

This is the peer reviewed version of the following article:

Influence of energy drinks on obesity: a preliminary experimental study / Mattioli, Anna Vittoria; Pennella, Sonia; Manenti, Antonio; Ballerini Puviani, Matteo; Farinetti, Alberto. - In: PROGRESS IN NUTRITION. - ISSN 1129-8723. - 19:4(2017), pp. 369-372. [10.23751/pn.v19i4.6438]

Terms of use:

The terms and conditions for the reuse of this version of the manuscript are specified in the publishing policy. For all terms of use and more information see the publisher's website.

23/04/2024 09:40

(Article begins on next page)

ORIGINAL ARTICLE

Influence of energy drinks on obesity: a preliminary experimental study

Anna Vittoria Mattioli¹, Sonia Pennella², Antonio Manenti¹, Matteo Ballerini Puviani², Alberto Farinetti¹

¹Surgical, Medical and Dental Department of Morphological Sciences related to Transplant, Oncology and Regenerative Medicine University of Modena and Reggio Emilia - E-mail: annavittoria.mattioli@unimore.it; ²Istituto Nazionale per le ricerche cardiovascolari, U.O. University of Modena and Reggio, Emilia, Modena, (Italy)

Summary. We performed an experimental research in Sprague-Dawley rats in order to evaluate the effects of different caffeinated beverages on obesity. Animals were divided in 4 groups and received beverages containing different concentration of caffeine: a commercial Energy Drink, a commercial Cola Soda, regular coffee and water. After 15 days we found that Energy Drink and Cola Soda induced body weight gain, on contrary sweetened coffee and water did not influence weight. Besides, an increased reactivity and motility was observed in the Energy Drink supplemented animals. Laboratory tests excluded obesity-correlated dysmetabolism. We supposed a central nervous action of some components of Energy Drink other than caffeine, even if their finest mechanisms are unknown. At long term, degeneration from a condition of body weight gain to obesity cannot be excluded.

Key words: obesity, energy drinks, caffeine

Introduction

In recent years the consumption of energy drinks (e.g. Red Bull®, Monster®, etc.) has increased constantly among young individuals (1-3) (Fig. 1).

The amount of caffeine varies widely in EDs and it is estimated between 80 to 114 mg/can (3).

Besides caffeine (with 80-160 mg/can), EDs contain several other psychoactive substances as taurin, ginseng, gluconolactone and guaranà, the concentration of which is often disparate and not well indicated (4).

The consumption of EDs among young persons is growing persistently and it has been recently linked with cardiac arrhythmia and young unexpected death (3, 5, 6) EDs are consumed by young because they enhance focus, attention and reactivity (7, 8). EDs do not show a great degree of toxicity when consumed

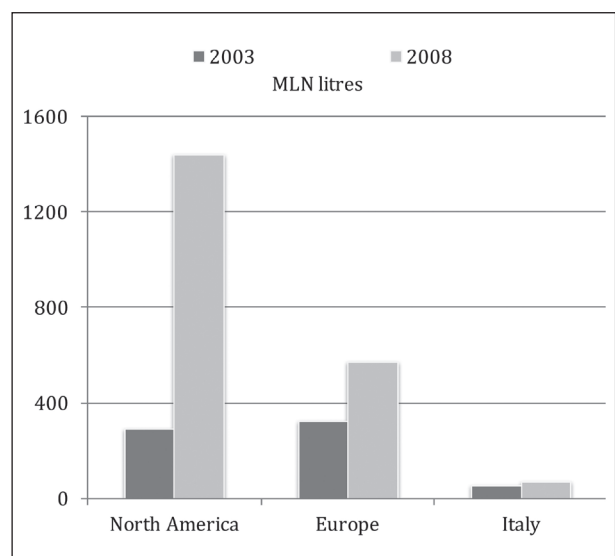


Figure 1.

by healthy subjects in moderate doses, however when consumed in high doses or together with alcoholic beverages, they may cause arrhythmia (3, 5, 6).

It is renowned that the reaction of individuals to caffeine consumption is variable. Caffeine, in fact, stimulates both the central and the peripheral nervous systems affecting the cardiocirculatory and breathing systems (8-11). Recently a paper from showed that caffeine can improve blood lipid and antioxidant levels, and effectively reduce rat serum leptin levels, inhibit the absorption of fatty acids, and markedly reduce the expression levels of the IL-6 and TNF- gene (12). Some effects of EDs are not known. The present study was design to evaluate the effects of different caffeinated beverages in an animal model.

Methods

We analyze the effect of EDs on body weight gain (BWG) and obesity comparing with different caffeinated beverages.

Forty Sprague-Dawley rats, weighting 150-170 g., were randomly divided into 4 groups. In addition to standard laboratory diet they were fed with different sweetened beverages containing caffeine and caffeine-like substances.

Group A: control group. Group B: a Commercial "Energy Drink" (ED) (80mg/500ml of caffeine and 110 Kcal for week). Group C: a commercial "Cola Soda" (34mg/ 500ml of caffeine plus 92 Kcal for week). Group D: commercial sweetened coffee (250 mg/500ml of caffeine plus 42 Kcal for week. The follow-up lasted 15 days.

The BWG of the animals was calculated every 5 days, and their behavior was daily observed. Changes in behavior were classified on the base of two parameters, strictly correlated each-other: "reactivity", that included energy, vivacity till to an over aggressive answer to nociceptive stimuli, and "motility" manifesting with an increased and quicker running and climbing up inside the cage. The following score was adopted:

Grade O: no change detectable; Grade 1: increased motility, and moderate reactivity; Grade 2:

Increased motility and reactivity with difficulty in manipulating the animals.

Normal laboratory diet consumption was measured every day in all the groups.

After 14 days animals were euthanized. Liver function, renal function, lipid, glucose and methaemoglobin were assessed. Liver and pancreas were macroscopically and histologically evaluated; thickness of white fatty of the subcutaneous space and inside the renal fascia was measured.

All animals received care in compliance with the European Convention on Animal Care. The same trained operator provided animal care in order to reduce stress. The Research Animal Care and Use Committee of our University approved the study.

Statistical analysis. SPSS software, version 14.0.1 (SPSS Inc., Chicago, Ill, USA), was used for statistical analysis. Comparison of data between groups was performed by ANOVA. The t-test was used to compare data within animals of the same group. $P < 0.05$ was considered statistically significant. All data are expressed as mean +SD.

Results

Animals treated with EDs (Group B) and Soda Cola (Group C) consumed the daily total amount of the supplemented beverages during the follow-up period. Animals treated with sweetened coffee (Group D) consumed the daily total amount of beverages during the first 6 days (mean 6 + 1 day) then tend to reduce intake at 70% of administered beverage.

At the end of the study, BWG was observed in Group B (+10%; $p < 0.01$), and in Group C (+5%; $p < 0.05$), while smaller changes were reported in Control Group (+2.50 %; $p = n.s.$) and in Group D (+2.72% $p = n.s.$). WE also reported an increase consumption of the normal laboratory diet in Group B and C, respectively + 15%, and +10%.

Behavioral changes were observed in Group B (EDs), and were scored as grade 1 in 2 animals, and grade 2 in 8 animals.

The fasting blood glucose test controls, and the other final metabolic parameters were in a normal range. Measures of the white fatty in the different groups did not demonstrate significant changes.

Histology did not show signs of organ damage;

in particular liver histology excluded signs of steatosis, typically found in obesity (13).

Discussion

The main finding of the present preliminary study is that animals supplemented with EDs and Soda Cola developed body gain weight as compared to animals treated with sweetened coffee and control group. Increase of body weight has been recently associated to sweetened beverages (14, 15).

We found that BWG was progressive, overcoming the small amount of carbohydrate calories added to the beverages, and can be correlated to an augmented appetite as shown by the increased consumption of the normal laboratory diet. This anabolic action of EDs and of Soda Cola can be referred to a digestive effect, direct or mediated, rather than to a simple caloric supplementation. We hypothesize a central neurologic stimulation, considering that caffeine and caffeine-like substances pass the blood-brain barrier (16-18). The disproportion between BWG and the greater caloric assumption can be explained considering the calories consumption, induced by the increased motility (19).

However the sole caffeine content in the EDs does not explain this effect, as demonstrated by the difference in BWG between group B and D that assumed sweetened coffee. The more vigorous action of EDs seems to be related to its composition that included several different substances, even if all the proper mechanisms of each component are not yet completely known (20-22). It well correlates with its neuro-motorial positive effect, which compensates a more caloric intake, directing the anabolic process to a prevalent muscular mass hypertrophy, assuring a metabolic compensation and avoiding an excessive fat accumulation through greater energy dissipation. The present mid-term study was tailored on the live expectancy of the animals; however we cannot exclude, at long-term, a possible degeneration from BWG to a real obesity.

The protocol allowed us to distinguish between a simple and physiological BWG and obesity. We also observed an ergogenic and positive metabolic of a prolonged EDs consumption, distinguishing it from

adverse effects, especially cardiac or neurological, often due to its acute overconsumption (1, 5, 23,24, 25). These data suggest more extended pharmacological researches on the different active components of EDs.

Acknowledgements

The authors would like to thank Molinari Caffè for providing the study material.

Funding

This research was supported by a grant from "Fondazione Vignola" (Italy)

References

1. Kumar GS, Park S, Onufrak S. Association between reported screening and counseling about energy drinks and energy drink intake among U.S. adolescents. *Patient Educ Couns*. 2014 Feb;94(2):250-4. doi: 10.1016/j.pec.2013.09.026.
2. Harris JL, Munsell CR. Energy drinks and adolescents: what's the harm? *Nutr Rev*. 2015 Apr;73(4):247-57. doi: 10.1093/nutrit/nuu061.
3. Avci S. Death of a young man after overuse of energy drink. *Am J Emerg Med* 2013;31:1624.
4. Flotta D. Consumption of Energy Drinks, alcohol and alcohol-mixed Energy Drinks among Italian adolescents. *Alcohol Clin Exp Res* 2014;38:1654.
5. Mc Lellan TM, Lieberman HR. Do Energy drinks contain active components other than caffeine? *Nutrition Rev* 2012; 70: 730-744.
6. Mattioli AV, Pennella S, Manenti A, Farinetti A. Energy drink overconsumption can trigger atrial fibrillation. *J Cardiovasc Med* 2016;17:902-904.
7. Emond JA, Gilbert-Diamond D, Tanski SE, Sargent JD. Energy drink consumption and the risk of alcohol use disorder among a national sample of adolescents and young adults. *J Pediatr* 2014; 165: 1194-1200.
8. Kumar G, Park S, Onufrak S. Perceptions about energy drinks are associated with energy drink intake among U.S. youth. *Am J Health Promot*. 2015 Mar-Apr;29(4):238-44. doi: 10.4278/ajhp.130820-QUAN-435. Epub 2014 Jan 24.
9. Mattioli A.V., Farinetti A., Miloro C., Pedrazzi. Influence of coffee and caffeine consumption on atrial fibrillation in hypertensive patients. *Nutr Metab Cardiovasc Dis*. 2011 Jun;21(6):412-7. Epub 2010 Feb 18.
10. Mattioli AV, Pennella S, Farinetti A. Lifestyle and atrial fibrillation *Progress in Nutrition* 2012; 14:87-99.
11. Mattioli AV, Miloro C, Pennella S, Pedrazzi P, Farinetti A. Adherence to Mediterranean diet and intake of antioxi-

- dants influence spontaneous conversion of atrial fibrillation. *Nutr Metab Cardiovasc Dis.* 2013 Feb;23(2):115-21. doi: 10.1016/j.numecd.2011.03.005.
12. Xu Y, Zhang M, Wu T, Dong Dai S, Xu J, Zhou Z. The anti-obesity effect of green tea polysaccharides, polyphenols and caffeine in rats fed with a high-fat diet. *Food Funct.* 2015 Jan;6(1):297-304. doi: 10.1039/c4fo00970c. Epub 2014 Nov 28.
 13. Beandrup Kristianse MN, Skovgard V, Rigbolt KTG, Sloth Tobol K, Roth JD, Jelesing J et al. Obese-diet induced mouse models of non-alcoholic steatohepatitis –tracking disease by liver biopsy. *World J Hepatol* 2016; 8: 673-684.
 14. Babwah TJ, Maharaj RG, Nunes P. Energy drinks and other dietary supplement use among adolescents attending secondary schools in Trinidad and Tobago. *Public Health Nutr.* 2014 Oct;17(10):2156-65. doi: 10.1017/S1368980013003339.
 15. Bahar Karadavut, Habibe ahin, Gül ah Kaner, Serhat Karadavut. Is beverage consumption associated with increased body weight among adolescents? *Progress in nutrition* 2017; 19:41-47.
 16. Schwartz MW, Woods SC, Porte D Jr, Seeley RJ, Baskin DG. Central nervous system control of food intake. *Nature* 2000; 404: 661-667.
 17. Kalin S, Heppner FL, Beckmann I, Prinz M, Tschop M, Yi CX. Hypothalamic innate immune reaction in obesity. *Nat Rev Endocrinol* 2015; 11: 339- 351.
 18. Mc Clelland J, Bozhilova N, Campbell I, Schmidt U. A systematic review of the effects of neuromodulation on eating and blood weight: evidence from human and animal studies. *Eur Eat Disorders Rev* 2013; 21: 436-455.
 19. Dungan CM, Li J, Williamson DL. Caloric restriction normalizes obesity-induced alterations on regulators of skeletal muscle growth signalling. *Lipids* 2016; Jun 11(Epub ahead of print).
 20. Gavrieli A, Karfopoulou E, Kardatou E, Spyreli E, Frago-poulou E, Mantzores CS, Yannakoulia M. Effects of different amounts of coffee on dietary intake and appetite of normal-weight and overweight/obese individuals. *Obesity* 2013; 21: 1127-1132.
 21. Huxtable RJ. Physiological action of taurine *Physiol Rev* 1992; 72: 101-163.
 22. Alsunni AA. Energy drink consumption: beneficial and adverse effects. *Int J Health Sciences* 2015;9: 46-474.
 23. Ali F, Rehman H, Babayan Z, Stapleton D, Joshi DD. Energy drink and their adverse health effects: a systematic review of the current evidence. *Potgrad Med* 2015; 17: 308-322.
 24. Pennella S, Mattioli AV. Energy drinks and atrial fibrillation in young adults. *Clin Nutr* (in press) 10.1016/j.clnu.2017.05.00.
 25. Mattioli AV. Effects of caffeine and coffee consumption on cardiovascular disease and risk factors. *Future Cardiol.* 2007 Mar; 3(2):203-12. doi: 10.2217/14796678.3.2.203.

Correspondence:

Anna Vittoria Mattioli, MD
 Department of Life Science-University of Modena and R.E.
 Via del pozzo, 71
 41100 Modena (Italy)
 Phone: 0039/59/4224043 Fax: 0039/59/4224323
 E-mail: annavittoria.mattioli@unimore.it