Review of: "Long-term beneficial effect of faecal microbiota transplantation on colonisation of multidrug-resistant bacteria and resistome abundance in patients with recurrent Clostridioides difficile infection"

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Potential competing interests: No potential competing interests to declare.

Review: Long-term beneficial effect of faecal microbiota transplantation on colonisation of multidrug-resistant bacteria and resistome abundance in patients with recurrent Clostridioides difficile infection.

Nooij et al studied the effect of FMT is patients with rCDI on colonisation with MDR bacteria and antibiotic resistance genes. Results showed that the colonisation with MDR bacteria decreased from a high 23% pre-FMT to about 11% 3 weeks post-FMT. Two years post-FMT the resistomes of the recipients normalised to that of the donor.

The study is well designed and written. The results are clearly outlined and conclusions firm. The few grammatical errors should be addressed.

There are however a few limitations of the study.

1. Pre-FMT samples were taken after vancomycin treatment of at least 4 days. Standard of care for rCDI is treatment with fidaxomicin and not vancomycin. It is not clear how treatment failure was defined and why 4 days were used as cut-off.
2. The authors must explain why fidaxomicin was not employed more often (only in 6) as treatment strategy following vancomycin failure.
3. Although MDR genes were identified, only 7 of 63 were resistant to vancomycin in the pre-FMT cohort. There is no mention of fidaxomicin resistance. What then drives resistance to treatment?
4. The presence of resistance genes does not equate to treatment failure as only 20% of those with resistomes required

FMT. Eighty percent of those failed medical therapy and required FMT never had resistance genes. So, what is the clinical relevance of resistomes?

5. The authors must elucidate the treatment population in a clearer fashion. For instance, how many patients were retreated and how many more had FMT more than once. What are their clinical and laboratory parameters? Does the answer to resistance genes lie there?

What is promising is that FMT restores the resistome to an acceptable level. FMT is the only recognised treatment strategy for rCDI, but more data is needed for resistance treatment. Does fixing resistomes improve medical therapy for rCDI?

Thus, although this data is very novel and promising, it creates more questions. Hopefully this will be answered with follow-up studies.