Ethyl Glucuronide (EtG) is a water-soluble, stable, non-volatile, direct metabolite of ethanol. Metabolism occurs as soon as alcohol enters the bloodstream. More than 90% of alcohol consumed is metabolised in the liver, via the action of the enzyme alcohol dehydrogenase (ADH). ADH catalyses the reaction in which alcohol is oxidised to acetaldehyde. This is then metabolised to acetyl CoA by acetaldehyde dehydrogenase and then to carbon dioxide. Other enzymes capable of metabolising alcohol include: catalase, the cytochrome P450 enzyme, CYP2E1 and microsomal oxidising systems (MEOS). Between 5-8% of unchanged alcohol is excreted in the urine, sweat and breath. The elimination of ethanol by enzymatic conjugation with glucuronic acid represents approximately 0.5 to 1.5% of the total ethanol elimination.

Whilst the detection period for alcohol is relatively short, EtG can be detected for up to 80 hours and peaks at approximately 4 hours after alcohol consumption. EtG offers several advantages over traditional markers of alcohol abuse such as gamma glutamyl transferase (GGT), mean corpuscular volume (MCV) and carbohydrate deficient transferrin (CDT). It is a direct, specific and sensitive marker of alcohol consumption, being present only if ethanol is consumed. It is not influenced by age, gender, medication or non-alcohol related diseases and is not dependant on chronic alcohol consumption. EtG does not accumulate during chronic alcohol intake.

For these reasons EtG is proving a promising biomarker for monitoring alcohol abstinence, where zero tolerance policies are in existence. Such examples include liver transplant recipients, recovering alcoholics in withdrawal treatment programmes and some fields of employment such as aviation or other automotive fuel. The alcohol in these products may enter the body via oral consumption. Absorption or inhalation. We investigated the effect of incidental alcohol consumption, from mouthwash, on urinary EtG concentrations.

**RESULTS & DISCUSSION**

All test and control samples were negative for ethanol (<10mg/dL). Four of the five subjects were negative for EtG after use of all mouthwashes tested. One subject yielded elevated results (84-180ng/mL) for the mouthwashes compared to the other patients. Previous studies have also shown this individual to give higher urinary EtG concentrations than other subjects tested. These findings may result from incidental alcohol consumption via another route or possibly EtG synthesis in infected urine. It is possible that inter-individual variations in EtG production exist. Whatever the source, it is apparent that EtG positives may result from incidental alcohol exposure in individuals who knowingly abstain from alcoholic beverage consumption. Until more is known about the effects every day alcohol containing products have on EtG production, care should be taken in applying cut offs and interpreting these results.

**EXPERIMENTAL**

Five commonly available, alcohol containing mouthwashes (table 1) were purchased and used as directed. Five female volunteers (<30 years old), abstained from alcohol for a minimum of 3 days. Urine samples were then obtained immediately before (control) and 4 hours (+/- 15 minutes) after use of the mouthwashes. These were analysed for EtG using the Microgenics DRI® EtG Enzyme Immunoassay on the Olympus AU400 platform. Assays were semi-quantitative (0, 100, 500, 1000, 2000 (ULOQ) ng/mL) with 4 QC levels employed (375, 625, 750, 1250ng/mL). The sensitivity of the assay is quoted as being 15.3ng/mL, but cut offs of 500ng/mL or 1000ng/mL are typically recommended for monitoring alcohol abuse in alcohol rehabilitation programs. Unlike urinary excretion of ethanol, EtG concentrations are highly influenced by water intake. Normalisation of EtG to creatinine is recommended.

Creatinine, measured using the Jaffe reaction on the Siemens Advia 2400 and ethanol concentrations measured by head space GC-FID on a Shimadzu GC 2014 coupled to a HTA, HT200H head space auto sampler, were used for comparison.

**REFERENCES**