
A Compartmentalized Microbial Consortium for the Detection and Degradation of Gastrointestinal PET

A. Mengane^{1*}, C. Shin², S. Parvaresh Rizi¹, X. Fu¹

[1] Division of Engineering Science, Faculty of Applied Science & Engineering, University of Toronto, Toronto, Ontario, M5S 1A4

[2] Department of Physiology, Schulich School of Medicine & Dentistry, Western University, London, Ontario, N6A 5C1

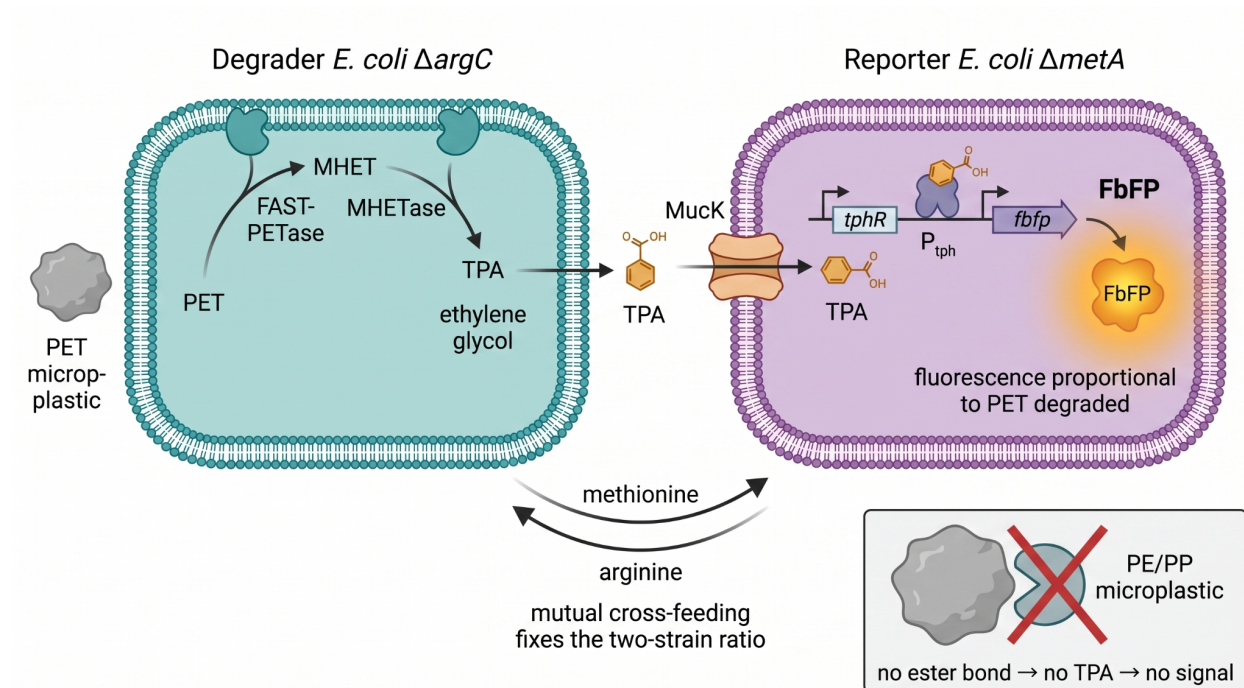
*Correspondence: claireshinxv@gmail.com

Abstract

Microplastic pollution is an emerging health concern, with polyethylene terephthalate (PET) plastic being one of the most abundant ester-bonded polymers detected in the human gastrointestinal tract (Schwabl et al., 2019). PET releases phthalates and antimony (Sax, 2010), degrading hormonal systems and causing reproductive issues (Dhaka et al., 2022). Furthermore, microplastics contribute to intestinal barrier disruption and chronic inflammation in tumoral colon tissue (Dzierżyński et al., 2025; Cetin et al., 2023; Gosavi et al., 2025), where individuals with higher concentrations of microplastics in their fecal matter are 12 times more likely to develop colorectal cancer (Xu et al., 2025). The discovery of *Ideonella sakaiensis* was promising through hydrolysis of PET into its constitutive monomers (Yoshida et al., 2016); however, advancements on PET degradation are limited regarding physiological environments.

This study proposes a two-strain engineered *Escherichia coli* Nissle 1917 consortium combining FAST-PETase-mediated PET degradation with terephthalic acid (TPA) responsive biosensing for real-time quantification of microplastic breakdown under anaerobic gut-like conditions. The study hypothesizes that PET hydrolysis by the Degradation Strain will generate TPA proportional to microplastic degradation, activating the Reporter Strain to produce a fluorescent readout. The consortium uses a division-of-labour approach, with a Degradation Strain expressing surface-displayed FAST-PETase/MHETase to hydrolyze PET microplastics into TPA (Kotnis et al., 2025). The Reporter Strain expresses a MucK-type TPA uptake transporter and a TphR-responsive biosensor driving oxygen-independent FbFP fluorescence to quantify degradation (Oh et al., 2025). Mutual auxotrophic regulation will maintain a stable degrader-to-reporter ratio (Grandel et al., 2025). We expect PET exposure to produce degradation-dependent FbFP fluorescence, correlating positively to HPLC-quantified TPA and gravimetric PET mass reduction. Non-PET substrates (e.g. polypropylene) will confirm substrate specificity, which are predicted to have minimal signal. Fluorescence intensity is predicted to correlate with TPA production and PET mass reduction, validating the consortium as a quantitative biosensor for monitoring degradation.

Although in vitro gut models cannot fully replicate the human gut, this experiment validates the consortium as a quantitative degradation biosensor. Since colorectal tumour tissues have high microplastic concentrations (Cetin et al., 2023), this platform could support future efforts in risk stratification by allowing patient-specific assessment of microplastic persistence and degradation, guiding preventative strategies to reduce microplastic accumulation (Mashayekhi-Sardoo et al., 2025).



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Figure 1. Proposed two-strain *Escherichia coli* Nissle 1917 consortium for real-time quantification of polyethylene terephthalate (PET) degradation under gut-relevant conditions (37 °C, pH approximately 7). Left: the Degradation strain ($\Delta argC$) surface-displays FAST-PETase and MHETase, which hydrolyze PET into mono(2-hydroxyethyl) terephthalate (MHET) and then terephthalic acid (TPA) and ethylene glycol (EG); MHETase is required to liberate the TPA that the sensor detects. Right: TPA is transferred to the Reporter strain ($\Delta metA$), imported through the MucK transporter, and bound by the TphR regulator, activating the promoter that drives oxygen-independent flavin-based fluorescent protein (FbFP) expression, so fluorescence increases with the amount of PET degraded. Bottom: reciprocal auxotrophic cross-feeding of arginine and methionine between the strains holds the consortium ratio constant, keeping the signal quantitatively proportional to degradation. Inset: polyethylene and polypropylene lack ester bonds, yield no TPA, and produce no signal, confirming PET specificity.

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