First draft statement on the interaction of caffeine and alcohol and their combined effects on health and behaviour

1. The Committee considered a review of the literature on caffeine and alcohol interactions at their meeting in March 2012. Members agreed to produce a statement outlining their views on the data available.

2. Members were interested in the effects of glucose and sucrose on gastric emptying and whether this would have an influence on the interactions between alcohol and caffeine. The vast majority of energy drink sales (78%) contain sucrose or glucose and fructose with the remaining 22% containing artificial sweeteners (BSDA, 2012).

3. Mono- and disaccharides have been shown to reduce the rate of gastric emptying compared to water alone or artificial sweeteners with the smaller monosaccharides having the greatest effect due to their osmolarity (Trout and Bernstein, 1986; Elias et al, 1968; Lavin et al, 2002; Ma et al, 2009; Wu et al, 2006). The co-consumption of alcohol and sugar-sweetened energy drinks is therefore likely to slow down gastric emptying and reduce the peak blood alcohol level compared to their artificially sweetened counterparts.

4. The Committee also noted that it would be useful to have some social science perspectives. The FSA Social Science Unit has noted that the evidence contained within the paper does not seem to consider the social or environmental factors that might be at play. Some questions might relate to:
   - Understanding who might drink such drinks, in what situations and why.
   - Do those factors that predispose someone to negative behaviours also mean they are driven towards energy drinks?
   - Who is the population most at risk.
   - All of the studies that looked at drinking habits linked to behaviours were carried out amongst students. Is this because they are the most at risk or because this was a ready population to research.
   - What about those from lower socio-economic backgrounds?
   - Who are the heavy consumers?
   - It would be useful to investigate the market profile of these drinks in the UK.

5. An energy drinks manufacturer has provided confidential information to the Secretariat which included an assessment of caffeine intakes from a large sample of individuals from Austria along with their socioeconomic profiles. Due to the confidential nature and the similarity of the results to those already publically available, the secretariat did not consider that this added much to the available literature.
6. Professor Peter Rogers, Head of the School of Experimental Psychology at the University of Bristol has made some comments on the paper which the Committee may wish to see. These have been included as annex 3.

Questions asked of the Committee

i. Members are invited to comment on the first draft statement on the interaction of caffeine and alcohol and their combined effects on health and behaviour in Annex 1 and do members wish to add anything in the light of Professor Rogers’ comments in annex 3?

ii. Members are asked if they wish to make a reference in the statement to the effects of sugar sweetened and artificially sweetened drinks on gastric emptying; given that sales of artificially sweetened drinks are limited compared to the sugar sweetened varieties.

iii. Should a conclusion be added with respect to need for social science research?

Secretariat
June 2012
References:


First draft statement on the interaction of caffeine and alcohol and their combined effects on health and behaviour

Introduction
1. The Committee was asked to comment on concerns over the potential for interaction between the caffeine in energy drinks and alcohol\(^1\) to result in adverse behavioural or toxic effects.

Background
2. Since the introduction of “Red Bull” in Austria in 1987, the sales of energy drinks have risen dramatically, with the average annual growth rate from 2002 to 2006 being 55% (Reissig et al, 2009). The popularity of energy drinks mixed with alcoholic beverages has also increased, especially amongst young males, with individuals who consume higher levels of energy drinks also consuming greater quantities of alcohol and seeming to engage in a greater degree of risk taking. These factors have given rise to concerns over the health effects of this combination of psychoactive substances in particular, a phenomenon described as “wide awake drunk” where the stimulatory effect of caffeine prevents the consumer from realising how intoxicated they are, with the potential for increased toxicological damage and adverse behavioural effects occurring (Reissig et al, 2009). In a report from the Scottish Prisons Service, other alcoholic beverages such as “Buckfast Tonic Wine”, which contains significant quantities of caffeine (375-550 mg/L) and 15% alcohol by volume, have been associated with violence in young offenders in Scotland; with 43.4% of young offenders from a sample of 172 admitting consumption of Buckfast Tonic Wine prior to their most recent offence (Scottish Prisons Service, 2009; Wikipedia, 2011). Most energy drinks contain levels of caffeine approximately equivalent to the levels found in coffee along with other substances such as sugar, taurine and glucuronolactone.

3. The Scientific Committee on Food (SCF), who advised the European Commission prior to the creation of the European Food Safety Authority (EFSA), looked at the safety of energy drinks in 1999 and 2003 (SCF, 1999; SCF, 2003). As part of their second assessment, the SCF looked at the evidence for an interaction between caffeine and alcohol. The Committee concluded that the majority of studies suggested that caffeine would not exacerbate the adverse effects of alcohol; at lower blood alcohol levels, and on simpler tasks, caffeine may improve performance (SCF, 2003).

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\(^1\) In this document, the term alcohol will refer to ethanol present in alcoholic beverages.
4. The SCF also looked at the evidence for interactions between alcohol and other constituents of energy drinks such as taurine and glucuronolactone. They observed that both taurine and alcohol inhibit the release of the antidiuretic hormone vasopressin, therefore they could act additively to increase water and sodium loss from the body in the short term increasing the risk of dehydration. In a 13-week study in rats, taurine has been shown to cause behavioural effects in animal studies including persistent increased activity, occasional chewing of limbs and a possible decrement in motor performance on a rotarod in all dose groups tested (300, 600 and 1000 mg/kg bw/day). The lowest dose was equivalent to 6 times the mean acute intake from energy drinks of 50mg/kg bw. The SCF concluded that some alcohol–taurine interactions are possible, including behavioural interactions, but these were neither marked nor consistent in human and animal studies. The SCF was of the opinion that focused neurological studies should be carried out in this area. The SCF concluded that glucuronolactone would not be expected to interact with alcohol or other constituents of energy drinks.

5. The COT was asked to consider the literature published since the SCF opinion of 2003, and advise on the potential for interactions between caffeine and alcohol.

Current European legislation on caffeine

6. Under European Directive 2002/67/EC on the labelling of foodstuffs containing quinine and foodstuffs containing caffeine, beverages containing more than 150 mg/l caffeine (other than those based on coffee or tea) must carry the statement ‘High caffeine content’ in the same field of vision as the name of the product followed by a reference in brackets to the caffeine content expressed in mg per 100ml. Under the new Food Information Regulation (EU 1169/2011), which comes into effect on the 13 December 2014, beverages containing more than 150 mg/l caffeine (other than those based on coffee or tea) must carry the statement ‘High caffeine content. Not recommended for children or pregnant or breast feeding women’ in the same field of vision as the name of the beverage followed by a reference in brackets to the caffeine content expressed in mg per 100ml.

Caffeine in beverages

7. Caffeine (1,3,7-trimethyl xanthine) is probably the most widely used psychoactive substance (Benowitz, 1990). The pharmacologically active dose can vary considerably between individuals as a tolerance is rapidly developed to the effects of caffeine, however levels of 2-3 mg/kg bw have been shown to stimulate central nervous system activity in humans (FDA, 1978). The structure of caffeine is shown in figure 1:
8. In 2004, the Dietary Caffeine and Health Study estimated a mean caffeine intake of 241mg/day for 6,000 individuals from the Bristol area who filled out a questionnaire designed to measure consumption of coffee, tea, chocolate products, cola drinks and energy drinks (FDA, 1978; Heatherley et al, 2006b; Heatherley et al, 2006a). This level of intake is similar to that identified from a Ministry of Agriculture, Fisheries and Food (MAFF) survey of 1988 of consumption of coffee, tea and colas, from which the mean caffeine intake was estimated to be 3.98 mg/kg body weight per day (i.e. 279 mg/day for a 70 kg person) for the general population and 3.43 mg/kg body weight per day (i.e. 240 mg/day for a 70 kg person) for pregnant women. In terms of instant coffee, this would be equivalent to 2-2.5 average sized mugs (260ml) assuming an average content of 100 mg caffeine per mug. The MAFF survey did not include other sources of caffeine such as chocolate, cold and flu remedies, headache treatments and energy drinks (COT, 2001; MAFF, 1998). Most energy drinks contain approximately 80mg caffeine per 250ml can (1.1mg/kg bw/can for a 70kg adult) although drinks with smaller volumes and higher caffeine levels have appeared on the market in recent years.

9. Recently, a preliminary estimate of caffeine intake for the UK was derived from the rolling National Dietary and Nutrition rolling Survey (NDNS)\(^2\). The estimates are from the first two years of the survey and only apply to respondents who reported the consumption of foods containing caffeine in 4-day un-weighed diaries. The intake assessment was restricted to 20 NDNS food groups related to coffee, tea, cocoa, soft drinks and dietary supplements. The caffeine content was estimated from a variety of sources including information received from food manufacturers and previous MAFF (MAFF, 1998) and more recent FSA surveys (FSA, 2004). The mean total caffeine intake in men and women age 19-64 from NDNS was estimated at 130 and 122 mg/d respectively. The corresponding figures for older men and women in the 65+ age range were 143 and 131 mg/d respectively. Boys and girls in the age range of 11-18 years had lower intakes (46 and 44 mg/d respectively) (MRC, 2011). The recent NDNS intake estimates for caffeine and their compatibility with older exposure data derived previously for the UK population will be considered in detail once further details emerge from the MRC preliminary consideration.

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\(^2\) The NDNS provides detailed, quantitative information on food consumption, nutrient intakes, nutritional status and related characteristics. The NDNS is, in its current form, a four year rolling survey. The sample size for the survey is 500 adults and 500 children per year, covering all ages from 1½ years upwards living in private households. People living in institutions are not covered and pregnant and lactating women are also excluded. The survey includes boosted samples in Scotland, Wales and Northern Ireland to enable cross-country comparisons to be made. So far, only results from the first two years of the survey have been published (Department of Health, 2011).
Psychopharmacology and biochemistry of caffeine

10. Caffeine enters the brain quickly after absorption, and metabolism is variable with a half life ranging from 2 to 12 hours in healthy adults. Caffeine’s primary biologically relevant mechanism is through its action as a non-specific adenosine antagonist. Adenosine receptors are found throughout the body and adenosine acts presynaptically to inhibit neuronal release of several neural transmitters, reduces spontaneous firing of neurons, produces sedation and has anticonvulsant activity (Benowitz, 1990).

11. Adenosine is involved in a number of fundamental processes such as ATP related energy metabolism and RNA synthesis but is also released in response to metabolic stress and acts to protect the brain by suppressing neural activity and increasing blood flow through adenosine receptors (Latini and Pedata, 2001). A2A receptors are largely concentrated in the basal ganglia region and may be involved in the dopamine system (which is involved in reward and arousal). Adenosine may also be involved in the sleep-wake cycle (Basheer et al, 2004; Latini and Pedata, 2001). Caffeine has secondary effects that may not be related to adenosine, since caffeine is also a competitive non-selective phosphodiesterase inhibitor, allowing the build up of cyclic AMP in cells (Essayan, 2001).

12. In the central nervous system, caffeine acts primarily as a stimulant, increasing arousal and vigilance, reducing fatigue and decreasing motor reaction times in some tasks. In higher doses, caffeine can induce insomnia, anxiety, tremors, and seizures (Benowitz, 1990). The ADORAA2A 1083TT genotype of the adenosine A2A receptor has been associated with lower caffeine intakes suggesting a genetic link to the degree of caffeine consumption (Cornelis et al, 2007).

13. Studies carried out in adults showed improvements in aerobic endurance, anaerobic performance, choice reaction time, concentration and memory following consumption of an energy drink (80 mg caffeine or 1.1 mg/kg bw for a 70 kg adult; (Alford et al, 2001)) or (0.58, 1.70 and 1.75mg/kg bw (Howard and Marczinski, 2010)) compared to controls consuming a dummy energy drink or water. Following a mental depletion task (designed to require significant concentration on a number of tasks at one time), caffeine was found to increase aggression compared to placebo, but no effects were observed in groups who did not undertake the mental depletion task (Denson et al, 2011).

14. Metabolism of caffeine takes place through 3 main pathways shown in Figure 2, with the percentages indicating the mean proportion of caffeine metabolised to a particular metabolite. Some of the metabolites of caffeine pictured below also have pharmacological activity (Casarett et al, 1996).
Consumption of alcohol

15. Alcohol is widely consumed in the UK with at least one alcoholic drink being consumed in the week prior to interview by 68% of men and 54% of women in the UK during 2009 according to the General Lifestyle Survey carried out by the Office of National Statistics. In the same report, mean weekly consumption of alcohol was 16.3 units for men and 8.0 units for women in the 12 months prior to interview, equivalent to 2.33 g/kg bw for an average 70kg man and 1.33 g/kg bw for an average 60kg woman (Office of National Statistics, 2009). However, these data are for the total population including those who do not drink alcoholic beverages and therefore mean consumption by consumers will be higher. The proportion of adults exceeding recommended limits of 4 units (32 g alcohol\(^3\)) in one day in the week prior to interview was reported to be 37% for men and 3 units (24 g alcohol) in one day in the week prior to interview was 29% for women.

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\(^3\) [www.drinkaware.co.uk](http://www.drinkaware.co.uk). One unit of alcohol equals 8g pure alcohol.
Psychopharmacology and biochemistry of alcohol

16. Alcohol is rapidly absorbed from the stomach and intestine and distributed widely through simple diffusion from blood into tissues. Alcohol is converted to acetaldehyde primarily through the action of alcohol dehydrogenase (ADH) using the co-enzyme nicotinamide adenine dinucleotide (NAD). Acetaldehyde is then converted to the corresponding carboxylic acid through the action of the NAD-dependent enzyme acetaldehyde dehydrogenase (AcDH) which is then released from the liver and oxidised peripherally (Casarett et al, 1996) (see Figure 3).

![Figure 3: Metabolism of ethanol](image)

17. It is thought that behaviour is governed by two distinct systems: one that activates a response and one that inhibits a response. The impaired ability to inhibit a response under the influence of alcohol has received considerable attention because of the social implications of excessive consumption (Marczinski and Fillmore, 2003). Doses of 0.62 g/kg bw absolute alcohol (43.4 g for a 70 kg adult) have been shown to reduce response inhibition under laboratory conditions (Fillmore and Vogel-Sprott, 1999).

18. Alcohol is a central nervous system depressant and its mode of action has not been fully elucidated. It is understood that alcohol acts in the central nervous system (CNS) by binding to the GABA-A receptor, which mediates rapid inhibitory neurotransmission throughout the CNS. The outward signs of alcohol intoxication, such as impaired sensory and motor function, slowed cognition and stupefaction, are a result of this receptor binding activity (Kumar et al, 2009). It is not clear if the binding of the GABA-A receptor is directly responsible for the effects on response inhibition.

19. Studies looking at the effects of alcohol on attention tasks indicate that attention concentrated on a single source of information is not impaired by alcohol, but in divided attention tasks, especially those where two tasks follow each other closely, reaction time is increased (Moskowitz and Burns, 1971).

Health effects of co-consumption of alcohol with caffeine

20. In its opinions of 1999 and 2003, the SCF noted the existence of anecdotal reports of serious cardiac outcomes in young individuals but stated that these reports were incomplete and that consumption of energy drinks and alcohol often occurred in connection with other drugs; limiting the ability to draw conclusions. The Committee looked at a number of papers published since the SCF opinion including one on cardiac effects of co-consumption of alcohol and caffeine (Wiklund et al,
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2009). The Committee did not consider that these studies contributed significantly to the evidence base.

21. It has been suggested that the combination of energy drinks and alcohol may have several effects (Weldy, 2010):

- Carbonation tends to increase the absorption of alcohol (although some non-carbonated energy drinks are available, the majority of sales are of carbonated products)
- Diluted concentrations of alcohol are emptied from the stomach into the faster absorbing small intestine more quickly than higher concentrations.
- Caffeine keeps one awake and blunts the sedative effects of alcohol
- Lengthened time awake theoretically allows greater consumption of alcohol before loss of consciousness
- At low blood alcohol levels, caffeine appears to decrease some of the physical and mental impairment resulting from alcohol. At higher blood alcohol levels no such effects are observed.
- Energy drink ingredients give the consumer a false sense of physical and mental competence and decrease the awareness of impairment

Higher caffeine intake is associated with higher alcohol intake

22. Four studies looking at the energy drink and alcohol consumption of university students in the US and Canada, showed a relationship between energy drink consumption and alcohol consumption. Some of these studies looked into alcohol related incidents and showed that high consumers of both energy drinks and alcohol were at greater risk from a range of alcohol related consequences than consumers of alcohol alone (Arria et al, 2011) (O'Brien et al, 2008; Price et al, 2010; Velazquez et al, 2011). One Canadian study showed a significant association between energy drink and alcohol consumption with risk taking including consumption of illicit drugs (Brache and Stockwell, 2011). Caffeine consumption in 12-13 year olds in high school was significantly associated with the use of caffeine, nicotine and alcohol a year later (Collins et al, 2011).

23. In a field study of bar patrons, individuals who consumed alcohol mixed with energy drinks were at a 3-fold increased risk of leaving a bar highly intoxicated as well as a 4-fold increased risk of intending to drive compared to other drinking patrons who did not consume alcohol mixed with energy drinks (Thombs et al, 2010).

24. The Committee concludes that there does seem to be a correlation between high energy drink consumption and high alcohol consumption but limitations in the study designs do not indicate whether or not this relationship is causal. Members noted that the majority of studies were carried out in college students and little work had been carried out in lower socio-economic groups.

Does caffeine counteract the neuro-cognitive effects of alcohol consumption?

25. There is some evidence that caffeine can ameliorate some of the effects of alcohol but the mechanisms for this activity are still unclear. There are also several studies that discount this hypothesis. In a review of the data up until 1988, the authors stated that the variation in dose of both caffeine and alcohol along with
performance indicators used and study design did not allow simple conclusions to be
drawn in this area (Fudin and Nicastro, 1988).

26. A number of studies have been published since the SCF opinion of 2003
investigating the effect of alcohol and caffeine consumption on various aspects of
neurological function. The doses ranged from 1.2 – 5.6 mg/kg bw caffeine and 0.18
– 1.07 g/kg bw alcohol. Many of these studies have used driving simulators and
doses of approximately 2-3 cups of coffee and 1-2 standard measures of vodka.
Results were mixed, with some studies concluding that caffeine did not antagonise
the physiological effects of alcohol and other studies showing that some key aspects
of alcohol intoxication were ameliorated, especially motor reaction time, mean
tracking performance and memory reaction time. In some studies the perception of
degree of intoxication appeared to be altered by caffeine with individuals consuming
alcohol and caffeine perceiving themselves to be less intoxicated that those
consuming an equivalent amount of alcohol alone (Azcona et al, 1995) (Burns and
Moskowitz, 1990) (Ferreira et al, 2006) (Ferreira et al, 2004) (Fillmore and Vogel-
Sprott, 1999) (Kerr et al, 1991) (Marczinski and Fillmore, 2006) (Marczinski and

27. In conclusion, the range of neurological end-points measured and protocols
used makes direct comparisons difficult; a meta-analysis of these studies would be
useful.

*Expectation of effect may be related to actual effect*

28. The Committee considered that there is evidence to suggest that the
expectation of behaviour of individuals following consumption of alcohol and/or
caffeine can influence their actual behaviour (Fillmore and Vogel-Sprott, 1995)
(Harrell and Juliano, 2009); but concluded that it is not clear whether the combination
of alcohol and caffeine are able to trigger a pre-disposition to a particular behaviour.

*“Polysubstance use” and genetic factors*

29. There has been some suggestion that high caffeine use may be a marker for
the use of other drugs, legal or not, and other addictive behaviours such as
excessive gambling or internet use (Kaminer, 2010; Pallanti et al, 2006) (Arria et al,
2010) (Istvan and Matarazzo, 1984). Some studies using a cohort of male and
female mono- and dizygotic twin pairs, looking at caffeine, alcohol and drug
consumption along with smoking and a range of disorders including depression,
anxiety, panic and anti-social behaviour, found the relationship between high
caffeine use and high alcohol consumption to be dependent on familial factors, which
were primarily genetic. Modelling identified two genetic factors – one linked to illicit
drug use and one to legal drug use including caffeine and alcohol (Kendler et al,

30. In another cohort of male di- and monozygotic twins, heavy consumption of
alcohol and heavy smoking were significantly associated, and heavy smoking and
heavy coffee consumption were significantly associated, whereas heavy coffee
consumption and heavy alcohol consumption were not (Swan et al, 1997).
Comparisons between di- and monozygotic twins showed that there was a
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statistically significant likelihood that the extent of individual consumption of coffee and alcohol had a genetic basis and that using a number of models to determine the best fit, co-consumption of alcohol, coffee and nicotine were also found to have a genetic basis (Swan et al, 1996).

31. In studies in patients who had voluntarily sought treatment for substance abuse disorders, combined caffeine, nicotine and alcohol consumption were found to be correlated, but not caffeine and nicotine and other substances of abuse such as heroin, cannabis and glue (Amit et al, 2004; Kozlowski et al, 1993).

Case reports following consumption of caffeine alone or in combination with alcohol

32. The National Programme on Substance Abuse Deaths has identified eight case studies in the UK through a literature search, in 7 of which the coroner has named caffeine alone (5 cases) or in combination with alcohol (3 cases) as a contributing factor to death. In the final case report, the parents had linked the death to caffeine consumption (Corkery, 2012). One study in the peer reviewed literature describes acute renal failure following consumption of 3 litres of energy drink mixed with vodka (Schoffl et al, 2011).

33. Analysis of phone calls to the New South Wales Poisons Information Service over a 7 year time period revealed that of 297 calls related to caffeinated energy drinks, 73% were a result of recreational exposure to energy drinks and the median age was 17 years. Co-ingestion of other substances was reported in 46% of calls relating to recreational exposure with the most popular substances to be co-ingested being alcohol (23%) and other caffeine containing products such as cola and caffeine tablets (20%). Signs of serious toxicity such as hallucinations, seizures and cardiac ischemia were described in 21 calls. Of the callers, 128 people sought or were advised to seek urgent medical attention of which 57 had not co-consumed other substances (Gunja and Brown, 2012).

34. The Committee noted the above case reports but concluded that it is not possible to determine if there is a causal relationship.

Conclusions

35. The increasing consumption of drinks containing caffeine mixed with alcohol has raised concerns over the physical and mental health effects of this combination of psychoactive substances. The phenomenon known as "wide awake drunk" has been described where the stimulatory effects of caffeine prevent the consumer from realising how intoxicated they are, with the potential for increased toxicological damage and adverse behavioural effects occurring including increased risk taking, violence and criminal activity.

36. A body of work has looked into the physiological effects of the combination of caffeine and alcohol. Results were mixed, with some studies concluding that caffeine did not antagonise the physiological effects of alcohol and other studies showing that some key aspects of alcohol intoxication were ameliorated, especially
motor reaction time, mean tracking performance and memory reaction time. In some studies the perception of degree of intoxication appeared to be altered by caffeine with individuals consuming alcohol and caffeine perceiving themselves to be less intoxicated that those consuming an equivalent amount of alcohol alone.

37. Surveys carried out in college-aged adults showed an increased propensity for risk taking in groups who consume higher levels of caffeine and alcohol combinations compared to those consuming lower levels of these substances. Expectation of effect may shape the subjective and behavioural response to alcohol and to caffeine.

38. A number of studies have looked at the use of caffeine and how this is associated with the use of other substances of abuse. There seems to be an association between severity of alcoholism and caffeine and nicotine use. Analysis of studies in monozygotic twins has shown that these effects do not appear to be causal with familial factors, partly genetic, predisposing for a higher intake of caffeine and dependence. Excessive consumption of caffeine also appears to be associated with an increased risk for a wide range of psychiatric disorders.

39. The COT concluded that the evidence suggests a correlation between high caffeine consumption and high alcohol consumption but limitations in the study designs do not allow conclusions on whether or not this relationship is causal. The range of neurological end-points measured and protocols used makes direct comparisons difficult. A meta-analysis of studies on the neuro-cognitive effects of alcohol and caffeine consumption would be useful. The majority of studies were carried out in college students and little work had been carried out in lower socio-economic groups.

40. The Committee concluded that there is evidence to suggest that the expectation of a behavioural effect following consumption of alcohol and/or caffeine can influence the actual behaviour. It is not clear whether the combination of alcohol and caffeine are able to trigger a pre-disposition to a particular behaviour. From the available case reports it is not possible to determine if the relationship is causal.

Secretariat
June 2012
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COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT (COT)

First draft statement on the interaction of caffeine and alcohol and their combined effects on health and behaviour

Annex Two: Search Criteria and databases used

As the Scientific Committee on Food (SCF) looked at alcohol and caffeine interactions in 2003, only references published after this time have been included in the literature review. Some references that were not included by the SCF but published prior to 2003 have come to light through searching the reference lists of published papers. When considered relevant, these have been included in this paper. Due to the availability of human studies, most animal studies have not been included in the paper unless considered extremely relevant.

Searches using Pubmed

Caffeine, alcohol, behaviour (limits 01/01/2003-present)
Caffeine, alcohol, interactions (limits 01/01/2003-present)
Energy drinks, alcohol, behaviour (limits 01/01/2003-present)
Energy drinks, alcohol, interactions (limits 01/01/2003-present)
Caffeine, alcohol, behaviour (limits 01/01/2003-present; human studies only)
Caffeine, alcohol, interactions (limits 01/01/2003-present; human studies only)
Energy drinks, alcohol, behaviour (limits 01/01/2003-present; human studies only)
Energy drinks, alcohol, interactions (limits 01/01/2003-present; human studies only)

Searches using Google Scholar

All in title: Caffeine, alcohol, (NOT rat, mice) (since 2003, articles excluding patents)
All in title: “Energy drinks”, alcohol, (NOT rat, mice) (since 2003, articles excluding patents)

Secretariat
March 2012
COMMITTEE ON TOXICITY OF CHEMICALS IN FOOD, CONSUMER PRODUCTS AND THE ENVIRONMENT (COT)

First draft statement on the interaction of caffeine and alcohol and their combined effects on health and behaviour

Comments received from Professor Peter Rogers and Dr Angela Attwood in response to the discussion paper entitled “The interaction of caffeine and alcohol and their combined effects on health and behaviour” (TOX/2012/10)

The contents of this annex are restricted and will not be available on the COT website.

Secretariat
June 2012