Surface Mediated Enzyme Sequestration by Ingested HDPE Macroplastics in a Biomimetic King Penguin Intestinal Model

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Macroplastic debris has been reported in the gastrointestinal tracts of King penguins (Aptenodytes patagonicus), yet the molecular mechanism by which ingested plastics disrupt digestion remains unresolved. This investigation tested whether hydrophobic polymer surfaces act as non-biological adsorption sites that sequester digestive enzymes, thereby reducing free enzyme availability under biomimetic intestinal conditions representative of King penguins (39 °C; pH 7.5–8.0). This frames plastic ingestion as a surface-chemistry-mediated disruption of enzyme-driven hydrolysis. Phase 1 tested how different polymers affected starch and lipid digestion. Polymer type significantly influenced starch breakdown at the 90-minute Benedict's endpoint (one-way ANOVA, $F(4,10) = 21.5$, $p < 0.001$, $?^2 = 0.90$). Tukey HSD showed that LDPE and PS reduced starch digestion compared with the positive control, while PET and PVC had no significant effect. Lipid digestion also differed significantly by polymer type, based on the change in pH after 120 minutes, ?pH120 ($F(4,10) = 5.30$, $p = 0.015$, $?^2 = 0.68$). Post hoc analysis showed that PET, PS, and LDPE reduced lipid digestion, whereas PVC did not. Phase 2 used a BCA assay to examine protein digestion under increasing HDPE exposure. Although protein digestion increased over time across all treatments, HDPE produced a clear dose-dependent reduction. Early-phase reaction rates declined as HDPE mass increased, and percent suppression analysis confirmed sustained reduction in digestive efficiency, with the greatest inhibition in the high-HDPE treatment. Together, these findings support enzyme sequestration as a plausible molecular mechanism by which hydrophobic macroplastics may impair digestion in marine endotherms.

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