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What are Biofilms?

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Keywords

biofilms; bacteria; spinal infection

Despite aggressive perioperative antibiotic treatments, between 1 in 20 and 1 in 100 patients undergoing spinal surgery develop an infection.¹ For patients with traumatic injuries, these rates easily exceed 10%.¹ Prevention of spinal infection depends on immune surveillance, stringent sterile measures to minimize bacterial bioburden, and antibiotic prophylaxis. As the topography of the hardware and the bone is complex, the intervertebral disc and fusion mass reside in a relatively hypoxic environment, retention of hardware is required for spinal stability, and antibiotics show attenuated efficacy against implant-associated bacteria², infections can cause life-threatening problems in spinal sites. In the presence of an implant, bacteria adhere and form biofilms (Figure 1), which are three-dimensional networks of bacteria that are encased in an extracellular polymeric substance, an encapsulating slime that contains polysaccharides and nucleic acids. The structures are semi-permeable and have channels for nutrient streaming where position within this three-dimensional structure may impact survival. Importantly, once bacteria are formed into biofilms, whether floating or associated with the implant or bone matrix, the bacteria undergo changes in metabolism and gene expression that reduce antibiotic susceptibility.³ This antibiotic recalcitrance occurs due to: (a) reduced metabolic rate —antibiotics generally target functions of rapidly growing cells; (b) secretion of factors that sequester antibiotics; and (c) decreased antibiotic diffusion rates in the biofilm, resulting in lower antibiotic concentrations and increased survival of biofilm bacteria.⁴ Importantly, the implant surface serves as the perfect host for these organisms. Specifically, it is rapidly coated with fibronectin, collagen, fibrin(ogen), and other serum proteins that facilitate bacterial adherence to the metal and biofilm formation (Figure 2). Finally, the biofilm matures and reintroduces non-adherent planktonic bacteria into the surrounding space, seeding new colonies from this original nidus of infection. These planktonic bacteria, whether from the biofilm or the original infection, can adhere to the bone extracellular matrix and most likely infiltrate the bone matrix (Figure 3). This localization provides a protected niche until cell death and/or osteolysis releases bacteria from their niches and clinical infection is reestablished. Therefore, with all of the problems

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associated with the eradication of established infection, it is critical to define new methods and technologies that can serve as successful preventatives.

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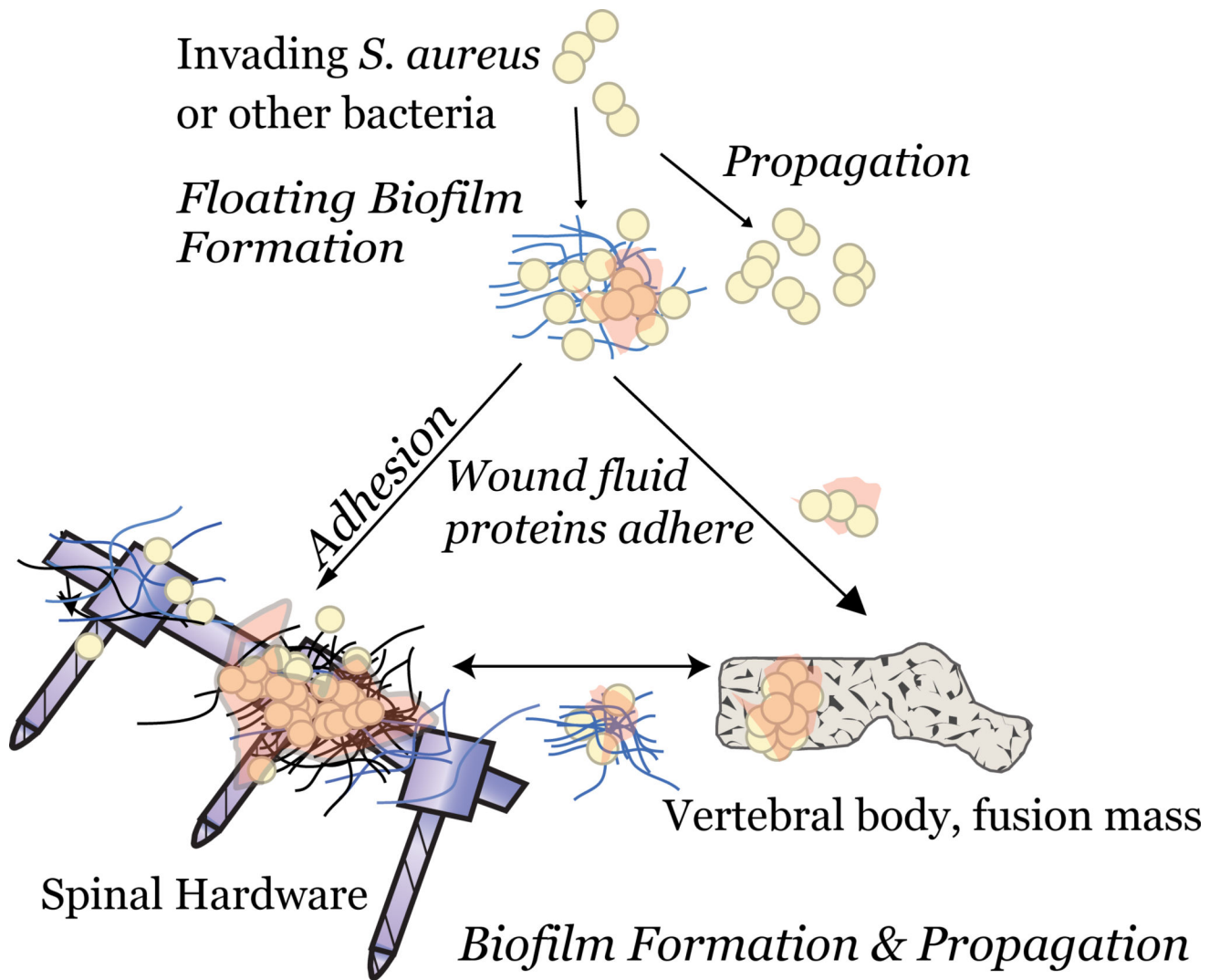


Figure 1. Etiology of spinal infection. Contaminating *Staphylococcus aureus* (*S. aureus*) aggregate into biofilms either in the wound fluid or on the surfaces of the protein-coated implant or bone.

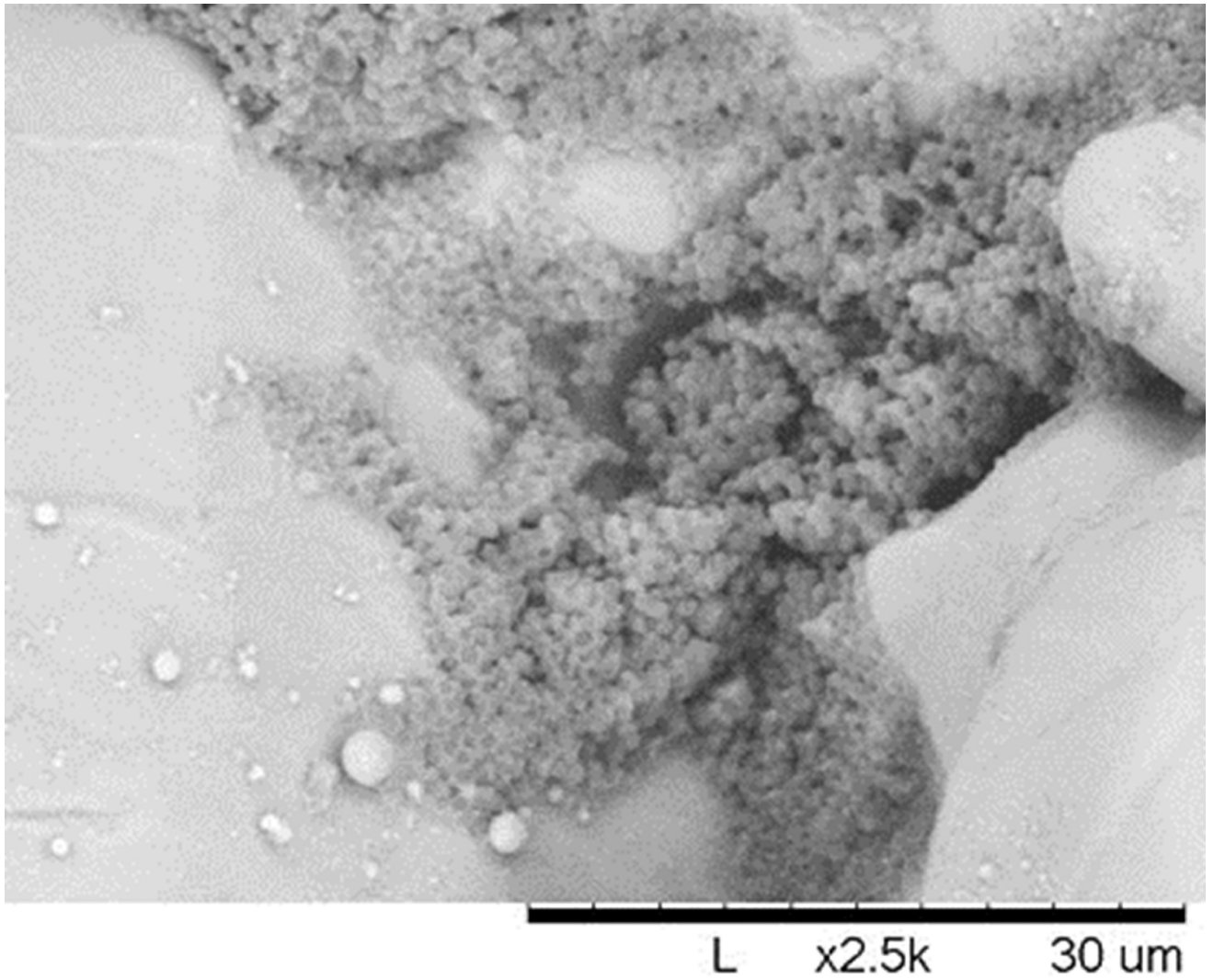


Figure 2.
S. aureus readily forms biofilms in topographic niches of implant metals, such as titanium alloy (shown here).

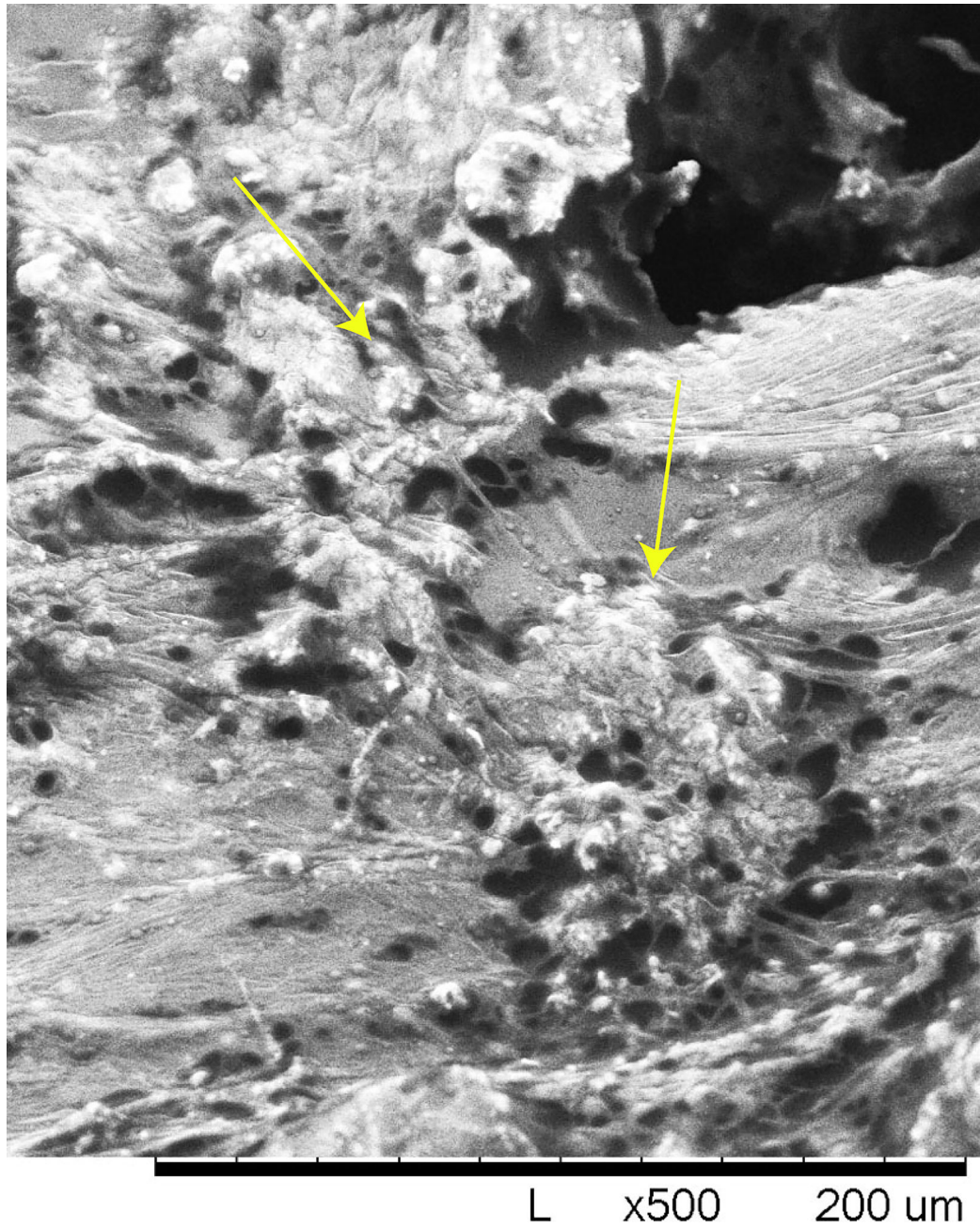


Figure 3. Bacteria form biofilm colonies on bone (yellow arrows). This bone sample was reamed from a periprosthetic joint infection operation and shows abundant biofilm coverage.