

Editorial

Leclercia adecarboxylata: A Pathogen for the New Age?

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Leclercia adecarboxylata was initially described by Leclerc in 1962 as *Escherichia adecarboxylata*, a member of the pigmented *Enterobacteriaceae* group (Enteric group 41) [1]. However, based on the detailed phenotypic and genotypic characterization of the species (Tamura et al.), it was renamed in 1986 to *L. adecarboxylata* [2]. *L. adecarboxylata* is a typical member of the Enterobacterales order: it is a Gram-negative, lactose-positive, motile rod [3]. This species is abundantly found in nature (e.g., aquatic environments, drinking water, soil and on the surface of various objects). *L. adecarboxylata* was previously considered an uncommon isolate from human clinical samples and scarce clinical data was available about its relevance in infectious processes. However, the significance of this species in clinical cases has recently been re-evaluated. This pathogen has been reported as a causative agent in bacteremia/sepsis, diarrhea, pneumonia, peritonitis, septic arthritis, skin and soft tissue infections, abscesses and urinary tract infections, both in immunocompromised (e.g., leukemia or other malignancy, AIDS) and immunocompetent individuals [4]. Microbiological laboratories, whose methods only include classical biochemical methods, may be inadequate in the correct identification of *L. adecarboxylata*, because this species shares many biochemical characteristics with its sibling species in the order Enterobacterales. This is especially true in conjunction with *Escherichia coli*, as *L. adecarboxylata* is phenotypically and biochemically very similar to this common gut bacteria [5].

Biochemical reaction-based identification systems (e.g., VITEK) and selective Chromogenic Media (e.g., CHROM ID[®] CPS[®] Elite Agar or CPSE) may offer a cost-effective solution to the correct identification of this species [4]. Biochemical reactions that may be useful in the differentiation of *E. coli* from *L. adecarboxylata* include lysine decarboxylase, malonate assimilation and acid production from arabinol and cellobiose, however, these are only reliable in the case of wild-type strains [5]. With the advent of rapid molecular methods (polymerase chain reaction) and typing techniques, Matrix-Assisted Laser Desorption/Ionization Time-Of-Flight Mass Spectrometry (MALDI-TOF MS) and Next Generation Sequencing (NGS) in clinical microbiology laboratories, the perceived incidence and significance of this pathogen has increased substantially, and it can no longer be rejected as ‘just another contaminant’ [4].

L. adecarboxylata strains are naturally sensitive to various antibiotics, such as penicillinase-stable penicillins and other beta-lactams, sulfamethoxazole/trimethoprim, tetracyclines, aminoglycosides, fluoroquinolones, chloramphenicol, azithromycin and nitrofurantoin [4]. In contrast, they have intrinsic resistance to penicillin G, oxacillin, glycopeptides, non-azithromycin macrolides (erythromycin, roxithromycin, clarithromycin, ketolides), clindamycin, streptogramins, linezolid, rifampicin, fusidinic acid and fosfomycin [4,5]. Some reports suggest that resistance to fosfomycin may be an additional differentiating factor between *E. coli* and *L. adecarboxylata*, however, due to the increase in fosfomycin resistance in *E. coli*, this may also lead to discrepant results. This species is also affected by the rapid emergence of drug resistance in Gram-negative pathogens, a case of a multidrug-resistant *L. adecarboxylata* isolate has already been described [6,7].

The clinical relevance of isolated *L. adecarboxylata* from various specimens in the microbiology laboratory need to be evaluated based on the clinical picture. For this reason, continuous communication between the clinicians and the diagnostic lab is crucial. Physicians (ranging from general practitioners to infectious disease specialists) should familiarize themselves with “old/new” pathogens, that may cause disease for the adequate diagnosis and therapy of their patients [8].

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