Table 1. Essential agreement (%) between M.I.C.E. or Etest™ strips and CLSI BMD (±1 $\log_2$ dilution)

	Enterobacteriaceae[a] (*n*=40)		Non-fermenters[b] (*n*=40)		Staphylococci[c] (*n*=40)		Enterococci[d] (*n*=40)	
	M.I.C.E.	Etest	M.I.C.E.	Etest	M.I.C.E.	Etest	M.I.C.E.	Etest
Ampicillin	100.0	100.0					95.0	100.0
Amoxicillin/clavulanic acid	90.0	97.5						
Ciprofloxacin	100.0	97.5	97.5	97.5	85.0	95.0		
Cefotaxime	85.0	82.5						
Erythromycin					100.0	100.0		
Gentamicin	100.0	97.5	95.0	92.5	95.0	97.5		
Gentamicin HL							100.0	100.0
Imipenem	97.5	100.0	100.0	100.0				
Linezolid					97.5	100.0	100.0	100.0
Oxacillin					90.0	95.0		
Vancomycin					72.5	90.0	80.0	87.5
Overall	95.4	95.8	97.5	96.7	90.0	96.3	93.8	96.9

[a]*K. pneumoniae* (*n*=20) and *E. coli* (*n*=20).
[b]*P. aeruginosa* (*n*=20) and *Acinetobacter* spp. (*n*=20).
[c]*S. aureus* (*n*=20) and coagulase-negative *Staphylococcus* (*n*=20).
[d]*E. faecalis* (*n*=20) and *E. faecium* (*n*=20).

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Transparency declarations

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Comment on: Rifaximin modulates the colonic microbiota of patients with Crohn's disease: an *in vitro* approach using a continuous culture colonic model system

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Keywords: immunity, gastroenterology, CD4+/CD103+, gut homing, uncomplicated diverticular disease

Sir,

We read with interest the paper by Maccaferri *et al*.[1] We appreciated their reference to our previous work.[2] However, their reference misrepresents our data. In fact, in our paper we did not show that rifaximin reduced release of inflammatory cytokines. Rather, we evaluated T cell subpopulations in uncomplicated diverticular disease (UDD) before and after a short course of rifaximin treatment. In the peripheral blood we demonstrated that CD103+ (gut homing T cells) were significantly increased at baseline and decreased after antibiotic treatment, suggesting a role for central immunity in the pathogenesis of the disease. More important and novel were our findings at the tissue level where CD4+/CD62L+ cells were increased before treatment, while CD4+/CD103+ cells increased after treatment. Our data strongly suggest that antibiotic treatment modified T cell subpopulation distributions both in the peripheral blood and at the tissue level and that the study of tissue infiltrating lymphocytes (TILs)[3,4] is an interesting new tool in the study of gut homing, especially when combined with the characterization of T cell subpopulations in the peripheral blood. We have previously suggested that the study of TILs, as compared with peripheral blood, better reflects in more detail the pathological process at the site of infection.[2] Accordingly, in our previous study of immunological modifications induced by rifaximin, we demonstrated that in UDD antibiotic treatment reduced the recruitment of CD4+/CD103+ cells in the peripheral blood and induced a significant increase in CD4+/CD103+ subpopulations at the tissue level, resulting in a redistribution of CD4+/CD103+. Although it is possible that these events may lead to altered cytokine production as suggested by Maccaferri *et al*.,[1] unfortunately this was not part of our study. Heterogeneity of CD4 cells has been shown in the past,[5] but only recently novel T cell subpopulations, such as T regulatory cells (Tregs), Th17 cells and CD4+/CD103+ gut homing cells, have been described in detail (as reviewed by Pandolfi *et al*.[6]).

We conclude that the study of CD4 subpopulations in the gastrointestinal mucosa is a valuable new tool to understand the effects of treatments for diseases causing inflammation. Our results were not acknowledged by Maccaferri's citation of our paper, while they incorrectly reported that our work showed a modification in cytokine production.

Transparency declarations

None to declare.

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Rifaximin modulates the colonic microbiota of patients with Crohn's disease: an *in vitro* approach using a continuous culture colonic model system—authors' response

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Keywords: inflammatory bowel disease, immune modulation, immune response

Sir,

We read with interest the comment by Cianci *et al*.,[1] referring to our recent publication in *JAC*.[2]

Cianci *et al*.[1] state that we cited their recent work[3] incorrectly. We appreciate the importance of the research of Cianci *et al*.[3] on gut-homing T cells for the understanding of inflammatory conditions in the bowel. Further, we regret that in our sentence concerning 'alternative mechanisms of action' of rifaximin, including '(iii) reduction of inflammatory cytokine release', we did not expand on the complexity of the involvement of the immune system. We are grateful for the detailed explanation and clarification by Cianci *et al*.[1] regarding their article[3] that we referred to.

We would like to emphasize that it was our intention to discuss how the therapeutic efficacy of the antibiotic rifaximin can be