inhaled insulin, and a large proportion of patients randomised to inhaled insulin chose to continue with that regimen. These findings held regardless of type of diabetes or whether inhaled insulin was added to subcutaneous insulin therapy or oral agents.[10–12]

Given the comparable glycaemic control and safety profile seen with the inhaled insulin preparation in comparison to subcutaneous administration, patient acceptance may prove to be the primary determinant of marketplace success. For some patients, doctors and budget holders, the issue of drug costs, cost effectiveness, and uncertainty regarding potential long term adverse effects related to pulmonary route of delivery will outweigh the perceived benefits of ease of administration.

Both considerations contributed to the recent decision by NICE (the National Institute for Health and Clinical Excellence) to decline funding for inhaled insulin for the NHS. This opinion will be revisited after several months of public commentary.[13] However, patients with type 1 diabetes or insulin dependent type 2 diabetes who are distressed by frequent injections may be able to improve their blood sugar control substantially and avoid complications from diabetes with this new product. For patients with type 2 diabetes with failing oral agents who are reluctant to start treatment with insulin, inhaled insulin offers the same efficacy as subcutaneous insulin, without multiple daily injections. When added to oral agents, it may obviate the need for subcutaneous insulin entirely.

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Passive smoking's role in diabetes

More evidence of the harmfulness of tobacco smoke

Smoking is the main cause of lung cancer, chronic obstructive pulmonary disease, and peripheral atherosclerosis and one of the most important risk factors for cardiovascular disease. In particular, the risk attributed to current smoking varies from 40% for coronary heart disease to more than 60% for cancers of the pharynx and oesophagus and more than 80% for lung cancer.[1] In addition, the exposure of non-smokers to environmental tobacco smoke has been associated with a substantial increase in their risk of coronary heart disease and cancer.[2 3] Several investigators have suggested that both active and passive smoking affects the cardiovascular system through endothelial dysfunction, increases in oxidised low density lipoprotein cholesterol, platelet adherence, inflammation, and mitochondrial and oxidative damage, as well as an acute deterioration in the elastic properties of the aorta.[4–7] Indeed, some of the effects of passive smoke on the cardiovascular systems of non-smokers are comparable to the effects of smoking on smokers.[3] Now comes another effect of active and passive smoking: an increased risk of glucose intolerance.

Research p 1064

In this issue (p 1064) Houston et al show an increased risk from tobacco smoke of glucose intolerance,[8] which is a precursor of diabetes and atherosclerotic disease.[9] In particular, they found a strong association between exposure to tobacco smoke and the incidence of glucose intolerance during the 15 year follow-up of young adults. The incidence of glucose intolerance was 22% among smokers, 17% among those who never smoked but had been exposed to smoke, 14% among former smokers, and 12% among those who had never smoked and had no passive smoke exposure. In addition, the authors reported that current and never smokers with passive smoke exposure experienced, respectively, 65% and 35% higher risks of developing glucose intolerance than never smokers without passive exposure, even after adjustment for various baseline sociodemographic, biological, and behavioural factors. Moreover, in the analyses stratified by race and sex the risks of glucose

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intolerance associated with tobacco exposure were greater in men than in women and in white people than in African-Americans.

The number of people with diabetes or glucose intolerance is rising owing to population growth, ageing, urbanisation, and the increasing prevalence of obesity and physical inactivity.[9] Though some studies have shown a dose-response association between smoking and the incidence of diabetes, others have failed to do so.[10 11] The study of Houston et al clearly showed that both active and passive smoking were associated with the development of glucose intolerance among young adults.[8] Their study has several strengths, but also some limitations. The strengths include the large sample size (about 4600 participants), stratification by sex and race, validation of passive smoking by serum cotinine concentrations, long term follow-up (15 years), an adequate participation rate (>74%), and controlling for potential sociodemographic, biological, and behavioural confounders. However, the effect of smoking on the incidence of glucose intolerance occurred irrespective of waist:hip ratio, baseline insulin, and C reactive protein levels, markers that have been associated with the development of diabetes and presence of smoking habits in previous studies.[9 10] Potentially, the measurement of these markers at various time points during the follow-up might clarify whether or not they constitute a causal pathway between smoking and glucose intolerance.

In addition, the authors reported that smokers and never smokers with passive smoke exposure were more often African-American and less often women than never smokers with no passive smoke exposure, while current smokers also had less education, drank more alcohol, and had higher fat intakes than never smokers with no passive smoke exposure. Although these sociodemographic and lifestyle characteristics were taken into account in the analyses, residual confounding might still exist, and the variability and misclassification usually observed in measuring healthcare, socioeconomic, and lifestyle variables might mask the true findings.

The tobacco industry has vigorously contested allegations that passive smoking is dangerous,[12] but the evidence for the harmful effects of passive smoking keeps growing, and this study by Houston et al provides evidence for a new risk from exposure to tobacco smoke. The finding needs confirming, but in the meantime most non-smokers wish not to be exposed to tobacco smoke against their will. The momentum for bans on smoking in public places continues.

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Community acquired pneumonia in primary care

Doctors cannot target antibiotics and reduce resistance until new diagnostic tests prove feasible and affordable at the point of care

Practice p 1077

In Europe 90-95% of antibiotic use occurs outside hospitals, and community acquired lower respiratory tract infections (LRTI) are the leading reason for prescribing antibiotics.[1] Few conditions in medicine are so controversial or have resulted in so much promiscuity in prescribing. The escalating resistance of common bacterial respiratory pathogens to antibiotics in the community[2] will be contained only by reducing prescribing in everyday practice and targeting antibiotics selectively. We have known this for a long time. But it is difficult to target antibiotics appropriately, particularly in LRTI.

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Several problems underlie this clinical uncertainty about which patients with LRTI benefit from antibiotics and which do not. The update on diagnosis and management of pneumonia by Hoare and Lim in this week's *BMJ* nicely illustrates this controversy (p 1077).[3] Results of trials indicate that most patients with initially uncomplicated infection will probably have limited benefit, but this evidence is scant: the relevant Cochrane review included only 750 patients.[4]

There are no comprehensive studies of sufficient size powered to assess benefit in clinical subgroups; the value of detecting microbial aetiology; the role of