Background
Nitric oxide (NO) has anti-platelet activity.1 Nitrate, nitrite and nitrosothiols (SNO) are NO intermediates.2 Exogenous NO sources include dietary inorganic nitrate and commercial products, Beet It (BI) juice and SIS® Go+ nitrate (SGN) gel.3 In the entero-salivary circulation nitrate reduces to nitrite via nitrate reductase (NR) from commensal oral bacteria.4 Nitrite reacts with gastric proteins to form R-SNO; clopidogrel may enhance production.5 Patients with coronary artery disease (CAD) have impaired endogenous NO generation.6 7

Methods
A model was created enabling in vitro sodium nitrate salt (SNS) reduction by NR from Aspergillus niger.8 SGN gel, placebo gel and BI were tested using this. Product was added to gastric medium to form R-SNO, with and without clopidogrel.5 NO metabolites were quantified using ozone-based chemiluminescence.9

Results
SGN gel and BI produced limited nitrite. Placebo gel (nitrate deficient) mixed with SNS also produced limited nitrite. Gel dilution improved yield. R-SNO production was greater from nitrite converted from SGN gel than SNS; clopidogrel did not enhance yield.

Key messages
A model was established for the reduction of nitrate from commercial agents. R-SNO formed in gastric medium in the presence of nitrite. This helps understand a clinical study assessing therapeutic effects of nitrate on platelet activity in CAD.1

REFERENCES

Conflict of interest None