

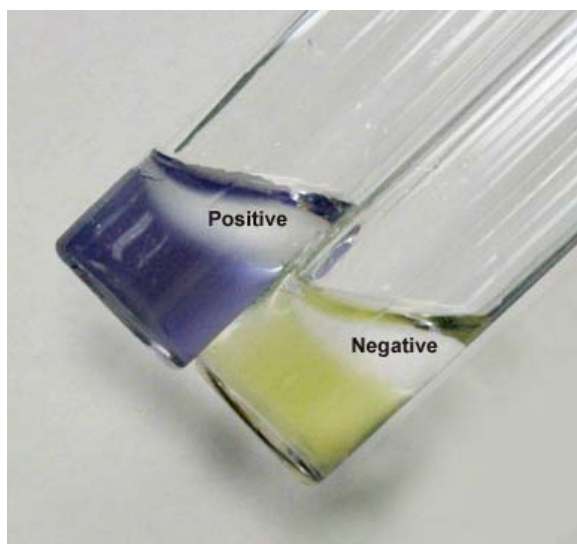
# The Slug Bug

By Barbara Fox, MS, MPH, MT(ASCP)

Coagulase negative Staphylococci (CoNS) are ubiquitous colonizers of both humans and animals. They tend to colonize skin in areas of increased surface moisture such as the groin, axilla, and perineum. The most common species is *Staphylococcus epidermidis*. In a clinical laboratory, the microbiologist's job is to decide when these CoNS may be agents of infection rather than harmless commensals. It is not always easy.

In years past, when looking at wound or abscess cultures where CoNS could be found as a contaminant, most microbiologists would not work them up, thinking they were not a pathogen in a non-sterile site. **In 1988, however, the CoNS *Staphylococcus lugdunensis* was characterized as an unusually virulent organism causing, among other things, endocarditis, septic arthritis, osteomyelitis, peritonitis, abscesses of the brain, breast and groin, and prosthetic medical device infections.** The organism has been found to exude an extracellular slime which facilitates colonization and inhibits phagocytosis.

Colonies are opaque white to slightly yellow on BAP, usually elaborating a narrow zone of beta hemolysis, and can be clumping factor positive, depending upon the testing kit used. They are [tube coagulase](#) negative. *S. lugdunensis* can be distinguished from other CoNS by their positive [Rapid Ornithine Decarboxylase](#) reaction within 2-4 hours (Hardy Diagnostics Cat. no. K279) and a positive [PYR](#). (Hardy Diagnostics Cat. no. Z75)



*Rapid Ornithine Test (Cat. no. K279) showing a positive and negative reaction at 4 hours.*

Combine the colony morphology and the positive rapid screening tests and you have a presumptive *S. lugdunensis* which can be worked up for definitive ID and sensitivity by automated ID/AST systems. Although it has been sensitive *in vitro* to all beta lactams in the past, resistance to penicillin and methicillin (via the *mecA* gene) has been detected recently.

This author has looked for and found the organism in numerous abscess cultures from both hospitalized and outpatient populations. As clinical microbiologists, we need to recognize clinical disease agents and assist in antibiotic resistance surveillance using our most current and best practice protocols.

**I urge all of you to look for and rule in or out *S. lugdunensis* when working on wound and abscess cultures, as well as other sterile sites where CoNS would normally be worked up as the pathogen. To NOT do so would be akin to missing a *S. aureus* in terms of clinical relevance.**

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