

## NEWSLETTER

Supporting the Derbyshire Health Community

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<b>Further in this issue</b>	<b>Page 2</b>	<b>SSRIs and the risk of fracture</b>
		<b>Issues with dopamine agonists in Parkinson's disease</b>
	<b>Page 3</b>	<b>Choice of phenytoin preparation</b>
	<b>Page 4</b>	<b>Self-monitoring of blood glucose – the DiGEM study</b>
	<b>Page 5</b>	<b>Anti-VEGF treatment</b>
	<b>Page 6</b>	<b>Lumiracoxib</b>
		<b>Glitazones and heart failure</b>
	<b>Page 7</b>	<b>Which insulin regime for type 2 diabetes?</b>
		<b>Driving and insulin-treated diabetes</b>
	<b>Page 8</b>	<b>Echinacea for the common cold</b>

### CEPPaC update

RED drugs are those where prescribing responsibility would normally lie with a hospital consultant or a specialist. AMBER drugs are those that although usually initiated within a hospital setting, could appropriately become the responsibility of the GP. This would normally be under a shared care agreement. GREEN drugs are regarded as routine for primary care prescribing. BROWN drugs are those that CEPPaC does not recommend for use (DARK BROWN) or only in restricted circumstances (LIGHT BROWN) due to lack of data on safety, effectiveness, and/or cost-effectiveness. *All BROWN drugs are non-formulary.*

The most recent updates are in the table below:

<b>Drug</b>	<b>Date considered</b>	<b>Decision</b>
Celluvisc eye drops	September 2007	GREEN (not first line)
Lymecycline 408mg caps	September 2007	GREEN (second line for acne)
N-acetylcysteine 600mg tabs	September 2007	RED
Minocycline	September 2007	DARK BROWN
Cerazette	August 2007	GREEN
Evra patch	August 2007	LIGHT BROWN (if oral COC not suitable)
Yasmin	August 2007	LIGHT BROWN (if other oral COC not suitable)
Rotigotine patches	August 2007	LIGHT BROWN (if oral dopamine agonists not suitable)
Eflornithine cream	July 2007	DARK BROWN
Etoricoxib/lumiracoxib/valdecoxib	July 2007	DARK BROWN
Ibandronate 150mg tablet	July 2007	DARK BROWN
Rimonabant	July 2007	DARK BROWN
Rosuvastatin	July 2007	DARK BROWN
Eplerenone	July 2007	LIGHT BROWN (when spironolactone not tolerated)
Fentanyl lozenges	July 2007	LIGHT BROWN (for breakthrough pain only when fentanyl patches indicated i.e. oral route not possible)
Metformin SR	June 2007	GREEN (2 <sup>nd</sup> line use only)

### Problems with fentanyl patches

Dr David Brooks, Macmillan Consultant in Palliative medicine, has reported several problems he has seen with the use of generic (and possibly parallel imported) fentanyl patches. The issues are patches not adhering well, achieving less pain relief, and plain patches i.e. no drug name or dose on the patch once removed from the packaging.

This has been discussed at CEPPaC and the advice is to prescribe fentanyl patches by brand name or if writing them generically, to make sure the prescription states 'matrix patch' to ensure a branded product is dispensed. Any problems with the patches should be reported to the MHRA.

## **SSRIs and the risk of fracture**

Further to the article in the April issue, two more papers have been published on this subject<sup>1,2</sup>. Both studies were based on national cohorts designed to study osteoporotic fractures and appear to be well conducted. The first study, in women with a mean age of 78.5 years, found that, after adjustment for a range of potential confounders, mean total hip BMD decreased by 0.82% per year in those using SSRIs (n=198) whilst it decreased by 0.47% per year in those who did not use antidepressants (n=2406). This result was statistically significant. TCAs were not associated with an increased rate of bone loss.

The second study, in men with a mean age of 73.7 years, found that, after adjustment for a range of potential confounders, BMD was 3.9% lower at the total hip and 5.9% lower at the lumbar spine in users of SSRIs (n=160) compared with BMD in men not using antidepressants (n=5708). These results were statistically significant. TCAs and trazodone were not associated with lower BMD.

Neither study can prove causability, for which a randomised controlled trial of SSRIs with BMD as an outcome would be needed. However, this is unlikely to occur. Both authors were careful to recommend further research to confirm their findings.

The accompanying editorial<sup>3</sup> "Mend the mind, but mind the bones!", suggests that there is some unnecessary use of SSRIs in the general medical community, and the indications for starting and continuing SSRI therapy now should be even more carefully scrutinised. However, those who truly need SSRIs should continue to receive them despite potential bone concerns. The author says that the growing evidence now supports at least preliminary recommendations – that depression and in particular SSRI use, should be added to the list of risk factors that prompt clinicians to more carefully consider bone health.

<sup>1</sup> Ann Intern Med 2007; 167: 1240-5

<sup>2</sup> Ann Intern Med 2007; 167: 1246-51

<sup>3</sup> Ann Intern Med 2007; 167: 1231-2

## **Issues with dopamine agonists in Parkinson's disease**

Earlier this year pergolide was voluntarily withdrawn from the market in the USA because of the risk of damage to heart valves. The MHRA issued a warning about this in 2003, and in late 2004/early 2005, the use of pergolide was restricted to use under specialist supervision in patients who had failed therapy with other drugs for Parkinson's disease.

Recent publications have reported a similar frequency of heart valve damage with cabergoline as with pergolide<sup>1,2</sup>. Clinically important regurgitation in any valve was found with significantly greater frequency in patients taking pergolide (23.4%) or cabergoline (28.6%) but not in patients taking non-ergot derived dopamine agonists (0%), as compared with control subjects (5.6%)<sup>1</sup>.

As a result, the MHRA has recommended that the same restrictions be applied to the use of cabergoline as for pergolide. The SPC for cabergoline has been updated to include:

- Restriction of the indication for use of cabergoline in the management of the signs and symptoms of Parkinson's disease (PD) to second line therapy in patients who are intolerant to or fail treatment with a non-ergot compound, as monotherapy, or as adjunctive treatment to levodopa plus dopa-decarboxylase inhibitor;
- Contraindication in patients with a history of pulmonary, pericardial, and retroperitoneal fibrotic disorders and/or anatomical evidence of cardiac valvulopathy of any valve;
- Warnings regarding fibrosis and cardiac valvulopathy, as well as patient monitoring requirements.

The monitoring requirements include:

- Before initiating treatment:  
All patients should undergo a cardiovascular evaluation, including echocardiogram, to assess the potential presence of asymptomatic valvular disease. It may be appropriate to perform baseline investigations of ESR or other inflammatory markers, lung function/chest x-ray and renal function prior to initiation of therapy. If fibrotic valvular disease is detected, the patient should not be treated with cabergoline.
- Clinical diagnostic monitoring for development of valvular disease or fibrosis, as appropriate, is recommended. Following treatment initiation, the first echocardiogram should occur within 3-6 months, thereafter, the frequency of echocardiographic monitoring should be determined by appropriate individual clinical assessment, but should occur at least every 6 to 12 months.

This has been discussed at CEPPaC and practices are advised to ensure that patients on pergolide or cabergoline are reviewed in secondary care. If they are not currently under the care of a PD specialist, the advice is to refer them for a review. The non-ergot oral dopamine agonists ropinirole and pramipexole are short-acting alternatives, and the rotigotine patch may be used in some patients requiring a longer acting agent. NICE clinical guideline 35 recommends that to ensure accurate diagnosis, people with suspected PD should be referred quickly and untreated to a specialist with expertise in the differential diagnosis of this condition. The specialist should review the diagnosis at regular intervals (6 – 12 months).

Patients treated with dopamine agonists for treatment of PD, including cabergoline, especially at high doses, have been reported as exhibiting signs of pathological gambling, increased libido and hypersexuality. A recent BMJ editorial discusses pathological gambling in PD<sup>3</sup>.

Pathological gambling is an impulse control disorder characterised by excessive gambling. The prevalence of pathological gambling in PD is about 3.4%, rising to 7.2% in patients taking dopamine agonists. In contrast, the lifetime prevalence of pathological gambling in the general population in the UK is 1%. People who develop Parkinson's disease at a younger age are reported to have a higher risk of pathological gambling.

It is not clear which dopamine agonist precipitates the disorder, as all such agonists have been implicated. Patients taking both a dopamine agonist and levodopa are at increased risk, although those who take either a single dopamine agonist or levodopa can be affected.

Patients and families often do not suspect drug treatment as the cause of pathological gambling and therefore do not mention it to the doctor. Better awareness of the problem among patients and carers, coupled with routine direct questioning by clinical staff about changes in behaviour and development of new compulsions and gambling, will help to identify the problem early.

Once recognised, several strategies may help. Reducing or stopping dopamine agonists may be considered, as anecdotal evidence suggests this helps improve or stop the pathological gambling behaviour.

1. N Engl J Med 2007; 356: 39-46
2. N Engl J Med 2007; 356: 29-38
3. BMJ 2007; 334:810-11

### **Choice of phenytoin preparation**

There is a huge difference in the cost of generic phenytoin tablets and phenytoin capsules (Epanutin).

From the August Drug Tariff:

Phenytoin 100mg tablets x 28	£53.51
Phenytoin 100mg capsules x 28	£0.94

For a patient on a daily dose of 300mg the expenditure would be:

Phenytoin tablets 300mg daily	£2,087 per annum
Phenytoin capsules 300mg daily	£37 per annum

This represents a 56-fold difference in basic NHS reimbursement cost. Using the latest ePACT data, extrapolated for a full year effect, switching from phenytoin tablets to capsules would save Derbyshire County PCT £557,356 per year and Derby City PCT £59,814 per year. Maintaining the existing pattern of phenytoin prescribing would severely curtail our ability to fund other key priority areas of prescribing.

There is an obvious need for caution when considering a switch between different formulations of anticonvulsant medication. The current BNF (March 2007; edition 53: p.248) states 'on the basis of single dose tests there are no clinically relevant differences in bioavailability between available phenytoin sodium tablets and capsules but there may be a pharmacokinetic basis for maintaining the same brand of phenytoin in some patients'.

Sheffield PCT has introduced a policy of switching phenytoin tablets to capsules after seeking advice from the senior neurology team at Sheffield Teaching Hospitals FT (who also cover north Derbyshire). They backed a policy promoting the routine use of the most cost-effective therapeutic option. The Derby neurologists also support the Sheffield approach.

Key clinical statements/considerations from the neurology teams include:

- Minor differences in pharmacokinetic properties of different formulations of phenytoin are unlikely to make any difference in clinical effectiveness
- The policy should be linked with advice that if the patient notices any symptoms of intoxication (usually ataxia, dizziness or sedation) or change in seizure frequency within 6 weeks of a change in formulation, the possibility that this might be caused by different bioavailability of the products should be considered. In practice it is more likely that other factors would be involved
- If patients are going to change formulation they should be warned of the possibility of intoxication and the small possibility of deterioration in seizure control. For a minority of patients who are seizure free, this may be a significant issue, particularly if they are driving.

This has been discussed at CEPPaC and the recommendation is to consider switching phenytoin tablets to phenytoin capsules at the next routine medication review in consultation with the patient. There is one exception – avoid the switch in seizure-free drivers, as a precautionary measure. If you wish to discuss this issue further please contact any member of the medicines management team who will be happy to help.

**Key message:** CEPPaC recommends switching patients from phenytoin tablets to capsules (Epanutin) where clinically appropriate to ensure the effective use of NHS resources. Any new prescriptions for phenytoin should be for the capsule formulation.

### **Self-monitoring of blood glucose – the DiGEM study**

Large trials of management of patients with type 1 diabetes have incorporated self-monitoring of blood glucose (SMBG) as an essential part of self-management and it is accepted practice. There is some evidence that self-monitoring for insulin treated patients with type 2 diabetes can be beneficial, but optimisation of SMBG may be possible. A systematic review did not find that SMBG in people with type 2 diabetes not using insulin improved glucose control by a clinically relevant degree<sup>1</sup>. Blood glucose monitoring is expensive and a large amount of scarce NHS resource is used up each year providing the strips. Consensus guidelines have based recommendations for SMBG on a theoretical potential to better self manage glycaemic control. A recent UK study tested whether SMBG, with or without instruction on incorporating findings into self-care, compared with standardised usual care can improve glycaemic control in patients with non-insulin treated diabetes<sup>2</sup>.

#### *Method*

- The DiGEM study was a four-year open, randomised, three arm, parallel group trial with sequential recruitment of patients from general practices in Oxfordshire and South Yorkshire.
- The primary aim was to determine whether HbA<sub>1c</sub> levels at 12 months were significantly different between patients with non-insulin treated type 2 diabetes receiving one of three allocated interventions:
  - standardised usual care with measurement of HbA<sub>1c</sub> levels by health professionals every 3 months (control group)
  - use of a blood glucose meter (3 times daily on 2 days per week), with advice for participants to contact their doctor for interpretation of results, in addition to usual care (less intensive self monitoring)
  - use of a blood glucose meter with training in self interpretation and application of the results to diet, physical activity, and drug adherence (more intensive self monitoring). Participants were encouraged to experiment with the frequency of monitoring.
- Secondary outcomes were blood pressure, weight, total cholesterol level, ratio of total cholesterol to HDL-C, and BMI.
- The intervention was initiated at the first visit after randomisation and continued at the scheduled visits at 1, 3, 6 and 9 months. Training and support for the research nurses delivering the intervention was designed to ensure adherence to the study protocol.
- Patients in each arm of the trial received feedback on glycaemic control, which was used to explore success of goals and to set new ones. The patient's doctor was notified of all HbA<sub>1c</sub> results and asked to consider changes in drugs in line with the NICE diabetes guidelines for all patients. The doctor was also notified if blood glucose readings were consistently greater than 15 mmol/l.
- They carried out a single intention to treat analysis of the main trial endpoints.
- The trial was funded by the NHS and the National Institute for Health Research health technology assessment programme.

## Results

- At 12 months no differences were found in HbA<sub>1c</sub> levels between the groups after adjustment for baseline HbA<sub>1c</sub> levels (p=0.12). There was no evidence of differences in levels between groups over the period of follow-up (p=0.38).
- No differences were found in the secondary outcomes except for a small difference in total cholesterol levels between the three groups (p=0.01). The mean difference in change in total cholesterol levels from baseline to 12 months between the control group and less intensive intervention group (not adjusted for baseline) was -0.06.mmol/l (-0.26 to 0.14) and between the control group and more intensive intervention group was -0.23 (-0.43 to -0.04).
- No differences were found between the groups in the proportions of patients prescribed an increase in hypoglycaemic drugs between baseline and 12 months. Also no differences were found in statin prescribing.

## Discussion/implications

- This appears to have been a robust study, performed in general practices in England, which has implications for guidelines. The participants were representative of well-controlled non-insulin treated patients with type 2 diabetes in the community who are the target group for current recommendations of up to twice daily self monitoring and testing after meals.
- No significant improvement in glycaemic control was found after 12 months in patients with non-insulin treated type 2 diabetes using self monitoring of blood glucose levels when compared to those not self monitoring. No evidence was found of a significantly different impact of self monitoring on glycaemic control when comparing subgroups of patients defined by duration of diabetes, therapy, diabetes related complications, and EQ-5D score. Also no evidence was found that more intensive compared with less intensive monitoring led to differences in glycaemic control.
- As the authors comment “Despite an intervention based on standards of best clinical practice and underpinned by appropriate psychological theory, we found no convincing evidence of an effect on glycaemic control”.
- The authors conclude “Routine self monitoring of blood glucose for patients with reasonably well controlled non-insulin treated type 2 diabetes seems to offer, at best, small advantages; is not well accepted; and the cost, effort, and time involved in the procedures may be better directed to supporting other health related behaviours. Current guidelines for the use of self monitoring of blood glucose among patients with reasonably well controlled non-insulin treated type 2 diabetes should be reviewed.”
- All our interventions should be effective, cost-effective and affordable. Current evidence suggests that the routine use of SMBG in non-insulin treated diabetes does not meet these criteria.
- A common reason given for prescribing blood glucose testing strips is that they empower patients. The participants in the intensive self-monitoring group were indeed empowered to make changes to their lifestyle but this did not lead to improved HbA<sub>1c</sub> levels.
- The accompanying editorial<sup>3</sup> concludes “**the results of this study should encourage clinicians to discuss the value of glucose testing with their patients and give them the confidence to discontinue it if it is providing no benefit**”.

Follow the local guidelines on SMBG and only use it when there is a clearly defined need, with an action plan on how to respond to the results.

1. Diabetes Care 2005; 28:1510-17
2. BMJ 2007; 335:132-6
3. BMJ 2007, 335:105-6

## Anti-VEGF treatment

We have received the following advice from the East Midlands Specialised Commissioning Group on the use of anti-VEGF treatments (Lucentis®, Macugen® and Avastin®) for diabetic retinopathy:

“It has come to the attention of the East Midlands SCG that Trusts may request funding for anti-VEGF treatment for the treatment of diabetic retinopathy. This potential pressure needs to be addressed very quickly. Whilst anti VEGFs may become important treatments in the future there are no robust clinical trials that have assessed their use in diabetic retinopathy. This is a large patient group and establishing effectiveness and cost-effectiveness will be extremely important. The decision to fund should eventually be on the basis of a) cost and clinical-effectiveness and b) providing access to all patients who would benefit in the patient population – not just the odd case.

It is therefore strongly advised that:

1. As there are no clinical trials to support anti-VEGFs for the treatment of diabetic retinopathy, it should not be considered for funding.
2. If and when there is evidence of clinical and cost effectiveness available, funding considerations should be categorised as a service development (given the potential number of patients) and considered within the LDP and until agreed as a service (i.e. provide access to all who would benefit) no cases should be funded.
3. Trusts should be informed that cases referred as individual funding requests will be refused.
4. Trusts should be advised to present a business case only when there is a sound evidence base for this treatment.”

Please bear this in mind when referring patients.

### **Lumiracoxib**

Lumiracoxib has been taken off the Australian market due to reports of hepatotoxicity. Novartis has issued a press release as a result. Following an EU interim assessment led by the Medicines and Healthcare products Regulatory Agency (MHRA) of the latest safety evidence new prescribing advice has been issued:

- Lumiracoxib is now contraindicated in patients
  - with any current hepatic disease;
  - with prior drug-induced significant (>3xULN) elevations of transaminases;
  - with liver transaminases >1.5xULN before treatment, or >3xULN during treatment (see below); or
  - taking other medicines associated with clinically significant hepatotoxicity
- The new Liver Function Test (LFT) monitoring advice is:
  - Perform baseline LFTs before starting treatment (lumiracoxib is contraindicated if transaminases >1.5xULN).
  - Where treatment is needed for longer than 1 month, repeat LFTs (monthly).
  - Stop treatment if transaminases >3xULN, repeat in 7 days if transaminases >2xULN
  - Conduct LFTs for patients reporting any systemic illness whilst taking lumiracoxib
- Patients already taking lumiracoxib should be reviewed at their next routine appointment. If continued treatment is considered appropriate (after consideration of overall benefit and risks, and after taking new contraindications into account), then LFTs should be taken.

*Reminder:* Treatment should be limited to the shortest duration necessary and should not exceed the recommended 100mg daily. The need for continued treatment should be frequently reassessed.

*Advice to Patients:* Physicians should counsel their patients for possible signs and symptoms of hepatic injury such as nausea, vomiting, anorexia, malaise, fatigue, dark urine and right upper abdominal discomfort, as well as specific symptoms such as itching or jaundice. Patients should be advised to stop treatment in the event of any symptoms and seek urgent advice from their doctor.

**Lumiracoxib is classified DARK BROWN in Derbyshire.**

### **Glitazones and heart failure**

There is already evidence that glitazones may increase the risk of heart failure. A recent analysis estimates the magnitude of the risk of heart failure with glitazones<sup>1</sup>. Data from RCTs, observational studies, and case reports were analysed in a teleo-analysis. A teleo-analysis attempts to determine the adverse effect of a drug by complementing information from different study designs across all grades of evidence.

Three RCTs of 10,731 patients provided numerical information on heart failure events. The pooled odds ratio (OR) for heart failure was 2.1 (95% CI 1.08 to 4.08; p = 0.03). The background incidence of heart failure in patients with diabetes is approximately 1.9% over 2.2 years. The estimated number needed to harm (NNH) with glitazones, based on an OR of 2.1, would be approximately 50 over a 2.2-year follow-up period.

Four observational studies of 67,382 patients gave a pooled OR for heart failure of 1.55 (1.33 to 1.80;  $p < 0.00001$ ). Heart failure occurred even among patients with no history of heart failure, was not confined to patients on insulin, and was not limited to the elderly (26% of reported cases occurred in patients aged < 60 years). Heart failure occurred at both high and low doses, usually weeks to months after initiation of glitazone. The occurrence of heart failure several months after initiation suggests a long-term effect of glitazones, which may not be avoided by slow-dose titration, and long-term vigilance would be prudent.

This analysis should be viewed in context with the recent meta-analysis on potential increased risk of MI with rosiglitazone (see PACE newsletter June 07), the increased risk of fracture with glitazones (newsletter April 07), and the lack of robust evidence for beneficial effects on hard clinical endpoints with glitazones.

**Local advice is to review treatment options for people with type 2 diabetes, particularly those with suspected or confirmed heart failure. Glitazones should only be used in line with the NICE guidance and if one is appropriate, then pioglitazone is the preferred choice.**

1. Diabetes Care 2007; 30:2148-53

### **Which insulin regime for type 2 diabetes?**

Insulin treatment in type 2 diabetes (T2DM) increases glycaemic control but is associated with significant weight gain. Which insulin regime is best able to optimise glycaemic control whilst minimising weight gain? A retrospective study with the aim of comparing glycaemic control and weight gain in patients with T2DM commenced on basal insulin or mixed insulin has been published<sup>1</sup>.

Subjects had T2DM for a mean duration of 7 years, median age of 60 years (range 30-97) and were poorly controlled while treated with oral hypoglycaemic agents. 65% had been commenced on basal insulin (BI) with NPH (Insulatard or Humulin I) and 35% as mixed insulin (MI) (Mixtard 30, Humulin M3, Novomix 30 or Humalog Mix25). Follow up was over a mean period of 3.8 years.

There was a significant improvement in HbA<sub>1c</sub> in both groups, which was not significantly different between them. During follow-up there was a clinical and statistical difference in weight gain between the two groups, with greater weight gain in the MI group. Subjects using BI used significantly lower doses of insulin and a greater proportion were taking metformin.

The authors conclude that basal NPH insulin, taken with metformin, is an effective treatment for patients with T2DM poorly controlled on oral hypoglycaemic agents. Compared to twice daily pre-mixed insulin over long-term follow up, the subjects had similar improvements in glycaemic control with less weight gain.

1. Pract Diab Int 2007; 24:212-16

### **Driving and insulin-treated diabetes**

Patients with diabetes who require insulin treatment are required to inform the DVLA and their motor insurance company. The reason regulations are stricter for patients with insulin-treated diabetes, compared to those controlled by tablet or diet alone, is that there is a greater risk of episodes of severe hypoglycaemia with insulin treatment. Hypoglycaemia is common in insulin-treated patients and can occur with or without symptoms; it can result in cognitive impairment, mood changes, cardiac arrhythmias, convulsions and coma in severe cases. Studies have shown that hypoglycaemia results in a major decline in all of the visual information processing tasks and driving performance is impaired at blood glucose levels as high as 3.4 – 4.0mmol/L.

In the UK, patients requiring insulin treatment are usually given a three-year driving licence and at the time of renewal will be asked to declare any problems with hypoglycaemia and a medical review may be sought. At this time, patients are issued with guidance for safe driving practices from DVLA, which is similar to the information provided by Diabetes UK. The key recommendations made are:

- Always keep fast-acting carbohydrate in the car
- Carry a blood glucose meter while driving
- Measure blood glucose before every journey
- Measure blood glucose during long journeys (every two hours)
- Do not drive if you feel hypoglycaemic or if blood glucose is under 4.0mmol/L

- Stop driving during hypoglycaemia
- Wait at least 45 minutes before resuming driving following an episode of hypoglycaemia
- Cease driving if loss of hypoglycaemic warning symptoms occurs; consult the diabetes care team and inform the DVLA
- Inform the DVLA of any episode of severe hypoglycaemia whilst driving

It is very important that patients are informed of these when they are first started on insulin therapy, and that this information is reinforced regularly.

The driving advice for insulin-treated patients as issued by the DVLA and Diabetes UK has been included in the Grampian Diabetes Guidelines for primary and secondary care since 2004. The purpose of a recently published study was to investigate whether patients and primary and secondary care health professionals in Grampian were aware of these recommendations and if patients recalled and adhered to the information they received<sup>1</sup>.

An anonymous questionnaire was given to all patients attending a secondary care diabetes clinic over a three-week period. A second web-based questionnaire was e-mailed to primary and secondary care health care professionals (HCPs) involved in diabetes care throughout Grampian.

In all, 117 patients with insulin-treated diabetes completed the questionnaire and 106 HCPs from primary and secondary care completed the web-based questionnaire. The majority of patients (95%) were aware that they were obliged to inform the DVLA of their insulin treatment and 92% had done so. Only 15% of patients always tested and 24% would never test a blood glucose prior to driving. Ninety-four percent of patients recognised symptoms of hypoglycaemia most of the time. Only 17% would wait the recommended 45 minutes before driving again after an episode of hypoglycaemia. All HCPs knew that patients are obliged to inform the DVLA of their insulin treatment. Sixty-two percent of HCPs knew patients should test before every journey. Thirteen percent of HCPs thought it safe to drive with blood glucose <4mmol/L. Eight percent of HCPs did not know that impaired awareness of hypoglycaemia might be a contraindication to driving.

The authors conclude that the blood testing habits of drivers with insulin-treated diabetes do not follow the guidance issued by the DVLA and Diabetes UK. Patients and many health professionals are not aware of many of the recommendations for safe driving, e.g. blood glucose testing before driving, not driving if blood glucose is too low, not resuming driving after a 'hypo' for 45 minutes, and ceasing driving if loss of hypoglycaemic warning symptoms occurs.

**They recommend that regular reinforcement of driving recommendations is required as part of routine diabetes care.**

1. Pract Diabet Int 2007; 24:201-6

### **Echinacea for the common cold**

The mechanism of action underlying the proposed immunostimulatory effects of echinacea are unclear and controversy exists about its benefit in the prevention and treatment of the common cold. A meta-analysis of 14 randomised placebo-controlled trials evaluating the effects of echinacea has recently been published<sup>1</sup>.

Incidence of the common cold was reported as an odds ratio (OR) and duration of the common cold as the weighted mean difference (WMD). Echinacea decreased the odds of developing the common cold by 58%, OR 0.42 (95% CI 0.25 to 0.71; p <0.001) and the duration of cold by 1.4 days, WMD -1.44 (-2.24 to -0.64; p=0.01).

The authors conclude that published evidence supports echinacea's benefit in decreasing the incidence and duration of the common cold. However they add, large-scale randomised prospective studies controlling for variables such as species, quality of preparation and dose of echinacea, method of cold induction, and objectivity of study endpoints evaluated, are needed before echinacea for prevention or treatment of the common cold can become standard practice.

1. Lancet Infect Dis 2007; 7:473-80