LETTER TO THE EDITOR

SEROGROUPING AS A VALID BUT INCOMPLETE METHOD TO DIAGNOSE DIARRHEAGENIC *ESCHERICHIA COLI*

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In the recent article by Codruța-Romanița Usein *et al*. published in the latest issue of the *Romanian Archives of Microbiology and Immunology* the authors alert about the insufficiency of commercial O antisera for a correct diagnosis of diarrheagenic *Escherichia coli* [1]. In their study, the authors show that additional procedures including amplification of virulence genes are essential for a correct identification of enteropathogenic *E. coli* (EPEC) and Shiga toxin-producing *E. coli* (STEC)/Enterohemorrhagic *E. coli* (EHEC).

Bacterial identification and characterization techniques have evolved fast since the introduction of molecular methods during the 1990s to the use of genomics in recent years. But economic and technical restraints prevent laboratories all over the world from joining the genetics/genomics race in a routine basis. Commercial O antisera remain then the best resource for detection of EPEC and EHEC.

A limitation of commercial kits for serogrouping is that they only target major groups of EPEC and EHEC, defined a long time ago [2]. But the diarrheagenic *E. coli* populations circulating in each country might evolve along time. That has been the case of O80 and O26, which are displacing O157 as the predominant serogroup of EHEC in France and other countries in Europe in recent years [3, 4]. O80:H2 EHEC was described as a hybrid pathotype, with both intestinal (*stx, eae, ehxA*) and extraintestinal virulence factors, and an unusual multidrug resistant profile (to aminopenicillins, aminoglycosides, nalidixic acid, cotrimoxazole, tetracycline, and phenicols). Severe extraintestinal infections caused by O80 were recently described in Europe [5, 6, 7]. The EHEC O26:H11 international outbreak in 2016 was a multi-strain event, with strains carrying different virulence genes – some strains carrying *stx1a, stx2a, eae*, while *stx2a* was missing in others. The outbreak, related to a dairy producer in Italy, affected patients in Romania and Italy, and it supposed the largest EHEC- associated HUS outbreak in Italy to date [8, 9, 10].

Also, epidemics can eventually be caused by infrequent serogroups, like O104, which caused an international outbreak in 2011, centred in Germany, with more than 3800 cases. This O104:H4 strain was also hybrid, characterized by the combination of EHEC typical virulence genes (*stx1, stx2, eae,* and *ehx*) and atypical ones as the *aggR* gene, which is the defining factor for enteroaggregative *E. coli* (EAEC) strains [11]. In order to detect outbreaks and epidemics like these, antisera will be insufficient to identify the new clones, for which virulence genes will have to be searched [12].

Eventually, strains belonging to the typical serogroups of EPEC and EHEC might not carry the expected virulence genes [1, 13, 14], so pathogenic clones can be overestimated and medical measures including antimicrobial therapy might be over-prescribed.

EPEC and enterotoxigenic *E. coli* (ETEC) are endemic in developing countries, while in developed countries major outbreaks are caused by EHEC. Molecular characterisation of *E. coli* strains targeting the adequate

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virulence genes is needed in order to prevent underestimation of those clones [15].

Therefore, efforts must be held to search for virulence factors in parallel to a correct identification of serogroups, to properly identify circulating and emerging diarrheagenic *E. coli* populations, thus establish treatment and public health measures.

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