedation
	■ Second generation antipsychotics	■ Olanzapine, ■ Risperidone ■ Aripiprazole ■ Quetiapine	
Benzodiazepines and related drugs	■ Benzodiazepines	■ Diazepam ■ Lorazepam ■ Chlordiazepoxide	Anxiety, sedation, alcohol withdrawal
	■ Z-drugs	■ Zopiclone ■ Zolpidem ■ Zaleplon	Insomnia
	■ Pregabalin and gabapentin		Anxiety, chronic pain
Mood stabilisers	■ Lithium		Bipolar affective disorder
	■ Drugs used in epilepsy	■ Sodium valproate ■ Carbamazapine ■ Lamotrigine	Bipolar affective disorder, epilepsy
	■ Some antipsychotics	■ Olanzapine	Bipolar affective disorder
Stimulants	■ methylphenidate (Ritalin)		Attention deficit hyperactivity disorder
	■ atomoxetine		
	■ amphetamine		

Dr Joanna Moncrieff, Dr Tom Stockmann, May 2019

APPG for Prescribed Drug Dependence
http://prescribeddrug.org/

REP132/12.19

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for Prescribed
Drug Dependence