Energy drinks and atrial fibrillation

Atrial fibrillation in healthy adolescents after highly caffeinated beverage consumption: two case reports.

Abstract

INTRODUCTION:
Energy drinks and highly caffeinated drinks comprise some of the fastest growing products of the beverage industry, often targeting teenagers and young adults. Cardiac arrhythmias in children related to high caffeine consumption have not been well described in the literature. This case series describes the possible association between the consumption of highly caffeinated drinks and the subsequent development of atrial fibrillation in the adolescent population.

CASE PRESENTATIONS:
We report the cases of two Caucasian adolescent boys of 14 and 16 years of age at the time of presentation, each without a significant cardiac history, who presented with palpitations or vague chest discomfort or both after a recent history of excessive caffeine consumption. Both were found to have atrial fibrillation on electrocardiogram; one patient required digoxin to restore a normal sinus rhythm, and the other self-converted after intravenous fluid administration.

CONCLUSION:
With the increasing popularity of energy drinks in the pediatric and adolescent population, physicians should be aware of the arrhythmogenic potential associated with highly caffeinated beverage consumption. It is important for pediatricians to understand the lack of regulation in the caffeine content and other ingredients of these high-energy beverages and their complications so that parents and children can be educated about the risk of cardiac arrhythmias with excessive energy drink consumption.

Sources of sexual inequality in the STEM fields

Sex differences on g and non-g intellectual performance reveal potential sources of STEM discrepancies
Abstract
The analysis of sex differences in cognitive abilities is largely confusing because these differences are masked by the pervasive influence of the general factor of intelligence (g). In this study a battery of five reasoning tests (abstract [AR], numerical [NR], verbal [VR], mechanical [MR], and spatial [SR]) was completed by a sample of 3233 young and old adolescents representative of the population. Using a latent variable approach, mean differences on the general factor were estimated after examining measurement invariance. Results show that the difference, favoring boys in latent g increases with age from two to four IQ points. Further, boys outperform girls in all the subtests and the observed differences were generally explained by g. However, mechanical reasoning is a systematic and strong exception to this finding. For the young adolescents, the observed difference in MR is equivalent to 10 IQ points, and this difference increases to 13 IQ points for the old adolescents. Only 1 (young) or 2 (old) IQ points of the sex difference in MR can be accounted for by g. The findings suggest that the persistent – and usually neglected average large advantage of boys in mechanical reasoning (MR) – orthogonal to g – might be behind their higher presence in STEM (science, technology, engineering, and math) disciplines. A new look at this relevant social issue is proposed in this study.

Highlights
► Sex differences in cognition are masked by the general factor of intelligence (g). ► The difference in favoring boys increases across adolescence up to 4 IQ points. ► Boys show an advantage in mechanical reasoning irrespective of latent g. ► This advantage might help to understand sex discrepancies in STEM disciplines.

Top cardiologists make $3 million a year

Top New York City Interventional Cardiologists Now Making $3 Million a Year

There’s no recession for top interventional cardiologists in New York City. Mt. Sinai’s Samin Sharma and New York-Presbyterian’s Jeffrey Moses now make at least $3 million a year, Sharma told CardioBrief.

Sharma will make his money staying in one place at Mt. Sinai. Moses, however, will have to travel between New York-Presbyterian’s uptown (Columbia University Medical Center) and east side (Weill
Cornell Medical Center) medical centers. New York-Presbyterian announced on Friday that Moses will now play an expanded role at New York-Presbyterian, serving as the director of interventional cardiology at both centers. Moses was previously located at the Columbia University site.

More power to them, and all that.

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**Calorie restriction monkeys fed 30% sugar**

This just boggles the mind. In an article in *Nature*, [Calorie restriction falters in the long run](http://www.nature.com/articles), we learn that calorie restriction effects on aging in monkeys may not be all that they’ve been cracked up to be. [A recent study](http://www.ncbi.nlm.nih.gov/pubmed/23154841) found no effect of calorie restriction on aging. In an earlier study, a connection was found. Why the difference?

In 2009, another study2, which began in 1989 at the Wisconsin National Primate Research Center (WNPRC) in Madison, concluded that caloric restriction did extend life in rhesus monkeys. The investigators found that 13% of the dieting group died from age-related causes, compared with 37% of the control group.

One reason for that difference could be that the WNPRC monkeys were fed an unhealthy diet, which made the calorie-restricted monkeys seem healthier by comparison simply because they ate less of it. The WNPRC monkeys’ diets contained 28.5% sucrose, compared with 3.9% sucrose at the NIA. Meanwhile, the NIA meals included fish oil and antioxidants, whereas the WNPRC meals did not. Rick Weindruch, a gerontologist at the WNPRC who led the study, admits: “Overall, our diet was probably not as healthy.”

I can barely imagine, in a study on longevity, feeding a diet that was almost 30% sugar. Shouldn’t that researcher lose his gerontologist’s license? Or is it just the case that, when the study started, they didn’t even think that that much sugar could be detrimental to the health of monkeys?
**Milk does a mind good?**

**Nations That Consume a Lot of Milk Also Win a Lot of Nobel Prizes**

Nations that consume a lot of milk and milk products also tend to have a lot of Nobel laureates among their populations, suggest the authors of a letter, published in Practical Neurology.

Research published last year in the New England Journal of Medicine reported a strong association between a nation’s chocolate consumption and Nobel laureate prowess, speculating that the flavonoid content of chocolate was behind the boost in brain power.

This got the letter authors thinking. As chocolate is often combined with milk, could it be the amount of milk/milk products consumed per head that fuels Nobel Prize success?

They looked at the 2007 data from the Food and Agriculture Organization on per capita milk consumption in 22 countries as well as the information provided by the author of the chocolate theory, and found a significant association.

Sweden has the most Nobel laureates per 10 million of its population (33). Although, it hosts the Nobel committee, which some might argue could introduce an element of bias; it also consumes the most milk per head of the population, getting through 340kg every year.

And Switzerland, which knocks back 300kg of the white stuff every year, has a Nobel haul of similar proportions (32).

At the other end of the scale, China has the lowest number of Nobel laureates in its population. But it also has the lowest milk consumption of the countries studied – at around 25kg a year.

There does seem to be a ceiling effect, however, note the authors, with no discernible impact beyond an annual per capita consumption of 350kg, as Finland’s Nobel haul seems to attest.

Is milk consumption therefore simply a reflection of a strong educational system, or do Nobel Prize winners celebrate by drinking it, query the authors?

But there is a plausible biological explanation for the link: milk is rich in vitamin D, and this may boost brain power, the evidence suggests.

“So to improve your chances of winning Nobel prizes you should not only eat more chocolate but perhaps drink milk too: or strive for synergy with hot chocolate,” conclude the authors, who highlight
their conflicts of interest, which include a tendency to take milk with cereal and coffee, and to eat chocolate whenever the opportunity arises.

Sleeping pills and death

Hypnotics’ association with mortality or cancer: a matched cohort study

Daniel F Kripke1, Robert D Langer2, Lawrence E Kline1
+ Author Affiliations

Abstract
Objectives An estimated 6%–10% of US adults took a hypnotic drug for poor sleep in 2010. This study extends previous reports associating hypnotics with excess mortality. [...]

Subjects Subjects (mean age 54 years) were 10 529 patients who received hypnotic prescriptions and 23 676 matched controls with no hypnotic prescriptions, followed for an average of 2.5 years between January 2002 and January 2007.

Main outcome measures Data were adjusted for age, gender, smoking, body mass index, ethnicity, marital status, alcohol use and prior cancer. Hazard ratios (HRs) for death were computed from Cox proportional hazards models controlled for risk factors and using up to 116 strata, which exactly matched cases and controls by 12 classes of comorbidity.

Results As predicted, patients prescribed any hypnotic had substantially elevated hazards of dying compared to those prescribed no hypnotics. For groups prescribed 0.4–18, 18–132 and >132 doses/year, HRs (95% CIs) were 3.60 (2.92 to 4.44), 4.43 (3.67 to 5.36) and 5.32 (4.50 to 6.30), respectively, demonstrating a dose–response association. HRs were elevated in separate analyses for several common hypnotics, including zolpidem, temazepam, eszopiclone, zaleplon, other benzodiazepines, barbiturates and sedative antihistamines. Hypnotic use in the upper third was associated with a significant elevation of incident cancer; HR=1.35 (95% CI 1.18 to 1.55). Results were robust within groups suffering each comorbidity, indicating that the death and cancer hazards associated with hypnotic drugs were not attributable to pre-existing disease.

Conclusions Receiving hypnotic prescriptions was associated with greater than threefold increased hazards of death even when
prescribed <18 pills/year. This association held in separate analyses for several commonly used hypnotics and for newer shorter-acting drugs. Control of selective prescription of hypnotics for patients in poor health did not explain the observed excess mortality.

### Antidepressant effect of resveratrol

**Antidepressant-like effect of trans-resveratrol: Involvement of serotonin and noradrenaline system.**

Abstract

The antidepressant-like effect of trans-resveratrol, a phenolic compound present in Polygonum cuspidatum, was evaluated through behavioral and neurochemical methods. trans-Resveratrol (20, 40 and 80 mg/kg, via gavage) significantly decreased the immobility time in mouse models of despair tests, but did not influence locomotor activity. Two behavioral models and neurochemical assays suggested that trans-resveratrol produced a significant increase in serotonin and noradrenaline levels at 40 or 80 mg/kg in brain regions. In addition, trans-resveratrol dose dependently inhibited MAO-A activity. These findings indicate that the antidepressant-like effect of trans-resveratrol might be related to serotonergic and noradrenergic activation.

### IBS and sensitivity to wheat

**Nonceliac IBS Might Be “Wheat Sensitivity”**

Some patients with irritable bowel syndrome but without celiac disease improved on a wheat-free diet. After a brief look at the research, we come across this:

Comment: As an allergist, I am hesitant to recommend diet avoidance without objective evidence of food allergy or celiac disease. Although this study suggests that many IBS patients actually might have gluten sensitivity that improves with a gluten-free diet, the study was retrospective, and 15% of patients reported worsening during their placebo challenge (personal communication from the
This is just short of insane, although the doctor more or less contradicts himself in the first and last sentences. Why hesitate? No harm whatsoever is done by avoiding wheat, and it may even be an actual cure, rather than ongoing medication or, God forbid, surgery. Unfortunately his attitude is all too common among the medical establishment.

Most patients aren’t going to know that dietary treatment might work unless their doctor tells them. But this doctor “hesitates” to tell them because… it might not work?

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The Art and Science of Low Carbohydrate Performance

Just read this book the other day, The Art and Science of Low Carbohydrate Performance, by Jeff Volek and Stephen Finney. Volek is a prominent dietary researcher, as well as an athlete, and Phinney is a physician who has done original research on ketogenic diets. The book is a follow-up to their previous book, The Art and Science of Low carbohydrate Living, which I also read and is very worthwhile. The present book is in Kindle format for 6 bucks.

The book addresses questions and issues for athletes who want to eat a low carb, ketogenic diet. The current dogma is that athletes must consume high amounts of carbs. The dogma also states that at high levels of intensity in exercise, cells burn chiefly carbohydrates. Well, they do if one eats a high carb diet. The authors demonstrate that on a VLCKD (very low carb ketogenic diet), cells adapt to fat burning and, after a period of time to allow for “keto-adaptation”, typically 2 or more weeks, athletes on this diet can perform as well or better than others.

The authors discuss the importance of mineral replacement, esp. of sodium and potassium, the lack of which has typically hindered performance on the VLCKD in the past. The body handles these minerals differently when carbs are nearly absent from the diet. (A VLCKD contains under 50 grams carb daily.)

One issue I would have liked to see the authors address is for bodybuilders, among whom current dogma states that carbs must be eaten to gain muscle. Research shows that the added insulin spike from carbs does not produce any
further growth than that produced by protein alone. OTOH, bodybuilders do like to go very low carb to shed fat.

All in all, quite a good book that can be read in a couple of hours.

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**Synthetic fecal transplants**

**Stool substitute transplant therapy for the eradication of Clostridium difficile infection: ‘RePOOPulating’ the gut**

**Abstract**

**Background**

Fecal bacteriotherapy (‘stool transplant’) can be effective in treating recurrent Clostridium difficile infection, but concerns of donor infection transmission and patient acceptance limit its use. Here we describe the use of a stool substitute preparation, made from purified intestinal bacterial cultures derived from a single healthy donor, to treat recurrent C. difficile infection that had failed repeated standard antibiotics. Thirty-three isolates were recovered from a healthy donor stool sample. Two patients who had failed at least three courses of metronidazole or vancomycin underwent colonoscopy and the mixture was infused throughout the right and mid colon. Pre-treatment and post-treatment stool samples were analyzed by 16S rRNA gene sequencing using the Ion Torrent platform.

**Results**

Both patients were infected with the hyper virulent C. difficile strain, ribotype 078. Following stool substitute treatment, each patient reverted to their normal bowel pattern within 2 to 3 days and remained symptom-free at 6 months. The analysis demonstrated that rRNA sequences found in the stool substitute were rare in the pre-treatment stool samples but constituted over 25% of the sequences up to 6 months after treatment.

**Conclusion**

This proof-of-principle study demonstrates that a stool substitute mixture comprising a multi-species community of bacteria is capable of curing antibiotic-resistant C. difficile colitis. This benefit correlates with major changes in stool microbial profile and these changes reflect isolates from the synthetic mixture.

It worked. This seems important: *C. difficile* infection is quite common in hospitalized patients, and fecal transplants have been successful. But, they must be treated like any other transplant in that the donor must be tested
for HIV, hepatitis B and C, and others. With this synthetic transplant, doctors could have an off-the-shelf treatment available whenever needed. Costs could drop significantly.

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**Alternative autism treatments**

**A Review of Complementary and Alternative Treatments for Autism Spectrum Disorders**

**Abstract**

Given the severe and chronic problems associated with Autism Spectrum Disorders (ASD) and the limitations of available treatments, there exists a large public health need for additional interventions. As more parents are inquiring about complementary and alternative treatments (CATs), both parents and practitioners require up-to-date information about them and whether and how to integrate them into treatment. After presenting data on CAT usage patterns for ASD, we review 13 ingestible (i.e., orally administered) and 6 noningestible (i.e., externally administered) CATs for ASD. For each CAT we briefly describe its definition; rationale for use; current research support, limitations, and future directions; safety issues; and whether we currently recommend, not recommend, or find it acceptable for the treatment of ASD. We conclude this paper with recommendations for future research and ten clinical recommendations for practitioners.

From the summary:

4. Summary

Nineteen CATs were reviewed, including 13 ingestible and 6 noningestible CATs. Research on these CATs is extremely varied, ranging from case studies to double-blind, sham-controlled RCTs, with and without significant results. Their safety, easy of use, sensibility, and expense (SECS) also vary considerably. Currently, we would only recommend two ingestible and one noningestible CAT, melatonin and RDA/RDI multivitamin/mineral (for those with a limited diet and/or poor appetite), and massage therapy, respectively. However, the following CATs are considered acceptable and worth considering for a short, monitored trial, if conventional Txs for ASD and the two recommended CATs have been given a reliable trial and found ineffective. For ingestible CATs: B6 and magnesium, multivitamin/mineral (even without a restricted/idiosyncratic diet.
and/or poor appetite, as long as no ingredient is above tolerable limit), folic acid, omega-3, L-Carnosine, probiotics and GI medication (only for ASD patients with GI symptoms), iron supplementation (only for those with low serum ferritin), and chelation (only for those with confirmed heavy metal toxicity). For noningestible CATs: Acupuncture, exercise, music therapy, and animal-assisted therapy.

Although published after our literature search, we feel it is important to mention N-Acetylcysteine (NAC) as an ingestible CAT that has great potential. NAC is a glutamatergic modulator and antioxidant and was recently examined in a 12-week, double-blind, randomized, placebo-controlled study in children with autistic disorder [116]. Thirty-three, 3–10 year-olds were randomized and NAC was initiated at 900 mg daily for 4 weeks, then 900 mg twice daily for 4 weeks and 900 mg three times daily for 4 weeks. Oral NAC was well tolerated with limited side effects and compared with placebo, resulted in significant improvements on the ABC irritability subscale (; ; ).

Is food addiction a cause of obesity?

Is food addiction a valid and useful concept? Short answer: probably not.

Short answer: probably not.

In this paper, we consider the concept of food addiction from a clinical and neuroscientific perspective. Food addiction has an established and growing currency in the context of models of overeating and obesity, and its acceptance shapes debate and research. However, we argue that the evidence for its existence in
humans is actually rather limited and, in addition, there are fundamental theoretical difficulties that require consideration. We therefore review food addiction as a phenotypic description, one that is based on overlap between certain eating behaviours and substance dependence. To begin, we consider limitations in the general application of this concept to obesity. We share the widely held view that such a broad perspective is not sustainable and consider a more focused view: that it underlies particular eating patterns, notably binge eating. However, even with this more specific focus, there are still problems. **Validation of food addiction at the neurobiological level is absolutely critical, but there are inconsistencies in the evidence from humans suggesting that caution should be exercised in accepting food addiction as a valid concept.** We argue the current evidence is preliminary and suggest directions for future work that may provide more useful tests of the concept.

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**Low-carb research round up**

Dr. Steve Parker has an excellent round-up of recent research on the efficacy and health benefits of low-carb and Mediterranean style diets, [here](#).

Added: Dr. Parker also has a post detailing why saturated fat does not cause heart disease, well worth reading.

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**Accuracy of Body Mass Index**

*Accuracy of Body Mass Index to Diagnose Obesity In the US Adult Population*

**Abstract**

**Background**

Body mass index (BMI) is the most widely used measure to diagnose obesity. However, the diagnostic accuracy of BMI to detect excess in body adiposity is largely unknown.

**Methods**

A cross-sectional design of 13,601 subjects (age 20–79.9 years; 48%
Results

BMI-defined obesity (≥ 30 kg/m²) was present in 21% of men and 31% of women, while BF % -defined obesity was present in 50% and 62%, respectively. A BMI ≥ 30 had a high specificity (95% in men and 99% in women), but a poor sensitivity (36% and 49%, respectively) to detect BF % -defined obesity. The diagnostic performance of BMI diminished as age increased. BMI had a good correlation with BF % in men (R² = 0.44) and women (R² = 0.71), but also with lean mass (R² = 0.50 and 0.55, respectively).

Conclusions

Despite the good correlation between BMI and BF %, the diagnostic accuracy of BMI to diagnose obesity is limited, particularly for individuals in the intermediate BMI ranges. A BMI cut-off of ≥ 30 kg/m² has a good specificity but misses more than half of people with excess fat. These results help to explain the U and J-shape association between BMI and outcomes.

Could this have something to do with the recent “revelation” that obesity is not generally bad for health? I think so. BMI-defined obesity missed many of the BF-defined obese. In other words, many of those with lower BMIs are actually obese, thus their health is statistically the same as the BMI-defined obese.

BCAAs and anxiety

Branched-chain amino acids alter neurobehavioral function in rats.

Obesity is associated with mood disorders, but underlying mechanisms are not well understood. We have recently described a strong association of branched-chain amino acids (BCAA) and aromatic amino acids (AAA) with obesity and insulin resistance. In the current study, we have investigated the potential impact of BCAA on behavioral functions. We demonstrate that supplementation of either a high sucrose or a high fat diet with BCAA induces anxiety-like behavior in rats compared to control groups fed on
unsupplemented diets. These behavioral changes are associated with a significant decrease in the concentration of tryptophan (Trp) in brain tissues and a consequent decrease in serotonin, but no difference in indices of serotonin synaptic function. The anxiety-like behaviors and decreased levels of Trp in the brain of BCAA-fed rats were reversed by supplementation of Trp in the drinking water, but not by administration of fluoxetine, a selective serotonin reuptake inhibitor, suggesting that the behavioral changes are independent of the serotonergic pathway of Trp metabolism. Instead, BCAA supplementation lowers the brain levels of another Trp-derived metabolite, kynurenic acid, and these levels are normalized by Trp supplementation. We conclude that supplementation of high energy diets with BCAA causes neurobehavioral impairment. Since BCAA are spontaneously elevated in human obesity, our studies suggest a potential mechanism for explaining the strong association of obesity and mood disorders.

This study used rats and an unknown (to me) amount of BCAAs, so its validity to weightlifters and other humans who supplement with BCAAs is unknown. However, based on some other studies I’ve read, relatively small amounts of BCAA could do this. For instance: The effects of a branched chain amino acid mixture supplemented with tryptophan on biochemical indices of neurotransmitter function and decision-making.

We have previously shown that a 60-g mixture of branched chain amino acids (BCAAs) lowers the plasma availability of the catecholamine precursors tyrosine (TYR) and phenylalanine (PHE) and produces biochemical and neuropsychological changes consistent with impaired dopamine neurotransmission. However, the BCAA mixture also lowers the ratio of tryptophan (TRP) to BCAA which could impair brain serotonin function.

OBJECTIVES:
To determine the biochemical and neuropsychological effects of a BCAA mixture supplemented with TRP.

METHODS:
We studied 32 healthy volunteers who were randomly and blindly allocated to either a single administration of amino acid mixture (60 g BCAA and 2 g TRP) or placebo. We carried out venous sampling to measure plasma levels of amino acids and performed selected cognitive tasks sensitive to monoamine manipulation 5 h after mixture ingestion.

RESULTS:
Relative to placebo, the BCAA/TRP mixture substantially lowered the ratio of TYR+PHE:BCAA and increased plasma prolactin. The ratio of TRP:BCAA was also lowered but to a lesser extent. The BCAA/TRP mixture produced significant changes in a task of decision-making where volunteers showed reduced discrimination between gambles with
CONCLUSIONS:
A 62 g BCAA/TRP mixture decreases the availability of TYR and PHE for brain catecholamine synthesis and increases plasma prolactin consistent with lowered brain dopamine function. Addition of 2 g TRP to the 60 g BCAA mixture does not prevent a reduction of the ratio TRP:BCAA relative to placebo. The effects of the BCAA/TRP mixture on decision-making suggest a general action of dopamine pathways on the processing of emotional information in risky choice, including punishment-related cues, consistent with suggestions that dopamine mechanisms mediate behavioural responses to aversive as well as appetitive stimuli in instrumental conditioning.

One might want to be careful when supplementing with BCAAs.