There is little doubt that abdominal adhesions form in response to peritoneal trauma. Although adhesions may result from events occurring during fetal development, the vast majority can be directly linked to surgery. However, adhesions have not been widely held to be a surgical complication, even though the impact of just one complication due to adhesions—that of small-bowel obstruction after colorectal surgery—equals, or surpasses, that of wound infection.1,2

Why is there this discrepancy between impact and acknowledgment of this complication? The answer is simple: the formation of intra-abdominal adhesions cannot be assessed unless the abdomen is reopened. Even then, adhesions are difficult to quantify. For years, in the treatment of female infertility, gynaecologists have dealt with the consequences of adhesions. The ineffectiveness of adhesiolysis in improving fertility propelled the development of in-vitro fertilisation. In surgery, however, the dominant complication due to adhesions (small-bowel obstruction) may not manifest until many years after the operation. Up to a fifth of first episodes of this complication occur two decades after the triggering procedure.1 Although clinically recognised to be a major problem, the lack of non-invasive means of quantifying adhesion formation, combined with the many years needed before a clinical trial yields results, have deterred research in this area. Adhesions have thus come to be perceived as an inevitable consequence of surgery, about which little can be done.

Although adhesions are far from completely investigated at the cellular and molecular level, a central pathophysiological mechanism leading to their development has emerged from animal studies.4–7 When the peritoneum is injured, bleeding and leakage of plasma proteins from damaged surfaces form a fibrinous deposit in the abdominal cavity (figure). This process is further fuelled by the subsequent post-traumatic inflammation. Fibrin, the function of which is restoration of injured tissues, is sticky, so the fibrinous exudate may attach to adjacent intra-abdominal structures. During the first few days after injury, this attachment seems to be reversible and the exudate undergoes enzymatic degradation by locally released fibrinolytic factors. As a part of the wound-healing response, fibrin deposition triggers a tissue-repair process that extends into the fibrin scaffolding. Within 5 days, the fibrin mesh is invaded by proliferating fibroblasts, which replace the fibrin with more durable components of the extracellular matrix (ie, collagen). Once this event has occurred, the adhesion is believed to be irreversible. Thus, the balance between fibrin deposition and degradation during the first few days is critical to the development of adhesions.

The pathogenesis of adhesions is difficult to study in human beings. The peritoneal cavity is inaccessible postoperatively, which restricts investigations of postoperative events. In recent years, investigations have been done intraoperatively. Such studies have shown a reduced peritoneal fibrinolytic capacity in conditions associated with the development of adhesion (peritonitis and surgery).8,9 Trauma has been reported to rapidly lower the main fibrinolytic stimulator, tissue-type plasminogen activator (t-PA),10 and subsequently to increase its inhibitor, plasminogen activator inhibitor type-1 (PAI-1).11 Patients with extensive adhesions were reported to have an overexpression of PAI–1 in peritoneum.12 Although there is no direct evidence of a causal relation between impaired fibrinolysis and formation of adhesions in human beings, the accumulated data strongly support the notion that a compromised peritoneal fibrin-clearing capacity favours the development of adhesions, and is in keeping with the current concept of the pathophysiology.

There is no treatment for adhesions other than surgery. Most patients who have undergone abdominopelvic
surgery develop adhesions, but the vast majority are symptom-free. Because surgically divided adhesions are extremely likely to form again, with recurrence of symptoms,19 adhesions should not be treated unless clinically prompted. Prevention of adhesions is thus of the utmost importance.

From the proposed mechanisms by which adhesions develop, several approaches to adjuvant therapy emerge—reduction of fibrin deposits by limitation of the inflammatory response; facilitation of the degradation of fibrin with fibrinolytic stimulators; and separation of surfaces during the time fibrin remains sticky. Results from animal experiments clearly show that all these approaches are effective. However, the possible side-effects of modulating the inflammatory response (infection, delayed wound healing) and administering fibrinolytic stimulators (bleeding) have made separation of tissue surfaces the most attractive option. Tissues can be physically separated during the critical period postoperatively by application of a biodegradable membrane or film at injured sites, which presumably prevents the formation of fibrin bridges and thus of adhesions. Clinical trials with barriers based on hyaluronic acid have shown reductions in quantity of adhesions of up to about 50% in abdominal12 and pelvic surgery.20 Other barrier formulations are being tested.

Why barriers have not proven 100% effective is not clear. Biological variability21 may be part of the explanation, but other factors may also be important. Because adhesions result from peritoneal trauma and inflammation, events occurring during surgery are obvious candidate factors. There is general agreement that surgical procedures and materials used should be "atraumatic." However, this ogyanor does not provide any guidelines as to what constitutes an atraumatic injury. It is therefore not surprising, albeit noteworthy, that surgical techniques, materials, devices, and other accessories used during the operation are not commonly tested for their potential to influence peritoneal tissue repair and adhesion formation. Laparoscopic surgery is commonly assumed, but has not been proven, to eliminate the problem with adhesions. In fact, reports have been conflicting, so measures for reducing the formation of adhesions will be needed with laparoscopic surgery.

Because of the difficulties in the assessment of the clinical consequences and size of adhesion-related complications, the epidemiological study in today's Lancet by Harold Ellis and co-workers is a cornerstone in delineating the problem. Perhaps this study will stimulate industry, administrators, and surgeons alike to acknowledge the size of the problem posed by adhesions and to act. Although the best option is prevention by use of a barrier based on hyaluronic acid, the ultimate question is whether reduction of adhesion formation translates into fewer clinical complications. This issue is of interest not only in benign diseases, but also especially in patients undergoing curative surgery for colorectal cancer, the largest group of patients undergoing open abdominal surgery.

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10 Holmåådahl L, Eriksson E, Eriksson BI, Risberg B. Depression of peritoneal fibrinolysis during surgery is a local response to trauma. Surgery 1998; 123: 539–44.

Lifestyle and atopy
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Epidemiological evidence of substantial variations in the risk of allergic sensitisation in relation to family structure and lifestyle in childhood has been accumulating throughout the past decade. Perhaps the most consistent observation is the inverse association between atopy and sibship size.1 Several studies have suggested that older siblings have a stronger protective effect than younger siblings, although the latter have some independent influence. These relations, first highlighted 10 years ago, led to speculation that infections in early childhood may reduce the risk of allergy. Further support for this “hygiene hypothesis” comes from a recent Lancet publication by Ursula Krämer and colleagues, who found that early entry to communal day care was associated with a lower prevalence of atopy among children from small families in former East Germany.

Socioeconomic variations in the prevalence of atopy are evident in countries as diverse as the UK,3 former East Germany,4 and Ghana.5 In each case it is the less affluent groups that are at lower risk. A recent study of Swiss children5 has shown that the prevalence of symptoms of seasonal rhinitis and of Aeroallergen-specific IgE antibodies is three times lower among the offspring of farmers than among other children in rural Switzerland. This effect was greater for families of full-time farmers than elsewhere. Another is that the diet of farming households, which includes a greater proportion of...