this goal. However, as funding for Quality Protects and Children First is mainstreamed in 2004, primary care trusts will need to show their commitment to invest in the future health of looked after children—otherwise the vision may prove to be only a mirage.

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Tobacco, coffee, and Parkinson's disease

Caffeine and nicotine may improve the health of dopaminergic systems

Parkinson's disease belongs to that small group of conditions that occur less often among cigarette smokers than in non-smokers. The observation was first made in a case-control study over 30 years ago,[1] but, as Hernán and colleagues have shown in their recent systematic review and meta-analysis,[2] the finding has been replicated many times. The protective effect is large—according to the pooled data, current smokers have a 60% reduction in risk compared with those who have never smoked—and consistent between studies in different settings. The fact that two very large prospective studies found a similar reduction in risk to that seen in retrospective studies rules out the possibility that the association can be accounted for by differential survival between smokers and non-smokers.[3] Coffee drinking too, seems to protect against Parkinson's disease. Here the pooled estimate is a 30% reduction in risk for coffee drinkers compared with non-drinkers.

In "An Essay on the Shaking Palsy," James Parkinson noted that his first case "had industriously followed the business of a gardener, leading a life of remarkable temperance and sobriety." Since then several small studies have implied that people with Parkinson's disease tend to exhibit traits such as inflexibility, cautiousness, and lack of novelty seeking even before they have developed motor symptoms.[4 5] This idea has never been tested in a large prospective study, but it does raise the possibility that people who will later develop Parkinson's disease are constitutionally less likely to feel the need for the type of stimulation provided by tobacco and coffee. This might occur if the genetic determinants of likelihood and intensity of behaviours such as cigarette smoking and coffee drinking were the same as or closely linked to the genes that determined susceptibility to Parkinson's disease. If so, any apparent protective effect might be the result of confounding. The authors of the systematic review explored this possibility in a sensitivity analysis. They made the fairly extreme assumption that such a genetic combination was present in a third of the population and conferred both a fivefold increase in risk of Parkinson's disease and, simultaneously, a fivefold decrease in likelihood of taking up smoking. Even after adjusting for a genetic influence of this strength, smoking still conferred more than a 30% reduction in risk.

If confounding by a genetic haplotype looks unlikely, what other reasons remain? A theoretical possibility is that the relation between cigarette smoking or coffee drinking and Parkinson's disease is operating in the reverse direction. In other words, Parkinson's disease makes people less likely to smoke or drink coffee. Of course these habits are usually acquired by early adult life, whereas symptoms of Parkinson's disease are rare before late middle age. So this explanation could be correct only if the subclinical phase of the disease is very much longer than we currently believe.

Perhaps it is more plausible that substances present in coffee and tobacco—caffeine and nicotine are obvious candidates—have a central action that improves the health of dopaminergic systems. Evidence in support of caffeine's role as a neuroprotectant has recently emerged from a study using a mouse model of Parkinson's disease. Mice that were pretreated with caffeine before exposure to the dopaminergic neurotoxin 1-methyl-4-phenyl-1,2,3,6-tetrahydropyridine (MPTP) lost less striatal dopamine and fewer dopamine transporter binding sites.[6] Caffeine's apparent neuroprotective effect may be due to its ability to block adenosine A_{2A} receptors that are concentrated in the dopamine rich areas of the brain.[7] Adenosine decreases dopaminergic neurotransmission by means of antagonistic interactions between A_{2A} receptors and dopamine receptors.[8] The blockade of these receptors can therefore facilitate dopaminergic transmission by stimulating dopamine release and by potentiating the effects of dopamine receptor stimulation. Knockout mice that lack functional adenosine A_{2A} receptors are also resistant to the dopamine depleting effects of MPTP.[6]

Like caffeine, nicotine has been found to reduce MPTP-induced dopaminergic toxicity in animal models of Parkinson's disease.[9 10] One mechanism under-

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lying this protective action may be its ability to increase the expression of neurotrophic factors that are known to promote survival of dopaminergic neurons.[9] But tobacco contains numerous other chemicals whose influence on biological processes may play a part. Smoking causes a reduction in activity of monoamine oxidase A and B, for example, which might protect against neuronal damage by inhibiting the enzymatic oxidation of dopamine.[11]

One unachieved goal in the treatment of Parkinson's disease is preventing it getting worse. If, as the epidemiological evidence implies, caffeine and nicotine are neuroprotective, some of the new pharmacological treatments currently being developed, such as adenosine A_{2A} receptor blockers and nicotinic agonists, might not only improve symptoms but slow the relentless progression of the disease.

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Massage treatment for back pain

Evidence for symptomatic relief is encouraging but not compelling

Throughout history different forms of massage treatment have been used in all medical cultures to alleviate a wide range of symptoms. This article focuses on the most common form, classic muscular (Swedish) massage, as a symptomatic treatment for back pain.[1] It will define the therapeutic modality, review the evidence for or against effectiveness and safety, and discuss possible mechanisms of action as well as the problems of conducting research in this area.

Swedish massage is a touch therapy that uses a range of techniques to manipulate the soft tissues of the body: effleurage (slow rhythmic stroking), kneading (circular compression), petrissage (forceful skin rolling), friction (penetrating pressure from the fingertips with circular or transverse movement), tapotement (percussive movements), vibration (trembling movement of both hands).[2] In most English speaking countries, massage is seen as an alternative or complementary treatment,[3] whereas on the European continent it is considered a conventional treatment, particularly for back pain. In Austria, for example, 87% of patients with back pain receive (and are usually reimbursed for) massage treatment.[4]

A recent Cochrane review of massage treatment for back pain summarised five randomised clinical trials on the subject, three of which were of high methodological quality.[5] One study compared massage with detuned laser therapy as placebo, and the other trials compared massage with various other physical treatments such as acupuncture or spinal manipulation. The review shows that massage is superior to placebo, relaxation treatment, acupuncture, or self care education; inferior to manipulation, shiatsu, or transcutaneous electrical stimulation; and no different from treatment with corsets or exercise. The benefit lasted at least one year. The authors concluded that massage "might" be beneficial for subacute and chronic non-specific low back pain.[5] In a further relevant trial, patients with "non-inflammatory rheumatic pain" (not just back pain) were randomised to receive either 10 sessions of classic massage or usual medical care for five weeks.[6] By the end of this period, both groups had improved similarly, and at three months' follow up more pain relief had occurred in the massage group.

These studies are not easy to interpret. Some are methodologically weak; most used control interventions with uncertain effectiveness; some tested massage other than Swedish massage; some allowed concomitant interventions; and one trial[6] was not conducted exclusively on patients with back pain. Back pain is not a disease entity but a symptom, and future studies should aim at determining whether certain types of patients respond better than others. The overall picture that seems to emerge implies that the evidence for massage as a symptomatic relief of back pain is encouraging but not compelling.[3] Similar conclusions would be reached if one looked at other conditions for which massage has been tested in controlled clinical trials.[3]

Most massage therapists are convinced that massage treatment is free of risk. This is not true. Too much force can cause fractures of osteoporotic bones; and even rupture of the liver and damage to nerves have been associated with massage.[7] These events are rarities and massage is relatively safe, provided that well trained therapists observe the contraindications:

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