# Editorial

The ancient Greeks believed that suppuration of a wound was an expected feature of its natural history, and the physician encouraged the flow of what the Romans later called pus bonum et laudabile. This affirmative view of purulence held sway until the 19th century when Lord Lister refused to play the bacteria's game and strove to achieve clean, uninfected surgical wounds. Lister's theories of antisepsis changed the course of medical history with regard to wound sepsis; however, it subsequently became clear that additional modalities were needed to avert postoperative wound infection, especially when the intestinal flora with its rich source of microbes was the fount of contamination.

In this issue Tzianabos et al. (1) extend the Listerian quest to prevent wound sepsis by attempting to fortify the host's immunological status. Surprisingly, their work has shown that protection against abscess caused by mixed aerobic-anaerobic flora is related to a T cell–dependent response which can be induced by immunization with a capsular polysaccharide complex from the major microbial component, *Bacteroides fragilis.* This work is important since it suggests that immunization, either actively with this polysaccharide or passively with T cell–derived soluble mediators, might protect against infection by this organism as well as a mixed flora.

Yet several questions remain unanswered. It is not clear that prevention of abscess, which is a complex structure requiring a collagen capsule, is necessarily related to prevention of wound infection, which is a complication five times more common than abscess after intraabdominal contamination. Does immunization with the capsular polysaccharide prevent wound infection with *B. fragilis* and its microbial running mates? Or is the protection related to the specific collagen-filled structure known as abscess?

Another issue is the three rather different clinical settings of abscess formation for which polysaccharide immunization might be applied. The first condition is an established intraabdominal abscess following sometime after peritonitis or a bowel injury. It seems unlikely that the approach of polysaccharide immunization would be effective here. Surgical intervention and antibiotics will probably be the deciding modalities in this situation. (An old definition of surgical intervention applies: "The swift and ready motion of steadfast hands with experience.") The second setting is elective bowel surgery which is frequently associated with inapparent bowel leakage and peritoneal soiling. In the modern era, using mechanical bowel preparation and effective antibiotic prophylaxis, the expected incidence of postoperative wound infection is ~ 5–7% with an abscess rate of ~ 1–2%. There would be considerable question whether these already respectably low figures can be reduced any further by preoperative polysaccharide immunization; surely, the size of a clinical trial to show a significant lowering of this abscess rate is an imposing prospect. Even in this form of elective surgery, postoperative abscess formation is seen more often in immunocompromised hosts who might not respond well to preoperative immunization.

The third clinical setting, which in fact carries the highest risk of postoperative intraabdominal sepsis, includes patients with unexpected bowel perforation, e.g., diverticulitis, appendicitis, and penetrating abdominal trauma. Because of the unpredictable nature of such events it would be impossible to administer prior polysaccharide immunization to these unfortunate individuals. In their paper, the experiment with polysaccharide immunization after the bacterial challenge showed that protection was seen only with immunization using the highest dose of polysaccharide and only with challenge by a single homologous organism, *B. fragilis.* It would be important to simulate the real-life situation by challenging animals with contamination by a mixed flora in high inoculum and subsequently giving them active or passive immunization. These reservations aside, there is a need for more information before knowing the potential for *B. fragilis* polysaccharide immunization in clinical settings, which the investigators are clearly intending to provide.

For practicing surgeons in the trenches the old adage "Never let the sun set on undrained pus" still applies. The studies by Tzianabos and colleagues are in the tradition of Lord Lister and the legions of researchers who followed with efforts in the laboratory to treat or prevent wound sepsis and abscess formation. By elucidating the immunological mechanisms of abscess formation through a T cell–mediated process and then showing that protection can be produced with the purified capsular polysaccharide of the major pathogen, *B. fragilis,* this group of investigators has opened a new window onto possible protection from the burden of good and laudable pus. These pathogens are still sensitive to the extant antibiotics, so new drugs of this class are not likely to lower the rates of intraabdominal infection. Bolstering the host's defenses has enormous potential to reduce even further these dread complications.

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## Reference

1. Tzianabos, A. O., D. L. Kasper, R. L. Cisneros, R. S. Smith, and A. B. Onderdonk. 1995. Polysaccharide-mediated protection against abscess formation in experimental intraabdominal sepsis. *J. Clin. Invest.* 96:2727–2731.

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