Over the past few decades various antidepressants have come onto the market. These treatments have had a substantial impact on the lives of thousands of people with depression, and have become the mainstay of treatment for the condition.

Antidepressants are not routinely recommended for initial treatment of patients with subthreshold or mild depression because evidence does not suggest that the benefits of treatment outweigh the risks. For these patients, preferred first-line management includes watchful waiting, guided self-help programmes (based on psychological therapies, such as cognitive behavioural therapy [CBT]) and structured supervised exercise programmes; discussion of these non-pharmacological therapies is beyond the scope of this article.

There is more evidence for the effectiveness of antidepressants for people with moderate-to-severe depression; the response rate for such patients is approximately 50%, compared with 30% for placebo. Some 55–65% of patients will continue to experience some symptoms despite antidepressant treatment; CBT and other psychological therapies should be considered alongside antidepressants for these patients.

The aim of antidepressant therapy is to achieve remission of acute symptoms (complete relief) and full functional recovery.

Antidepressant choice
There are nearly 30 antidepressants licensed for use in the UK. With a couple of exceptions (namely reboxetine and agomelatine) all clinically effective antidepressants augment serotonergic activity in the brain. Although evidence is conflicting, it does not consistently demonstrate that any specific antidepressants are significantly better than others. Therefore, all antidepressants are considered to be of a similar general efficacy, when used at therapeutic doses for sufficient time. The tolerability and acceptability of different antidepressants vary among patients.

Generally, selective serotonin reuptake inhibitors (SSRIs; see Box 1, p229) are recommended first line because they have a more favourable risk-benefit ratio than other classes, such as tricyclic antidepressants (TCAs; see Box 2, p230), monoamine-oxidase inhibitors (MAOIs; see Box 3, p231) or other antidepressants (see Box 4, p232), which should be reserved for situations where first-line antidepressants have failed.

The following should be considered when choosing an antidepressant:

- Clinical presentation, including any associated psychiatric disorders or general medical problems (eg, liver impairment)
- Previous response to antidepressants
- How well a patient tolerated any previously used antidepressants, including any adverse effects experienced
- Suicide risk and likelihood of overdose
- Potential side effect profile of the medicines
- Concurrent medication and risk of drug interactions (eg, tramadol and SSRIs, which can cause serotonin syndrome; see p232)

Prescribers should give patients the opportunity to contribute to decisions around antidepressant selection; the choice should be based on an informed discussion of the relative benefits and side effect profiles of the appropriate antidepressant options.

Response to antidepressants
People’s responses to antidepressants vary considerably. Currently there is little evidence to guide prescribing according to subtypes of depression or personal
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characteristics. It is also not possible to predict the patients who will respond best to antidepressants, or from which antidepressants they will derive the most benefit.

Patients who show an early response (within the first two weeks) are more likely to respond fully and those without an early response are unlikely to respond later. Patients with depression who have comorbidities show poorer responses to antidepressants, as do those who have been depressed for over one year.

If a person shows little or no improvement after adherence to a treatment dose of an antidepressant for around two to four weeks they are unlikely to respond fully to that antidepressant. If the first antidepressant a patient uses is ineffective, the dose can be increased (if it is an antidepressant with a suspected dose-response relationship) or it can be stopped and an alternative antidepressant tried, either from the same or a different class (see below for details about switching between therapies).

**Maintenance treatment**

Once a person finds an antidepressant that works and is well tolerated it should be continued, at the same treatment dose, for at least six months after remission from the acute episode. This maintenance treatment period should be extended to 12 months for older adults and to 24 months for people who have recently had two or more depressive episodes that have caused considerable functional impairment.

**Stopping or changing treatment**

Stopping an antidepressant, or changing from one to another, should not be done abruptly. Clinicians should advise patients only to stop their antidepressants in a planned manner, in consultation with their prescriber, to avoid symptoms of withdrawal or discontinuation syndrome. Symptoms of abrupt withdrawal can include gastrointestinal and influenza-like symptoms, sensory and sleep disturbances, agitation, dizziness, anxiety and irritability. Such symptoms are usually mild and self-limiting, and last for about a week, but for some people they can be more severe and long-lasting.

Specific circumstances where it is appropriate to stop an antidepressant abruptly include if a patient develops hypomania or experiences an intolerable adverse reaction.

Antidepressants are not addictive; they are not associated with tolerance and craving.

**Treatment-resistant depression**

If a person does not respond to monotherapy (and has tried two different antidepressants), despite good adherence and appropriate treatment doses, he or she is diagnosed with treatment-resistant depression (the chance of a patient responding to subsequent antidepressants decreases with each failed therapy). In such cases, psychological therapies should be offered in combination with antidepressants. Pharmacological approaches include the use of the MAOIs phenelzine as mono-therapy; however, the dietary restrictions required and its toxicity in overdose mean that this is not a suitable option for many patients. Other strategies include augmentation of existing antidepressant treatment with lithium, an atypical antipsychotic or another antidepressant (eg, mirtazapine) — see Box 4 (p232) and Box 5 (p233).

For depression with psychotic symptoms adjunct antipsychotic therapy is recommended; antipsychotics should not be used as monotherapy.

**Electroconvulsive therapy**

Electroconvulsive therapy (ECT) is a treatment option for acute severe depression that has not responded to medicines. It is also an option if a person’s depressive illness is life-threatening and a rapid improvement in clinical condition is required.
In-depth discussion of ECT is beyond the scope of this article.

Antidepressants for older people
Due to pharmacokinetic and pharmacodynamic changes, older adults (>65 years of age) usually require lower antidepressant doses, are more sensitive to adverse effects and take longer to respond to treatment. Moreover, the consequences of side effects, such as sedation, can be profound for older people (eg, increased risk of falls), so this should be considered carefully when selecting an antidepressant.

Another factor to consider is that many older adults take numerous medicines, therefore increasing the potential for drug interactions and drug-disease interactions. The principles of treatment in older adults with dementia or Parkinson’s disease should be the same as for patients without these comorbidities; SSRIs are the antidepressants of choice.

Antidepressants for youths
Fluoxetine is the only antidepressant licensed in the UK for the treatment of depression in children under 18 years of age. It should only be used for the treatment of those eight years of age or older with moderate-to-severe episodes of depression that are unresponsive to psychological therapy. The use of CBT with fluoxetine has been shown to be more effective than using fluoxetine alone, CBT alone or placebo. The dose of fluoxetine for children is usually 10mg daily, for which the syrup (20mg/5ml) must be used.

The little evidence that exists around the treatment of moderate-to-severe depression in younger children (<13 years) suggests that antidepressants show a small benefit over placebo (although this is not statistically significant).

For all children, if treatment with fluoxetine is unsuccessful or not tolerated, another antidepressant can be considered, eg, off-label use of sertraline or citalopram. TCAs are not useful for the treatment of prepubertal children and there is only marginal evidence to suggest a slight benefit in adolescents. Because of this, and the poor tolerability of TCAs and their toxicity in overdose, they are not recommended for use in children or adolescents.

The European Medicine Agency’s Committee for Medicinal Products for Human Use conducted a review into the use of SSRIs and SNRIs for paediatric patients. It concluded that these medicines increased the risk of non-fatal suicide-related behaviour (suicide attempt or suicidal thoughts), self-harm and hostility (predominantly aggression, oppositional behaviour or anger) compared with placebo. Therefore, if antidepressants are used for children and adolescents, they should be monitored carefully for the appearance of suicidal behaviour, self-harm or hostility, particularly at the beginning of treatment.

Pregnancy and breastfeeding
Certain medicines, including psychotropics, when taken during pregnancy can increase the risk of fetal

Box 2: A closer look at tricyclic antidepressants
Tricyclic antidepressants (TCAs) block the presynaptic reuptake of serotonin and noradrenaline (to different extents depending on the individual drugs). It should be noted that there is not a common mechanism of action among TCAs — they are classified as TCAs based on chemical structure.

TCAs have varying degrees of antimuscarinic side effects, ie, dry mouth, constipation, blurred vision and urinary retention. Other adverse effects include dizziness and sweating.

With the exception of lofepramine, TCAs are more cardiotoxic in overdose than other equally effective antidepressants (eg, SSRIs). Consequently TCAs, except lofepramine, are generally considered unsuitable for older adults.

TCAs can be roughly divided into those with marked sedative properties and those that are less sedating. These, and other properties of the individual TCAs, are outlined below:

- Amitriptyline — active metabolite is nortriptyline; sedating and particularly dangerous in overdose
- Clomipramine — predominantly selective for serotonin; sedating and moderately toxic in overdose
- Dosulepin — not recommended because of increased cardiac risk and pro-convulsive effects, which

- Imipramine — more selective for noradrenaline than other TCAs; few sedative properties; marked antimuscarinic side effects
- Lofepramine — few antimuscarinic or sedative effects; low risk of toxicity in overdose
- Nortriptyline — less sedating than other TCAs
- Trimipramine — sedating
- Trazodone — TCA-related antidepressant with sedative properties; fewer antimuscarinic and cardiotoxic effects than other TCAs

account for toxicity in overdose; sedating
malformations. However, assessing this risk is difficult because of a paucity of data and confounding factors, such as physical and mental comorbidities, concurrent medication and lifestyle choices (eg, diet, smoking, substance misuse). Furthermore, it is impossible to be sure that any medicine is safe in pregnancy because it is unethical to conduct the randomised placebo-controlled trials that would be necessary to demonstrate this fact unequivocally.

Although the use of antidepressants during pregnancy can affect fetal neurodevelopment, and has been associated with a small increase in spontaneous abortion and preterm labour, it remains important to treat a pregnant woman's depression satisfactorily.1,2,3

TCAs, such as amitriptyline, imipramine and nortriptyline, are often considered the first choice when starting an antidepressant in pregnancy, based on cumulative data demonstrating no evidence of increased birth defects.4,5 Fluoxetine and paroxetine have been associated with a small increased risk of congenital cardiac defects.6

All antidepressants carry the risk of neonatal withdrawal or toxicity so, in the first few weeks after delivery, infants should be monitored for signs of withdrawal and toxicity.

The World Health Organization advocates breastfeeding for at least four to six months post-partum and this has implications for antidepressant choice.7 Each case should be assessed on an individual basis because it may not be appropriate or necessary to change the antidepressant for a woman who is already stable on therapy. In any circumstance, close monitoring of infants is advised.

Data around the use of SSRIs by breastfeeding mothers are limited. Drug levels present in breast milk vary — levels of sertraline are low so it is usually recommended first line1 and levels of citalopram and fluoxetine are high so use of these medicines is generally avoided.8 TCAs, such as imipramine and nortriptyline, can be used by breastfeeding mothers since levels in breast milk are low.9,10 There are some limited data addressing possible neurodevelopmental delay in antidepressant-exposed infants; however, infant exposure to TCAs or SSRIs via breast milk has not been definitively linked with any significant long-term effects.11,12

High levels of lithium are excreted into breast milk so breastfeeding when this drug is not recommended.13

Other considerations

Hyponatraemia

Hyponatraemia has been linked with all types of antidepressant but has been reported most frequently with SSRIs, and more often in older adults. Hyponatraemia can also be caused by the syndrome of inappropriate antidiuretic hormone secretion (SIADH).

When induced by antidepressants, hyponatraemia usually develops near the beginning of therapy and is unrelated to dose. It should be suspected in all patients who develop drowsiness, confusion or convulsions while taking an antidepressant.13 The exact mechanism of this reaction is unknown but it is thought to be related to SIADH.

### Box 3: Specific considerations for MAOIs

<table>
<thead>
<tr>
<th>Monoamine-oxidase inhibitors (MAOIs) increase the amount of monoamine neurotransmitters (ie, adrenaline, dopamine, noradrenaline and dopamine) in synapses. All MAOIs are toxic in overdose. They should only be used for treatment-resistant depression and be started by specialist services or GPs with a special interest in mental health.1</th>
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</thead>
<tbody>
<tr>
<td>The use of MAOIs can potentiate the pressor effect of tyramine, causing a dangerous rise in blood pressure (an early warning symptom of which is a throbbing headache). For this reason patients taking MAOIs should be advised to avoid foods that are high in tyramine (eg, matured cheese, game, broad bean pods, Bovril, Oxo, Marmite — or any similar meat, yeast extract or fermented soya bean extract). Also recommended is that patients eat fresh foods only and avoid alcohol and food that is suspected of being stale or “going off” (particularly meat, fish, poultry or offal). MAOIs also inhibit the metabolism of indirect-acting sympathomimetics, contained in many cough and decongestant preparations, which can have an increased pressor effect. The danger of these interactions persists for up to two weeks after MAOI treatment is discontinued. When switching between MAOIs and other antidepressants, patients should not start taking a new antidepressant until two weeks after an MAOI has been stopped. Conversely, an MAOI should not be started until at least seven to 14 days after a TCA or related antidepressant has been stopped. Regarding specific MAOIs:</td>
</tr>
<tr>
<td>Isocarboxazid — is rarely used because it is poorly tolerated</td>
</tr>
<tr>
<td>Phenelzine — probably the safest irreversible MAOI</td>
</tr>
<tr>
<td>Tranylcypromine — the MAOI with the greatest stimulant action; most likely to cause a hypertensive crisis</td>
</tr>
<tr>
<td>Moclobemide — reversibly inhibits MAOI-A (unlike other MAOIs) and is therefore less toxic than other MAOIs; dietary restrictions do not need to be followed as stringently</td>
</tr>
</tbody>
</table>

If a patient taking an antidepressant develops hyponatraemia, the suspected medicine should be withdrawn and the patient's serum sodium monitored carefully until it returns to normal. If he or she still requires an antidepressant, one from a different class should be selected.

Cardiovascular disease

SSRIs are generally safer than TCAs with respect to cardiovascular side effects. SSRIs have negligible or no effect on blood pressure or heart rhythm and they are therefore the antidepressants of choice for people with coronary heart disease.1,14

Citalopram and escitalopram can cause prolongation of the QT interval. This effect is dose-dependent and, consequently, their maximum recommended doses have been reduced.15 Additionally, these medicines are contraindicated in patients with known QT-interval prolongation or congenital long QT syndrome, and in...
combination with other medicines known to prolong the QT interval.

Sertraline is the antidepressant of choice for patients with a recent myocardial infarction (MI) or unstable angina because it has been shown to be safer for such patients. Venlafaxine can exacerbate cardiac arrhythmias at higher doses (ie, 2200mg daily) so is contraindicated in patients at high risk of ventricular arrhythmias or with uncontrolled hypertension. When higher doses are used, blood pressure should be monitored routinely. 

TCAs generally cause postural hypotension, increased heart rate and slowing of cardiac conduction. With the exception of lofepramine, TCAs can cause arrhythmias (this effect is dose related) and may be associated with increased incidence of MI. Therefore, these medicines should be avoided in patients at risk of serious arrhythmias and are contraindicated for people who have recently suffered an MI. The arhythmogenic potential of TCAs means that they can be fatal if taken in overdose and this has contributed to a decline in their use.

**Bleeding disorders** SSRIs block the uptake of circulating serotonin into platelets, which leads to a reduction in platelet aggregation, thereby prolonging bleeding time. This effect is thought to be the underlying mechanism for the increased risk of gastrointestinal bleeding with SSRI use. Where possible SSRIs should be avoided in patients at risk of gastrointestinal bleeding (eg, those taking aspirin or non-steroidal anti-inflammatory drugs [NSAIDs]) or those with a history of gastrointestinal bleeding. If a patient requires both an SSRI and an NSAID (or aspirin) a gastrosoprotecive medicine should also be offered (eg, a proton pump inhibitor). Patients prescribed heparin or warfarin should not be started on an SSRI.

**Alcohol misuse** Alcohol can contribute to a depressed mood. Therefore, for patients who misuse alcohol and have depression or an anxiety disorder, the alcohol misuse should be treated first. This can lead to considerable improvement in depression and anxiety.

Symptoms of depression and anxiety are common in alcohol withdrawal. Such symptoms generally subside within three to four weeks of abstinence. Stopping alcohol consumption is crucial in determining its role in a patient’s depression; if depression continues after abstinence then treatment for depression should be started. Antidepressants with mixed pharmacological activity (eg, imipramine) appear to be more effective than SSRIs for these patients. However, their toxicity should be borne in mind and use of other, safer antidepressants might be advisable. Whether or not antidepressants are effective for the management of substance misuse has yet to be determined.

**Epilepsy** People with epilepsy are more prone to depression than those without the condition. In addition, almost all antidepressants (with the exception of agomelatine) are known to decrease the seizure threshold to varying extents; seizures have been reported in people taking antidepressants, with or without pre-existing epilepsy. This effect is often dose-related so it is recommended that antidepressants are only started for patients with epilepsy once their seizures are well controlled; antidepressants should be started at low doses and increased gradually.

The SSRIs are the antidepressants thought to be least pro-convulsive and are therefore considered first line (although agomelatine is not known to lower the seizure threshold, there are fewer data for its use in patients with epilepsy than for other antidepressants). Sertraline and citalopram are preferred because of their low risk of interacting with other medicines, notably anticonvulsants.

**Suicide** Overall, antidepressants, including SSRIs, are not directly associated with completed suicide and do not appear to be linked with a significantly increased risk of suicidal behaviour in adults. However, it is impossible to rule out the sensitivity of an individual to this particular effect of antidepressants.

When starting SSRIs, clinicians should warn patients of the possible risk of suicidal behaviour and, in the early stages of medication, should monitor patients closely because the risk of attempted suicide is highest during the first few weeks of treatment. Generally, suicidal patients should not be given large quantities of antidepressants during this stage of treatment. Also see “Antidepressants for youths” (p.30).

**Serotonin syndrome** Serotonin syndrome is an acute, rare and potentially life-threatening condition. Symptoms include agitation, restlessness, confusion, neuromuscular hyperactivity, autonomic instability, sweating, diarrhoea, tremors, shivering and hyperthermia.

As the name suggests, the syndrome relates to the regulation of serotonin, specifically excess central serotonin activity. It is usually caused by the use of one or more serotonergic medicines (eg, certain antidepressants, triptans or tramadol) leading to excess serotonin in the brain.

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**Box 4: Other antidepressants**

**Agomelatine** Agomelatine is a melatonin receptor agonist and a selective serotonin-receptor antagonist. It does not affect the uptake of serotonin, noradrenaline or dopamine.

Agomelatine can cause nausea, diarrhoea, constipation, abdominal pain, headache, sleep disturbances, dizziness, drowsiness, agitation, fatigue, anxiety, back pain and sweating. It can also cause hepatic dysfunction, so should not be used in people with risk factors for liver dysfunction. However, this reaction is unpredictable and anyone can be affected. Hence, liver function tests should be conducted for all patients at baseline and after three, six, 12 and 24 weeks of treatment. If liver transaminases exceed three times the upper limit of normal, the medicine should be stopped.

**Mirtazapine** A presynaptic alpha₂-antagonist, mirtazapine increases central noradrenergic and serotonergic neurotransmission. Common side effects include sedation (which can be considerable during initial treatment), oedema, increased appetite and substantial weight gain.

**Reboxetine** Reboxetine is a selective noradrenaline reuptake inhibitor. It has no effect on serotonin transmission or activity. Adverse effects of reboxetine include insomnia, sweating, dizziness and anticholinergic effects. The drug must be given twice daily.
Addition of lithium or antipsychotics to antidepressant treatment should only be done by specialist services and GPs with a special interest in mental health.1

**Lithium**

The mode of action of lithium in the treatment of mood disorder is not fully understood. It modifies the production and turnover of neurotransmitters such as serotonin; it may also block dopamine receptors. Lithium reduces the risk of suicide compared with antidepressants alone, but should not be used as monotherapy in depression.2

Common adverse effects of the drug include gastrointestinal disturbances, fine tremor, impaired urinary concentration and polyuria, polydipsia, leucocytosis, weight gain, oedema, hyperparathyroidism, hyperthyroidism and hyperglycaemia. Lithium salts have a narrow therapeutic range and can cause toxicity so monitoring of serum levels of lithium should be performed routinely every three months (target level 0.4–1mmol/L). Signs of toxicity are blurred vision, anorexia, muscle weakness, vomiting, diarrhoea, arthralgia, myalgia, mild drowsiness and sluggishness, increasing to giddiness with ataxia, coarse tremor, lack of co-ordination and dysarthria. The need for continued therapy should be assessed regularly and if signs of toxicity develop lithium should be stopped immediately. Preparations vary widely in bioavailability; changing the preparation requires the same precautions as initiation of treatment.

**Atypical antipsychotics**

Atypical antipsychotics (for example, aripiprazole, olanzapine, quetiapine, risperidone) act predominantly by blocking dopamine (D2) receptors in the brain, but also have activity at a range of other receptors (for example muscarinic or serotoninergic). These medicines are the preferred option for adjuvant treatment of psychotic depression (in addition to antidepressant therapy).4

The onset of symptoms is usually within 24 hours of a change in serotonergic medicines, and the syndrome usually lasts for around 24 hours (although it can persist for longer). Treatment is immediate discontinuation of suspected causative medicines.

If not done with due caution, switching between antidepressants can precipitate serotonin syndrome. Specific guidance around switching depends on the antidepressants that a patient is switching from and to, and the doses of the medicines.

**Complementary medicines**

St John’s wort is a herbal remedy that can be purchased in the UK for treating mild depression.1 Although it has demonstrated similar efficacy to prescribed antidepressants in acute mild-to-moderate depression,1 according to the British National Formulary it should not be prescribed or recommended for people with depression. In the UK it is not regulated as a medicine and preparations are not standardised, so the amount of active ingredient varies between preparations.18 St John’s wort can induce hepatic enzymes, thereby inducing the metabolism of certain conventional medicines (eg, antidepressants and the contraceptive pill).18

Omega-3 supplements have been widely assessed for use in depression and for those at risk of the condition. It is thought that eicosapentaenoic acid (one type of omega-3) has some efficacy in treating depression, but there are no agreed guidelines around its use.19 Omega-3 supplements are not recommended as monotherapy for depression but can have some use as an add-on therapy.19

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This month’s Lifelong Learning questions are based on the CLINICAL FOCUS articles on depression, which were commissioned from an independent author. The information in the Box (adjacent) is there to help you identify knowledge gaps and undertake continuing professional development. This online module will close on 1 November 2012.

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1 (a) F, (b) T, (c) T, (d) F, (e) T
2 (a) F, (b) T, (c) T, (d) F, (e) T
3 (a) T, (b) F, (c) T, (d) T, (e) T
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8 (a) T, (b) T, (c) T, (d) F, (e) T
9 (a) T, (b) F, (c) T, (d) F, (e) T
10 (a) T, (b) T, (c) T, (d) F, (e) F

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