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Antimicrobial activity of apple cider vinegar against methicillin resistant *Staphylococcus aureus* and resistant *E. coli*

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Introduction: Globally, there has never been a more desperate need for novel anti-microbial agents to target microbes and multi drug resistance from bacterial infections especially to combat methicillin resistant *Staphylococcus aureus* (MRSA).

Aim: The aim of this study was to investigate the potential anti-microbial effects of apple cider vinegar (ACV). We used resistant *E. coli* and MRSA microbial strains purchased from ATCC. We tested the effect of commercial ACV directly on microbial cultures over a 24-hour period, measuring inhibition zones. We also looked at whether ACV could have an anti-inflammatory effect *in vitro*. This was tested using human blood derived monocytes which were incubated with microbes and AVC. Collected supernatants were analysed for pro-inflammatory cytokine secretion by ELISA.

Results: ACV was able to significantly inhibit resistant *E. coli* and MRSA growth demonstrated by the results of direct co-culture with each of the microbial inoculums and ACV in varying concentrations. The zone of inhibition with the addition of ACV to each of the microbes varied dose dependently on ACV concentration. For MRSA 100mg/ml of ACV had the strongest effect, whereas on resistant *E. coli* cultures, the most potent effect was visible at 200mg/ml concentration ($p<0.05$). When monocytes were cultured with both microbes they secreted inflammatory cytokines (TNFα, IL-1β). ACV was effective in significantly inhibiting inflammatory cytokine secretion in human peripheral blood monocytes cultured with *E. coli* and MRSA.

Conclusion & Significance: ACV displayed potent anti-microbial and anti-inflammatory activity against resistant *E. coli* and MRSA. We propose that ACV could be potentially therapeutic in clinical antibiotic resistance.

Biography

Darshna Yagnik is a lecturer in Immunology and Biomedical Sciences at Middlesex University. Her research is based on human *in vitro* models of mononuclear cell differentiation and their role in inflammatory pathways and particularly the resolution phase of inflammation.

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