sional and Linguistics Assessments Board (PLAB). When appropriate, the opportunities for taking the PLAB examination in their own country should be made clear, and the NHS Executive has plans to make such information available over the internet. These moves are to be welcomed if they deter those overseas doctors who are ill prepared from coming to the United Kingdom only to spend months or years in attempting the PLAB without any hope of a post at the end. The GMC also recommends that overseas doctors should be encouraged to develop their educational objectives before applying to train in the United Kingdom, and to go on organised training programmes. Meanwhile, trusts have the freedom to appoint senior house officers to educationally approved posts and are intent on ensuring that vacancies are filled with doctors who are competent to do the work, whatever their educational objectives. Overseas senior house officers tend to be older and more experienced than UK graduates,[11] and they fill an important service need. While they continue to come, in the hope (however unrealistic) of gaining a place in the specialist registrar training programme of their choice, they will continue to be welcomed to fill these vacancies.

The document identifies the purpose of the grade as developing and applying skills and knowledge; demonstrating the ability to carry out responsibilities; clarifying career intentions; and beginning training in the chosen career. Most doctors have developed career aims by the end of their preregistration year, and success then depends on getting the right jobs, gaining supportive references, and passing college examinations. Some senior house officers achieve all this and still fail to gain entry to an oversubscribed specialty. They are then faced with starting all over again, marked as failures. A closer match between the training opportunities at senior house officer and specialist registrar levels would obviate much of this wasted time. It is no wonder that junior doctors' representatives would like to see the grade abolished and replaced by a single specialist training grade,[5] an option that the GMC intended to consider[13] but which is not addressed in the document.

Despite the difficulties, the grade does serves important purposes. It gives an opportunity for young doctors to experience working in different specialties and environments before committing themselves to a specialty. They can demonstrate their aptitude to those who select for the career of their choice. It provides a mechanism for gaining generic training until their aptitudes, opportunities, and choices are clearer. To help in these choices, senior house officers should be able to obtain direct access to up to date information about training opportunities in each specialty and each part of the country and trends in consultant numbers. Opportunities in basic specialist training should match training opportunities at specialist registrar level.

The introduction of clinical governance and the drive to improve the quality of health care[14] will probably result in trusts reconsidering the role of senior house officers in delivering their services. Providing cover for the hospital out of hours should surely be the responsibility of an appropriately led and skilled multidisciplinary team in which senior house officers learn while working under supervision. Trusts and those commissioning health care will wish to review the place of the education and training of all doctors in the context of improving patient care, both today and tomorrow.

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Do dietary lectins cause disease?

The evidence is suggestive—and raises interesting possibilities for treatment

In 1988 a hospital launched a "healthy eating day" in its staff canteen at lunchtime. One dish contained red kidney beans, and 31 portions were served. At 3 pm one of the customers, a surgical registrar, vomited in theatre. Over the next four hours 10 more customers suffered profuse vomiting, some with diarrhoea. All had recovered by next day. No pathogens were isolated from the food, but the beans contained an abnormally high concentration of the lectin phytohaemagglutinin.[1] Lectins are carbohydrate binding proteins present in most plants, especially seeds and tubers like cereals, potatoes, and beans. Until recently their main use was as histology and blood transfusion reagents, but in the past two decades we have realised that many lectins are (*a*) toxic, inflammatory, or both; (*b*) resistant to cooking and digestive enzymes; and (*c*) present in much of our food.[2] It is thus no surprise that they sometimes cause "food poisoning." But the really disturbing finding came with the discovery in 1989 that some food lectins get past the gut wall and deposit themselves in distant organs.[3 4] So do they cause real life diseases?

This is no academic question because diet is one part of the environment that is manipulable and

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because lectins have excellent antidotes, at least in vitro. Because of their precise carbohydrate specificities, lectins can be blocked by simple sugars and oligosaccharides. Wheat lectin, for example, is blocked by the sugar N-acetyl glucosamine and its polymers.[5] These natural compounds are potentially exploitable as drugs should lectin induced diseases be identified.

Wheat gliadin, which causes coeliac disease, contains a lectin like substance that binds to human intestinal mucosa,[6] and this has been debated as the "coeliac disease toxin" for over 20 years.[7] But coeliac disease is already managed by gluten avoidance, so nothing would change were the lectin hypothesis proved. On the other hand, wheat lectin also binds to glomerular capillary walls, mesangial cells, and tubules of human kidney and (in rodents) binds IgA and induces IgA mesangial deposits. This suggests that in humans IgA nephropathy might be caused or aggravated by wheat lectin; indeed a trial of gluten avoidance in children with this disease reported reduced proteinuria and immune complex levels.[8]

Of particular interest is the implication for autoimmune diseases. Lectins stimulate class II HLA antigens on cells that do not normally display them, such as pancreatic islet and thyroid cells.[9] The islet cell determinant to which cytotoxic autoantibodies bind in insulin dependent diabetes mellitus is the disaccharide N-acetyl lactosamine,[10] which must bind tomato lectin if present and probably also the lectins of wheat, potato, and peanuts. This would result in islet cells expressing both class II HLA antigens and foreign antigen together—a sitting duck for autoimmune attack. Certain foods (wheat, soya) are indeed diabetogenic in genetically susceptible mice.[11] Insulin dependent diabetes therefore is another potential lectin disease and could possibly be prevented by prophylactic oligosaccharides.

Another suspect lectin disease is rheumatoid arthritis. The normal human IgG molecule possesses carbohydrate side chains, which terminate with galactose. In rheumatoid arthritis much of the galactose is missing, so that the subterminal sugar—N-acetyl glucosamine—is exposed instead. These deficient IgG molecules feature strongly in the circulating immune complexes that cause fever and symptoms.[12] In diet responsive rheumatoid arthritis one of the commonest trigger foods is wheat, and wheat lectin is specific for N-acetyl glucosamine—the sugar that is normally hidden but exposed in rheumatoid arthritis. This suggests that N-acetyl glucosamine oligomers such as chitotetraose (derived from the chitin that forms crustacean shells) might be an effective treatment for diet associated rheumatoid arthritis. Interestingly, the health food trade has already siezed on N-acetyl glucosamine as an antiarthritic supplement.[13]

Among the effects observed in the small intestine of lectin fed rodents is stripping away of the mucous coat to expose naked mucosa and overgrowth of the mucosa by abnormal bacteria and protozoa.[14] Lectins also cause discharge of histamine from gastric mast cells,[15] which stimulates acid secretion. So the three main pathogenic factors for peptic ulcer—acid stimulation, failure of the mucous defence layer, and abnormal bacterial proliferation (*Helicobacter pylori*) are all theoretically linked to lectins. If true, blocking these effects by oligosaccharides would represent an attractive and more physiological treatment for peptic ulcer than suppressing stomach acid. The mucus stripping effect of lectins[16] also offers an explanation for the anecdotal finding of many allergists that a "stone age diet," which eliminates most starchy foods and therefore most lectins, protects against common upper respiratory viral infections: without lectins in the throat the nasopharyngeal mucus lining would be more effective as a barrier to viruses.

But if we all eat lectins, why don't we all get insulin dependent diabetes, rheumatoid arthritis, IgA nephropathy, and peptic ulcers? Partly because of biological variation in the glycoconjugates that coat our cells and partly because these are protected behind a fine screen of sialic acid molecules, attached to the glycoprotein tips.[10] We should be safe. But the sialic acid molecules can be stripped off by the enzyme neuraminidase, present in several micro-organisms such as influenza-viruses and streptococci. This may explain why diabetes and rheumatoid arthritis tend to occur as sequelae of infections. This facilitation of lectins by micro-organisms throws a new light on postinfectious diseases and makes the folklore cure of fasting during a fever seem sensible.

Alternative medicine popularisers are already publishing articles about dietary lectins,[17] often with more enthusiasm than caution, so patients are starting to ask about them and doctors need to be armed with facts. The same comment applies to entrepreneurs at the opposite end of the commercial spectrum. Many lectins are powerful allergens, and prohevein, the principal allergen of rubber latex, is one. It has been engineered into transgenic tomatoes for its fungistatic properties,[18] so we can expect an outbreak of tomato allergy in the near future among latex sensitive individuals. Dr Arpad Pusztai lost his job for publicising concerns of this type (20 February, p 483).

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