Drug Treatments in Psychiatry

Introduction

Since the 1950's effective drug treatments have revolutionised the management of many psychiatric disorders. However, there is no such thing as a "miracle cure" and the use of drugs in psychiatry requires psychological as well as pharmacological skills. Because of the sensitivity and complexity of the central nervous system interactions and adverse effects with psychotropic (i.e. psychologically active) drugs are common. The pharmacokinetics and pharmacodynamics of these compounds must therefore be understood. It is not obvious to patients that drugs can improve their psychological difficulties, especially if insight is impaired, and steps to encourage compliance are vital.

Psychosocial effects of taking medication may be positive (e.g. placebo effect) or negative (e.g. less motivation in psychotherapy because tablets are seen as a panacea). Medication is often just one component of a treatment plan that also includes psychosocial therapies. The place of drugs in this combined approach should be explained to the patient.

Compliance is increased by attention to the following:

- Empathy: the patient feels understood.
- Education: about the diagnosis and its implications.
- Explanation: about the drug, its intended action and the likely adverse effect.
- Efficiency: simplify the drug regime as much as possible and avoid polypharmacy.

Pharmacokinetics

As psychotropic drugs need to be lipophilic to cross the blood brain barrier they are readily absorbed and widely distributed. Almost all are metabolised in the liver to hydrophilic compounds excreted by the kidney. Many have long half-lives and therefore only need to be administered once a day.

Interactions:

Many interactions occur between psychotropic drugs and other psychotropics as well as other classes of drugs. It is important that you are aware that both adverse and advantageous interactions can occur. An example of an advantageous interaction may include the use of multiple anticonvulsants in the treatment of epilepsy. There is emerging evidence regarding the use of multiple mood stabilisers, antipsychotics and antidepressants. However these are strictly for use by specialists and the general rule of avoiding polypharmacy should be adhered to as far as possible. The list of interactions below only gives examples – you should consult a source such as the British National Formulary for specific information. Interactions can be divided into:

Pharmacokinetic

These interactions commonly result from induction or competitive inhibition of hepatic enzymes, for example antimuscarinics induce metabolism of phenothiazines and reduce serum level; phenothiazines compete for metabolism with tricyclics and may increase serum levels; some SSRIs inhibit the enzymes that metabolise tricyclics and phenothiazines.

Pharmacodynamic

These interactions occur when compounds act at the same receptor, synapse or system, e.g. alcohol potentiates other sedatives.

Adverse Effects:

Important adverse events for specific drug groups are detailed under each of the groups described below. However you should consult a source such as the British National Formulary for more comprehensive information. When considering adverse effects generally, it is helpful to use a simple checklist:

Dose dependent (e.g. via receptor mechanism):

- **Autonomic**: e.g. Antimuscarinic (phenothiazines, tricyclics) namely: dry mouth, blurred vision, closed angle glaucoma, tachycardia, constipation, ileus, urinary retention.
- Cardiovascular: e.g. Hypotension, Arrhythmia (phenothiazines, tricyclics)
- Psychiatric: e.g. Delirium (phenothiazines, tricyclics)
- Neurological: e.g. Seizures (phenothiazines, tricyclics)
- Endocrine: e.g. Amenorrhoea, weight gain (phenothiazines)

Dose independent (e.g. via autoimmune mechanism):

- Hepatitis: (phenothiazines, tricyclics)
- Blood dyscrasias: (phenothiazines, tricyclics, N.B. clozapine)
- Skin rashes: (phenothiazines, tricyclics)

High risk situations (Pregnancy, breast feeding, the elderly and the physically ill):

Lipophilic drugs readily cross the placenta and also enter breast milk. Teratogenesis (e.g. carbamazepine) and neonatal toxicity (e.g. sedation with benzodiazepines) may occur. Whenever possible psychotropics should be avoided in early pregnancy or when there is a risk of pregnancy and many are contraindicated in late pregnancy or breast-feeding. When they are required their use should only be used under the supervision of a specialist. An evidence based approach can be aided with use of information from local Drug Information Centres or organisations such as Motherisk.

The elderly may have low body mass, reduced hepatic metabolism and a less adaptable central nervous system, rendering them prone to adverse effects (e.g. delirium). Reduced dosages are often advisable. Beware interactions with concurrent physical illness and its treatment.

Drug Groups

Psychotropic drugs can be considered in six main classes. Examples of commonly used compounds are given but the British National Formulary or Data Sheet Compendium should be consulted for dosages and more detailed information regarding adverse effects and interactions before prescribing.

Hypnotics and Anxiolytics

There is no clear distinction between hypnotics and anxiolytics. Short half-life compounds tend to be used as hypnotics because of the reduced risk of "hangover" effects. Like alcohol, they probably act on GABA receptors and, like alcohol impair ones ability to drive or operate machinery and, like alcohol, cause tolerance with physical and/or psychological dependence. It is important to understand that other than for short-term amelioration of anxiety, antidepressants are the treatment of choice for the pharmacological management of anxiety.

Benzodiazepines

Mechanism of action:

It is believed that benzodiazepines act by enhancing the action of GABA at GABA_A receptors. These receptor complexes have been identified as having specific benzodiazepine (as well as barbiturate and alcohol) binding sites on them.

Examples:

Diazepam, because of its long half-life it is used as an anxiolytic.

Temazepam has a short half-life and is used as a hypnotic.

Lorazepam and Triazolam are more potent short half-life benzodiazepines and generally should be avoided as they cause intense withdrawal phenomena and dependence. Lorazepam can be given intramuscularly and is used for rapid tranquillisation of severely disturbed patients in an in-patient setting.

Cautions:

Respiratory failure, breast feeding, previous addiction.

Adverse effects:

Drowsiness, confusion, disinhibition or aggression.

Interactions: Potentiate other sedatives, e.g. alcohol.

Tolerance:

May develop within 3 to 14 days.

Withdrawal:

Insomnia, anxiety, sweating, tremor, perceptual disturbance, delirium and seizures. Can manifest within hours of last dose or take up to 3 weeks depending on drug half-life and may persist for months. Because of dependency and withdrawal problems current guidelines limit their use to a maximum of 2-4 weeks for severe, disabling anxiety or insomnia. Alternatives include advice about insomnia, counselling, cognitive or behavioural psychotherapy and antidepressants.

Others

Alternative hypnotics and anxiolytics are similar to benzodiazepines in terms of indications, tolerance and withdrawal but they have some different adverse effects.

Examples:

Barbiturates; these should be avoided as they are dangerous in overdose.

Chlormethiazole; causes conjunctival, nasal and gastric irritation. Its stimulant properties are addictive.

Zopiclone; short acting drug and hence used as a hypnotic. Addictive properties probably similar to benzodiazepines.

Buspirone: This drug is occasionally used as an anxiolytic. It has a totally different mechanism of action compared to all of the other drugs described above, being a $5-HT_{1A}$ receptor agonist. There is no evidence of addiction or abuse potential associated with its use. However it may be less potent at reducing anxiety than benzodiazepines. In addition, its therapeutic effects take 2-3 weeks to develop, rather than occurring rapidly as seen following a single dose of a benzodiazepine. Buspirone is also mildly antidepressant.

Antidepressants

It is important to remember that antidepressants do not alter normal mood. This group of drugs is effective in the treatment and prophylaxis of depressive disorders. Antidepressant action takes 2-4 weeks to develop, therefore it is important to encourage persistence. Some antidepressant may also be used to treat anxiety disorders. Whilst it is important to emphasise that they are not addictive some are very toxic in overdose and a discontinuation syndrome may occur unless a gradual withdrawal programme is used.

Tricyclics (TCAs)

This group of antidepressants used to be the main stay of treatment of depressive illness. However they are characterised by a number of drawbacks including a high rate of side effects that may lead to lowered compliance. Selection of a drug from within the class tends to be on the basis of adverse effect profile. Long half-lives allow single daily dose regime and they are often given at night when their sedative effect may be helpful. Most are too dangerous for unsupervised supply if the patient is a suicide risk.

Mechanism of action:

The therapeutic effect of TCAs derives from their ability to block the re-uptake of 5-HT and/or noradrenaline into neurones. This effect occurs after a single dose of the drug, while the therapeutic benefit takes some weeks to occur (see above). It is believed that this requires adaptive changes need to occur in various receptor systems as a result of the increased levels of 5-HT and noradrenaline. Note that some TCAs are relatively 5-HT selective (e.g. clomipramine) and some relatively noradrenaline selective (e.g. lofepramine). It is not know if sub groups of patients respond better to one type rather than another.

Examples:

Amitriptyline and Dothiepin are more sedative and used to be used particularly for agitated depression.

Imipramine is less sedative and used to be used particularly for retarded patients.

Lofepramine has less marked anticholinergic effects and is less likely to cause dangerous cardiac arrhythmia in overdose.

Clomipramine is the most serotonergic selective TCA and the only one that is effective in treating OCD. It may also be more effective than the others for treatment resistant depressed patients.

Indications:

Depressive disorders, anxiety disorders.

Cautions:

Cardiac disease, glaucoma (closed angle), prostatism, epilepsy, hepatic impairment.

Adverse effects:

Large number due to multiple pharmacological actions of the drugs:

Muscarinic M_1 receptor antagonism - anticholinergic effects including dry mouth, blurred vision, constipation, urinary retention and impotence. Also delirium at high doses or in susceptible patients.

Histamine H₁ receptor antagonism – sedation and weight gain.

Adrenergic α receptor antagonism – postural hypotension.

Direct membrane effects – reduced seizure threshold, arrhythmia.

Serotonin 5-HT₂ receptor antagonism – weight gain (and reduced anxiety).

5-HT and NA reuptake blockade – mania (and antidepressant effects).

Interactions:

Hypertensive crisis with adrenaline or MAOIs.

Monoamine Oxidase Inhibitors (MAOIs)

Traditional MAOIs non-selectively and irreversibly inhibit monoamine oxidase (MAO-A and MAO-B). MAO-A metabolises noradrenaline and 5-HT, MAO-B phenylethylamine and benzylamine, while dopamine and tyramine and metabolised by either. MAOIs may have a role in atypical and treatment resistant depressions, though nowadays their use is limited due to the dietary restrictions, interactions and toxicity in overdose of this group of drugs.

Mechanism of action:

MAOIs led to increases in 5-HT and noradrenaline (and to a certain extent dopamine) due to blocking their metabolism. However, like other antidepressants, the therapeutic response is delayed and depends on neuronal adaptation to increases in monoamine levels.

Examples:

Tranylcypromine is an amphetamine analogue and is the only antidepressant that causes dependency. It is best avoided.

Phenelzine is safer because it is less stimulant.

Moclobemide is a non-traditional MAOI. It is both reversible and selective, just blocking MAO-A. As a result there is less of a problem with dietary restrictions.

Cautions:

Cardiac disease, epilepsy, hepatic impairment, ECT.

Adverse effects:

Anticholinergic effects, hypotension, oedema, fits, neuropathy, drowsiness, delirium, mania, hepatitis, leucopenia.

Interactions:

Hypertensive crisis with sympathomimetics and tricyclics due to monoamine excess, potentiates CNS depression with opiates.

Tyramine reaction: Tyramine is a naturally occurring sympathomimetic normally inactivated by MAO in the gut. Matured protein foods (e.g. cheese, meat or yeast extract, game, alcoholic or de-alcoholised drinks) are tyramine rich and therefore cause hypertensive crisis. A warning card must be given to patients on traditional MAOIs. Remember these restrictions apply for at least 2 weeks after MAOIs are stopped (the time taken for MAO to regenerate following their irreversible blockade by traditional MAOIs).

Selective Serotonin Re-uptake Inhibitors (SSRIs)

These newer antidepressants are no more effective than TCAs or MAOIs but they have relatively few adverse effects and are much safer in overdose. As a result they have become the mainstay for the treatment of depression. In addition they are effective in treating anxiety disorders, including OCD, and possibly bulimia nervosa and impulse control disorders.

Mechanism of action:

These drugs selectively block the re-uptake of 5-HT leading to neuronal adaptive processes that produce the therapeutic effect.

Examples:

Fluoxetine has a long half-life (takes 5 weeks to be excreted fully). This is a disadvantage if patients require the drug to be swapped to another, but is useful in patients who occasionally forget their medication. Also helps to prevent any discontinuation syndrome on stopping.

Paroxetine has a short half-life so its advantages and disadvantages are the converse of fluoxetine. It can still be prescribed once a day.

Citalopram is the pharmacologically cleanest drug. Its half-life is slightly longer than paroxetine.

Indications:

Depressive disorders, anxiety disorders, panic disorder, OCD, impulse control disorders.

Cautions:

Renal or hepatic impairment, pregnancy, epilepsy (though may be better than TCAs).

Adverse effects:

These can be divided into:

Short lived. Last for 3 or 4 days following institution or increases in dose. Include nausea, anorexia, and increased anxiety. It is especially important to warn patients that these drugs may transiently increase their anxiety symptoms before they improve.

Long term. Last throughout the course of treatment. Include headache, insomnia, and sexual dysfunction (delayed ejaculation, anorgasmia).

Interactions:

Toxicity with MAOIs and anti-migraine drugs such as sumatriptan.

Other antidepressants

There are an increasing number of newer antidepressants that do not fall into the categories above. They are increasing in usage and may carry some specific advantages. They include:

Reboxetine:

Selective inhibitor of noradrenaline re-uptake (NARI). May be indicated for depression with anergia, poor motivation and concentration though definitive research awaited.

Caution in renal and hepatic impairment, urinary retention and glaucoma. Adverse effects mainly "anticholinergic" (because of increased noradrenergic activity) – see under TCAs. Interacts with MAOIs

Venlafaxine:

Inhibits 5-HT and noradrenaline re-uptake (like TCAs) but doesn't have other pharmacological actions of TCAs therefore much better tolerated and much safer. Some research and a lot of anecdote that it is effective in severe and treatment resistant depression. Also effective for anxiety disorders including panic disorder and OCD

Caution if history of myocardial infarction. Adverse events essentially the same as for SSRIs. Can also cause increases in blood pressure at higher doses. Avoid use with MAOIs

Trazodone

Primary therapeutic action appears to derive from potent 5-HT₂ receptor antagonism and weak 5-HT re-uptake inhibition. Particularly used for generalised anxiety or as a non-specific sedative – it is well tolerated, non-addictive and safe in overdose. It is not infrequently added in small doses to an SSRI to help improve sleep.

Adverse effects are primarily sedation (histamine H_1 antagonism). A rare serious side effect is priapism.

Mirtazepine

Complex pharmacological actions. Its therapeutic effect is believed to derive from adrenergic α_2 receptor antagonism which leads to increased 5-HT and noradrenergic neurotransmission, since these receptors inhibit both systems. In addition: blocks 5-HT₂ receptors which helps treat anxiety; blocks 5-HT₃ receptors which helps prevent sexual dysfunction with the drug; blocks histamine H₁ receptors which produces sedation. May be of benefit in severe and treatment resistant depression due to potent effects on both 5-HT and noradrenergic systems (c.f. clomipramine and venlafaxine). Also useful where sedation is required.

Caution in epilepsy, hepatic and renal impairment, cardiac disorders, urinary retention, diabetes mellitus. Adverse effects include weight gain, sedation and rarely a reversible agranulocytosis.

Mood Stabilisers

This group of drugs' primary indication is bipolar affective disorder where they are useful for prophylaxis. There is an expanding number of drugs in this group and some may be effective in the treatment of mania and others in the treatment of depressive episodes. In addition some may be useful as adjuncts to antidepressants in resistant unipolar affective disorder.

Lithium

Lithium is an inorganic, hydrophilic substance distributed and excreted via sodium transport mechanisms. As a result changes in sodium balance may alter excretion. Its therapeutic range is very close to toxic levels and it is therefore important to check serum levels at least 3 monthly (initially weekly). Blood samples should be taken 12 hours after last dose (time for distribution of drug) and a maintenance level of between 0.4 and 1.0 millimoles per litre should be aimed for.

Mechanism of action:

It is unknown how lithium exerts its therapeutic effects. One possibility relates to its ability to block steps in the inositol phosphate second messenger system.

Indications:

The acute treatment of mania, prophylaxis of bipolar disorder and antidepressant augmentation in treatment resistant depression.

Cautions:

Renal or cardiac impairment, pregnancy, breast-feeding.

Interactions:

Diuretics, NSAIDs, ACE inhibitors (increased levels); MAOIs, carbamazepine, high dose antipsychotics.

Adverse effects:

Polyuria and polydipsia, fine tremor, gastrointestinal disturbances, oedema, weight gain, hypothyroidism and goitre, ECG changes.

Intoxication:

Rare below levels of 1.5 millimoles per litre but can occur within the therapeutic range. Anorexia, vomiting, diarrhoea, weakness, ataxia, drowsiness, confusion and coma may progress to renal and circulatory failure, convulsions and death.

Preparation for lithium:

Renal function, electrolytes, thyroid status and ECG must all be checked. Patients must know signs of intoxication and risk that a low sodium diet, dehydration or other drugs may dangerously increase levels. Initially check levels weekly. A warning card should be given.

Other Mood Stabilisers

These are all anticonvulsants. Recently there have been several new anticonvulsants introduced to treat epilepsy. Their utility in bipolar disorder is generally unknown. However it should be noted that not all anticonvulsants are mood stabilisers (for example phenytoin).

Mechanism of action: How anticonvulsants are mood stabilisers is unknown. It has been hypothesised that it relates to their ability to prevent 'kindling' the process by which epilepsy may develop. This is because the life time course of bipolar disorder is for an increase in the frequency of episodes over time.

Examples:

Carbamazepine is used at levels therapeutic in epilepsy. It is not as effective as lithium but is less toxic. Adverse effects include drowsiness or neutropenia. Interaction with lithium or MAOIs may cause neurotoxicity.

Sodium valproate may be as effective as lithium. In addition it may be better for patients who have very frequent episodes of illness (3 or 4 episodes a year or more). It may be most appropriate for patients who have a high proportion of manic episodes. Adverse effects include hepatic dysfunction, blood disorders, nausea, ataxia and tremor.

Lamotrigene has recently been shown to be helpful in depressed bipolar patients and for prophylaxis. It is associated with serious and sometimes fatal skin disorders (Stevens-Johnson syndrome) The drug needs to be introduced slowly and patients warned to look out for flu like symptoms and skin rashes. Other adverse effects include headaches, ataxia, nausea. It can alter the levels of other anticonvulsants so great care is needed if used in combination with other mood stabilisers.

Gabapentin. This drug has been increasingly used due to early reports of benefit and good tolerability. Recent research suggests it is ineffective in bipolar disorder.

Antipsychotics

These are also called neuroleptics or major tranquillisers. They are indicated in schizophrenia, mania and psychotic depression and may be useful for short-term sedation in aggression or agitation. Their antipsychotic effect may take several weeks to develop. Antipsychotics also have important prophylactic effect in schizophrenia. Generally, because of their long half-lives a single dose may be prescribed. Antipsychotics are divided into "typical" and "atypical".

Typical Antipsychotics

These drugs have been in use for around 5 decades and their introduction was a major advance in the treatment of schizophrenia.

Mechanism of action:

The therapeutic effect of typical antipsychotics depends on their ability to block dopamine D_2 receptors. Indeed the clinical dose of these drugs correlated highly with their affinity for these receptors. This is a major argument in favour of the "dopamine hypothesis" of schizophrenia. Some of these drugs are available in depot form that can be administered to patients by intramuscular injection every 1-4 weeks. This is a great advantage in-patients with poor compliance.

Cautions:

Cardiovascular and cerebrovascular disease, parkinsonism, epilepsy, pregnancy and breast feeding, renal and hepatic impairment, prostatism, glaucoma.

Adverse effects:

Pharmacologically they are similar to TCAs without significant monoamine re-uptake blockade (and so they are not antidepressant) but with dopamine antagonism (so that they are antipsychotic). As a general rule therefore their side effects are similar to TCAs. However they have important additional extrapyramidal effects (EPS) due to the dopamine antagonism in the basal ganglia:

- An immediate effect may be acute dystonia often of jaw, neck or external ocular muscles.
- After days or weeks of treatment; parkinsonism with the classical triad of akinesia, rigidity and tremor may manifest. Both of these earlier effects respond to antimuscarinic drugs (e.g. procyclidine, orphenadrine).
- Akathisia, a subjective restlessness of the legs often leading to pacing, may appear within weeks of starting treatment but does not respond to antimuscarinics.
- Tardive dyskinesia, appears later, after at least several months. It manifests in involuntary, choreiform movements mainly of mouth and face but sometimes also limbs and trunk. The condition is often irreversible despite neuroleptic withdrawal.
- Dopamine blockade in hypothalamus also leads to hyperprolactinaemia with gynaecomastia, galactorrhoea and amenorrhoea.

Other adverse effects include:

- Neuroleptic Malignant Syndrome (NMS): A rare but potentially fatal syndrome of rigidity, hyperpyrexia and clouding of consciousness. Serum creatinine phosphokinase levels are elevated. There is a higher risk in hot weather or pyrexial patients.
- Blood dyscrasias, hepatitis, skin rashes and photosensitivity

Interactions:

Increased effect of other hypotensives or sedatives.

Examples:

These drugs may be usefully grouped according to adverse effective profile:

- Sedation +++; anticholinergic ++; Extrapyramidal ++ *Chlorpromazine*; an aliphatic phenothiazine also a potent alpha adrenergic antagonist causing marked hypotension.
- Sedation ++; anticholinergic +++; extrapyramidal + *Thioridazine*; a piperidine phenothiazine. The intrinsic anticholinergic activity of these drugs limits their extrapyramidal effects by the same mechanisms that anticholinergic medication is effective in Parkinson's disease. However thioridazine has a high risk of leading to ECG changes and in the future this may severely limit its use.

 Sedation +; anticholinergic +; extrapyramidal +++ *Trifluoperazine*; a piperazine phenothiazine *Haloperidol*; a butyrophenone *Flupenthixol decanoate*; *Fluphenazine decanoate*; depot preparations

Atypical Antipsychotics

In recent years there has been the development of several new antipsychotics. These have often been referred to as "atypical". A confusing aspect is that the word "atypical" has developed two different meanings. Some people use it to refer to drugs that have a low incidence of extrapyramidal side effects (EPS). However others use it to refer to a slightly different pharmacology of some drugs that includes a lower potency at blocking D_2 receptors but also a high potency at blocking 5-HT₂ receptors. Such drugs are 'modelled' on clozapine (see below) and have led to hypothesises that the ratio between D_2 and 5-HT₂ receptor blockade is important in the treatment of schizophrenia. All these drugs tend to be better tolerated than typical antipsychotics. However they are considerably more expensive. There is an ongoing debate about whether they should be prescribed "first line" or reserved for patients who fail to respond or do not tolerate typical antipsychotics.

Sulpiride

Substituted benzamide that is a 'clean' dopamine antagonist therefore without many of the side effects of typical antipsychotics. In addition it has low affinity for basal ganglia dopamine receptors and therefore causes less EPS (hence it's classification by some people as atypical).

Should be avoided in renal impairment. Less sedating than some typical antipsychotics, though still can be used for acute psychosis

Clozapine

Mixed $5-HT_2/D_2$ antagonist. The only drug that has been conclusively shown to have higher efficacy than typical antipsychotics in regard to treatment resistant schizophrenic patients. Also some evidence that it is able to treat 'negative' symptoms. However it is associated with potentially fatal agranulocytosis. As a result it is only indicated in schizophrenia which has not responded to other antipsychotics. In addition patients MUST have regular (initially weekly) blood counts performed before being prescribed the medication.

Other adverse events include marked sedation, hypersalivation, anticholinergic effects, but less EPS than typical antipsychotics.

Risperidone, Olanzapine, Quetiapine

All different but they have varying degrees of $5-HT_2/D_2$ antagonism which is believed to produce their therapeutic benefit. It remains unclear whether they have advantages over typical with regard to efficacy in the same way that clozapine does. They may have some efficacy with regard to negative symptoms.

All are associated with marked weight gain but less EPS compared to typical antipsychotics.

- Risperidone can cause EPS at higher doses. Can also cause anticholinergic effects and hyperprolactinaemia. Given once or twice a day.
- Olanzapine is reasonably sedative and so of use in agitated patients. It is contraindicated in glaucoma and should be used with caution in myeloproliferative disease.
- Quetiapine needs to be administered twice a day.

Anti-dementia drugs

Currently there are two anticholinesterase drugs available to treat Alzheimer's disease. They are believed to act by increasing levels of acetylcholine. They are effective in treating mild to moderately impaired patients and appear to slow the rate of cognitive decline, rather than reversing it.

Examples:

Donepezil, Rivastigmine.

Cautions:

Cardiac conduction problems, asthma, Chronic Obstructive Airways Disease, peptic ulceration.

Adverse effects:

Nausea, vomiting, diarrhoea, bradycardia and AV block, peptic ulceration.

Interactions:

Antiarrhythmics and beta-blockers.

Drugs used in the treatment of addictions

There are a few drugs in this class at present. They are all very different and include:

Disulfiram

An adjunct treatment for alcohol dependency along with psychological treatments. The patient must be motivated to take the drug regularly for it to be helpful.

Produces an unpleasant systemic reaction (e.g. tachycardia, palpitations, flushing and headache) if alcohol is also taken. Large dose of alcohol can produce a severe reaction that potentially can be fatal. Can cause drowsiness, nausea and vomiting.

Acamprosate

Is believed to aid abstinence in combination with counselling.

Can cause gastrointestinal disturbance. Continued alcohol consumption negates the therapeutic effect.

Buproprion

Noradrenaline and dopamine re-uptake inhibitor that is used in the USA as an antidepressant. Recently launched in the UK to aid withdrawal from smoking.