PEER REVIEW

FOOD ADDITIVES, PRESERVATIVES & COLORINGS

In 1000s of Peer Reviewed Reports

a Free Jeff Prager Publication

APRIL 2016

This free eMagazine uses peer reviewed reports from hundreds of medical journals representing many 100s of PhD clinicians, medical professionals and research scientists to confirm, and to do so without question, that a conventional diet can and usually does lead to more than 100 disease and disorder states in the human species. A conventional diet is a causative factor in most human illnesses. Eating an organic diet is a crucial component towards remaining disease-free as we age. It's a diet we should all have adhered to from birth, but we didn't know, did we?

TO SERVE MAN

At the Soylent Corporation our Prime Directive is to serve the needs of all mankind. That means YOU. And we take this responsibility seriously, especially with the extraordinary number of epidemics plaguing mankind—obesity, cancer, stroke, dementia, autism and more—far too many to list here. For those reasons we bring you the most advanced artificial formulations available in Soylent Yellow, Soylent Red and now, because we listen, we bring you Soylent Green! Our newest formulation is exactly what you've been asking for. No carbs, no protein, no nutrients at all, just pure, unadulterated artificially and laboratory created chemically inert food-stuff so you can eat all day, every day. Because we care!

From your friends at
SOYLENT CORPORATION

**Taste may vary from person to person*

PEER REVIEW

FOOD ADDITIVES, PRESERVATIVES & COLORINGS

And The Severe Health Damage Associated With A Conventional Diet

A Free Jeff Prager Publication created for Camy, Syrena and Illiana

This eBook was made possible through generous tax-free donations from the World Health Organization (WHO), the FDA, the CDC, Monsanto, Syngenta and other major multinational chemical manufacturing company's who collectively want to protect your health so you can avoid spending your hard earned money on a lifetime of health care. In particular I want to thank Monsanto, in advance, for their tireless, decades-long, multibillion dollar effort at producing organic fruits and vegetables and organic pesticides to clean up our earths environment (*which they will eventually be forced to do*), an ecosystem they and the others have usurped, ravaged and despoiled for more than a century. Thank you, all.

This eMagazine is free and is specifically designed for reading in full-screen mode using the arrow keys to turn the pages on a 21-inch monitor or larger. Some small parts or portions of this eMag may be parody or satire. Other, much larger parts, are an honest and tireless effort at telling the truth as I see it using the available peer reviewed literature. To be honest, I have a blast making these PDFs because for me it's a truly fun and exciting experience to both do the research and create the pages. Yet once they're finished I think everyone needs to know the same critically important information that I've learned. This is because my opinion, now molded, even armed, with 1000s of peer reviewed reports is in complete opposition to that of every single word we hear on the mainstream media.

So I believe these eMags really need to be freely accessible, don't you think so too? We're being lied to. Someone, in this case me, needs to compile and dispense the truth. My website is on the next-to-last page with some of my 70+ free eMagazines, images of some of their covers and brief descriptions with a link. Thank you again for downloading this eMagazine. I sincerely hope it helps.

Publishing the complete free eMagazine right around the first of May, 2016

Runaway Slaves LLC™

Food additives, preservatives and colorings were a time-bomb, and this one blew up decades ago. We're just now seeing the ever-so-slow explosion, watching as unwilling participants. The result? A horror show. Early death, disorders and the loss of proper brain function is the net result for us all. The majority of us ate these chemicals every single day. The majority of us still do. This eMagazine presents a massive collection of peer reviewed evidence to support the extraordinary harm that food additives are causing, not only to certain susceptible individuals, but to everyone that's eaten them. No one report or study of the more than 1000 reports in this volume verifies these concepts, no, it's purely and solely the totality of the evidence presented here that makes this all so uncomfortably true. And the evidence presented here is overwhelming. If you're eating a conventional diet of foods purchased at any grocers that's not 100% organic, you're eating yourself to a certain, painful and avoidable early death. Needlessly. The peer review proves it. The conventional food supply is poisoned. All of it.

~ Jeff Prager • April 2016 ~

THE FUTURE

By Leonard Cohen

Give me back my broken night
My mirrored room, my secret life
It's lonely here
There's no one left to torture
Give me absolute control
Over every living soul
And lie beside me, baby
That's an order!

Give me crack and anal sex
Take the only tree that's left
And stuff it up the hole
In your culture
Give me back the Berlin wall
Give me Stalin and St. Paul
I've seen the future, brother:
It is murder

Things are going to slide in all directions
Won't be nothing, won't be, nothing you can measure anymore
The blizzard, the blizzard of the world
Has crossed the threshold
And it has overturned
The order of the soul

When they said, they said repent,
Repent, repent
I wonder what they meant

You don't know me from the wind
You never will, you never did
I'm the little jew
Who wrote the Bible
I've seen the nations rise and fall
I've heard their stories, heard them all
But love's the only engine
Of survival

Your servant here, he has been told
To say it clear, to say it cold:

It's over, it ain't going
Any further
And now the wheels of heaven stop
You feel the devil's riding crop
Get ready for the future:
It is murder
Things are going to slide, slide in all directions
Won't be nothing, won't be nothing you can measure anymore
The blizzard, the blizzard of the world
Has crossed the threshold
And it has overturned
The order of the soul

When they said. they said repent, repent, repent, repent
I wonder what they meant

There'll be the breaking of the ancient western code
Your private life will suddenly explode
There'll be phantoms, there'll be fires on the road
And the white man dancing
You'll see your woman hanging upside down
Her features covered by her fallen gown
And all the lousy little poets coming round
Trying to sound like Charlie Manson
Yeah the white man dancing

Give me back the Berlin wall
Give me Stalin and St. Paul
Give me Christ
Or give me Hiroshima
Destroy another fetus now
We don't like children anyhow
I've seen the future, baby:
It is murder

Things are going to slide, slide in all directions
Won't be nothing, won't be nothing you can measure anymore
The blizzard, the blizzard of the world
Has crossed the threshold
And it has overturned
The order of the soul

When they said they said repent, repent, repent, repent
I wonder what they meant

If we as a society here in America continue to eat these laboratory manufactured foods filled with drugs of all sorts then Mr. Cohen might be quite right, the future will be nothing less than **murder.**

This eMagazine is NOT an advertisement vehicle for Michelob, Kelloggs, Hunts, Heinz, Wonder Bread, Natures Own, Coca Cola products, PepsiCo products or any of the other chemical laden, disease causing products pictured herein. These images are included to convey the extraordinary lengths taken regarding product design, shape, color, labeling, etc.
The corporate products pictured are the ones you should NOT buy.
The peer review and product ingredients listed show you why.
And with remarkable clarity.
Eat Organic, 100% Organic.

We may all have to use their worthless paper 'money'
But Don't Eat Their Food,
Don't Drink Their Water,
And Certainly Don't Buy Their Stuff

Food Additives, Preservatives, Flavorings, Colors and Emulsifiers And ALL Chemicals Added To Food And Drink

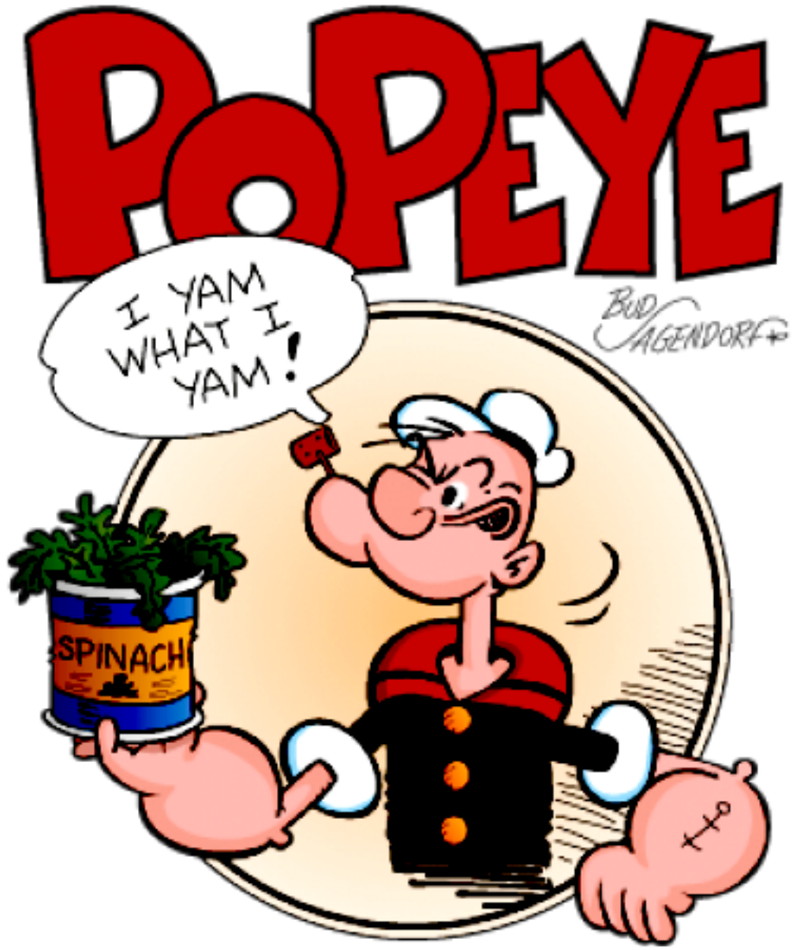
ARE DRUGS

This eMagazine contains 1000+ peer reviewed reports on DRUGS.

This eMagazine is about DRUGS

To be fair and honest this collection of peer review includes reports and studies addressing serious harm caused by drugs added to our food supply yet it also includes reports and studies on potential life-saving qualities that some of these food drugs may also have. Some food additives cause cancer and cure it, both. Eating them in our food causes cancer. Using them in infinitesimally small amounts therapeutically in the medical setting can have miraculous disease fighting effects.

You'll read about both.



MORE LOOPHOLE THAN LAW
The Food Additives Testing and Approval Process

Although consumers likely presume that a federal agency ensures the safety of ingredients in the food supply, in reality, this isn't the case.

First, many additives have not been thoroughly tested. And the vast majority of safety testing of food additives is done by food manufacturers (or by people hired by manufacturers), not the government or independent laboratories. Second, because of a loophole in the law, companies can declare on their own that an additive is "Generally Recognized As Safe" (GRAS), and start adding it to food without even informing the government. Such ingredients are required to be listed on labels although in some cases they appear simply as "artificial flavorings" or "artificial coloring".

The chapters on Povidone, a hospital and industrial disinfectant used since the 1930s, and Bisphenol A are examples of one of the latest ingredients to enter the food chain (Povidone) and one of the latest ingredients exiting (BPA). Povidone wasn't in food just a few years ago and I suspect, as the overwhelming supply of peer review mounts and public outcry builds, BPA will eventually be banned.

Some additives do undergo a more formal government approval process, but even that is no guarantee of safety. There are approved additives that have been shown in subsequent independent studies to harm health, and are in the "Avoid" category in Chemical Cuisine. But the FDA rarely reviews the safety of additives (including GRAS substances) once they enter the food supply.

BANNED ADDITIVES

The food and chemical industries have said for decades that all food additives are well tested and safe. This is not true. The history of food additives is riddled with additives that, after many years of use, were found to pose serious health risks and they are now banned. There are additives—drugs added to the food supply—that have been in use for 1, 2 and 3 decades or more and we're just now recognizing their harm medically, in the peer review. The moral of the story is that when someone says that all food additives are well tested and safe, they aren't. The peer review you're about to read proves this unequivocally.

THE BOTTOM LINE

If you regularly consume trans fats, food colorings, additives, preservatives, sweeteners, flavorings and the laboratory created chemicals added to our food supply, repeatedly and across decades you will develop diseases and disorders directly related to that diet. Bipolarism, neurological deficits in executive functioning, early onset dementia, cancers, heart and lung dysfunction and more diseases and disorders than can be listed here. They're described in the following 1000+ pages. Please heed the advice. This eMagazine was designed to motivate the reader to change her/his diet. I hope it works otherwise an early death is inevitable and my time, while not valuable, was wasted nevertheless.
Eat right and enjoy a long, healthy life!

> “The FDA’s reliance on industry studies in determining BPA’s safety must be re-evaluated in light of clear signs industry is willing to mislead the American people on this public-health issue.”

US Representative Bart Stupak, D-Michigan
Chairman of the House Energy and Commerce Committee’s
Oversight and Investigations Subcommittee

> “The makers of food additives, preservatives and colorings have been lying to us for decades about every additive known to man.”

Jeff Prager
Publisher & Activist

INGREDIENTS LIST
www.tensoda.com
on February 7, 2016

Canada Dry Ginger Ale Ten
Carbonated Water, **High Fructose Corn Syrup**, Citric Acid, Sodium Citrate, Malic Acid, **Sodium Benzoate** (Preservative), **Aspartame**, Natural Flavors, **Acesulfame Potassium, Caramel Color.**

A&W Root Beer Ten
Carbonated Water, **High Fructose Corn Syrup**, **Caramel Color, Sodium Benzoate** (Preservative), **Natural And Artificial Flavors**, **Aspartame**, **Acesulfame Potassium**, Malic Acid, Quillaia Extract.

7-Up Lemon Line Soda Ten
Filtered Carbonated Water, **High Fructose Corn Syrup**, Citric Acid, **Potassium Citrate, Potassium Benzoate** (Preservative), Natural Flavors, **Aspartame**, **Acesulfame Potassium, Calcium Disodium EDTA** (To Protect Flavor).

Sunkist Orange Soda Ten
Carbonated Water, **High Fructose Corn Syrup**, Citric Acid, **Sodium Citrate, Malic Acid, Sodium Benzoate** (Preservative), **Aspartame**, **Modified Food Starch**, Natural Flavors, **Acesulfame Potassium**, Caffeine, **Ester Gum, Yellow 6, Red 40**.

RC Cola Ten
Carbonated Water, **High Fructose Corn Syrup, Caramel Color, Phosphoric Acid, Potassium Citrate**, **Aspartame**, Natural And **Artificial Flavors, Potassium Benzoate** (Protects Flavor), **Caffeine**, Citric Acid, **Acesulfame Potassium**, **Acacia Gum, Sucralose**.

Not Listed On Labels
Bisphenol-A (BPA) is found lining most soda cans and is a component of the plastic bottles. It can and does leach into whatever’s in the container especially when it’s not kept cold—for example cases of soda stored in a warehouse or garage in the summer—or with acidic foods and drinks like tomato sauce or paste. As you’ll read in the peer review, we all have BPA in our blood, our breast milk and our urine and it has devastating effects at doses lower than those found in frequent users of canned and plastic containers. BPAs effects are dose dependent and regular users of BPA lined food products can expect both immediate and long term effects. Some effects are related to behavior and neurological deficits that are difficult to see or observe. It’s all in the peer review. All of the ingredients in **bold** above are either carcinogens, mutagens, neurotoxins or damaging to the health of the human species. You’re about to read the peer review on all of them and considerably more.

AN INTRODUCTION TO THE PEER REVIEW

by Jeff Prager

The pages of this eMagazine are divided into chapters for each of dozens of the most frequently used food additives. Additives included are additives that are, or are likely to be, mutagens, carcinogens, endocrine disruptors , neurotoxins or any other type of harmful industrial processed food additive. Often times these additives are harmful in much lower doses than one might expect and they can be profoundly harmful even in the very small doses in the foods they're found in. Some of us are consuming these various chemicals every single day and the peer review does not look good for those maintaining that style of conventional diet.

Genetics, lifestyle, stress factors and several other important components make up the complete gamut of elements that affect our health. The part that we can control most is the food we ingest. What we put into our bodies is without any question the single most important factor affecting our health. And yes, we can take control. In fact, we can exert significant control over many different health factors if we choose to. It's just very easy to choose not to. There are dozens if not hundreds of reasons we can choose not to eat for health and longevity, including health well into our later years. Many of us use them. It's an unfortunate part of the human condition—denial—or it's equally well known formal name, 'cognitive dissonance'. It's ubiquitous with food drug additives.

One of the most important concepts I hope you'll take away from this eMag is not just that it's never too late to make slow changes to your own diet but even more important than you and I and our health is the health of our children and grandchildren.

They are the future of the world we'll all too soon leave behind. The elite educate their children critically. We each need to start doing the same beginning with educating our children about the critical importance of food—what isn't food and what is food and how to eat for health—so that our progeny can lead healthy lives and possess the energy, desire and intellectual capacity to carry on their own personal Class War crusades using what we've taught them. Hopefully everyone reading here will recognize and understand class warfare. It begins at birth and continues on well past your death, and then forever on. Our world is simply composed of class warfare in every way, shape and form. It's a fact many of us knowingly live with but a fact unknown to the majority. If everyone knew we'd have won decades and even centuries ago. Unfortunately the subject is never discussed much, if at all, on major media and alternative media is a little thin on class warfare too. It's a part of your life. It is your life, start to end, whether you know it or not. Everything is Class Warfare. Spend some time Googling it if the concept is new to you.

Our species success requires raising "clean" and unadulterated children. As much as possible of course, because it's a difficult, tedious task. We're assaulted daily by everything from food colors and additives to car exhaust and industrial air pollution. There's Bisphenol A and Glyphosate in the air we breathe because we've manufactured and used so many millions of tons of the stuff for so many years now.

Speak to your children about diet and its critical importance, repeatedly, for the years to come, and teach them its direct and significant connection to either health or disease. Their particular diet will be their personal choice one day and you can impact those choices right now. This eMagazine gives you all of the tools you'll ever need to understand and teach the devastating damage that's already been done and often also how to clean up the filthy mess where possible.

Not one person I know would knowingly eat a petroleum product or derivative such as toluene, ink, cleaning fluids and defoamers, liquid plastics, emulsifiers or chemical glues, yet the majority of the human species will eat Hydrogenated Oil (E949), TBHQ, Aspartame (E951), Sodium Stearoyl Lactate (E481), Fluoride, Polysorbate 80, 65, 60 and 20, High Fructose Corn Syrup, Monosodium Glutamate (E621), Trans Fats, Brilliant Blue FCF (E133) and Blue #2, both banned in France, Finland and Norway because they can cause chromosomal damage and even Yellow Tartrazine (E102), banned in Sweden and Norway because it causes kidney and adrenal gland tumors and, of course, chromosomal damage as well. Americans aren't so lucky. They're not only legal here, these lab created drugs are sold to us, by the corporate drug mafia and they're used prolifically. We all eat them, some of us well more than others.

The peer review shows that repeatedly consuming food drugs of any and all types can cause depression, bipolar disorder, mania, bone loss, neurological disorders, Crohn's disease, hearing loss, skin cancer, various other cancers and too many more diseases to list here. You'll read the peer review and learn for yourself from the professionals that know about it, the authors. After all, they write these papers for us, for you and I.
Our western species' blind consumerism, and this disease causing diet being faithfully and scientifically designed and provided to us to sate our brains most inner childhood and child-like 'food' desires has been globally exported to every country on every continent by a very few multinational global industrial feed corporations. We're their cattle. They're feeding us sugar, sodium and a list of chemicals literally a mile long. Well, several pages long anyway because I included them here ... all of them.

If you walk through any conventional grocery store you'll see the big bucks that went into the fancy packaging, the images and vivid colors that make up our feed choices—pre-cooked everything—just heat and serve. On the run? Two minutes in the microwave! We've all done that, right? I have, too many times to count and it's something I'd prefer to forget, like we prefer to forget bad dreams. Yet this one's real, and it's as real as it ever gets.

The human species now mindlessly consumes Potassium Bromate (E924), Sodium Sulfite (E221), Sodium Nitrate (E251) and Sodium Nitrite (E250), something called BHA because no one can remember or pronounce butylated hydroxyanisole and BHT (E320), the largely also unpronounceable and easily forgettable butylated hydroxytoluene (E321) as it's known. We eat these chemicals in our cereals, our chewing gum, our breads, chips and crackers, our vegetable oils and everything else we put into our mouths bought as conventional food. We even wipe them all over our sponge-like, all absorbing skin in cosmetics. The peer review addresses repeated consumption of these various chemicals, even at extremely low doses and sometimes especially at low doses, and it's frightening. And repetitive over-consumption, for example, 4-6 cans of beer, coke, pepsi or any other soda each day, and especially the carbonated, caramel colored drinks, causes extreme harm rapidly.

Again, for example, you'll read that over-consumption of alcohol over a 6-year period, with MRI images taken every 2 years, causes a loss of about 10%, maybe slightly more, of gray brain matter. You'll see the MRI photos. This disturbs what's called "executive functioning" or "executive decision making" meaning we become unable to put incoming information together correctly and make the proper and very important life decisions that we face, and decisions we each face almost daily. As a result, we can often and repeatedly make bad decisions concerning critically important issues.

The large volume of categorized peer reviewed reports and studies collected here proves every last word I've typed here and more. Much more. The conventional food supply is poisoned and it will, undoubtedly and without question, lead to disease and disorder for anyone that either unknowingly or foolishly eats the diet repetitively and continuously.

Eventually the chemicals will have their impact both alone and more so synergistically and the peer review explains the molecular mechanics, so-to-speak, of how chemical food drugs make the human species ill and cause not just brain dysfunction but system-wide disruption and 100s of disorders. The very same manufacturers or subsidiaries then make the drugs that treat or ease these very same disorders. Don't forget that. It's a Drug Sales Racket.

Take notice that the older peer review often uses far more harsh language in its criticism of food additives than the newer studies might employ, sometimes. There are also a number of foreign countries that have banned many of the additives we still use here in the USA, and for good reason, they're deadly. However, in my ever present effort to be honest and seek the truth, there are about an equal number of chemicals that we've banned here in the US that are legal in many other countries. It's a Drug Sales Racket if there ever was one.

This is because the alleged governmental protection system —FDA, EPA, USDA, et al.—both here and abroad consist of haphazardly designed and managed programs, they are corrupted to the core, fully industry "owned," or much more likely, all three. You'll read about all of this and so much more right here in the 1000+ peer reviewed reports and studies.

Our American-made food supply and disease are ubiquitous with one another, whether you believe it's by design or not which is immaterial but worth mentioning, and as universal as the poisoning is, this is a concept that is not universally recognized so it's important that you share this eMag with the people you really love and care for. Let's try to educate the rest of the world or at the very least let's try to have an impact on the ones we care about the most. With 100% peer review, all linked and written by the worlds most respected and regarded medical researchers from almost every known and recognized specialty that exists. These are medical scientists from countries across the globe that often specialize in these subjects, all have PhD's and many are tenured professors. This is a collection of the best of the best.

Food and what we eat is the single most overlooked societal issue in this 21st century and it's the single most important contributing factor to almost every disease known to our species. If we don't personally educate our children on this issue I can promise you, no one else will. They'll age like many of us—with diseases and disabling disorders needlessly caused by 20, 30 and 40 years or more of eating proven human mutagens, carcinogens and neurotoxins—needlessly. We didn't know. So teach your children what's in this eMag and share it with your friends and family. Download and read this eMag slowly, over the days and weeks to come because you care about the future of our children and their children. Share it with everyone you know, please. That's why it's free.

~ Jeff Prager

Chapter Fifty

Artificial Sweeteners

The Peer Review

Aspartame
Sucralose
Saccharin
Acesulfame Potassium or
Acesulfame K
Neotame
Cyclamate
Erythritol
Glycerol
Lactitol
Maltitol
Mannitol
Polydextrose
Sorbitol
Xylitol
HFCS
Sugar

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“Histological findings showed that Aspartame 2 and Aspartame 3 caused cyto-architectural changes such as degeneration, monocytes infiltration and necrotic lesions in brain, kidney and liver of rats.”

Journal Of Basic Clinical And Physiological Pharmacology • January 2016

Effects of long-term administration of aspartame on biochemical indices, lipid profile and redox status of cellular system of male rats

Adaramoye OA, Akanni OO.

Abstract

BACKGROUND

Aspartame (N-L-a-aspartyl-L-phenylalanine-1-methyl ester) (ASP) is a synthetic sweetener used in foods and its safety remains controversial. The study was designed to investigate the effects of long-term administration of aspartame on redox status, lipid profile and biochemical indices in tissues of male Wistar rats.

METHODS

Rats were assigned into four groups and given distilled water (control), aspartame at doses of 15 mg/kg (ASP 1), 35 mg/kg (ASP 2) and 70 mg/kg (ASP 3) daily by oral gavage for consecutive 9 weeks.

RESULTS

Administration of ASP 2 and ASP 3 significantly increased the weight of liver and brain, and relative weight of liver of rats. Lipid peroxidation products significantly increased in the kidney, liver and brain of rats at all doses of ASP with concomitant depletion of antioxidant parameters, viz. glutathione-s-transferase, glutathione peroxidase, superoxide dismutase, catalase and reduced glutathione. Furthermore, ASP 2 and ASP 3 significantly increased the levels of gamma glutamyl transferase by 70% and 85%; alanine aminotransferase by 66% and 117%; aspartate aminotransferase by 21% and 48%; urea by 72% and 58% and conjugated bilirubin by 63% and 64%, respectively. Also, ASP 2 and ASP 3 significantly increased the levels of total cholesterol, triglycerides and low-density lipoprotein cholesterol in the rats. Histological findings showed that ASP 2 and ASP 3 caused cyto-architectural changes such as degeneration, monocytes infiltration and necrotic lesions in brain, kidney and liver of rats.

CONCLUSIONS

Aspartame may induce redox and lipid imbalance in rats via mechanism that involves oxidative stress and depletion of glutathione-dependent system.

http://www.ncbi.nlm.nih.gov/pubmed/26247507

THE SUGAR

Langers Pomegranite Cranberry contains **10 grams of sugar** per 8 ounce serving. The bottle pictured is a 64 ounce bottle with **80 grams of sugar** in it. It also contains the following ingredients: Filtered water, Apple, Pomegranite and Cranberry Juice Concentrates, Calcium Gluconate, Calcium Lactate, Citric Acid, Pectin, **Natural Flavors, Sucralose (Splenda Brand) and Acesulfame Potassium (K).**

The PediaSure is a smaller bottle, just 8 ounces, and packs a whopping **18 grams of sugar** in the bottle of Strawberry pictured below. Pediasure Strawberry contains Water, **Sugar (Sucrose), Corn Maltodextrin** listed as the first 3 ingredients so it’s water that’s the primary carrier of the much smaller additions of any vitamins, minerals or micro-nutrients. **FD&C Red No. 3** as a coloring agent. I would avoid this product. There are better ways to quickly get even greater amounts of the same vitamins and minerals listed on the Pediasure label, without the **sugar**, the **Red #3** or the **presumably GMO corn-derived maltodextrin** and we teach you how to make your own homemade, organic pediasure, without the chemicals and with a significantly higher nutrient profile in the chapter on coconut oil and for a lot less money than pediasure.

The Mountain Dew has a whopping **46 grams of sugar** in a 12 ounce container with **Red #40, Yellow #5 and Blue #1**. There are also a few more disorder-causing ingredients in the dew so please, don’t do the dew. Or the Pediasure. Or Langers.

Critical Reviews In Clinical Laboratory Sciences • February 2016

Sugar consumption, metabolic disease and obesity: The state of the controversy

Author information

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Abstract

The impact of sugar consumption on health continues to be a controversial topic. The objective of this review is to discuss the evidence and lack of evidence that allows the controversy to continue, and why resolution of the controversy is important. There are plausible mechanisms and research evidence that supports the suggestion that consumption of excess sugar promotes the development of cardiovascular disease (CVD) and type 2 diabetes (T2DM) both directly and indirectly. The direct pathway involves the unregulated hepatic uptake and metabolism of fructose, leading to liver lipid accumulation, dyslipidemia, decreased insulin sensitivity and increased uric acid levels. The epidemiological data suggest that these direct effects of fructose are pertinent to the consumption of the fructose-containing sugars, sucrose and high fructose corn syrup (HFCS), which are the predominant added sugars. Consumption of added sugar is associated with development and/or prevalence of fatty liver, dyslipidemia, insulin resistance, hyperuricemia, CVD and T2DM, often independent of body weight gain or total energy intake. There are diet intervention studies in which human subjects exhibited increased circulating lipids and decreased insulin sensitivity when consuming high sugar compared with control diets. Most recently, our group has reported that supplementing the ad libitum diets of young adults with beverages containing 0%, 10%, 17.5% or 25% of daily energy requirement (Ereq) as HFCS increased lipid/lipoprotein risk factors for CVD and uric acid in a dose-response manner. However, un-confounded studies conducted in healthy humans under a controlled, energy-balanced diet protocol that enables determination of the effects of sugar with diets that do not allow for body weight gain are lacking. Furthermore, recent reports conclude that there are no adverse effects of consuming beverages containing up to 30% Ereq sucrose or HFCS, and the conclusions from several meta-analyses suggest that fructose has no specific adverse effects relative to any other carbohydrate. Consumption of excess sugar may also promote the development of CVD and T2DM indirectly by causing increased body weight and fat gain, but this is also a topic of controversy. Mechanistically, it is plausible that fructose consumption causes increased energy intake and reduced energy expenditure due to its failure to stimulate leptin production. Functional magnetic resonance imaging (fMRI) of the brain demonstrates that the brain responds differently to fructose or fructose-containing sugars compared with glucose or aspartame. Some epidemiological studies show that sugar consumption is associated with body weight gain, and there are intervention studies in which consumption of ad libitum high-sugar diets promoted increased body weight gain compared with consumption of ad libitum low- sugar diets. However, there are no studies in which energy intake and weight gain were compared in subjects consuming high or low sugar, blinded, ad libitum diets formulated to ensure both groups consumed a comparable macronutrient distribution and the same amounts of fiber. There is also little data to determine whether the form in which added sugar is consumed, as beverage or as solid food, affects its potential to promote weight gain. It will be very challenging to obtain the funding to conduct the clinical diet studies needed to address these evidence gaps, especially at the levels of added sugar that are commonly consumed. Yet, filling these evidence gaps may be necessary for supporting the policy changes that will help to turn the food environment into one that does not promote the development of obesity and metabolic disease.

http://www.ncbi.nlm.nih.gov/pubmed/26376619

Water Research • February 2016

Photocatalytic transformation of acesulfame: Transformation products identification and embryotoxicity study

Author information

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Abstract

Artificial sweeteners have been recognized as emerging contaminants due to their wide application, environmental persistence and ubiquitous occurrence. Among them, acesulfame has attracted much attention. After being discharged into the environment, acesulfame undergoes photolysis naturally. However, acesulfame photodegradation behavior and identity of its transformation products, critical to understanding acesulfame's environmental impact, have not been thoroughly investigated. The present study aimed to fill this knowledge gap by a laboratory simulation study in examining acesulfame transformation products and pathways under UV-C photolysis in the presence of TiO2. Photodegradation products of acesulfame were isolated and analyzed using the LC-IM-QTOF-MS coupled with LC Ion Trap MS in the MS(n) mode. Our results show six new transformation products that have not been previously identified. The molecular structures and transformation pathways were proposed. Further embryotoxicity tests showed that acesulfame transformation products at the low g L(-1) level produced significant adverse effects in tail detachment, heart rate, hatching rate and survival rate during fish embryo development. The identification of additional transformation products with proposed transformation pathways of acesulfame, the increased toxicity of acesulfame after photolysis, and the fact that the accumulation of acesulfame transformation products is increasingly likely make acesulfame contamination even more important. Water resource control agencies need to consider legislation regarding acesulfame and other artificial sweeteners, while further studies are carried out, in order to protect the safety of this most vital resource.

http://www.ncbi.nlm.nih.gov/pubmed/26630044

“Artificial sweeteners have been recognized as emerging contaminants due to their wide application, environmental persistence and ubiquitous occurrence. Among them, acesulfame has attracted much attention. After being discharged into the environment, acesulfame undergoes photolysis naturally. However, acesulfame photodegradation behavior and identity of its transformation products, critical to understanding acesulfame's environmental impact, have not been thoroughly investigated. The present study aimed to fill this knowledge gap ... Our results show six new transformation products that have not been previously identified. The molecular structures and transformation pathways were proposed. Further embryotoxicity tests showed that acesulfame transformation products at the *low g L(-1) level produced significant adverse effects in tail detachment, heart rate, hatching rate and survival rate during fish embryo development ... the increased toxicity of acesulfame ... and the fact that the accumulation of acesulfame transformation products is increasingly likely make acesulfame contamination even more important.”

“These results suggest that aspartame and saccharin could be toxic to the human circulation system as well as embryonic development via impairment of lipoprotein function.”

Cardiovascular Toxicology • January 2015

Modified high-density lipoproteins by artificial sweetener, aspartame, and saccharin, showed loss of anti-atherosclerotic activity and toxicity in zebrafish

Author information

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Abstract

Safety concerns have been raised regarding the association of chronic consumption of artificial sweeteners (ASs) with metabolic disorders, especially in the heart and brain. There has been no information on the in vivo physiological effects of AS consumption in lipoprotein metabolism. High-dosage treatment (final 25, 50, and 100 mM) with AS (aspartame, acesulfame K, and saccharin) to human high-density lipoprotein (HDL) induced loss of antioxidant ability along with elevated atherogenic effects. Aspartame-treated HDL3 (final 100 mM) almost all disappeared due to putative proteolytic degradation. Aspartame- and saccharin-treated HDL3 showed more enhanced cholesteryl ester transfer activity, while their antioxidant ability was disappeared. Microinjection of the modified HDL3 exacerbated the inflammatory death in zebrafish embryos in the presence of oxLDL. These results show that AS treatment impaired the beneficial functions of HDL, resulting in loss of antioxidant and anti-atherogenic activities. These results suggest that aspartame and saccharin could be toxic to the human circulation system as well as embryonic development via impairment of lipoprotein function.

http://www.ncbi.nlm.nih.gov/pubmed/25142179

“While they are considered safe, there is increasing controversy regarding their potential ability to promote metabolic derangements in some humans. We recently demonstrated that NAS consumption could induce glucose intolerance in mice and distinct human subsets ...”

Gut Microbes • April 2015

Non-caloric artificial sweeteners and the microbiome: findings and challenges

Author information

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Abstract

Non-caloric artificial sweeteners (NAS) are common food supplements consumed by millions worldwide as means of combating weight gain and diabetes, by retaining sweet taste without increasing caloric intake. While they are considered safe, there is increasing controversy regarding their potential ability to promote metabolic derangements in some humans. We recently demonstrated that NAS consumption could induce glucose intolerance in mice and distinct human subsets, by functionally altering the gut microbiome. In this commentary, we discuss these findings in the context of previous and recent works demonstrating the effects of NAS on host health and the microbiome, and the challenges and open questions that need to be addressed in understanding the effects of NAS consumption on human health.

http://www.ncbi.nlm.nih.gov/pubmed/25831243

International Journal Of Immunopathology And Pharmacology • June 2015

Impact of aspartame and saccharin on the rat liver: Biochemical, molecular, and histological approach

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Abstract

The current work was undertaken to settle the debate about the toxicity of artificial sweeteners (AS), particularly aspartame and saccharin. Twenty-five, 7-week-old male Wistar albino rats with an average body weight of 101 ± 4.8 g were divided into a control group and four experimental groups (n = 5 rats). The first and second experimental groups received daily doses equivalent to the acceptable daily intake (ADI) of aspartame (250 mg/Kg BW) and four-fold ADI of aspartame (1000 mg/Kg BW). The third and fourth experimental groups received daily doses equivalent to ADI of saccharin (25 mg/Kg BW) and four-fold ADI of saccharin (100 mg/Kg BW). The experimental groups received the corresponding sweetener dissolved in water by oral route for 8 weeks. The activities of enzymes relevant to liver functions and antioxidants were measured in the blood plasma. Histological studies were used for the evaluation of the changes in the hepatic tissues. The gene expression levels of the key oncogene (h-Ras) and the tumor suppressor gene (P27) were also evaluated. In addition to a significant reduction in the body weight, the AS-treated groups displayed elevated enzymes activities, lowered antioxidants values, and histological changes reflecting the hepatotoxic effect of aspartame and saccharin. Moreover, the overexpression of the key oncogene (h-Ras) and the downregulation of the tumor suppressor gene (P27) in all treated rat groups may indicate a potential risk of liver carcinogenesis, particularly on long-term exposure.

http://www.ncbi.nlm.nih.gov/pubmed/26015492

“In addition to a significant reduction in the body weight, the Artificial Sweetener-treated groups displayed elevated enzymes activities, lowered antioxidants values, and histological changes reflecting the hepatotoxic effect of aspartame and saccharin.

Moreover, the overexpression of the key oncogene (h-Ras) and the downregulation of the tumor suppressor gene (P27) in all treated rat groups may indicate a potential risk of liver carcinogenesis, particularly on long-term exposure.”

“Long-term consumption of sugar substitutes aggravated cerebral ischemic injury in mice ...”

Stroke • June 2015

Dietary intake of sugar substitutes aggravates cerebral ischemic injury and impairs endothelial progenitor cells in mice

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Abstract

BACKGROUND AND PURPOSE

In our current food supply, sugar substitutes are widely used in beverages and other food products. However, there is limited information about the link between dietary consumption of sugar substitutes and stroke to date. This study sought to determine the effect of various sugar substitutes on the cerebral ischemic injury and endothelial progenitor cells, which have been implicated to play an important role in vascular repair and revascularization in ischemic brain tissues, in mice.

METHODS

After treatment with sucrose and various sugar substitutes (the doses are in the range of corresponding acceptable daily intake levels) and vehicle for 6 weeks, mice were subjected to permanent left middle cerebral artery occlusion, and the infarct volumes, angiogenesis, and neurobehavioral outcomes were determined. In addition, the number and function of endothelial progenitor cells were also examined.

RESULTS

After long-term treatment with fructose, erythritol (sugar alcohols), acesulfame K (artificial sweeteners), or rebaudioside A (rare sugars), the cerebral ischemic injury (both infarct volumes and neurobehavioral outcomes) was significantly aggravated, angiogenesis in ischemic brain was reduced, and endothelial progenitor cell function was impaired in mice compared with control. However, the similar impairments were not found in sucrose (with the same dose as fructose’s)-treated mice.

CONCLUSIONS

Long-term consumption of sugar substitutes aggravated cerebral ischemic injury in mice, which might be partly attributed to the impairment of endothelial progenitor cells and the reduction of angiogenesis in ischemic brain. This result implies that dietary intake of sugar substitutes warrants further attention in daily life.

http://www.ncbi.nlm.nih.gov/pubmed/25908458

American Journal Of Clinical Nutrition • June 2015

A dose-response study of consuming high-fructose corn syrup-sweetened beverages on lipid/lipoprotein risk factors for cardiovascular disease in young adults

Author information

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Abstract

BACKGROUND

National Health and Nutrition Examination Survey data show an increased risk of cardiovascular disease (CVD) mortality with an increased intake of added sugar.

OBJECTIVE

We determined the dose-response effects of consuming beverages sweetened with high-fructose corn syrup (HFCS) at zero, low, medium, and high proportions of energy requirements (Ereq) on circulating lipid/lipoprotein risk factors for CVD and uric acid in adults [age: 18-40 y; body mass index (in kg/m(2)): 18-35].

DESIGN

We conducted a parallel-arm, nonrandomized, double-blinded intervention study in which adults participated in 3.5 inpatient days of baseline testing at the University of California Davis Clinical and Translational Science Center's Clinical Research Center. Participants then consumed beverages sweetened with HFCS at 0% (aspartame sweetened, n = 23), 10% (n = 18), 17.5% (n = 16), or 25% (n = 28) of Ereq during 13 outpatient days and during 3.5 inpatient days of intervention testing at the research center. We conducted 24-h serial blood collections during the baseline and intervention testing periods.

RESULTS

Consuming beverages containing 10%, 17.5%, or 25% Ereq from HFCS produced significant linear dose-response increases of lipid/lipoprotein risk factors for CVD and uric acid: postprandial triglyceride (0%: 0 ± 4; 10%: 22 ± 8; 17.5%: 25 ± 5: 25%: 37 ± 5 mg/dL, mean of Δ ± SE, $P < 0.0001$ effect of HFCS-dose), fasting LDL cholesterol (0%: -1.0 ± 3.1; 10%: 7.4 ± 3.2; 17.5%: 8.2 ± 3.1; 25%: 15.9 ± 3.1 mg/dL, $P < 0.0001$), and 24-h mean uric acid concentrations (0%: -0.13 ± 0.07; 10%: 0.15 ± 0.06; 17.5%: 0.30 ± 0.07; 25%: 0.59 ± 0.09 mg/dL, $P < 0.0001$). Compared with beverages containing 0% HFCS, all 3 doses of HFCS-containing beverages increased concentrations of postprandial triglyceride, and the 2 higher doses increased fasting and/or postprandial concentrations of non-HDL cholesterol, LDL cholesterol, apolipoprotein B, apolipoprotein CIII, and uric acid.

CONCLUSIONS

Consuming beverages containing 10%, 17.5%, or 25% Ereq from HFCS produced dose-dependent increases in circulating lipid/lipoprotein risk factors for CVD and uric acid within 2 wk. These results provide mechanistic support for the epidemiologic evidence that the risk of cardiovascular mortality is positively associated with consumption of increasing amounts of added sugars. This trial was registered at clinicaltrials.gov as NCT01103921.

“These results provide mechanistic support for the epidemiologic evidence that the risk of cardiovascular mortality is positively associated with consumption of increasing amounts of added sugars.”

http://www.ncbi.nlm.nih.gov/pubmed/25904601

Abstract

CONTEXT

Sugar overconsumption and chronic stress are growing health concerns because they both may increase the risk for obesity and its related diseases. Rodent studies suggest that sugar consumption may activate a glucocorticoid-metabolic-brain-negative feedback pathway, which may turn off the stress response and thereby reinforce habitual sugar overconsumption.

OBJECTIVE

The objective of the study was to test our hypothesized glucocorticoid-metabolic-brain model in women consuming beverages sweetened with either aspartame of sucrose.

DESIGN

This was a parallel-arm, double-masked diet intervention study.

SETTING

The study was conducted at the University of California, Davis, Clinical and Translational Science Center's Clinical Research Center and the University of California, Davis, Medical Center Imaging Research Center.

PARTICIPANTS

Nineteen women (age range 18-40 y) with a body mass index (range 20-34 kg/m(2)) who were a subgroup from a National Institutes of Health-funded investigation of 188 participants assigned to eight experimental groups.

INTERVENTION

The intervention consisted of sucrose- or aspartame-sweetened beverage consumption three times per day for 2 weeks.

MAIN OUTCOME MEASURES

Salivary cortisol and regional brain responses to the Montreal Imaging Stress Task were measured.

RESULTS

Compared with aspartame, sucrose consumption was associated with significantly higher activity in the left hippocampus (P = .001). Sucrose, but not aspartame, consumption associated with reduced (P = .024) stress-induced cortisol. The sucrose group also had a lower reactivity to naltrexone, significantly (P = .041) lower nausea, and a trend (P = .080) toward lower cortisol.

CONCLUSION

These experimental findings support a metabolic-brain-negative feedback pathway that is affected by sugar and may make some people under stress more hooked on sugar and possibly more vulnerable to obesity and its related conditions.

http://www.ncbi.nlm.nih.gov/pubmed/25879513

Journal Of Clinical Endocrinology And Metabolism • June 2015

Excessive Sugar Consumption May Be a Difficult Habit to Break: A View From the Brain and Body

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“These experimental findings support a metabolic-brain negative feedback pathway that is affected by sugar and may make some people under stress more hooked on sugar and possibly more vulnerable to obesity and its related conditions.”

> “Aspartame and saccharin also decreased the survival and behaviour of adult flies and may be toxic to (or contribute to poor nutrition in) B. dorsalis. These sweeteners could therefore be developed as additive ingredients in baits.”

Pest Management Science • July 2015

Effect of sweeteners on the survival and behaviour of Bactrocera dorsalis (Hendel) (Diptera: Tephritidae)

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Abstract

BACKGROUND
The oriental fruit fly Bactrocera dorsalis (Hendel) (Diptera: Tephritidae) causes serious damage that affects fruit production. Chemical insecticides have been widely used for the prevention and control of this destructive pest. However, the resistance of B. dorsalis to these compounds has become a serious problem. This study tested six sweeteners for their toxicity to B. dorsalis.

RESULTS:
B. dorsalis fed on erythritol, aspartame and saccharin exhibited significantly higher mortality than those fed on sucrose. Flies fed on erythritol died faster than did the control flies (water). However, no dose-dependent effects were observed at the concentrations tested. These three sweeteners decreased the climbing ability of B. dorsalis. Notably, adults fed on saccharin exhibited significantly decreased climbing ability after 12 h compared with those fed on sucrose. Additionally, these three sweeteners had a negative effect on the frequency and duration of the flies' behaviour patterns (flying, walking, grooming and inactivity). Saccharin not only induced a marked reduction in the frequency of flights and walks but also induced decreases in the time spent flying and walking and increases in inactivity compared with sucrose. Erythritol induced a reduction in movement and increased the time spent inactive compared with the control and other treatments.

CONCLUSION:
Three sweeteners had significant negative effects on the survival of B. dorsalis. Erythritol was toxic to B. dorsalis. Aspartame and saccharin also decreased the survival and behaviour of adult flies and may be toxic to (or contribute to poor nutrition in) B. dorsalis. These sweeteners could therefore be developed as additive ingredients in baits.

http://www.ncbi.nlm.nih.gov/pubmed/26177595

Aspartame & Saccharin Are Both So Toxic To Insects That Researchers At The College Of Agriculture Recommend Adding These Sweeteners To Commercial Pesticides!

Journal Of Toxicology And Environmental Health, Part A • August 2015

Nonnutritive Sweeteners in Breast Milk

Author information

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Abstract

Nonnutritive sweeteners (NNS), including saccharin, sucralose, aspartame, and acesulfame-potassium, are commonly consumed in the general population, and all except for saccharin are considered safe for use during pregnancy and lactation. Sucralose (Splenda) currently holds the majority of the NNS market share and is often combined with acesulfame-potassium in a wide variety of foods and beverages. To date, saccharin is the only NNS reported to be found in human breast milk after maternal consumption, while there is no apparent information on the other NNS. Breast milk samples were collected from 20 lactating volunteers, irrespective of their habitual NNS intake. Saccharin, sucralose, and acesulfame-potassium were present in 65% of participants' milk samples, whereas aspartame was not detected. These data indicate that NNS are frequently ingested by nursing infants, and thus prospective clinical studies are necessary to determine whether early NNS exposure via breast milk may have clinical implications.

http://www.ncbi.nlm.nih.gov/pubmed/26267522

“Saccharin, sucralose, and acesulfame-potassium were present in 65% of participants' milk samples, whereas aspartame was not detected. These data indicate that non-nutritive sweeteners are frequently ingested by nursing infants, and thus prospective clinical studies are necessary to determine whether early non-nutritive sweetener exposure via breast milk may have clinical implications.”

BMJ • August 2015

Fetal exposure to dietary carcinogens and risk of childhood cancer: what the NewGeneris project tells us

Author Information

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Jos Kleinjans and colleagues summarise the evidence from a European study using biomarkers to assess how maternal diet affects the child

Abstract

Cancer in childhood is rare. Globally, there are around 175 000 new cancer cases a year among children aged 0-14 years. However, in Europe, since the 1950s the incidence of cancer in this age group has increased by about 1% a year, with leukaemia, brain tumours, and lymphomas accounting for most cases. The increases in incidence of lymphoid leukaemia, in particular, are more apparent in European than in Asian or African children. The development of childhood cancer thus seems to be affected by both genetic and environmental factors. Given that the highest incidences of childhood leukaemia are reported in children younger than 6-7 years, that the latency period of leukaemia in children is relatively short, and that adverse genetic events in utero have been shown to give rise to leukaemia in childhood, we hypothesised that fetal exposure to environmental carcinogens may be an underlying cause. Diet is an important source of carcinogenic compounds because of the accumulation of environmental carcinogens within the food chain (dioxins, polychlorinated biphenyls (PCBs), polycyclic aromatic hydrocarbons (PAHs)), as well as of formation of carcinogens such as PAHs, heterocyclic amines, and acrylamide during baking, frying, and grilling of food. The New Generis (Newborns and Genotoxic Exposure Risks) project therefore set out to investigate whether intake of dietary carcinogens by the pregnant mother leads to exposures of the fetus and initiates adverse biological responses that can induce cancer in later childhood.

Investigation of this hypothesis in an epidemiological study would require a huge sample size because of the relatively low incidence of childhood cancer (about 140 cases per million children). Case-control studies have been informative but also have limitations.

Key messages:

- An estimated 175,000 new cancer cases occur globally among children aged 0-14
- The fetus is exposed to dietary carcinogens consumed by the mother during pregnancy
- Exposure of the fetus causes perturbations of the molecular pathways linked with markers of carcinogenicity
- Genetic polymorphisms and male sex may predispose to susceptibility to carcinogen exposure before birth
- This increases risks of developing leukaemia, in particular
- Fetal exposure to dietary carcinogens may also affect birth weight and cause immune suppression

http://www.bmj.com/content/351/bmj.h4501.long

According to this peer reviewed report and many others just like it, women intending to become pregnant should adopt a diet that is 100% organic before, during and after the pregnancy. An organic diet is just as important after pregnancy so that your breast milk is as additive-free—poison-free—as possible. The manufacturers represented above, along with 100s more, all use harmful food additives and it's your responsibility to avoid them or perhaps suffer consequences.

> “Aspartame exposure resulted in detectable methanol even after 24 hours. Methanol and its metabolites may be responsible for the generation of oxidative stress in brain regions. The observed alteration in aspartame fed animals may be due to its metabolite methanol and elevated formate.”

Journal Of Biomedical Research • September 2015

Acute effect of aspartame-induced oxidative stress in Wistar albino rat brain

Author information

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Abstract

The present study was carried out to investigate the acute effect of aspartame on oxidative stress in the Wistar albino rat brain. We sought to investigate whether acute administration of aspartame (75 mg/kg) could release methanol and induce oxidative stress in the rat brain 24 hours after administration. To mimic human methanol metabolism, methotrexate treated rats were used to study aspartame effects. Wistar strain male albino rats were administered with aspartame orally as a single dose and studied along with controls and methotrexate treated controls. Blood methanol and formate level were estimated after 24 hours and rats were sacrificed and free radical changes were observed in discrete regions by assessing the scavenging enzymes, reduce dglutathione (GSH), lipid peroxidation and protein thiol levels. There was a significant increase in lipid peroxidation levels, superoxide dismutase activity (SOD), glutathione peroxidase levels (GPx), and catalase activity (CAT) with a significant decrease in GSH and protein thiol. Aspartame exposure resulted in detectable methanol even after 24 hours. Methanol and its metabolites may be responsible for the generation of oxidative stress in brain regions. The observed alteration in aspartame fed animals may be due to its metabolite methanol and elevated formate. The elevated free radicals due to methanol induced oxidative stress.

http://www.ncbi.nlm.nih.gov/pubmed/26445572

Albino Wistar Rat named “Groggy” used in scientific experiments

> “We found that the ranking of developmental toxicity was Saccharin > Caffeine > Aspartame > Sucrose, and there was a cumulative effect when Caffeine was combined with the sweeteners.”

Springerplus • September 2015

Assessing developmental toxicity of caffeine and sweeteners in medaka (Oryzias latipes)

Author information

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Abstract

The use of artificial sweeteners (ASWs) has increased and become more widespread, and consequently ASWs have appeared in aquatic environments around the world. However, their safety to the health of humans and wildlife remains inconclusive. In this study, using medaka embryos (Oryzias latipes), we investigated developmental toxicity of aspartame (ASP) and saccharin (SAC). Since ASWs are often consumed with caffeine (CAF) and CAF with sucrose (SUC), we tested biological activities of these four substances and the mixtures of CAF with each sweetener. The embryos were exposed to ASP at 0.2 and 1.0 mM, SAC at 0.005 and 0.050 mM, CAF at 0.05 and 0.5 mM, or SUC at 29 and 146 mM, starting from less than 5 h post fertilization until hatch. Control embryos were treated with embryo solution only. Several endpoints were used to evaluate embryonic development. Some of the hatchlings were also tested for anxiety-like behavior with the white preference test. The results showed that all four substances and the mixtures of CAF with the sweeteners affected development. The most sensitive endpoints were the heart rate, eye density, and hatchling body length. The hatchlings of several treatment groups also exhibited anxiety-like behavior. We then used the Integrated Biological Response (IBR) as an index to evaluate the overall developmental toxicity of the substances. We found that the ranking of developmental toxicity was SAC > CAF > ASP > SUC, and there was a cumulative effect when CAF was combined with the sweeteners.

Full text, graphs and charts with 42 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4562911/

Drug Chemistry And Toxicology • October 2015

The effect of five artificial sweeteners on Caco-2, HT-29 and HEK-293 cells

Author information

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Abstract

CONTEXT

Artificial sweeteners (AS) have been associated with tumor development (including colon cancer) in both animals and humans although evidence has been conflicting.

OBJECTIVES

Additional research was thus conducted by studying the effects of 5 AS on the morphology, cell proliferation and DNA in cells by utilizing Caco-2, HT-29 (colon) and HEK-293 (kidney) cell lines.

MATERIALS AND METHODS

Cells were exposed to sodium cyclamate, sodium saccharin, sucralose and acesulfame-K (0-50 mM) and aspartame (0-35 mM) over 24, 48 and 72 hours. Morphological changes were presented photographically and % cell viability was determined by using the MTT cell viability assay. Possible DNA damage (comet assay) induced by the AS (0.1, 1 and 10 mM, treated for 24, 48 and 72 hours) was studied. The appearance of "comets" was scored from no damage to severe damage (0-4).

RESULTS

Cells became flatter and less well defined at higher AS concentrations (>10 mM). At concentrations >10 mM, decreased cell viability was noted with both increasing concentration and increasing incubation time for all cell lines tested. In general, HEK-293 cells seemed to be less affected then the colon cancer cells. Sucralose and sodium saccharin seemed to elicit the greatest degree of DNA fragmentation of all the sweeteners tested in all the cell lines used.

DISCUSSION

Morphological cell alterations, cell viability and DNA fragmentation seemed to be more in the colon cancer cells.

CONCLUSIONS

Further studies have to be performed to clarify mechanisms involved causing these alterations in mammalian cells.

http://www.ncbi.nlm.nih.gov/pubmed/25317478

"Sucralose and sodium saccharin seemed to elicit the greatest degree of DNA fragmentation of all the sweeteners tested in all the cell lines used. Morphological cell alterations, cell viability and DNA fragmentation seemed to be more in the colon cancer cells."

> “... a scientific consensus has emerged suggesting that consumption of sugar-sweetened products, especially beverages, is casually linked to increases in risk of chronic, debilitating diseases including type 2 diabetes, cardiovascular disease, hypertension and stroke.”

Appetite • October 2015

Artificial sweeteners are not the answer to childhood obesity

Author information

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Abstract

While no single factor is responsible for the recent, dramatic increases in overweight and obesity, a scientific consensus has emerged suggesting that consumption of sugar-sweetened products, especially beverages, is casually linked to increases in risk of chronic, debilitating diseases including type 2 diabetes, cardiovascular disease, hypertension and stroke. One approach that might be beneficial would be to replace sugar-sweetened items with products manufactured with artificial sweeteners that provide sweet tastes but with fewer calories. Unfortunately, evidence now indicates that artificial sweeteners are also associated with increased risk of the same chronic diseases linked to sugar consumption. Several biologically plausible mechanisms may explain these counterintuitive negative associations. For example, artificial sweeteners can interfere with basic learning processes that serve to anticipate the normal consequences of consuming sugars, leading to overeating, diminished release of hormones such as GLP-1, and impaired blood glucose regulation. In addition, artificial sweeteners can alter gut microbiota in rodent models and humans, which can also contribute to impaired glucose regulation. Use of artificial sweeteners may also be particularly problematic in children since exposure to hypersweetened foods and beverages at young ages may have effects on sweet preferences that persist into adulthood. Taken as a whole, current evidence suggests that a focus on reducing sweetener intake, whether the sweeteners are caloric or non-caloric, remains a better strategy for combating overweight and obesity than use of artificial sweeteners.

http://www.ncbi.nlm.nih.gov/pubmed/25828597

Environmental Science And Pollution Research International • November 2015

Antibiotics and sweeteners in the aquatic environment: biodegradability, formation of phototransformation products, and in vitro toxicity

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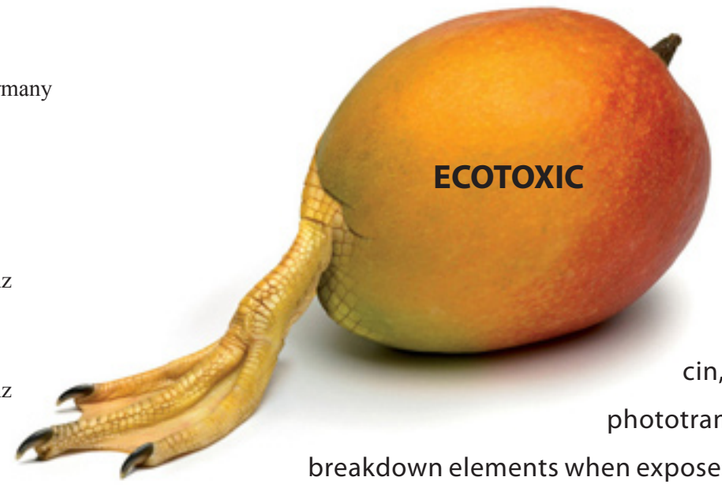
Abstract

In the present study, in vitro toxicity as well as biopersistence and photopersistence of four artificial sweeteners (acesulfame, cyclamate, saccharine, and sucralose) and five antibiotics (levofloxacin, lincomycin, linezolid, marbofloxacin, and sarafloxacin) and of their phototransformation products (PTPs) were investigated. Furthermore, antibiotic activity was evaluated after UV irradiation and after exposure to inocula of a sewage treatment plant. The study reveals that most of the tested compounds and their PTPs were neither readily nor inherently biodegradable in the Organisation for Economic Co-operation and Development (OECD)-biodegradability tests. The study further demonstrates that PTPs are formed upon irradiation with an Hg lamp (UV light) and, to a lesser extent, upon irradiation with a Xe lamp (mimics sunlight). Comparing the nonirradiated with the corresponding irradiated solutions, a higher chronic toxicity against bacteria was found for the irradiated solutions of linezolid. Neither cytotoxicity nor genotoxicity was found in human cervical (HeLa) and liver (Hep-G2) cells for any of the investigated compounds or their PTPs. Antimicrobial activity of the tested fluoroquinolones was reduced after UV treatment, but it was not reduced after a 28-day exposure to inocula of a sewage treatment plant. This comparative study shows that PTPs can be formed as a result of UV treatment. The study further demonstrated that UV irradiation can be effective in reducing the antimicrobial activity of antibiotics, and consequently may help to reduce antimicrobial resistance in wastewaters. Nevertheless, the study also highlights that some PTPs may exhibit a higher ecotoxicity than the respective parent compounds. Consequently, UV treatment does not transform all micropollutants into harmless compounds and may not be a large-scale effluent treatment option.



http://www.ncbi.nlm.nih.gov/pubmed/26169816

In Palatable Terms

In the present study, in vitro toxicity as well as biopersistence and photopersistence of four artificial sweeteners—acesulfame, cyclamate, saccharine, and sucralose, and five antibiotics levofloxacin, lincomycin, linezolid, marbofloxacin, and sarafloxacin—and of their phototransformation products or PTPs—their chemical breakdown elements when exposed to ultraviolet light from the sun—were investigated. The study reveals that most of the tested compounds and their phototransformation products were neither readily nor inherently biodegradable ... the study also highlights that some phototransformation products may exhibit a higher ecotoxicity than the respective parent compounds. Consequently, UV waste water treatment does not transform all micropollutants into harmless compounds and may not be a large-scale effluent treatment option for community treatment plants.

MANNITOL

Mannitol is a white, crystalline solid that looks and tastes sweet like sucrose. Medically it is used to treat increased intracranial pressure. It reduces pressure in the brain. It also has several industrial uses. In plants its purpose is to alleviate osmotic stress. Serious side effects may include worsening heart failure, electrolyte abnormalities, or low blood volume. It is unclear if it is safe in pregnancy. Mannitol is classified as a sugar alcohol; that is, it is derived from a sugar (mannose) by reduction. Other sugar alcohols include xylitol and sorbitol.

It is on the World Health Organization's List of Essential Medicines, the most important medications needed in a basic health system. It was originally isolated from the flowering ash and called manna after its supposed resemblance to the Biblical food.

Medical Uses

Mannitol is used to reduce acutely raised intracranial pressure until more definitive treatment can be applied, e.g., after head trauma. It may also be used for certain cases of kidney failure with low urine output, decreasing pressure in the eye, to increase the elimination of certain toxins, ciguatera fish poisoning, and to treat fluid build up. Mannitol acts as an osmotic laxative in oral doses larger than 20 grams and is sometimes sold as a laxative for children. Mannitol is also commonly used in the circuit prime of a heart lung machine during cardiopulmonary bypass. The presence of mannitol preserves renal function during the times of low blood flow and pressure, while the patient is on bypass. The solution prevents the swelling of endothelial cells in the kidney, which may have otherwise reduced blood flow to this area and resulted in cell damage.

Mannitol can also be used to temporarily encapsulate a sharp object (such as a helix on a lead for an artificial pacemaker) while it is passed through the venous system. Because the mannitol dissolves readily in blood, the sharp point will become exposed at its destination. Mannitol is the primary ingredient of Mannitol Salt Agar, a bacterial growth medium, and is used in other laboratory growth mediums. Mannitol is also the first drug of choice for the treatment of acute glaucoma in veterinary medicine. It is administered as a 20% solution IV. It dehydrates the vitreous humor and, therefore, lowers the intraocular pressure.

Researchers from Tel Aviv University describe experiments that could lead to a new approach for treating Parkinson's disease using the common sweetener, mannitol. These findings were confirmed by a second study which measured the impact of mannitol on mice engineered to produce human α-synuclein, developed by Dr. Eliezer Masliah of the University of California, San Diego. After four months, the researchers found that the mice injected with mannitol also showed a dramatic reduction of α-synuclein in the brain possibly making it an effective Parkinson's Disease treatment. All chemicals added to our food are actually drugs and they were always drugs, first.

Food Uses

Mannitol increases blood glucose to a lesser extent than sucrose (thus having a relatively low glycemic index) and is therefore used as a sweetener for people with diabetes, and in chewing gums. Although mannitol has a higher heat of solution than most sugar alcohols, its comparatively low solubility reduces the cooling effect usually found in mint candies and gums. However, when mannitol is completely dissolved in a product, it induces a strong cooling effect. It has a very low hygroscopicity – it does not pick up water from the air until the humidity level is 98%. This makes mannitol very useful as a coating for hard candies, dried fruits, and chewing gums, and it is often included as an ingredient in candies and chewing gum. The pleasant taste and mouthfeel of mannitol also makes it a popular excipient for chewable tablets. You'll find Mannitol in the very Pepto Bismol and many dozens if not hundreds of additional over-the-counter drugs. Mannitol is a drug. The terms "Food Additive" are a misnomer. All chemicals added to food are drugs.

MANNITOL IN PEPTO BISMAL

Chewable Tablets

Each peppermint-flavoured chewable tablet contains bismuth subsalicylate 262 mg. Nonmedicinal ingredients: calcium carbonate, magnesium stearate, **mannitol, peppermint flavour, PVP, Red 27 Lake, sodium saccharin, and talc**.

Caplets

Each caplet contains bismuth subsalicylate 262 mg. Nonmedicinal ingredients: calcium carbonate, magnesium stearate, **mannitol, microcrystalline cellulose, polysorbate 80, PVP, Red 27 Lake, silica, and sodium carboxymethyl starch.**

“Mannitol administration at peripheral hospitals is prone to dosing error”

Journal Of Neurosurgery • November 2015

Mannitol dosing error during inter-facility transfer for intracranial emergencies

Author information

Elliott CA1, MacKenzie M2, O’Kelly CJ1.

Abstract

OBJECT

Mannitol is commonly used to treat elevated intracranial pressure (ICP). The authors analyzed mannitol dosing errors at peripheral hospitals prior to or during transport to tertiary care facilities for intracranial emergencies. They also investigated the appropriateness of mannitol use based on the 2007 Brain Trauma Foundation guidelines for severe traumatic brain injury.

METHODS

The authors conducted a retrospective review of the Shock Trauma Air Rescue Society (STARS) electronic patient database of helicopter medical evacuations in Alberta, Canada, between 2004 and 2012, limited to patients receiving mannitol before transfer. They extracted data on mannitol administration and patient characteristics, including diagnosis, mechanism, Glasgow Coma Scale score, weight, age, and pupil status.

RESULTS

A total of 120 patients with an intracranial emergency received a mannitol infusion initiated at a peripheral hospital (median Glasgow Coma Scale score 6; range 3-13). Overall, there was a 22% dosing error rate, which comprised an underdosing rate (<0.25 g/kg) of 8.3% (10 of 120 patients), an overdosing rate (>1.5 g/kg) of 7.5% (9 of 120), and a nonbolus administration rate (>1 hour) of 6.7% (8 of 120). Overall, 72% of patients had a clear indication to receive mannitol as defined by meeting at least one of the following criteria based on Brain Trauma Foundation guidelines: neurological deterioration (11%), severe traumatic brain injury (69%), or pupillary abnormality (25%).

CONCLUSIONS

Mannitol administration at peripheral hospitals is prone to dosing error. Strategies such as a pretransport checklist may mitigate this risk.

http://www.ncbi.nlm.nih.gov/pubmed/26077141

“... several epidemiological studies have found that consumption of non-nutritive sweeteners (NNSs), mainly in diet sodas, is associated with increased risk to develop obesity, metabolic syndrome, and type 2 diabetes ... recent findings from our laboratory .. support the notion that non-nutritive sweeteners have [medically undesirable] metabolic effects.”

Physiology And Behavior • December 2015

Metabolic effects of non-nutritive sweeteners

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Abstract

Until recently, the general belief was that non-nutritive sweeteners (NNSs) were healthy sugar substitutes because they provide sweet taste without calories or glycemic effects. However, data from several epidemiological studies have found that consumption of NNSs, mainly in diet sodas, is associated with increased risk to develop obesity, metabolic syndrome, and type 2 diabetes. The main purpose of this article is to review recent scientific evidence supporting potential mechanisms that explain how “metabolically inactive” NNSs, which have few, if any, calories, might promote metabolic dysregulation. Three potential mechanisms, which are not mutually exclusive, are presented: 1) NNSs interfere with learned responses that contribute to control glucose and energy homeostasis, 2) NNSs interfere with gut microbiota and induce glucose intolerance, and 3) NNSs interact with sweet-taste receptors expressed throughout the digestive system that play a role in glucose absorption and trigger insulin secretion. In addition, recent findings from our laboratory showing an association between individual taste sensitivity to detect sucralose and sucralose’s acute effects on metabolic response to an oral glucose load are reported. Taken as a whole, data support the notion that NNSs have metabolic effects. More research is needed to elucidate the mechanisms by which NNSs may drive metabolic dysregulation and better understand potential effects of these commonly used food additives.

http://www.ncbi.nlm.nih.gov/pubmed/26095119

> “Here we argue that Non Nutritive Sweeteners are not physiologically inert compounds and consider the potential biological mechanisms by which Non Nutritive Sweetener consumption may impact energy balance and metabolic function, including actions on oral and extra-oral sweet taste receptors, and effects on metabolic hormone secretion, cognitive processes (e.g. reward learning, memory, and taste perception), and gut microbiota.”

Physiology & Behavior • December 2015

Physiological mechanisms by which non-nutritive sweeteners may impact body weight and metabolism

Author information

By M.V. Burke MV[1] and D.M. Small[2]

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Abstract

Evidence linking sugar-sweetened beverage (SSB) consumption to weight gain and other negative health outcomes has prompted many individuals to resort to artificial, non-nutritive sweetener (NNS) substitutes as a means of reducing SSB intake. However, there is a great deal of controversy regarding the biological consequences of NNS use, with accumulating evidence suggesting that NNS consumption may influence feeding and metabolism via a variety of peripheral and central mechanisms. Here we argue that NNSs are not physiologically inert compounds and consider the potential biological mechanisms by which NNS consumption may impact energy balance and metabolic function, including actions on oral and extra-oral sweet taste receptors, and effects on metabolic hormone secretion, cognitive processes (e.g. reward learning, memory, and taste perception), and gut microbiota.

http://www.ncbi.nlm.nih.gov/pubmed/26048305

> “These results indicate that aspartame induces apoptosis mainly via mitochondrial pathway involved in apoptosis due to oxygen toxicity.”

Environmental Toxicology And Pharmacology • January 2014

Aspartame-induced apoptosis in PC12 cells

Author information

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Abstract

Aspartame is an artificial sweetner added to many low-calorie foods. The safety of aspartame remains controversial even though there are many studies on its risks. In this study, to understand the physiological effects of trace amounts of artificial sweetners on cells, the effects of aspartame on apoptosis were investigated using a PC12 cell system. In addition, the mechanism of apoptosis induced by aspartame in PC12 cells and effects on apoptotic factors such as cytochrome c, apoptosis-inducing factor, and caspase family proteins were studied by Western blotting and RT-PCR. Aspartame-induced apoptosis in PC12 cells in a dose-dependent manner. In addition, aspartame exposure increased the expressions of caspases 8 and 9, and cytochrome c. These results indicate that aspartame induces apoptosis mainly via mitochondrial pathway involved in apoptosis due to oxygen toxicity.

http://www.ncbi.nlm.nih.gov/pubmed/24355796

PLoS One • March 2014

Rate of atherosclerosis progression in ApoE-/- mice long after discontinuation of cola beverage drinking

Author information

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Abstract

This study was conducted in order to evaluate the effect of cola beverages drinking on atherosclerosis and test the hypothesis whether cola beverages consumption at early life stages might affect the development and progression of atherosclerosis later in life. ApoE-/- C57BL/6J mice (8 week-old) were randomized in 3 groups (n=20 each) according to free access to water (W), sucrose sweetened carbonated cola drink(C) or aspartame-acesulfame K sweetened carbonated 'light' cola drink (L) for the next 8 weeks. Drinking treatment was ended by switching C and L groups to drinking water. Four mice per group and time were sequentially euthanized: before treatment (8 weeks-old), at the end of treatment (16 weeks-old) and after treatment discontinuation (20 weeks-old, 24 weeks-old, 30 week-old mice). Aortic roots and livers were harvested, processed for histology and serial cross-sections were stained. Aortic plaque area was analyzed and plaque/media-ratio was calculated. Early consumption of cola drinks accelerated atherosclerotic plaque progression favoring the interaction between macrophages and myofibroblasts, without the participation of either T lymphocytes or proliferative activity. Plaque/media-ratio varied according to drink treatment ($F_{2,54}=3.433$, $p<0.04$) and mice age ($F_{4,54}=5.009$, $p<0.03$) and was higher in C and L groups compared with age-matched W group ($p<0.05$ at 16 weeks and 20 weeks, $p<0.01$ at 24 weeks and 30 weeks). Natural evolution of atherosclerosis in ApoE-/- mice (W group) evidenced atherosclerosis acceleration in parallel with a rapid increase in liver inflammation around the 20 weeks of age. Cola drinking within the 8-16 weeks of age accelerated atherosclerosis progression in ApoE-/- mice favoring aortic plaque enlargement (inward remodeling) over media thinning all over the study time. Data suggest that cola drinking at early life stages may predispose to atherosclerosis progression later in life in ApoE-/- mice.

Full Text with Graphs, Charts and 27 References

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3966732/

“Data suggest that cola drinking at early life stages may predispose to atherosclerosis progression later in life ...”

Neurotoxicology And Teratology • March 2014

Cognitive and biochemical effects of monosodium glutamate and aspartame, administered individually and in combination in male albino mice

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Abstract

The present study was designed to investigate the in vivo effects of monosodium glutamate (MSG) and aspartame (ASM) individually and in combination on the cognitive behavior and biochemical parameters like neurotransmitters and oxidative stress indices in the brain tissue of mice. Forty male Swiss albino mice were randomly divided into four groups of ten each and were exposed to MSG and ASM through drinking water for one month. Group I was the control and was given normal tap water. Groups II and III received MSG (8 mg/kg) and ASM (32 mg/kg) respectively dissolved in tap water. Group IV received MSG and ASM together in the same doses. After the exposure period, the animals were subjected to cognitive behavioral tests in a shuttle box and a water maze. Thereafter, the animals were sacrificed and the neurotransmitters and oxidative stress indices were estimated in their forebrain tissue. Both MSG and ASM individually as well as in combination had significant disruptive effects on the cognitive responses, memory retention and learning capabilities of the mice in the order (MSG+ASM)>ASM>MSG. Furthermore, while MSG and ASM individually were unable to alter the brain neurotransmitters and the oxidative stress indices, their combination dose (MSG+ASM) decreased significantly the levels of neurotransmitters (dopamine and serotonin) and it also caused oxidative stress by increasing the lipid peroxides measured in the form of thiobarbituric acid-reactive substances (TBARS) and decreasing the level of total glutathione (GSH). Further studies are required to evaluate the synergistic effects of MSG and ASM on the neurotransmitters and oxidative stress indices and their involvement in cognitive dysfunctions.

http://www.ncbi.nlm.nih.gov/pubmed/24556450

“Both MSG and ASM individually as well as in combination had significant disruptive effects on the cognitive responses, memory retention and learning capabilities of the mice in the order (MSG+ASM)>ASM>MSG. Furthermore, while MSG and ASM individually were unable to alter the brain neurotransmitters and the oxidative stress indices, their combination dose (MSG+ASM) decreased significantly the levels of neurotransmitters (dopamine and serotonin) and it also caused oxidative stress ...”

SYNERGY

Synergy is the interaction or cooperation of two or more organizations, substances, or other agents to produce a combined effect greater than the sum of their separate effects. So, for example, when aluminum and mercury molecules meet within the body their negative neurological effects can be as much as 100 times greater than with either element alone. So what’s the synergy, at a molecular scale, of aspartame and MSG? What about nicotine, air pollution, fluoridated water and sucralose? What’s the synergy between those 4 elements? We don’t know and we’re just now beginning to investigate drug synergies and you’ll see several reports here that discuss synergy. It’s about time. Yet it’s an exhaustively complex science combined with a real lack of scientific interest so don’t expect much peer review on synergy anytime soon. A smattering here and there, like this one and just a few more.

“These joint responses support our hypothesis and suggest that exposure to sucralose may induce neurological and oxidative mechanisms with potentially important consequences for animal behaviour and physiology.”

PLoS One • April 2014

Sucralose induces biochemical responses in Daphnia magna

Author information

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Abstract

The intense artificial sweetener sucralose has no bioconcentration properties, and no adverse acute toxic effects have been observed in standard ecotoxicity tests, suggesting negligible environmental risk. However, significant feeding and behavioural alterations have been reported in non-standard tests using aquatic crustaceans, indicating possible sublethal effects. We hypothesized that these effects are related to alterations in acetylcholinesterase (AChE) and oxidative status in the exposed animals and investigated changes in AChE and oxidative biomarkers (oxygen radical absorbing capacity, ORAC, and lipid peroxidation, TBARS) in the crustacean Daphnia magna exposed to sucralose (0.0001-5 mg L(-1)). The sucralose concentration was a significant positive predictor for ORAC, TBARS and AChE in the daphnids. Moreover, the AChE response was linked to both oxidative biomarkers, with positive and negative relationships for TBARS and ORAC, respectively. These joint responses support our hypothesis and suggest that exposure to sucralose may induce neurological and oxidative mechanisms with potentially important consequences for animal behaviour and physiology.

Full text, graph and 63 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3974716/

Smucker’s Strawberry Preserves - Sugar Free
INGREDIENTS

Water, Strawberries, **Polydextrose, Maltodextrin,** Fruit Pectin, **Locust Bean Gum**, **Natural Flavor**, Citric Acid, **Potassium Sorbate , Sucralose, Calcium Chloride , Red 40.**

Contains controversial artificial sweeteners

There is controversy as to the safety of artificial sweeteners consumed over a long period of time. Some studies have linked artificial sweeteners to cancer and other diseases.

Contains controversial artificial colors

Once upon a time, there were no food colorings. Then folks figured out that food looks better and sells more when it can be enlivened through dyes. For most of food history, the dyes were from natural sources – beet juice for red, turmeric for yellow, etc… However, in the quest to increase color intensity and lower manufacturing costs, cheap artificial petroleum based dyes were introduced to market. Unfortunately they pose a risk for hyperactivity in children, cancer, and allergic reactions.

Highly Processed!

This product is highly processed. If you’ll take a look at its ingredient list, you might discover new words to add to your vocabulary. When that happens it’s time to put that product back on the store shelf where it belongs. Many of theses ingredients are required to increase the shelf life of the product and improve the flavor that disappears when food is not fresh. Most of these ingredients have negative reviews in the peer reviewed literature.

“Taken together, the studies performed by G.D. Searle in the 1970s and other chronic bioassays do not provide adequate scientific support for Aspartame safety. In contrast, recent results of life-span carcinogenicity bioassays on rats and mice published in peer-reviewed journals, and a prospective epidemiological study, provide consistent evidence of Aspartame’s carcinogenic potential.”

American Journal Of Industrial Medicine • April 2014

The carcinogenic effects of aspartame: The urgent need for regulatory re-evaluation

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Abstract

Aspartame (APM) is an artificial sweetener used since the 1980s, now present in >6,000 products, including over 500 pharmaceuticals. Since its discovery in 1965, and its first approval by the US Food and Drugs Administration (FDA) in 1981, the safety of APM, and in particular its carcinogenicity potential, has been controversial. The present commentary reviews the adequacy of the design and conduct of carcinogenicity bioassays on rodents submitted by G.D. Searle, in the 1970s, to the FDA for market approval. We also review how experimental and epidemiological data on the carcinogenic risks of APM, that became available in 2005 motivated the European Commission (EC) to call the European Food and Safety Authority (EFSA) for urgent re-examination of the available scientific documentation (including the Searle studies). The EC has further requested that, if the results of the evaluation should suggest carcinogenicity, major changes must be made to the current APM specific regulations. Taken together, the studies performed by G.D. Searle in the 1970s and other chronic bioassays do not provide adequate scientific support for APM safety. In contrast, recent results of life-span carcinogenicity bioassays on rats and mice published in peer-reviewed journals, and a prospective epidemiological study, provide consistent evidence of APM’s carcinogenic potential. On the basis of the evidence of the potential carcinogenic effects of APM herein reported, a re-evaluation of the current position of international regulatory agencies must be considered an urgent matter of public health.

http://www.ncbi.nlm.nih.gov/pubmed/24436139

All Trident Gum Contains Aspartame

Trident
Ingredients
Strawberry Twist

SORBITOL, GUM BASE, **XYLITOL, GLYCERIN, NATURAL AND ARTIFICIAL FLAVORING, MANNITOL**; LESS THAN 2% OF **ACESULFAME POTASSIUM**, **ASPARTAME**, **BHT** (TO MAINTAIN FRESHNESS), **RED 40 LAKE**, **SOY LECITHIN. CONTAINS SOY. PHENYLKETONURICS: CONTAINS PHENYLALANINE.**

Trident Ingredients
Trident Bubble Gum

SORBITOL, GUM BASE, **XYLITOL, GLYCERIN; LESS THAN 2% OF ACESULFAME POTASSIUM, ASPARTAME, BHT (TO MAINTAIN FRESHNESS), MANNITOL, NATURAL AND ARTIFICIAL FLAVORING, RED 40 LAKE, SOY LECITHIN, SUCRALOSE. CONTAINS SOY. PHENYLKETONURICS: CONTAINS PHENYLALANINE**.

Trident Ingredients Splashing Fruit

SORBITOL, GUM BASE, **XYLITOL, NATURAL AND ARTIFICIAL FLAVORING, GLYCERIN, MANNITOL; LESS THAN 2% OF: ACESULFAME POTASSIUM, ASPARTAME, BHT (TO MAINTAIN FRESHNESS), BLUE 1, PECTIN, RED 40, SOY LECITHIN, SUCRALOSE, YELLOW 5, YELLOW 5 LAKE, YELLOW 6 AND YELLOW 6 LAKE.**

> “It is clear that long term aspartame exposure could alter the brain antioxidant status, and can induce apoptotic changes in [the] brain.”

Redox Biology • April 2014

Biochemical responses and mitochondrial mediated activation of apoptosis on long-term effect of aspartame in rat brain

Author information
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Abstract

Aspartame, an artificial sweetener, is very widely used in many foods and beverages. But there are controversies about its metabolite which is marked for its toxicity. Hence it is believed to be unsafe for human use. Previous studies have reported on methanol exposure with involvements of free radicals on excitotoxicity of neuronal apoptosis. Hence, this present study is proposed to investigate whether or not chronic aspartame (FDA approved Daily Acceptable Intake (ADI),40 mg/kg bwt) administration could release methanol, and whether or not it can induce changes in brain oxidative stress status and gene and protein expression of anti-apoptotic Bcl-2 and pro-apoptotic Bax and caspase-3 in the rat brain region. To mimic the human methanol metabolism, Methotrexate (MTX)-treated Wistar strain male albino rats were used and after the oral administration of aspartame, the effects were studied along with controls and MTX-treated controls. Aspartame exposure resulted with a significant increase in the enzymatic activity in protein carbonyl, lipid peroxidation levels, superoxide dismutase, glutathione-S-transferase, glutathione peroxidase and catalase activity in (aspartame MTX)-treated animals and with a significant decrease in reduced glutathione, glutathione reductase and protein thiol, pointing out the generation of free radicals. The gene and protein expression of pro apoptotic marker Bax showed a marked increase whereas the anti-apoptotic marker Bcl-2 decreased markedly indicating the aspartame is harmful at cellular level. It is clear that long term aspartame exposure could alter the brain antioxidant status, and can induce apoptotic changes in brain.

Full text, graphs, charts and 85 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4085354/

BUGS BUNNY
Vitamin Products

Ingredients: **Dicalcium Phosphate, Sorbitol, Magnesium Phosphate, Sodium Ascorbate, Gelatin, Ferrous Fumarate, Natural and Artificial Flavors (including fruit acids), Starch, Stearic Acid, FD&C Red #40 Lake, Vitamin E Acetate, Carrageenan**, Niacinamide, **Magnesium Stearate, Hydrogenated Vegetable Oil**, **Zinc Oxide, FD&C Yellow #6 Lake, FD&C Blue #2 Lake**, Calcium Pantothenate, **ASPARTAME**** (a sweetener), **Cupric Oxide, Pyridoxine Hydrochloride**, Vitamin A Acetate, Riboflavin, Thiamine Mononitrate, Beta Carotene, Folic Acid, Potassium Iodide, Vitamin D, Biotin, Magnesium Oxide, Vitamin B 12 .

“... pharmacological perturbations during perinatal growth can cause persistent effects on the function of white adipose tissue, altering susceptibility to obesity later in life. Accordingly, the current study established a developmental period in which saccharin at high concentrations reduces adiposity and increases lean and bone mass in male mice while decreasing generalized growth in female mice.”

Endocrinology • April 2014

Administration of saccharin to neonatal mice influences body composition of adult males and reduces body weight of females

Author information

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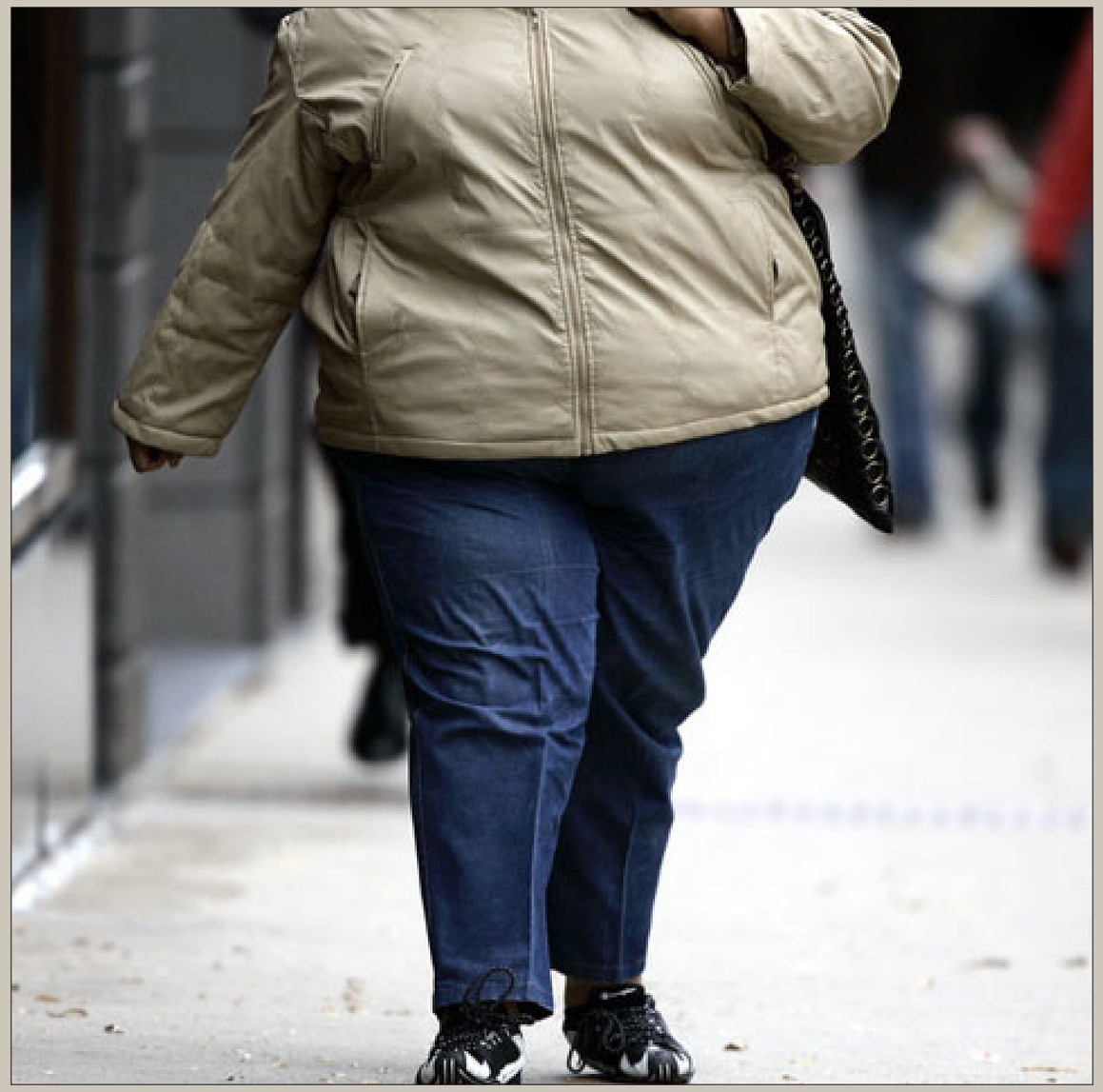
Abstract

Nutritional or pharmacological perturbations during perinatal growth can cause persistent effects on the function of white adipose tissue, altering susceptibility to obesity later in life. Previous studies have established that saccharin, a nonnutritive sweetener, inhibits lipolysis in mature adipocytes and stimulates adipogenesis. Thus, the current study tested whether neonatal exposure to saccharin via maternal lactation increased susceptibility of mice to diet-induced obesity. Saccharin decreased body weight of female mice beginning postnatal week 3. Decreased liver weights on week 14 corroborated this diminished body weight. Initially, saccharin also reduced male mouse body weight. By week 5, weights transiently rebounded above controls, and by week 14, male body weights did not differ. Body composition analysis revealed that saccharin increased lean and decreased fat mass of male mice, the latter due to decreased adipocyte size and epididymal, perirenal, and sc adipose weights. A mild improvement in glucose tolerance without a change in insulin sensitivity or secretion aligned with this leaner phenotype. Interestingly, microcomputed tomography analysis indicated that saccharin also increased cortical and trabecular bone mass of male mice and modified cortical bone alone in female mice. A modest increase in circulating testosterone may contribute to the leaner phenotype in male mice. Accordingly, the current study established a developmental period in which saccharin at high concentrations reduces adiposity and increases lean and bone mass in male mice while decreasing generalized growth in female mice.

Full text, graphs, charts and 92 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3959603/



Water Research • April 2014

Evaluating the environmental impact of artificial sweeteners: a study of their distributions, photodegradation and toxicities

Author information

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Abstract

While having a long tradition as safe food additives, artificial sweeteners are a newly recognized class of environmental contaminants due to their extreme persistence and ubiquitous occurrence in various aquatic ecosystems. Resistant to wastewater treatment processes, they are continuously introduced into the water environments. To date however, their environmental behavior, fate as well as long term ecotoxicological contributions in our water resources still remain largely unknown. As a first step in the comprehensive study of artificial sweeteners, this work elucidates the geographical/seasonal/hydrological interactions of acesulfame, cyclamate, saccharin and sucralose in an open coast system at an estuarine/marine junction. Higher occurrence of acesulfame (seasonal average: 0.22 μg L(-1)) and sucralose (0.05 μg L(-1)) was found in summer while saccharin (0.11 μg L(-1)) and cyclamate (0.10 μg L(-1)) were predominantly detected in winter. Seasonal observations of the four sweeteners suggest strong connections with the variable chemical resistance among different sweeteners. Our photodegradation investigation further projected the potential impact of persistent acesulfame and sucralose compounds under prolonged exposure to intensive solar irradiation. Real-time observation by UPLC-ESI/MS of the degradation profile in both sweeteners illustrated that formation of new photo by-products under prolonged UV irradiation is highly viable. Interestingly, two groups of kinetically behaved photodegradates were identified for acesulfame, one of which was at least six times more persistent than the parent compound. For the first time, acute toxicity for the degradates of both sweeteners were arbitrarily measured, revealing photo-enhancement factors of 575 and 17.1 for acesulfame and sucralose, respectively. Direct comparison of photodegradation results suggests that the phototoxicity of acesulfame degradation products may impact aquatic ecosystems. In an attempt to neutralize this prolonged environmental threat, the feasibility of UV/TiO2 as an effective mineralization process in wastewater treatment was evaluated for both sweeteners. Under an environmental and technical relevant condition, a >84% removal rate recorded within 30 min and complete photomineralization was achieved within 2 h and delivering the best cost efficiency comparing to existing removal methods. A compilation of distribution, degradation, toxicity and attenuation results presented in this paper will go through critical discussions to explore some current issues and to pinpoint solutions for a better control in the emergent contamination of artificial sweeteners.

http://www.ncbi.nlm.nih.gov/pubmed/24289948

“... artificial sweeteners are a newly recognized class of environmental contaminants due to their extreme persistence and ubiquitous occurrence in various aquatic ecosystems.”

FULL THROTTLE 16oz

This garbage contains **58 grams—14.5 teaspoons—of sugar in one can, almost 12 times the recommended daily allowance of 5 grams of sugar per person per day.** It also contains the following ingredients:

Carbonated Water, **High Fructose Corn Syrup**, Citric Acid, **Sugar, Natural And Artificial Flavors,** Sodium Citrate, Acacia, Niacinamide (Vitamin B3), Calcium Pantothenate (Vitamin B5), **Glycerol Ester of Rosin, Yellow 5**, Pyridoxine Hydrochloride (Vitamin B6), and Cyanocobalamin (Vitamin B12).

To be sure, NOS High Performance Energy Drink to the left of Full Throttle is just as bad.

“Cerebellar cortex is considered target areas of Aspartame neurotoxicity, while P. anisum oil [Anise Oil], when used in combination with Aspartame displays a protective action against neurotoxicity.”

Ultrastructural Pathology • May 2014

Ameliorative effect of Pimpinella anisum oil on immunohistochemical and ultrastuctural changes of cerebellum of albino rats induced by aspartame

Author information

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Abstract

The study aims to investigate the protective effect of Pimpinella anisum oil on aspartame (ASP) which resulted in cerebellar changes. The rats were divided into four equal groups: Group 1: (control group): served as control animals. Group 2: control P. anisum oil received .5 mL/kg/d/b wt. once daily. Group 3 (ASP group): received daily 250 mg/kg/b wt. of ASP dissolved in distilled water and given orally to the animals by intra-gastric tube for 2 months. Group 4: received .5 mL/kg/b wt. of prophylactic P. anisum oil once daily, followed by ASP after 2 h for 2 months. The histopathological approach revealed marked changes in the Purkinje cells, myleinated nerve fibers and granular cells of ASP-treated animals. Some of these cells appeared with deeply stained cytoplasm. Ultrastructural examination showed Purkinje cells with dilated rough endoplasmic reticulum and condensed mitochondria. Granular cells appeared with less c nuclei and surrounded by dissolution of most Mossy rosettes structures. Most myelinated nerve fibers showed thickening of myelinated sheath and others showed splitting of their myelin sheath. The histopathological, immunohistochemical and ultrastructural alterations were much less observed in concomitant use of P. anisum oil with ASP. Cerebellar cortex is considered target areas of ASP neurotoxicity, while P. anisum oil, when used in combination with ASP displays a protective action against neurotoxicity.

http://www.ncbi.nlm.nih.gov/pubmed/24684500

P. ANISUM OIL • ANISE

Anise, also called aniseed, is a flowering plant in the family Apiaceae native to the eastern Mediterranean region and Southwest Asia. Its flavor has similarities with some other spices, such as star anise, fennel, and licorice. Anise is sweet and very aromatic, distinguished by its characteristic flavor. The seeds, whole or ground, are used for preparation of tea (alone or in combination with other aromatic herbs), as well as in a wide variety of regional and ethnic confectioneries, including black jelly beans, British aniseed balls, Australian humbugs, New Zealand aniseed wheels, Italian pizzelle, German Pfeffernüsse and Springerle, Austrian Anisbögen, Dutch muisjes, New Mexican bizcochitos, and Peruvian picarones. It is a key ingredient in Mexican atole de anís and champurrado, which is similar to hot chocolate, and it is taken as a digestive after meals in India. The Ancient Romans often served spiced cakes with aniseed called mustaceoe at the end of feasts as a digestive aid. This tradition of serving cake at the end of festivities is the basis for the tradition of serving cake at weddings.

Anise is used to flavor Middle Eastern arak; Colombian aguardiente; French absinthe, anisette, and pastis; Greek ouzo; Bulgarian and Macedonian mastika; German Jägermeister; Swiss Appenzeller Alpenbitter; Italian sambuca; Dutch Brokmöpke; Portuguese, Peruvian, and Spanish anísado and Herbs de Majorca; Mexican Xtabentún; and Turkish rakı. These liquors are clear, but on addition of water become cloudy, a phenomenon known as the ouzo effect. It is believed to be one of the secret ingredients in the French liqueur Chartreuse. It is also used in some root beers, such as Virgil’s in the United States.

The main use of anise in traditional European herbal medicine was for its carminative effect (reducing flatulence), as noted by John Gerard in his Great Herball, an early encyclopedia of herbal medicine: “*The seed wasteth and consumeth winde, and is good against belchings and upbraidings of the stomacke, alaieth gripings of the belly, provoketh urine gently, maketh abundance of milke, and stirreth up bodily lust: it staieth the laske (diarrhea), and also the white flux (leukorrhea) in women*.” Anise has also been used to treat menstrual cramps and colic. The essential oil has reportedly been used as an insecticide against head lice and mites. Pictured at right, a bowl of Anise seed.

“Acesulfame K may increase Intestinal Fat Deposition in presence of insulin resistance.”

Chemico-biological Interactions • May 2014

Effects of three intense sweeteners on fat storage in the C. elegans model

Author information

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Abstract

Beverages sweetened with caloric sweeteners (CS), glucose, sucrose or high-fructose corn syrup, are associated with weight gain. Beverages sweetened with intense sweeteners (IS) are marketed as low-calorie substitutes to prevent beverages-associated weight gain. Using Caenorhabditis elegans, the effects on intestinal fat deposition (IFD) and pharyngeal pumping rate (PPR) of cola beverages sweetened with glucose, aspartame, or aspartame plus acesulfame-potassium (AceK) were compared. Control groups received Escherichia coli (OP50) only. Study I: the nematodes received additional glucose- or IS-sweetened beverages. Study II: the nematodes received additional glucose, aspartame, or aspartame plus AceK (AAK). Beverages containing CS or IS (aspartame or AAK) did not alter IFD in wild type (N2) or in daf-16 deficiency. The CS cola increased IFD in sir-2.1 deficiency ($P<0.05$). The AAK-cola increased IFD in daf-16/daf-2 deficiency and sir-2.1 deficiency ($P<0.05$). Glucose increased IFD in N2 and daf-16 deficiency ($P<0.05$). Aspartame showed a tendency towards reduced IFD in N2 and decreased IFD in daf-16/daf-2 deficiency ($P<0.05$). AAK increased IFD in daf-16 deficiency and sir-2.1 deficiency ($P<0.05$), and reversed the aspartame-induced reduction in IFD. The aspartame-sweetened cola increased the PPR in daf-16/daf-2 deficiency and daf-16 deficiency ($P<0.05$); similar results were obtained in N2 with both IS ($P<0.05$). AAK increased the PPR in daf-16/daf-2, daf-16, and sir-2.1 deficiencies ($P<0.05$). Thus, IS increased the PPR, a surrogate marker of lifespan. Aspartame may have an independent effect in reducing IFD to assist humans desiring weight loss. AceK may increase IFD in presence of insulin resistance.

http://www.ncbi.nlm.nih.gov/pubmed/24632416

“Saccharin consumption may have negative effects on sperm parameters, and increases the rate of sperm DNA fragmentation and apoptosis in mice.”

Iranian Journal Of Reproductive Medicine • May 2014

Saccharin consumption increases sperm DNA fragmentation and apoptosis in mice

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Abbasi Sarcheshmeh A1, Omidi M1.

Author information

Abstract

BACKGROUND

Saccharin is an artificial non-caloric sweetener that used to sweeten products such as drinks, candies, medicines, and toothpaste, but our bodies cannot metabolize it. Sodium saccharin is considered as an important factor in tumor promotion in male rats but not in humans.

OBJECTIVE

The objective of this study was to investigate the effect of saccharin consumption on sperm parameters and apoptosis in adult mice.

MATERIALS AND METHODS

Totally 14 adult male mice were divided into 2 groups. Group 1 served as control fed on basal diet and group 2 or experimental animals received distilled water containing saccharin (0.2% w/v) for 35 days. After that, the left cauda epididymis of each mouse was cut and placed in Ham’s F10. Swimmed-out spermatozoa were used to analyze count, motility, morphology (Pap-staining) and viability (eosin-Y staining). Sperm DNA integrity, as an indicator of apoptosis, was assessed by SCD (sperm chromatin dispersion) and terminal deoxynucleotidyl transferase (TUNEL) assay.

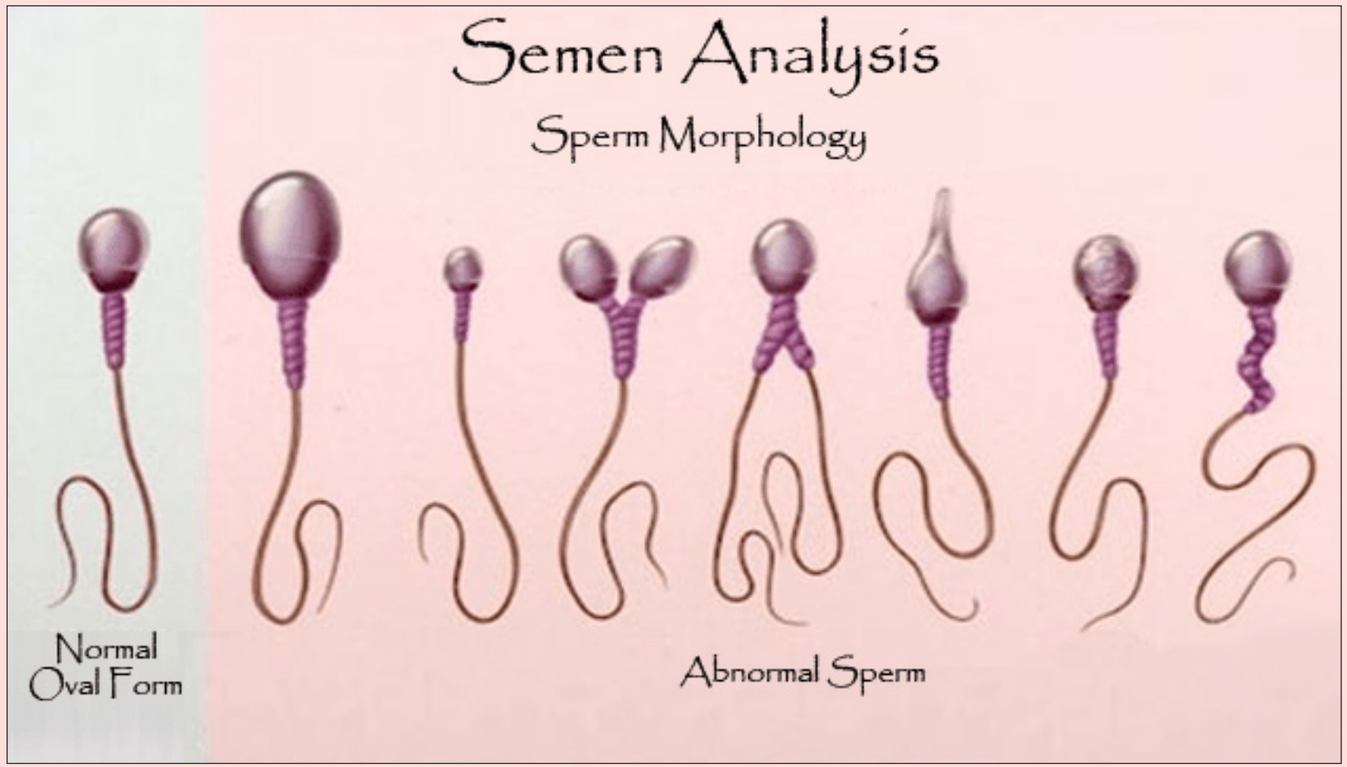
RESULTS

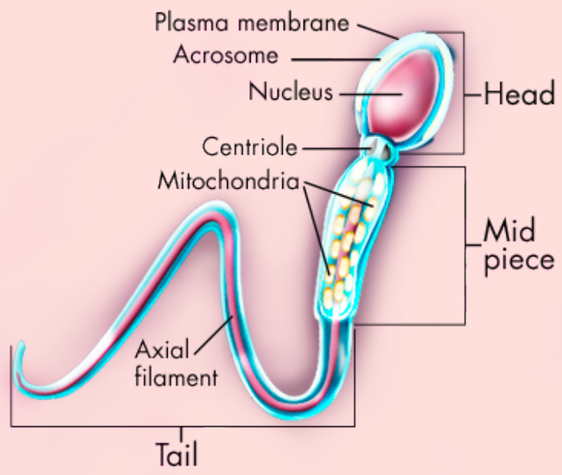
Following saccharin consumption, we had a reduction in sperm motility with respect to control animals (p=0.000). In addition, the sperm count diminished (17.70±1.11 in controls vs. 12.80±2.79 in case group, p=0.003) and the rate of sperm normal morphology decreased from 77.00±6.40 in control animals into 63.85±6.81 in saccharin-treated mice (p=0.001). Also, we saw a statistically significant increase in rates of sperm DNA damage and apoptosis in experimental group when compared to control one (p=0.001, p=0.002 respectively).

CONCLUSION

Saccharin consumption may have negative effects on sperm parameters, and increases the rate of sperm DNA fragmentation and apoptosis in mice.

http://www.ncbi.nlm.nih.gov/pubmed/25031574





“aspartame treatment significantly alters the tyrosine hydroxylase activity and amino acids levels in the brain. Our data suggest that chronic use of aspartame may affect electrolyte homeostasis and monoamine neurotransmitter synthesis dose dependently, and this might have a possible effect on cognitive functions.”

International Journal Of Toxicology • May 2014

Chronic Effect of Aspartame on Ionic Homeostasis and Monoamine Neurotransmitters in the Rat Brain

Author information

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Abstract

Aspartame is one of the most widely used artificial sweeteners globally. Data concerning acute neurotoxicity of aspartame is controversial, and knowledge on its chronic effect is limited. In the current study, we investigated the chronic effects of aspartame on ionic homeostasis and regional monoamine neurotransmitter concentrations in the brain. Our results showed that aspartame at high dose caused a disturbance in ionic homeostasis and induced apoptosis in the brain. We also investigated the effects of aspartame on brain regional monoamine synthesis, and the results revealed that there was a significant decrease of dopamine in corpus striatum and cerebral cortex and of serotonin in corpus striatum. Moreover, aspartame treatment significantly alters the tyrosine hydroxylase activity and amino acids levels in the brain. Our data suggest that chronic use of aspartame may affect electrolyte homeostasis and monoamine neurotransmitter synthesis dose dependently, and this might have a possible effect on cognitive functions.

http://www.ncbi.nlm.nih.gov/pubmed/24872471

EQUATE
Children's Multivitamin
Ingredients

Ingredients: **Dicalcium Phosphate**, **Sorbitol, Choline Bitartrate,** Sodium Ascorbate, Ferrous Fumarate, Magnesium Oxide, Mono & Diglycerides**, Gelatin**, Dl-Alpha Tocopheryl Acetate, **Talc, Natural and Artificial Flavors**, Stearic Acid, Niacinamide, Zinc Oxide, Calcium Pantothenate, Magnesium Stearate, **Silicon Dioxide, Aspartame (A Sweetener)**, **Hydrogenated Vegetable Oil (Cottonseed), Malic Acid, Fd&C Red #40 Lake, Fd&C Yellow #6 Lake,** Citric Acid**, Fd&C Blue #2 Lake**, Pyridoxine Hydrochloride, Cupric Oxide, Thiamin Mononitrate, Riboflavin, Monoammonium Glycyrrhizinate, Vitamin A Acetate, Beta-Carotene, Folic Acid, Potassium Iodide, Biotin, Cholecalciferol, Quassia Solid Extract, Cyanocobalamin, Gentian Solid Extract. **Phenylketonurics: Contains Phenylalanine.**

***Items in bold have been proven to cause harm to almost all humans in general and infants and children in particular.**

“Aspartame is one of the most widely used artificial sweeteners globally. Data concerning acute neurotoxicity of aspartame is controversial, and knowledge on its chronic effect is limited.”

Abstract

“Non-caloric artificial sweeteners” (NAS) are among the most widely used food additives worldwide, regularly consumed by lean and obese individuals alike. Non-caloric artificial sweetener consumption is considered safe and beneficial owing to their low caloric content, yet supporting scientific data remain sparse and controversial. Here we demonstrate that consumption of commonly used Non-caloric artificial sweetener formulations drives the development of glucose intolerance through induction of compositional and functional alterations to the intestinal microbiota. These Non-caloric artificial sweetener-mediated deleterious metabolic effects are abrogated by antibiotic treatment, and are fully transferrable to germ-free mice upon faecal transplantation of microbiota configurations from Non-caloric artificial sweetener-consuming mice, or of microbiota anaerobically incubated in the presence of Non-caloric artificial sweeteners. We identify Non-caloric artificial sweetener-altered microbial metabolic pathways that are linked to host susceptibility to metabolic disease, and demonstrate similar Non-caloric artificial sweetener-induced dysbiosis and glucose intolerance in healthy human subjects. Collectively, our results link Non-caloric artificial sweetener consumption, dysbiosis and metabolic abnormalities, thereby calling for a reassessment of massive Non-caloric artificial sweetener usage.”

http://www.ncbi.nlm.nih.gov/pubmed/25231862

Nature • October 2014

Artificial sweeteners induce glucose intolerance by altering the gut microbiota

Author information

Suez J1, Korem T2, Zeevi D2, Zilberman-Schapira G3, Thaiss CA1, Maza O1, Israeli D4, Zmora N5, Gilad S6, Weinberger A7, Kuperman Y8, Harmelin A8, Kolodkin-Gal I9, Shapiro H1, Halpern Z10, Segal E7, Elinav E1.

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“Suez et al. (2014) recently demonstrated that artificial sweeteners alter gut microbial communities, leading to glucose intolerance in both mice and humans.”

Cell Metabolism • November 2014

A bitter aftertaste: unintended effects of artificial sweeteners on the gut microbiome

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ROBINSONS LEMON BARLEY WATER
INGREDIENTS

Lemon (pictured below, at left): Water, **Glucose-Fructose Syrup**, Water and Concentrated Lemon Juice, **Sugar**, Barley Flour (2.5%), Citric Acid, **Preservatives (Potassium Sorbate, Sodium Metabisulfate)**, Ascorbic Acid, **Artificial Flavor, Saccharin (10mg/Fl Oz.) or 280 milligrams of saccharin per 28 ounce bottle.**

Abstract

Intestinal microbial communities regulate a range of host physiological functions, from energy harvest and glucose homeostasis to immune development and regulation. Suez et al. (2014) recently demonstrated that artificial sweeteners alter gut microbial communities, leading to glucose intolerance in both mice and humans.

Full text, graphs, charts and 10 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC4494042/

“Preliminary anti-cancer activity has been assessed against A549 human alveolar type-II like cell lines with the thiosaccharinate complex showing good activity.”

Journal Of Inorganic Biochemistry • December 2014

Combining anti-cancer drugs with artificial sweeteners: synthesis and anti-cancer activity of saccharinate (sac) and thiosaccharinate (tsac) complexes cis-[Pt(sac)2(NH3)2] and cis-[Pt(tsac)2(NH3)2]

Author information

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Abstract

The new platinum(II) complexes cis-[Pt(sac)2(NH3)2] (sac=saccharinate) and cis-[Pt(tsac)2(NH3)2] (tsac=thiosaccharinate) have been prepared, the X-ray crystal structure of cis-[Pt(sac)2(NH3)2] x H2O reveals that both saccharinate anions are N-bound in a cis-arrangement being inequivalent in both the solid-state and in solution at room temperature. Preliminary anti-cancer activity has been assessed against A549 human alveolar type-II like cell lines with the thiosaccharinate complex showing good activity.

http://www.ncbi.nlm.nih.gov/pubmed/25216367

“Results revealed that consuming non-caloric-sweetened beverages influences psychological processes in ways that - over time - may increase calorie intake ...”

[causing weight gain, precisely what they were allegedly laboratory designed to prevent]

Appetite • December 2014

The effect of non-caloric sweeteners on cognition, choice, and post-consumption satisfaction

Author information

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Abstract

Consumers often turn to non-caloric sweeteners (NCS) as a means of promoting a healthy body weight. However, several studies have now linked their long-term use to increased weight gain, raising the question of whether these products produce unintended psychological, physiological, or behavioral changes that have implications for weight management goals. In the following, we present the results of three experiments bearing on this issue, testing whether NCS-consumption influences how individuals think about and respond to food. Participants in each of our three experiments were randomly assigned to consume a sugar-sweetened beverage, an unsweetened beverage, or a beverage sweetened with NCS. We then measured their cognition (Experiment 1), product choice (Experiment 2), and subjective responses to a sugar-sweetened food (Experiment 3). Results revealed that consuming NCS-sweetened beverages influences psychological processes in ways that - over time - may increase calorie intake.

http://www.ncbi.nlm.nih.gov/pubmed/25128835

PLoS One • February 2013

Consumption of artificially-sweetened soft drinks in pregnancy and risk of child asthma and allergic rhinitis

Author information

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Abstract

BACKGROUND

Past evidence has suggested a role of artificial sweeteners in allergic disease; yet, the evidence has been inconsistent and unclear.

OBJECTIVE

To examine relation of intake of artificially-sweetened beverages during pregnancy with child asthma and allergic rhinitis at 18 months and 7 years.

METHODS

We analyzed data from 60,466 women enrolled during pregnancy in the prospective longitudinal Danish National Birth Cohort between 1996 and 2003. At the 25th week of gestation we administered a validated Food Frequency Questionnaire which asked in detail about intake of artificially-sweetened soft drinks. At 18 months, we evaluated child asthma using interview data. We also assessed asthma and allergic rhinitis through a questionnaire at age 7 and by using national registries. Current asthma was defined as self-reported asthma diagnosis and wheeze in the past 12 months. We examined the relation between intake of artificially-sweetened soft drinks and child allergic disease outcomes and present here odds ratios with 95% CI comparing daily vs. no intake.

RESULTS

At 18 months, we found that mothers who consumed more artificially-sweetened non-carbonated soft drinks were 1.23 (95% CI: 1.13, 1.33) times more likely to report a child asthma diagnosis compared to non-consumers. Similar results were found for child wheeze. Consumers of artificially-sweetened carbonated drinks were more likely to have a child asthma diagnosis in the patient (1.30, 95% CI: 1.01, 1.66) and medication (1.13, 95% CI: 0.98, 1.29) registry, as well as self-reported allergic rhinitis (1.31, 95% CI: 0.98, 1.74) during the first 7 years of follow-up. We found no associations for sugar-sweetened soft drinks.

CONCLUSION

Carbonated artificially-sweetened soft drinks were associated with registry-based asthma and self-reported allergic rhinitis, while early childhood outcomes were related to non-carbonated soft drinks. These results suggest that consumption of artificially-sweetened soft drinks during pregnancy may play a role in offspring allergic disease development.

Full Text, Graphs, Charts and 46 References

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3584110/

“These results suggest that consumption of artificially-sweetened soft drinks during pregnancy may play a role in offspring allergic disease development.”

Behavioral Neuroscience • April 2013

Adverse effects of high-intensity sweeteners on energy intake and weight control in male and obesity-prone female rats

Author information

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Abstract

The use of high-intensity sweeteners has been proposed as a method to combat increasing rates of overweight and obesity in the human population. However, previous work with male rats suggests that consumption of such sweeteners might contribute to, rather than ameliorate, weight gain. The goals of the present experiments were to assess whether intake of high-intensity sweeteners is associated with increased food intake and body weight gain in female rats; to evaluate whether this effect depends on composition of the maintenance diet (i.e., standard chow compared with diets high in energy, fat, and sugar [HE diets]); and to determine whether the phenotype of the rats with regard to propensity to gain weight on HE diets affects the consequences of consuming high-intensity sweeteners. The data demonstrated that female rats fed a low-fat, standard laboratory chow diet did not gain extra weight when fed yogurt dietary supplements sweetened with saccharin compared with those fed glucose-sweetened dietary supplements. However, female rats maintained on a "Westernized" diet high in fat and sugar (HE diet) showed significant increases in energy intake, weight gain, and adiposity when given saccharin-sweetened compared with glucose-sweetened yogurt supplements. These differences were most pronounced in female rats known to be prone to obesity prior to the introduction of the yogurt diets. Both selectively bred Crl:OP[CD] rats and outbred Sprague-Dawley rats fed an HE diet showing high levels of weight gain (diet-induced obese [DIO] rats) had increased weight gain in response to consuming saccharin-sweetened compared with glucose-sweetened supplements. However, in male rats fed an HE diet, saccharin-sweetened supplements produced extra weight gain regardless of obesity phenotype. These results suggest that the most negative consequences of consuming high-intensity sweeteners may occur in those most likely to use them for weight control, females consuming a "Westernized" diet and already prone to excess weight gain.

Full Text with Graphs, Charts and 74 References

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3985091/

"These results suggest that the most negative consequences of consuming high-intensity sweeteners may occur in those most likely to use them for weight control, females consuming a "Westernized" diet and already prone to excess weight gain."

Science Progress • April 2013

Averting comfortable lifestyle crises

Author information

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Abstract

How have climate change and diet shaped the evolution of human energy metabolism, and responses to vitamin C, fructose and uric acid? Through the last three millennia observant physicians have noted the association of inappropriate diets with increased incidence of obesity, heart disease, diabetes and cancer, and over the past 300 years doctors in the UK observed that overeating increased the incidence of these diseases. Anthropological studies of the Inuit culture in the mid-nineteenth century revealed that humans can survive and thrive in the virtual absence of dietary carbohydrate. In the 1960s, Cahill revealed the flexibility of human metabolism in response to partial and total starvation and demonstrated that type 2 diabetics were better adapted than healthy subjects to conserving protein during fasting. The potential role for brown adipose tissue thermogenesis in temperature maintenance and dietary calorie control was suggested by Rothwell and Stock from their experiments with 'cafeteria fed rats' in the 1980s. Recent advances in gene array studies and PET scanning support a role for this process in humans. The industrialisation of food processing in the twentieth century has led to increases in palatability and digestibility with a parallel loss of quality leading to overconsumption and the current obesity epidemic. The switch from animal to vegetable fats at the beginning of the twentieth century, followed by the rapid increase in sugar and fructose consumption from 1979 is mirrored by a steep increase in obesity in the 1980s, in the UK and USA. Containment of the obesity epidemic is compounded by the addictive properties of sugar which involve the same dopamine receptors in the pleasure centres of the brain as for cocaine, nicotine and alcohol. Of the many other toxic effects of excessive sugar consumption, immunocompromisation, kidney damage, atherosclerosis, oxidative stress and cancer are highlighted. The WHO and guidelines on sugar consumption include: alternative non-sugar sweeteners; toxic side-effects of aspartame. Stevia and xylitol as healthy sugar replacements; the role of food processing in dietary health; and beneficial effects of resistant starch in natural and processed foods. The rise of maize and soya-based vegetable oils have led to omega-6 fat overload and imbalance in the dietary ratio of omega-3 to omega-6 fats. This has led to toxicity studies with industrial trans fats; investigations on health risks associated with stress and comfort eating; and abdominal obesity. Other factors to consider are: diet, cholesterol and oxidative stress, as well as the new approaches to the chronology of eating and the health benefits of intermittent fasting.

http://www.ncbi.nlm.nih.gov/pubmed/24547668

PLoS One • August 2013

Long-term artificial sweetener acesulfame potassium treatment alters neurometabolic functions in C57BL/6J mice

Author information

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Abstract

With the prevalence of obesity, artificial, non-nutritive sweeteners have been widely used as dietary supplements that provide sweet taste without excessive caloric load. In order to better understand the overall actions of artificial sweeteners, especially when they are chronically used, we investigated the peripheral and central nervous system effects of protracted exposure to a widely used artificial sweetener, acesulfame K (ACK). We found that extended ACK exposure (40 weeks) in normal C57BL/6J mice demonstrated a moderate and limited influence on metabolic homeostasis, including altering fasting insulin and leptin levels, pancreatic islet size and lipid levels, without affecting insulin sensitivity and bodyweight. Interestingly, impaired cognitive memory functions (evaluated by Morris Water Maze and Novel Objective Preference tests) were found in ACK-treated C57BL/6J mice, while no differences in motor function and anxiety levels were detected. The generation of an ACK-induced neurological phenotype was associated with metabolic dysregulation (glycolysis inhibition and functional ATP depletion) and neurosynaptic abnormalities (dysregulation of TrkB-mediated BDNF and Akt/Erk-mediated cell growth/survival pathway) in hippocampal neurons. Our data suggest that chronic use of ACK could affect cognitive functions, potentially via altering neuro-metabolic functions in male C57BL/6J mice.

Full Text, Graphs, Charts and 89 References

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3737213/

“Our data suggest that chronic use of acesulfame K could affect cognitive functions, potentially via altering neuro-metabolic functions in male C57BL/6J mice.”

“This hypothesis suggested that both ulcerative colitis and Crohn’s disease are caused by weakening of the gut barrier due to damage of the protective mucus layer and the underlying tissue by the poorly inactivated digestive proteases resulting from a reduction of gut bacteria by dietary chemicals like saccharin and sucralose.”

World Journal Of Gastrointestinal Pathophysiology • August 2013

Why is damage limited to the mucosa in ulcerative colitis but transmural in Crohn’s disease?

Author information

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Abstract

It has been a big puzzle as why the inflammation of ulcerative colitis (UC) is limited to the mucosa, while in Crohn’s disease (CD) the inflammation is transmural and can be seen in all layers of the gut. Here, I give a tentative explanation extended from the unified hypothesis I proposed on the etiology of inflammatory bowel disease. This hypothesis suggested that both UC and CD are caused by weakening of the gut barrier due to damage of the protective mucus layer and the underlying tissue by the poorly inactivated digestive proteases resulting from a reduction of gut bacteria by dietary chemicals like saccharin and sucralose. However, the large amounts of bacteria in the colon make the recruitment of neutrophils and formation of crypt abscess the main manifestation of UC, while the infiltration of antigens and dietary particles in the small and large intestine mainly cause the recruitment of macrophages and formation of granulomas as the main manifestations in CD. The fast reacting and short life span of neutrophils make the fight and damage limited to the surface of the mucosa. In contrast, the long life span and constant movement of macrophages may bring the harmful agents deep into the tissue. Therefore, the pathogenesis of UC may be more like bacterial pneumonia, while CD may be more like pneumoconiosis or tuberculosis of the lung.

Full text with 7 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3740262/

“The results of this experiment indicate that long-term consumption of aspartame leads to an imbalance in the antioxidant/pro-oxidant status in the brain ...”

Drug And Chemical Toxicology • April 2013

Long-term consumption of aspartame and brain antioxidant defense status

Author information

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Abstract

The present study investigated the effect of long-term intake of aspartame, a widely used artificial sweetener, on antioxidant defense status in the rat brain. Male Wistar rats weighing 150-175 g were randomly divided into three groups as follows: The first group was given aspartame at a dose of 500 mg/kg body weight (b.w.); the second group was given aspartame at dose of 1,000 mg/kg b.w., respectively, in a total volume of 3 mL of water; and the control rats received 3 mL of distilled water. Oral intubations were done in the morning, daily for 180 days. The concentration of reduced glutathione (GSH) and the activity of glutathione reductase (GR) were significantly reduced in the brain of rats that had received the dose of 1,000 mg/kg b.w. of aspartame, whereas only a significant reduction in GSH concentration was observed in the 500-mg/kg b.w. aspartame-treated group. Histopathological examination revealed mild vascular congestion in the 1,000 mg/kg b.w. group of aspartame-treated rats. The results of this experiment indicate that long-term consumption of aspartame leads to an imbalance in the antioxidant/pro-oxidant status in the brain, mainly through the mechanism involving the glutathione-dependent system.

http://www.ncbi.nlm.nih.gov/pubmed/22385158

RED BULL SUCCESSFULLY MARKETS ASPARTAME, AN ACKNOWLEDGED POISON

Red Bull is valued at $6.5 billion and commands 43 percent of the white-hot energy drinks market. Considering how poorly it does in taste tests, that’s doubly impressive. It’s not really taste that Red Bull sells, but a promise: “Red Bull Gives You Wings.” And it’s particularly appealing to time-crunched college students facing both academic and social pressures. The ethical gray area of advertising energy drinks is tricky to navigate because the FDA can’t regulate energy drinks the way it does other potentially unsafe products like alcohol and tobacco. Unlike its rivals Monster and Five-Hour Energy, which are considered dietary supplements, Red Bull markets itself in the category of “conventional beverages.” As the New York Times reported in 2012, “*while producers of energy drinks that market them as dietary supplements... must notify the FDA about death and injuries claiming a possible link to their products, companies that market energy drinks as beverages do not. On one hand, we want to be protected from unscrupulous marketers, but on the other hand, we are a country of individualists who ... often resent the intrusion of government into private decisions*.” University administrators on college campuses can’t openly criticize companies because they risk claims of defamation. When reached for comment, a Red Bull PR representative said, “*We never talk about our marketing practices*.”

Water Research • September 2013

Occurrence of seven artificial sweeteners in the aquatic environment and precipitation of Tianjin, China

Author information

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Abstract

Seventy water samples, including wastewaters, tap waters, fresh surface waters, coastal waters, groundwaters, and precipitation samples, from Tianjin, China, were analyzed for seven commonly used artificial sweeteners (ASs). The concentrations of the investigated ASs were generally in the order of wastewater treatment plant (WWTP) influent > WWTP effluent > surface water > tap water > groundwater ≈ precipitation, while the composition profiles of ASs varied in different waters. Acesulfame, sucralose, cyclamate, and saccharin were consistently detected in surface waters and ranged from 50 ng/L to 0.12 mg/L, while acesulfame was the dominant AS in surface and tap waters. Aspartame was found in all of the surface waters at a concentration up to 0.21 μg/L, but was not found in groundwaters and tap waters. Neotame and neohesperidin dihydrochalcone were less frequently detected and the concentrations were low. The concentrations of the ASs in some of the surface waters were of the same order with those in the WWTP influents, but not with the effluents, indicating there are probably untreated discharges into the surface waters. The ASs were detected in precipitation samples with high frequency, and acesulfame, saccharin, and cyclamate were the predominant ASs, with concentrations ranging from 3.5 ng/L to 1.3 μg/L. A gross estimation revealed that precipitation may act as a source for saccharin and cyclamate in the surface environment of Tianjin city. Moreover, the presence of ASs in the atmosphere was primarily assessed by taking 4 air samples to evaluate their potential source in precipitation.

http://www.ncbi.nlm.nih.gov/pubmed/23866151

“Acesulfame, sucralose, cyclamate, and saccharin were consistently detected in surface waters and ranged from 50 ng/L to 0.12 mg/L, while acesulfame was the dominant artificial sweetener in surface and tap waters. Aspartame was found in all of the surface waters at a concentration up to 0.21 μg/L, but was not found in groundwaters and tap waters. Neotame and neohesperidin dihydrochalcone were less frequently detected and the concentrations were low. The concentrations of the artificial sweeteners in some of the surface waters were of the same order with those in the Waste Water Treatment Plants influents, but not with the effluents, indicating there are probably untreated discharges into the surface waters. The artificial sweeteners were detected in precipitation [rain] samples with high frequency, and acesulfame, saccharin, and cyclamate were the predominant artificial sweeteners, with concentrations ranging from 3.5 ng/L to 1.3 μg/L.”

> “accumulating evidence suggests that frequent consumers of these sugar substitutes may also be at increased risk of excessive weight gain, metabolic syndrome, type 2 diabetes, and cardiovascular disease.”

Trends In Endocrinology And Metabolism • September 2013

Artificial sweeteners produce the counterintuitive effect of inducing metabolic derangements

Author information

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Abstract

The negative impact of consuming sugar-sweetened beverages on weight and other health outcomes has been increasingly recognized; therefore, many people have turned to high-intensity sweeteners like aspartame, sucralose, and saccharin as a way to reduce the risk of these consequences. However, accumulating evidence suggests that frequent consumers of these sugar substitutes may also be at increased risk of excessive weight gain, metabolic syndrome, type 2 diabetes, and cardiovascular disease. This paper discusses these findings and considers the hypothesis that consuming sweet-tasting but noncaloric or reduced-calorie food and beverages interferes with learned responses that normally contribute to glucose and energy homeostasis. Because of this interference, frequent consumption of high-intensity sweeteners may have the counterintuitive effect of inducing metabolic derangements.

Full Text with Graphs, Charts and 54 References

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3772345/

“... for infants and 95th percentile high-level consumers (especially those who choose sucralose containing foods), the estimated daily intakes of sucralose were very close to and higher than the acceptable daily intakes. Therefore, the sucralose concentration in sweetened beverages should be reduced; this would benefit the health of both high level consumers and infants.”

Mountain Dew Diet Soda
INGREDIENTS

Carbonated Water, Concentrated Orange Juice, Citric Acid, **Natural Flavor, Citrus Pectin, Potassium Benzoate (Preserves Freshness), Aspartame, Potassium Citrate, Caffeine, Sodium Citrate, Acesulfame Potassium (K), Sucralose, Gum Arabic, Sodium Benzoate (Preserves Freshness), Calcium Disodium EDTA (to Protect Flavor), Brominated Vegetable Oil, Yellow 5.** Total nutritional value of Diet Mountain Dew comes in 35 milligrams of sodium, 2% of the daily recommended allowance. No vitamins, no minerals, no nutrients, this drink is a combination of carbonated water and deadly chemicals.

There is controversy as to the safety of artificial sweeteners consumed over a long period of time. Some studies have linked artificial sweeteners to cancer and other diseases. Aspartame is a popular artificial sweetener, both as a stand alone product and as a food and beverage ingredient. Several rat studies have shown aspartame to cause leukemia and tumors. Recent studies (Denmark, 2010) linked consumption of artificial sweeteners like aspartame to pre-term deliveries. Many consumers attribute their headaches to the consumption of aspartame. Bottom line: Until high-quality studies by independent scientists will be conducted, it is best to stay away from Aspartame.

Once upon a time, there were no food colorings. Then folks figured out that food looks better and sells more when it can be enlivened through dyes. For most of food history, the dyes were from natural sources – beet juice for red, turmeric for yellow, etc… However, in the quest to increase color intensity and lower manufacturing costs, cheap artificial petroleum based dyes were introduced to market. Unfortunately they pose a risk for hyperactivity in children, cancer, allergic reactions and more. Mountain Dew also contains sodium benzoate/benzoic acid. Sodium benzoate/benzoic acid are used to prevent the growth of microorganisms in acidic foods. They are natural substances. However, in beverages with ascorbic acid (vitamin C), a chemical reaction creates a small amount of benzene, a carcinogen. Don’t do the Dew.

International Journal Of Food Science & Nutrition • September 2013

Assessment of exposure of Korean consumers to acesulfame K and sucralose using a stepwise approach

Author information

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http://www.ncbi.nlm.nih.gov/pubmed/23631357

Abstract

Using a stepwise assessment of the exposure of Korean consumers to acesulfame K and sucralose, theoretical maximum daily intakes of the sweeteners were calculated using the Budget screening method, which resulted in values greater than the acceptable daily intakes (ADIs). Accordingly, the daily intakes of the sweeteners based on food consumption data and concentrations determined by instrumental analysis of 605 food samples were estimated for the more refined approach. The estimated daily intakes (EDIs) of all ordinary consumers were lower than the ADI, which was considered safe. However, for infants and 95th percentile high-level consumers (especially those who choose sucralose-containing foods), the EDIs of sucralose were very close to and higher than the ADI. Therefore, the sucralose concentration in sweetened beverages should be reduced; this would benefit the health of both high-level consumers and infants.

Don’t Do The Dew!

“In this context, obligatory ecotoxicity testing and stricter environmental regulations regarding food additives appear to be necessary.”

Environment International • October 2013

Ecotoxicity of artificial sweeteners and stevioside

Author information

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Abstract

Produced, consumed and globally released into the environment in considerable quantities, artificial sweeteners have been identified as emerging pollutants. Studies of environmental concentrations have confirmed the widespread distribution of acesulfame (ACE), cyclamate (CYC), saccharin (SAC) and sucralose (SUC) in the water cycle at levels that are among the highest known for anthropogenic trace pollutants. Their ecotoxicity, however, has yet to be investigated at a larger scale. The present study aimed to fill this knowledge gap by systematically assessing the influence of ACE, CYC and SAC and complementing the data on SUC. Therefore we examined their toxicity towards an activated sewage sludge community (30min) and applying tests with green algae Scenedesmus vacuolatus (24h), water fleas Daphnia magna (48h) and duckweed Lemna minor (7d). We also examined the effects caused by the natural sweetener stevioside. The high No Observed Effect Concentrations (NOECs) yielded by this initial evaluation indicated a low hazard and risk potential towards these aquatic organisms. For a complete risk assessment, however, several kinds of data are still lacking. In this context, obligatory ecotoxicity testing and stricter environmental regulations regarding food additives appear to be necessary.

http://www.ncbi.nlm.nih.gov/pubmed/24036324

Honest Fizz Zero Calorie Soda
INGREDIENTS
carbonated water, **erythritol, organic caramel color, natural flavors, stevia leaf extract**, citric acid, **caffeine**

“Sucralose and one of its hydrolysis products were found to be mutagenic at elevated concentrations in several testing methods. Cooking with sucralose at high temperatures was reported to generate chloropropanols, a potentially toxic class of compounds. Both human and rodent studies demonstrated that sucralose may alter glucose, insulin, and glucagon-like peptide 1 (GLP-1) levels. Taken together, these findings indicate that sucralose is not a biologically inert compound.”

The Journal Of Toxicology And Environmental Health Part B
Critical Reviews • December 2013

Sucralose, a synthetic organochlorine sweetener: overview of biological issues

Author information
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Abstract

Sucralose is a synthetic organochlorine sweetener (OC) that is a common ingredient in the world’s food supply. Sucralose interacts with chemosensors in the alimentary tract that play a role in sweet taste sensation and hormone secretion. In rats, sucralose ingestion was shown to increase the expression of the efflux transporter P-glycoprotein (P-gp) and two cytochrome P-450 (CYP) isozymes in the intestine. P-gp and CYP are key components of the presystemic detoxification system involved in first-pass drug metabolism. The effect of sucralose on first-pass drug metabolism in humans, however, has not yet been determined. In rats, sucralose alters the microbial composition in the gastrointestinal tract (GIT), with relatively greater reduction in beneficial bacteria. Although early studies asserted that sucralose passes through the GIT unchanged, subsequent analysis suggested that some of the ingested sweetener is metabolized in the GIT, as indicated by multiple peaks found in thin-layer radiochromatographic profiles of methanolic fecal extracts after oral sucralose administration. The identity and safety profile of these putative sucralose metabolites are not known at this time. Sucralose and one of its hydrolysis products were found to be mutagenic at elevated concentrations in several testing methods. Cooking with sucralose at high temperatures was reported to generate chloropropanols, a potentially toxic class of compounds. Both human and rodent studies demonstrated that sucralose may alter glucose, insulin, and glucagon-like peptide 1 (GLP-1) levels. Taken together, these findings indicate that sucralose is not a biologically inert compound.

With full text, graphs, charts and 374 incredible references, this is one great article!

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3856475/

Propel Zero Enhanced Water, Berry
INGREDIENTS

Water, Citric Acid, **Sodium Hexametaphosphate** (to protect flavor), **Natural Flavor, Potassium Sorbate** (preserves freshness), Ascorbic Acid (Vitamin C), **Sucralose**, Sodium Citrate, Potassium Citrate, **Acesulfame Potassium (K)**, Niacinamide (Vitamin B3), **Calcium Disodium EDTA** (to protect flavor), Vitamin E Acetate, Calcium Pantothenate (Vitamin B5), Pyridoxine Hydrochloride (Vitamin B6).

Has EDTA, on FDA’s toxicity watchlist. Ethylenediaminetetraacetic acid (EDTA) is used as a preservative to retain color. It may irritate the skin, cause skin rash, asthma and more. It is on FDA’s list of food additives to be studied for toxicity.

Folia Neuropathologia • December 2013

Effects of aspartame metabolites on astrocytes and neurons

Author information

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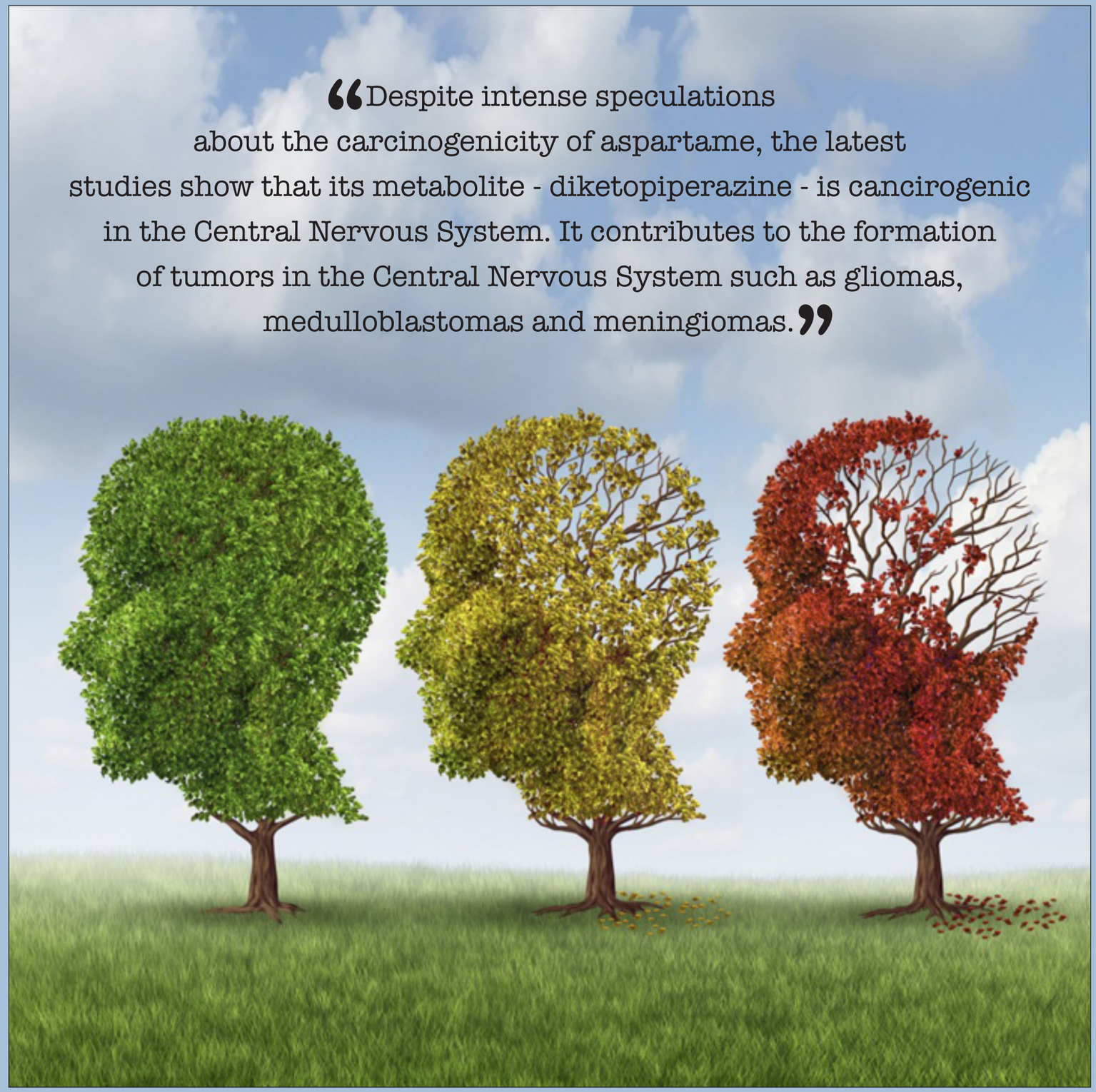
Abstract

Aspartame, a widespread sweetener used in many food products, is considered as a highly hazardous compound. Aspartame was discovered in 1965 and raises a lot of controversy up to date. Astrocytes are glial cells, the presence and functions of which are closely connected with the central nervous system (CNS). The aim of this article is to demonstrate the direct and indirect role of astrocytes participating in the harmful effects of aspartame metabolites on neurons. The artificial sweetener is broken down into phenylalanine (50%), aspartic acid (40%) and methanol (10%) during metabolism in the body. The excess of phenylalanine blocks the transport of important amino acids to the brain contributing to reduced levels of dopamine and serotonin. Astrocytes directly affect the transport of this amino acid and also indirectly by modulation of carriers in the endothelium. Aspartic acid at high concentrations is a toxin that causes hyperexcitability of neurons and is also a precursor of other excitatory amino acid - glutamates. Their excess in quantity and lack of astrocytic uptake induces excitotoxicity and leads to the degeneration of astrocytes and neurons. The methanol metabolites cause CNS depression, vision disorders and other symptoms leading ultimately to metabolic acidosis and coma. Astrocytes do not play a significant role in methanol poisoning due to a permanent consumption of large amounts of aspartame. Despite intense speculations about the carcinogenicity of aspartame, the latest studies show that its metabolite - diketopiperazine - is cancirogenic in the CNS. It contributes to the formation of tumors in the CNS such as gliomas, medulloblastomas and meningiomas. Glial cells are the main source of tumors, which can be caused inter alia by the sweetener in the brain. On the one hand the action of astrocytes during aspartame poisoning may be advantageous for neuro-protection while on the other it may intensify the destruction of neurons. The role of the glia in the pathogenesis of many CNS diseases is crucial.

http://www.ncbi.nlm.nih.gov/pubmed/23553132



International Journal Of Molecular Science • December 2013

Xylitol affects the intestinal microbiota and metabolism of daidzein in adult male mice

Author information

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Abstract

This study examined the effects of xylitol on mouse intestinal microbiota and urinary isoflavonoids. Xylitol is classified as a sugar alcohol and used as a food additive. The intestinal microbiota seems to play an important role in isoflavone metabolism. Xylitol feeding appears to affect the gut microbiota. We hypothesized that dietary xylitol changes intestinal microbiota and, therefore, the metabolism of isoflavonoids in mice. Male mice were randomly divided into two groups: those fed a 0.05% daidzein with 5% xylitol diet (XD group) and those fed a 0.05% daidzein-containing control diet (CD group) for 28 days. Plasma total cholesterol concentrations were significantly lower in the XD group than in the CD group ($p < 0.05$). Urinary amounts of equol were significantly higher in the XD group than in the CD group ($p < 0.05$). The fecal lipid contents (% dry weight) were significantly greater in the XD group than in the CD group ($p < 0.01$). The cecal microbiota differed between the two dietary groups. The occupation ratios of Bacteroides were significantly greater in the CD than in the XD group ($p < 0.05$). This study suggests that xylitol has the potential to affect the metabolism of daidzein by altering the metabolic activity of the intestinal microbiota and/or gut environment. Given that equol affects bone health, dietary xylitol plus isoflavonoids may exert a favorable effect on bone health.

Full text, graphs, charts and 48 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3876090/

DAIDZEIN

Daidzein can be found in food such as soybeans and soy products (and other foods) like tofu and textured vegetable protein. Soy isoflavones are a group of compounds found in and isolated from the soybean. Of note, total isoflavones in soybeans are—in general—37 percent daidzein, 57 percent genistein and 6 percent glycitein, according to USDA data. Soy germ contains 41.7 percent daidzein

BIOLOGICAL ACTIVITIES

Daidzein can be converted to its end metabolite S-equol in some humans based on the presence of certain intestinal bacteria. Based on several decades of research, S-equol has potential for significant health benefits. Daidzein has no classification in the United States, where it is not considered to be generally recognized as safe (GRAS), and has not been approved as a drug for any indication. It is a component of foods and dietary supplements derived from soy. Dietary supplements are not regulated as drugs in the U.S., and the labeling of dietary supplements in the U.S. may not describe the supplement as having any drug activity or effectiveness.

Scientists have studied some of the activities of daidzein in their laboratories, working with cells or with animals such as mice. Studies in cells and in animals sometimes give hints as to what a chemical might do when given to humans, but no one can know what a chemical does in humans until the chemical is tested in a clinical trial.

CELL PROLIFERATION STUDIES

Daidzein has both estrogenic and anti-estrogenic effects. Experiments in cells and in animals showed that lower concentrations stimulate breast tumor growth in in vitro and in vivo, and ihibits the antitumor effect of the cancer drug tamoxifen, but higher concentrations (above 10 μM) have the contrary effect. T47D:A18/PKC alpha tumor growth was demonstrated to be stimulated by genistein, but partially inhibited by daidzein; however, coadministration of tamoxifen with either daidzein or genistein produced tumors of greater size.

ANTIOXIDANT PROPERTIES

Scientific studies of daidzein's antioxidant properties have given contradictory results: some studies have shown antioxidant properties in laboratory experiments on cells, but in other experiments daidzein has caused oxidative stress on cells.

DAIDZEIN METABOLITE S-equol ACTIVITIES

Daidzein, when consumed from soy, is transformed in some humans, but not all, to produce S-equol [7-hydroxy-3-(49-hydroxyphenyl)-chroman]. Because it is a metabolite of daidzein, S-equol is not of plant origin. The molecular and physical structure of S-equol is similar to that of estradiol, the main sex hormone found in women. The ability to transform daidzein into S-equol is based on the presence of certain intestinal bacteria. In fact, several studies indicate that only 25 to 30 percent of the adult population of Western countries produces S-equol after eating soy foods containing isoflavones, significantly lower than the reported 50 to 60 percent frequency of equol-producers in adults from Japan, Korea, or China. Although still under investigation, the ability to produce S-equol may be associated with other health benefits, according to data from epidemiological and clinical trials. Studies in both animal models and humans have yielded data about the potential of S-equol use in menopause breast and prostate cancer and bone health.

According To The Peer Review We've Polluted Our Once Pristine Earth With Man-Made Chemicals—Well Beyond Repair—In Just Over 100 Years

> “Our results show that both physiology and locomotion behaviour were affected by exposure to sucralose. In Daphnia magna, the behavioural response was manifested as altered swimming height and increased swimming speed, whereas in gammarids the time to reach food and shelter was prolonged.”

Chemosphere • January 2012

Sucralose— an ecotoxicological challenger?

Author information

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Abstract

The non-calorie sweetener sucralose - sucrose containing three chlorine atoms - is intensively sweet and has become a popular substitute for sugar. Its widespread use, exceptional stability in combination with high water solubility have thus resulted in contamination of recipient waters. Earlier studies on sucralose in aquatic organisms indicate low bioaccumulation potential and negligible acute/chronic toxicity, but the close structural resemblance with sucrose in combination with the importance of sugar in nature, warrant a more detailed ecotoxicological assessment. The aim of this investigation was therefore to study behavioural and physiological effects of sucralose in crustaceans. Our results show that both physiology and locomotion behaviour were affected by exposure to sucralose. In Daphnia magna, the behavioural response was manifested as altered swimming height and increased swimming speed, whereas in gammarids the time to reach food and shelter was prolonged. Regardless if these behavioural responses were initiated via traditional toxic mechanisms or stimulatory effects, they should be considered as a warning, since exposed organisms may diverge from normal behaviour, which ultimately can have ecological consequences.

http://www.ncbi.nlm.nih.gov/pubmed/21955350

What Is electrolyte drink Pedialyte and why do we drink it?

It's a drink formulation that is supposed to restore mineral loss after vomiting and diarrhea. An electrolytic drink is especially helpful for little ones and the elderly, who are more susceptible to dehydration and mineral loss than the general population.

Pedialyte ingredients: Water, Dextrose. Less than 2% of the Following: Citric Acid, **Potassium Citrate, Sodium Chloride, Sodium Citrate, Sucralose, Acesulfame Potassium**, Zinc Gluconate, and **Artificial Flavor.**

In other words, Pedialyte is sugar water with minor amount of synthetic minerals added in, along with a generous dose of artificial colors and flavors.

Pedialyte Nutrition Facts

Electrolytes are minerals and nutrients found in the blood and other body fluids that carry an electric charge. So let's look at the mineral content of Pedialyte. See the chart at far right.

Now, let's take a look at coconut water.

When you open up a coconut, you have the meat, milk and water. The water is the highest in natural sugars and minerals, which are the most important to replenish after you've been sick. Interesting to note: coconut water's composition is very similar to human plasma. *So much so that, in wartime, coconut water has been used in blood transfusions! See the left chart of the two charts at right.

*(Source: Campbell-Falck, Darilyn et al. "The Intravenous Use of Coconut Water" American Journal of Emergency Medicine 18.1 (2000): 108-111.)

And it's loaded with nutrition.

As you can see, coconut water is very high in potassium (600 mg) when compared to its sodium content (200 mg). This is why we want to add high-quality sea or pink Himalayan salt to balance the mineral profile to be closer to a 1:1 ratio, so depending on your salt, 1/8-1/4 tsp. should do the trick. Any brand of coconut water, organic or not, with a little Pink Himalayan Salt or sea salt added for mineralization and you have a homemade product that's both cheaper and higher quality than Pedialyte, Pediasure and all of the competitors but what's far more important is that your electrolytic fluid is safe and has no chemicals in it. Easy and simple.

AVOID CONSUMING CHEMICALS.
LEAVE PEDIALYTE AND SIMILAR CHEMICAL LADEN EDIBLES IN THE STORE WHERE THEY BELONG. IF WE STOP BUYING THEIR GARBAGE THEY'LL STOP MAKING IT.

COCONUT WATER

Minerals		
Amounts Per Selected Serving	8 Ounces	%DV
Calcium	57.6 mg	6%
Iron	0.7 mg	4%
Magnesium	60.0 mg	15%
Phosphorus	48.0 mg	5%
Potassium	600 mg	17%
Sodium	252 mg	11%
Zinc	0.2 mg	2%
Copper	0.1 mg	5%
Manganese	0.3 mg	17%
Selenium	2.4 mcg	3%
Fluoride	~	

PEDIALYTE

Minerals		
Amounts Per Selected Serving	8 Ounces	%DV
Calcium	25.0 mg	2%
Iron	0.0 mg	0%
Magnesium	2.5 mg	1%
Phosphorus	25.0 mg	2%
Potassium	192 mg	5%
Sodium	253 mg	11%
Zinc	0.1 mg	0%
Copper	0.0 mg	1%
Manganese	~	~
Selenium	0.0 mcg	0%
Fluoride	~	

COST COMPARISON - JAN. 2016

The two largest bottles of Pedialyte pictured below, 1 liter bottles which are almost exactly 1 quart, average $13.97 each on Amazon.com. A comparable coconut water free of chemicals, Zico for example, runs $21.84 for a 12-pack of 14 ounce bottles. So Pedialyte gets you about 32 ounces for 14 bucks and Zico gets you 168 ounces for 22 dollars. This isn't rocket science is it?

American Heart Association • April 2012

Sweetened beverage consumption, incident coronary heart disease, and biomarkers of risk in men

Author information

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Harvard School of Public Health
665 Huntington Ave, Boston, MA 02115, USA

Abstract

BACKGROUND

Sugar-sweetened beverage consumption is associated with weight gain and risk of type 2 diabetes mellitus. Few studies have tested for a relationship with coronary heart disease (CHD) or intermediate biomarkers. The role of artificially sweetened beverages is also unclear.

METHODS AND RESULTS

We performed an analysis of the Health Professionals Follow-Up Study, a prospective cohort study including 42 883 men. Associations of cumulatively averaged sugar-sweetened (eg, sodas) and artificially sweetened (eg, diet sodas) beverage intake with incident fatal and nonfatal CHD (myocardial infarction) were examined with proportional hazard models. There were 3683 CHD cases over 22 years of follow-up. Participants in the top quartile of sugar-sweetened beverage intake had a 20% higher relative risk of CHD than those in the bottom quartile (relative risk=1.20; 95% confidence interval, 1.09-1.33; P for trend <0.01) after adjustment for age, smoking, physical activity, alcohol, multivitamins, family history, diet quality, energy intake, body mass index, pre-enrollment weight change, and dieting. Artificially sweetened beverage consumption was not significantly associated with CHD (multivariate relative risk=1.02; 95% confidence interval, 0.93-1.12; P for trend=0.28). Adjustment for self-reported high cholesterol, high triglycerides, high blood pressure, and diagnosed type 2 diabetes mellitus slightly attenuated these associations. Intake of sugar-sweetened but not artificially sweetened beverages was significantly associated with increased plasma triglycerides, C-reactive protein, interleukin-6, and tumor necrosis factor receptors 1 and 2 and decreased high-density lipoprotein, lipoprotein(a), and leptin (P<0.02).

CONCLUSIONS

Consumption of sugar-sweetened beverages was associated with increased risk of CHD and some adverse changes in lipids, inflammatory factors, and leptin. Artificially sweetened beverage intake was not associated with CHD risk or biomarkers.

Full Text, Graphs, Charts and 38 References

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3368965/

“Consumption of sugar-sweetened beverages was associated with increased risk of Coronary Heart Disease and some adverse changes in lipids, inflammatory factors, and leptin.”

“This paper aims to provide a detailed description of a unified hypothesis regarding the etiology of Inflammatory Bowel Disease, including the cause and mechanism of Inflammatory Bowel Disease, as well as the relationship between Ulcerative Colitis and Crohn’s Disease.”

World Journal Of Gastroenterology • April 2012

Etiology of inflammatory bowel disease: a unified hypothesis

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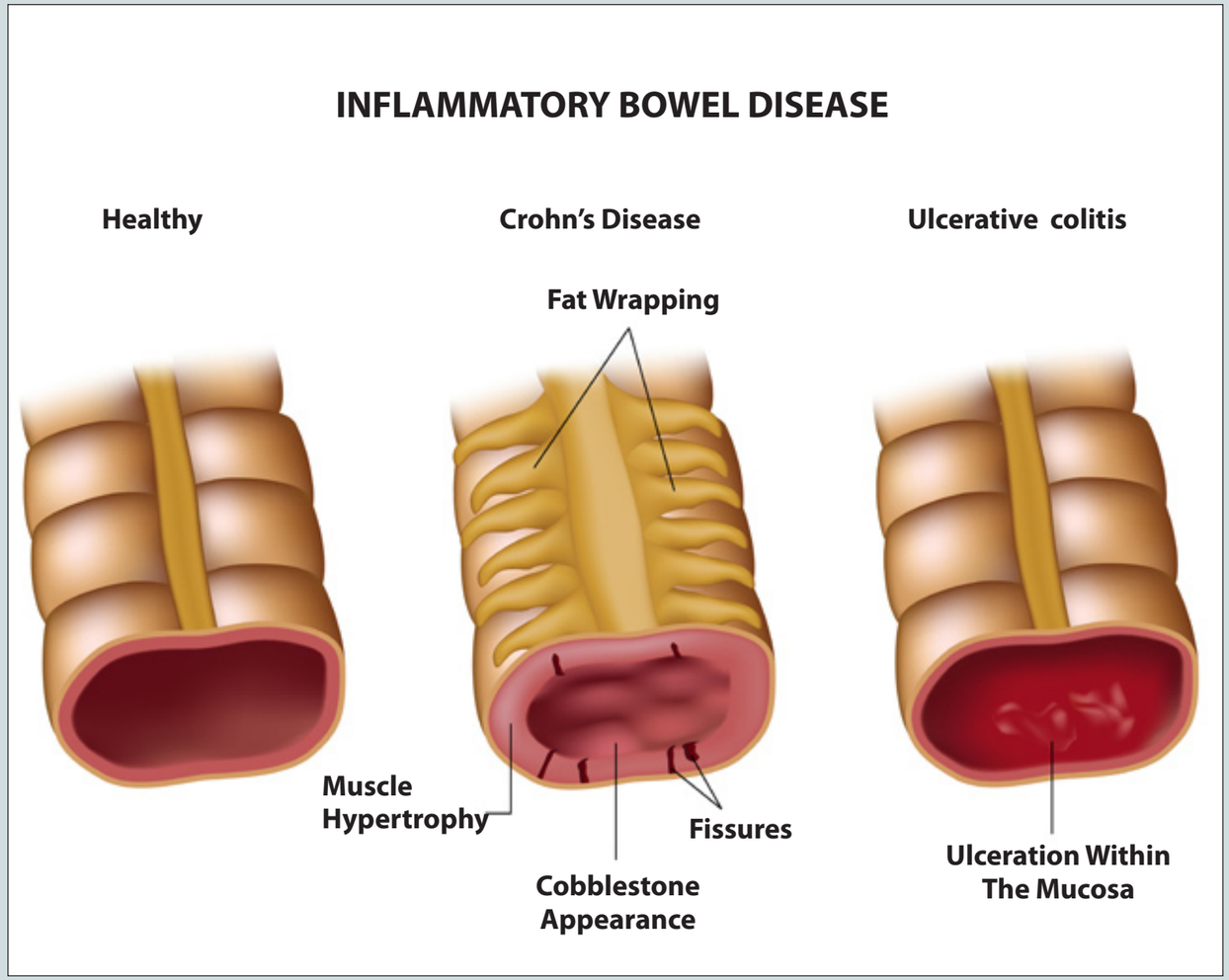
Abstract

Inflammatory bowel disease (IBD), including both ulcerative colitis (UC) and Crohn’s disease (CD), emerged and dramatically increased for about a century. Despite extensive research, its cause remains regarded as unknown. About a decade ago, a series of findings made me suspect that saccharin may be a key causative factor for IBD, through its inhibition on gut bacteria and the resultant impaired inactivation of digestive proteases and over digestion of the mucus layer and gut barrier (the Bacteria-Protease-Mucus-Barrier hypothesis). It explained many puzzles in IBD such as its emergence and temporal changes in last century. Recently I further found evidence suggesting sucralose may be also linked to IBD through a similar mechanism as saccharin and have contributed to the recent worldwide increase of IBD. This new hypothesis suggests that UC and CD are just two symptoms of the same morbidity, rather than two different diseases. They are both caused by a weakening in gut barrier and only differ in that UC is mainly due to increased infiltration of gut bacteria and the resultant recruitment of neutrophils and formation of crypt abscess, while CD is mainly due to increased infiltration of antigens and particles from gut lumen and the resultant recruitment of macrophages and formation of granulomas. It explained the delayed appearance but accelerated increase of CD over UC and many other phenomena. This paper aims to provide a detailed description of a unified hypothesis regarding the etiology of IBD, including the cause and mechanism of IBD, as well as the relationship between UC and CD.

Full text, graphs, charts and 171 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3332284/



Analytical And Bioanalytical Chemistry • July 2012

Artificial sweeteners— a recently recognized class of emerging environmental contaminants: a review

Author information

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Abstract

An overview is given of existing trace analytical methods for the determination of seven popular artificial sweeteners [acesulfame (ACE), aspartame, cyclamate (CYC), neotame, neohesperidine dihydrochalcone, saccharin (SAC), and sucralose (SUC)] from aqueous environmental samples. Liquid chromatography-electrospray ionization tandem mass spectrometry and liquid chromatography-electrospray ionization high-resolution mass spectrometry are the methods most widely applied, either directly or after solid-phase extraction. Limits of detection and limits of quantification down to the low nanogram per liter range can be achieved. ACE, CYC, SAC, and SUC were detected in wastewater treatment plants in high microgram per liter concentrations. Per capita loads of individual sweeteners can vary within a wide range depending on their use in different countries. Whereas CYC and SAC are usually degraded by more than 90% during wastewater treatment, ACE and SUC pass through wastewater treatment plants mainly unchanged. This suggests their use as virtually perfect markers for the study of the impact of wastewater on source waters and drinking waters. In finished water of drinking water treatment plants using surface-water-influenced source water, ACE and SUC were detected in concentrations up to 7 and 2.4 µg/L, respectively. ACE was identified as a precursor of oxidation byproducts during ozonation, resulting in an aldehyde intermediate and acetic acid. Although the concentrations of ACE and SUC are among the highest measured for anthropogenic trace pollutants found in surface water, groundwater, and drinking water, the levels are at least three orders of magnitude lower than organoleptic threshold values. However, ecotoxicology studies are scarce and have focused on SUC. Thus, further research is needed both on identification of transformation products and on the ecotoxicological impact of artificial sweeteners and their transformation products.

http://www.ncbi.nlm.nih.gov/pubmed/22543693

“Although the concentrations of Acesulfame K and Sucralose are among the highest measured for anthropogenic trace pollutants found in surface water, groundwater, and drinking water, the levels are at least three orders of magnitude lower than organoleptic threshold values. However, eco-toxicology studies are scarce and have focused on Sucralose. Thus, further research is needed both on identification of transformation products and on the eco-toxicological impact of artificial sweeteners and their transformation products.”

Diet Sodas Polluting Grand River With Artificial Sweeteners

Good news! You no longer have to sweeten your coffee or tea to get the flavour you're looking for. Just use the water from the Grand River to make your beverage. Apparently it is so polluted with artificial sweeteners that scientists can use this fact to follow the movement of treated waste in the region's water supply.

The artificial sweeteners found were cyclamate (banned in North America), saccharin, sucralose (Splenda), and acesulfame (known as Acesulfame-K or Acesulfame Potassium). They all provide no calories, mainly because they are not broken down by the human digestive system. What is startling about this study is that the amounts found in the river were much higher than what has been found in other water systems in other parts of the world.

Twenty-three sites along the Grand River were part of the study conducted jointly by Environment Canada and the University of Waterloo. At one site, researchers calculated that the consumption would have to be 90,000 to 190,000 cans of diet soda a day to account for the amount of acesulfame they found. The surrounding area is home to about 1,000,000 people. The purpose of the study was to see if chemicals could be used to trace when human waste enters into the water system, both from treatment plants and from septic tanks where waste may be seeping into groundwater.

Now, you did read that correctly. They were not looking to see, or concerned with, how much we are polluting the water with all the artificial crap that is in our food and elsewhere. No, they were looking for chemicals that could be used to track human waste. Who knew pollution could have such a purpose. I suppose that's just a sign of the times where chemicals in the water are so normal that scientists see them as helpers for determining other issues.

However, researchers did point out that the long term effects to both human and aquatic life are "unknown". This should at least be alarming to all of us as "unknown" health effects never turn out to be a good thing. So remember this information the next time someone makes fun of you for drinking bottled water or filtered water (hopefully re-mineralized). And think twice before using tap water for cooking or making tea and coffee.

More importantly, if you are someone who actually thinks diet soda is going to help you lose weight or prevent weight gain, then perhaps you need to really take a hard look at your thought processes. Your body needs nutrients and that includes those found in natural sweeteners. Pure honey, pure maple syrup, sucanat, coconut sweetener and palm sugar are all nutrient–dense and low glycemic. Providing the body with sufficient nutrients will provide steady energy, stable blood sugar, and prevent cravings which will do a lot more for proper weight management than any artificial sweetener could ever do. And they taste a lot better than an artificial sweetener and they aren't poisons.

Source:

Artificial Sweeteners in a Large Canadian River Reflect Human Consumption in the Watershed, John Spoelstra, Sherry L. Schiff, Susan J. Brown, Published: December 11, 2013,PLOS One, DOI: 10.1371/journal.pone.0082706

The Grand River, Canada

PLoSOne • December 11, 2013

Artificial Sweeteners in a Large Canadian River Reflect Human Consumption in the Watershed

Author information
By John Spoelstra, Sherry L. Schiff and Susan J. Brown

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Abstract

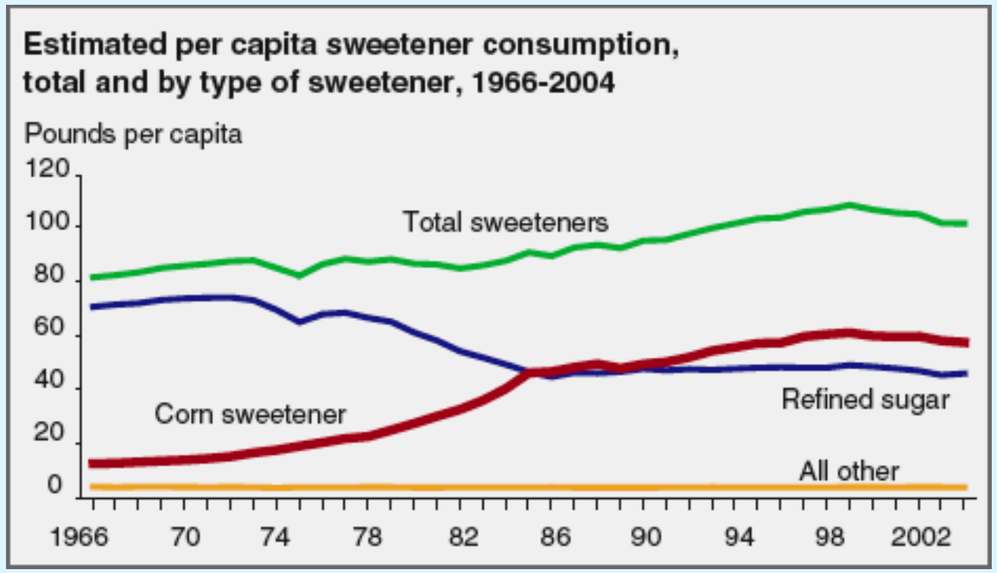
Artificial sweeteners have been widely incorporated in human food products for aid in weight loss regimes, dental health protection and dietary control of diabetes. Some of these widely used compounds can pass non-degraded through wastewater treatment systems and are subsequently discharged to groundwater and surface waters. Measurements of artificial sweeteners in rivers used for drinking water production are scarce. In order to determine the riverine concentrations of artificial sweeteners and their usefulness as a tracer of wastewater at the scale of an entire watershed, we analyzed samples from 23 sites along the entire length of the Grand River, a large river in Southern Ontario, Canada, that is impacted by agricultural activities and urban centres. Municipal water from household taps was also sampled from several cities within the Grand River Watershed. Cyclamate, saccharin, sucralose, and acesulfame were found in elevated concentrations despite high rates of biological activity, large daily cycles in dissolved oxygen and shallow river depth. The maximum concentrations that we measured for sucralose (21 µg/L), cyclamate (0.88 µg/L), and saccharin (7.2 µg/L) are the highest reported concentrations of these compounds in surface waters to date anywhere in the world. Acesulfame persists at concentrations that are up to several orders of magnitude above the detection limit over a distance of 300 km and it behaves conservatively in the river, recording the wastewater contribution from the cumulative population in the basin. Acesulfame is a reliable wastewater effluent tracer in rivers. Furthermore, it can be used to assess rates of nutrient assimilation, track wastewater plume dilution, separate human and animal waste contributions and determine the relative persistence of emerging contaminants in impacted watersheds where multiple sources confound the usefulness of other tracers. The effects of artificial sweeteners on aquatic biota in rivers and in the downstream Great Lakes are largely unknown.

PLOS ONE Staff • August 2014

Correction:
Artificial Sweeteners in a Large Canadian River Reflect Human Consumption in the Watershed

The maximum value for the concentration of cyclamate appears incorrectly throughout the paper as 0.88 µg/L. The correct maximum value for the concentration of cyclamate is 2.4 µg/L. This error appears in the Abstract, the first and second paragraph of the Results and Discussion and the last row of Table 1. **Cyclamate was almost 2.8 (2.7727) times higher than originally reported.**

Source:
http://journals.plos.org/plosone/article?id=10.1371%2Fjournal.pone.0082706



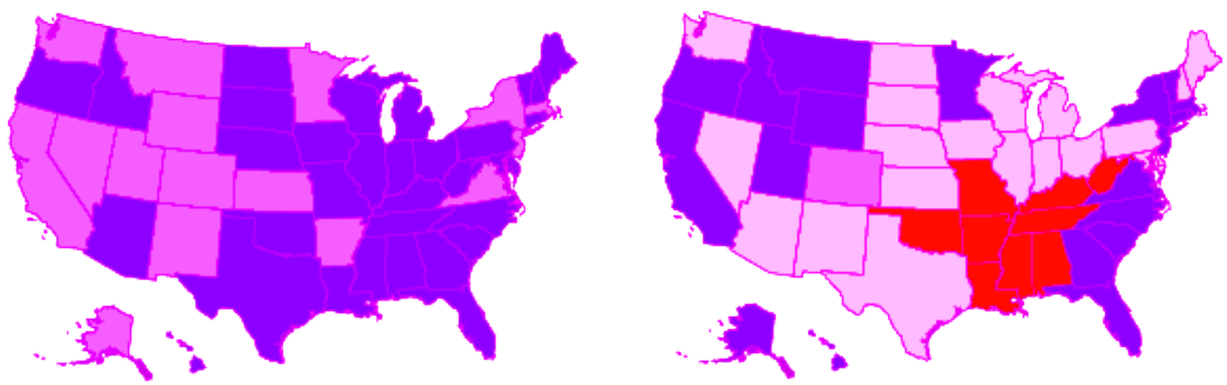
72 MILLION OBESE ADULTS IN THE UNITED STATES

In 2010, more than 60 percent of the United States was overweight or obese. It is estimated that if the current trend continues, 50 percent of the population will be obese by 2030.

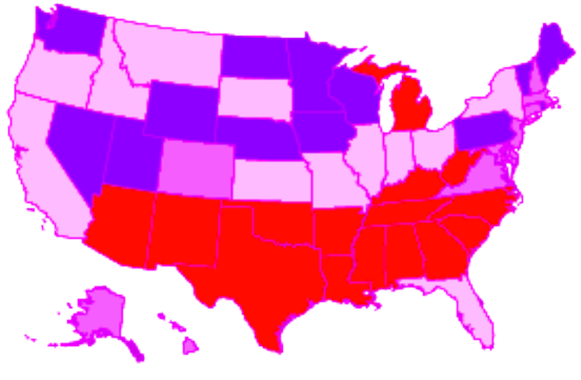
OBESITY RATES

STATE OBESITY RATES VS. STATE POVERTY RATES

1990 Vs. 2009 State Obesity Rates



2009 Poverty Rates



MISSISSIPPI

Has the highest obesity rate in the United States and is also the state with the highest percentage of its population living in poverty

States With The Lowest Obesity Rates	States With The Highest Obesity Rates
Colorado – 18.6%	Mississippi – 34.4%
District of Columbia – 19.7%	Louisiana – 33%
Connecticut – 20.6%	Tennessee – 32.3%

THE COST

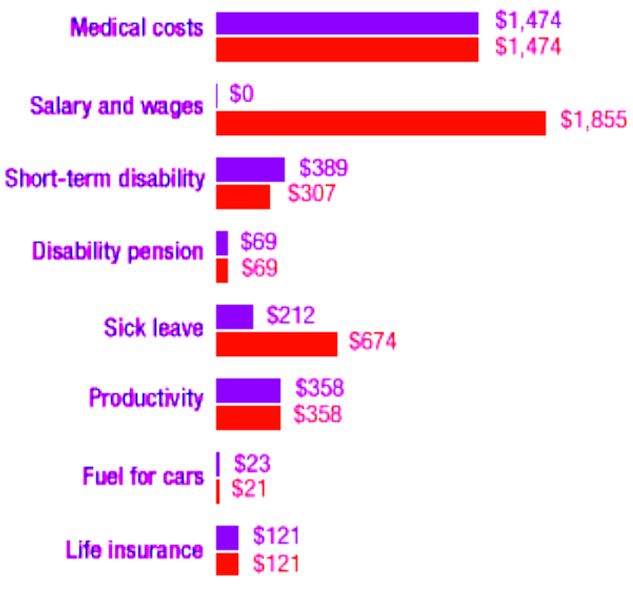
THE COST OF BEING AN OBESE INDIVIDUAL

The amount of money lost annually as a result of being obese

Total Losses By Gender

Women – $4,879 Men – $2,644

Breakdown



THE TYPICAL REVOLVING DOOR HAD TO BE WIDENED FROM 10 FEET TO 12 FEET

PORTION SIZES

CHANGING PORTION SIZES IN AMERICA

Movie Popcorn

1950
3 cups
174 calories

2004
21 cups (buttered)
1,700 calories

Hershey Bar

1900
2 ounces
297 calories

2011
7 ounces
900 calories

FAST FOOD

FAST-FOOD SALES, IN BILLIONS

2000 — $107.147

2010 — $164.836

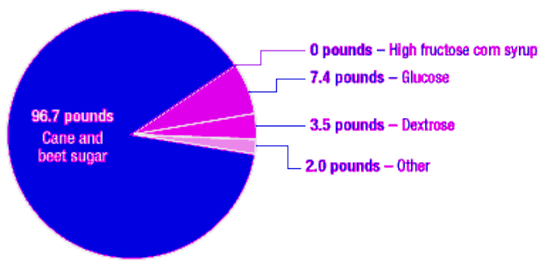
SWEET TOOTH

America's sugar consumption increased by 39 percent between the 1950s and 2000

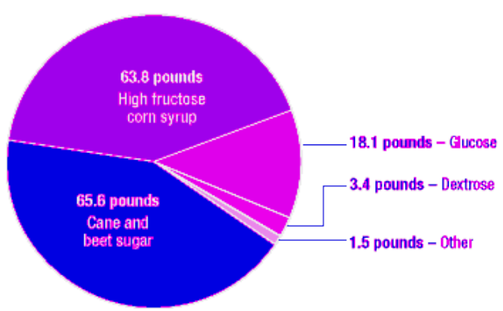
10 Teaspoons of added sugars Americans are advised not to exceed daily

20 Teaspoons of added sugars Americans actually consume

1950 - 1959
Total Caloric Sweeteners – 109.6 pounds, per capita, annually



2000
Total Caloric Sweeteners – 152.4 pounds, per capita, annually



EATING HABITS

AMERICANS ARE DRINKING LESS MILK, EATING MORE CHEESE

Annual American Cheese Consumption, per capita

1950s – 7.7 pounds

2000 – 29.8 pounds

287% Increase in annual cheese consumption since the 1950s

38% Decrease in annual milk consumption since the 1950s

Annual Milk Consumption, per capita

The current milk trend is toward low-fat milk as opposed to whole milk, which represented 92 percent of milk consumption in the 1950s but in 2000 represented only 36 percent.

■ Whole milk ■ Skim milk

1950s – 36.4 gallons

2000 – 22.6 gallons

AMERICANS ARE EATING MORE MEAT AND FEWER EGGS

In 2000, Americans consumed an average of 57 pounds more meat per capita than they did annually in the 1950s, and a third fewer eggs.

□ 1950s ■ 2000

Each animal equals 5 pounds per capita

BEEF
52.8 pounds
64.4 pounds

PORK
45.4 pounds
47.7 pounds

LAMB / VEAL
8.5 pounds
1.4 pounds

POULTRY
16.4 pounds
52.9 pounds

TURKEY
4.1 pounds
13.6 pounds

EGGS
Each egg equals 10 eggs per capita
374 eggs
250 eggs

SUGAR TOO MUCH OF A SWEET THING

The American Heart Association recommends that women consume no more than 6 teaspoons and men no more than 9 teaspoons of added sugars per day. Even one 20 oz. soda contains far more than that.

6 teaspoons added sugars for **women** per day

9 teaspoons added sugars for **men** per day

VS

15 teaspoons of sugars in a 20 oz. Coke

23 teaspoons *Actual* added sugars consumed by average American per day

Chemical Senses • October 2012

Rationale for further medical and health research on high-potency sweeteners

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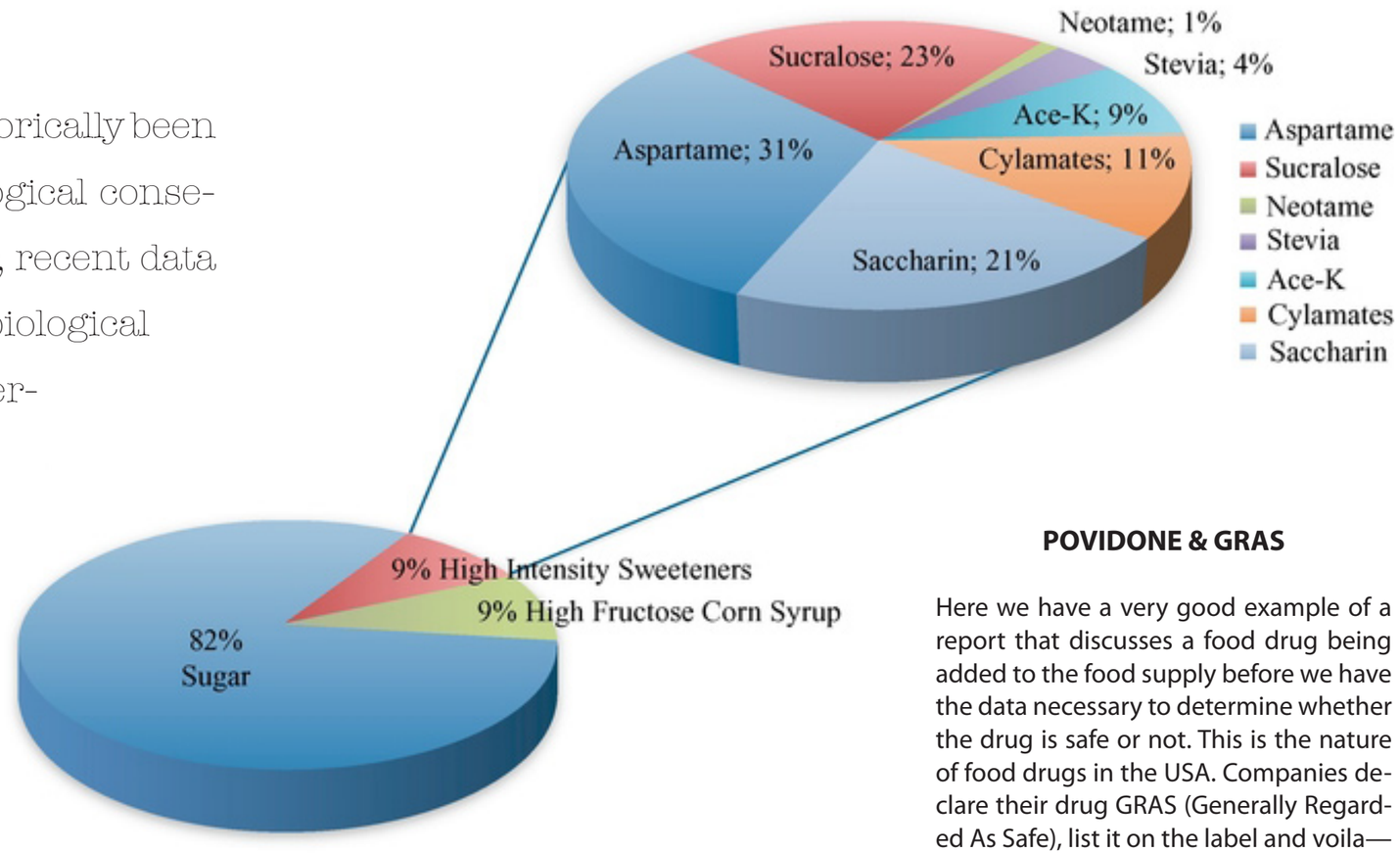
Abstract

High-potency or artificial sweeteners have historically been considered inert compounds without physiological consequences other than taste sensations. However, recent data suggest that some of these sweeteners have biological effects that may impact human health. Furthermore, there are significant gaps in our current knowledge of the pharmacokinetics of these sweeteners, their potential for "sweetener-drug interactions" and their impact on appetite and body weight regulation. Nine research needs are described that address some of the major unknown issues associated with ingestion of high-potency sweeteners.

Full text, graphs, charts and 86 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3440882/

THE GLOBAL HIGH INTENSITY SWEETENER MARKET



POVIDONE & GRAS

Here we have a very good example of a report that discusses a food drug being added to the food supply before we have the data necessary to determine whether the drug is safe or not. This is the nature of food drugs in the USA. Companies declare their drug GRAS (Generally Regarded As Safe), list it on the label and voila—a new food drug enters the market and your body—one like Povidone, a hospital disinfectant seen this year in some over-the-counter medications (Advil) and some foods and beverages.

“In conclusion, the consumption of aspartame leads to histopathological lesions in the liver and alterations of the genetic system in the liver and bone marrow of mother albino rats and their offspring. These toxicological changes were directly proportional to the duration of its administration and improved after its withdrawal.”

Pakistan Journal Of Biological Sciences • October 2012

Cytotoxic effect of aspartame (diet sweet) on the histological and genetic structures of female albino rats and their offspring

Abd Elfatah AA1, Ghaly IS, Hanafy SM.

Author information

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Abstract

The present study evaluated the effect of aspartame intake on the histological and genetic structures of mother albino rats and their offspring. Sixty adult female albino rats and 180 of their offspring were equally divided into two groups (control and treated), each group divided into three subgroups. Each subgroup consisted of 10 pregnant rats and 30 of their offspring. The experimental design divided into three periods: (1) the gestation period (subgroup one), (2) the gestation period and three weeks after delivery (subgroup two) and (3) animals in the third subgroup treated as subgroup two then left till the end of the ninth week after delivery. Each pregnant rat in the treated subgroups was given a single daily dose of 1 mL aspartame solution (50.4 mg) by gastric gavage throughout the time intervals of experimental design. At the end of each experimental period for control and treated subgroups, the liver of half of both control and treated groups were subjected for histological study while the liver and bone marrow of the other halves were subjected for cytogenetic studies. Body weight of both groups were recorded individually twice weekly in the morning before offering the diet. The results revealed that the rats and their offspring in the subgroups of control animals showed increases in body weight, normal histological sections, low chromosomal aberration and low DNA fragmentation. The treated animals in the three subgroups rats and their offspring revealed decreases in body weight, high histological lesions, increases in the chromosomal aberration and DNA fragmentation compared with control groups. In conclusion, the consumption of aspartame leads to histopathological lesions in the liver and alterations of the genetic system in the liver and bone marrow of mother albino rats and their offspring. These toxicological changes were directly proportional to the duration of its administration and improved after its withdrawal.

http://www.ncbi.nlm.nih.gov/pubmed/24159687

FROM THE RED BULL WEBSITE

Aspartame and Acesulfame K are among the most-tested and most-used sugar substitutes worldwide. Acesulfame K is a non-caloric sweetener. It is used worldwide in over a thousand different products including foods and drinks like chewing gum, dairy products, baked goods, etc. Aspartame is a low-calorie sweetener that is produced synthetically. It is used worldwide in over 5,000 different products (such as soft drinks, yogurts, candy, chewing gum, etc.).

Both, Aspartame and Acesulfame K, have an excellent safety profile. Numerous scientific studies demonstrate that these substances are safe for use as sweetening ingredients. The safety of these sweeteners has been evaluated by regulatory bodies all over the world (e.g. FDA in the US). Health Authorities rely on the safety evaluations of independent scientific advisory bodies such as the European Food Safety Authority (EFSA), the United States Food and Drug Administration (FDA), and the Joint FAO/WHO Expert Committee on Food Additives (JECFA).

The Red Bull “Road To Rampage” logo on the can at right is accurate. Drink enough of this poison and you’ll be rampaging for sure.

European Review For Medical And Pharmacological Sciences • December 2012

Studies on the effects of aspartame on memory and oxidative stress in brain of mice

Author information

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Abstract

OBJECTIVE

The dipeptide aspartame (N-L-alpha-aspartyl-Lphenylalanine, 1-methyl ester; alpha-APM) is one of the most widely used artificial sweeteners. The present study aimed to investigate the effect of repeated administration of aspartame in the working memory version of Morris water maze test, on oxidative stress and brain monoamines in brain of mice.

MATERIALS AND METHODS

Aspartame (0.625, 1.875 or 5.625 mg/kg) was administered once daily subcutaneously for 2 weeks and mice were examined four times a week for their ability to locate a submerged plate. Malondialdehyde (MDA), reduced glutathione (GSH), nitric oxide levels (the concentrations of nitrite/nitrate) and glucose were determined in brain.

RESULTS

Only at the highest dose of 5.625 mg/kg, did aspartame significantly impaired water maze performance. The mean time taken to find the escape platform (latency) over 2 weeks was significantly delayed by aspartame 5.625 mg/kg, compared with the saline-treated control group. Significant differences occurred only on the first trial to find the escape platform. Significant increase in brain MDA by 16.5% and nitric oxide by 16.2% and a decrease in GSH by 25.1% and glucose by 22.5% occurred after treatment with aspartame at 1.875 mg/kg. Aspartame administered at 5.625 mg/kg significantly increased brain MDA by 43.8%, nitric oxide by 18.6% and decreased GSH by 32.7% and glucose by 25.8%. Aspartame caused dose-dependent inhibition of brain serotonin, noradrenaline and dopamine.

CONCLUSIONS

These findings suggest impaired memory performance and increased brain oxidative stress by repeated aspartame administration. The impaired memory performance is likely to involve increased oxidative stress as well as decreased brain glucose availability.

http://www.ncbi.nlm.nih.gov/pubmed/23280025

Human And Experimental Toxicology • February 2011

Effect of saccharin on albino rats' blood indices and the therapeutic action of vitamins C and E

Author information

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Abstract

The present work aimed to study some blood indices of rats as affected by saccharin and the therapeutic action of vitamins C and E. The used adult female Rattus norvegicus albino rats in the present study were weighing 100-120 g. Administration of saccharin at a dose of 35 mg kg body weight (b.wt.) day for 35 days significantly decreased serum glucose, triglycerides, cholesterol, total protein and albumin values. These decrements were by 20.16%, 22.76%, 44.92%, 20.16% and 40.44%, respectively, compared to control level (p value < 0.01). But it increased levels of kidney function indices. The effect of saccharin was more pronounced on creatinine. Activities of Alanine aminotranferease (ALT), aspartate aminotransferase (AST) and Alkaline phosphatase (ALP) increased significantly following saccharin treatment to rats. Concerning hematoligical parameters, the more obvious changes were observed in the increment of white blood cell (WBC), mean corpuscular volume (MCV) and platelets (PLT) and the decrease in hematocrit, hemoglobin (Hb) and red blood cells (RBCs) count in response to the administration of saccharin. In general, vitamin C or E (150 mg kg b.wt. day for 35 days) was able to reduce the effects of saccharin intake. Both vitamins, however, generally have beneficial effects in reducing the changes in the studied parameters.

http://www.ncbi.nlm.nih.gov/pubmed/20382728

Public Health & Nutrition • March 2011

To what extent have sweetened beverages contributed to the obesity epidemic?

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Abstract

OBJECTIVE

A systematic literature review was conducted to determine whether sweetened beverage intake increases the risk for obesity, and the extent to which it has contributed to recent increases in energy intake and adiposity in the USA.

DESIGN

The search included studies published between 1970 and 2010 that examined secular trends, mechanisms, observational associations and intervention outcomes. Observational and intervention studies were abstracted and systematically evaluated for quality.

SETTING

Trends in obesity prevalence in the USA and studies from industrialized (developed) countries were included.

SUBJECTS

Studies were included for all ages, genders, ethnic and socio-economic groups for which data were available.

RESULTS

Obesity rates and sweetened beverage intake have increased in tandem in the USA. Studies consistently show that higher intake of sweetened beverages is associated with higher energy intake. Energy in liquid form is not well compensated for by reductions in the intake of other sources of energy. Well-designed observational studies consistently show a significant positive relationship between sweetened beverage intake and adiposity. More importantly, several well-conducted randomized controlled trials have shown statistically significant changes in adiposity as a result of corresponding changes in sweetened beverage intake.

CONCLUSIONS

All lines of evidence consistently support the conclusion that the consumption of sweetened beverages has contributed to the obesity epidemic. It is estimated that sweetened beverages account for at least one-fifth of the weight gained between 1977 and 2007 in the US population. Actions that are successful in reducing sweetened beverage consumption are likely to have a measurable impact on obesity.

http://www.ncbi.nlm.nih.gov/pubmed/20860886

“It is estimated that sweetened beverages account for at least one-fifth of the weight gained between 1977 and 2007 in the US population.”

“These results suggest that long-term consumption of Artificial Sweeteners might accelerate atherosclerosis and senescence ...”

Molecules And Cells • May 2011

Modified apolipoprotein (apo) A-I by artificial sweetener causes severe premature cellular senescence and atherosclerosis with impairment of functional and structural properties of apoA-I in lipid-free and lipid-bound state

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Abstract

Long-term consumption of artificial sweeteners (AS) has been the recent focus of safety concerns. However, the potential risk of the AS in cardiovascular disease and lipoprotein metabolism has not been investigated sufficiently. We compared the influence of AS (aspartame, acesulfame K, and saccharin) and fructose in terms of functional and structural correlations of apolipoprotein (apo) A-I and high-density lipoproteins (HDL), which have atheroprotective effects. Long-term treatment of apoA-I with the sweetener at physiological concentration (3 mM for 168 h) resulted in loss of antioxidant and phospholipid binding activities with modification of secondary structure. The AS treated apoA-I exhibited proteolytic cleavage to produce 26 kDa-fragment. They showed pro-atherogenic properties in acetylated LDL phagocytosis of macrophages. Each sweetener alone or sweetener-treated apoA-I caused accelerated senescence in human dermal fibroblasts. These results suggest that long-term consumption of AS might accelerate atherosclerosis and senescence via impairment of function and structure of apoA-I and HDL.

Full Text, Graphs, Charts and 40 References

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3887604/

“It can be concluded from these observations that long term consumption of aspartame leads to hepatocellular injury and alterations in liver antioxidant status ...”

Food And Chemical Toxicology • June 2011

Effect of long term intake of aspartame on antioxidant defense status in liver

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Abstract

The present study evaluates the effect of long term intake of aspartame, the artificial sweetener, on liver antioxidant system and hepatocellular injury in animal model. Eighteen adult male Wistar rats, weighing 150-175 g, were randomly divided into three groups as follows: first group was given aspartame dissolved in water in a dose of 500 mg/kg b.wt.; the second group was given a dose of 1000 mg/kg b.wt.; and controls were given water freely. Rats that had received aspartame (1000 mg/kg b.wt.) in the drinking water for 180 days showed a significant increase in activities of alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP) and □-glutamyl transferase (GGT). The concentration of reduced glutathione (GSH) and the activity of glutathione peroxidase (GPx), and glutathione reductase (GR) were significantly reduced in the liver of rats that had received aspartame (1000 mg/kg b.wt.). Glutathione was significantly decreased in both the experimental groups. Histopathological examination revealed leukocyte infiltration in aspartame-treated rats (1000 mg/kg b.wt.). It can be concluded from these observations that long term consumption of aspartame leads to hepatocellular injury and alterations in liver antioxidant status mainly through glutathione dependent system.

http://www.ncbi.nlm.nih.gov/pubmed/21376768

> “... it would be worthwhile to investigate whether possible links between sucralose intake and Inflammatory Bowel Disease exist, before it is too late.”

Canadian Journal Of Gastroenterology • September 2011

What made Canada become a country with the highest incidence of inflammatory bowel disease: Could sucralose be the culprit?

By Xiaofa Qin, MD PhD, Department of Surgery, University of Medicine and Dentistry of New Jersey, New Jersey Medical School, Newark, NJ, USA

Abstract

Inflammatory bowel disease (IBD) (which includes both ulcerative colitis and Crohn’s disease [CD]) emerged and dramatically increased in the past century (1). Early studies revealed that IBD was most prevalent in countries such as the United Kingdom, the United States and those in northern Europe (1). Compared with these countries, the prevalence of IBD in Canada was much lower. This was demonstrated in early epidemiological studies conducted in Canada. According to a review by Mayberry and Rhodes (2), a study published in 1972 reported the incidence and prevalence of CD in Sherbrooke, Quebec, at only 0.7 and 6.3 per 100,000 population, respectively, which were much lower than countries such as the United Kingdom, the United States, Sweden, Denmark, and even Israel and New Zealand at the same time or even decades earlier. However, studies in recent years have suddenly found that Canada has become a country with the highest incidence of IBD (3). For example, the prevalence of CD in Alberta in 1981 was only 44 per 100,000 population (4), compared with 91 per 100,000 in Olmsted County, Minnesota (USA) on January 1, 1980 (5). However, the prevalence of CD in Alberta increased to 283 per 100,000 on July 1, 2000 (3), compared with 174 per 100,000 in Olmsted County in January 1, 2001 (6). It would be valuable to know what caused the dramatic increase of IBD in Canada because it may provide critical information regarding its etiology.

A decade ago, a series of accidental findings made me suspect that the impaired inactivation of digestive proteases due to the inhibition of gut bacteria by dietary chemicals, such as saccharin, play a causative role in IBD as a result of the accelerated degradation of the mucous layer and underlying endothelium (7). It provided an explanation for many puzzles in IBD such as the dramatic increase of IBD in the 1950s and 1960s, and its levelling off since the latter part of the 1970s, as observed in many western countries including Canada (4,7). However, this hypothesis was challenged by the failure to provide an explanation for the recent high incidence of IBD in Canada, which had adopted more stringent standards for the use of saccharin than most other western countries after the finding of carcinogenic effects of saccharin on the bladders of experimental animals in 1977.

If not saccharin, then what caused the remarkable increase of IBD in Canada? I suggest that sucralose may be the culprit. Sucralose is a new, non-nutrient, high-intensity sweetener that has many superior properties. It is approximately 600 times sweeter than sucrose (thus two times sweeter than saccharin). Similar to saccharin, sucralose is heat and pH stable, but without the bitter aftertaste (8). In 1991, Canada was the first country to approve the use of sucralose, and it was allowed to be used as a tabletop sweetener in breakfast cereals, beverages, desserts, toppings, fillings, chewing gum, breath mints, fruit spreads, salad dressings, confectionary, bakery products, processed fruits and vegetables, alcoholic beverages, puddings and table syrups (8). Interestingly, the study by Wrobel et al (9) reported that the incidence of pediatric IBD in Southern Alberta was 2.3 (per 100,000 population) between 1983 and 1987, 2.5 between 1988 and 1992, 5.0 between 1993 and 1998, and 6.5 between 1999 and 2005 (9), indicating a dramatic increase in the early 1990s. Could sucralose cause the increase of IBD in Canada? How?

Similar to saccharin, sucralose can also exert potent inhibition of gut bacteria (10). However, it may have a more pronounced effect on gut bacteria than saccharin in that approximately 65% to 95% of sucralose is excreted through the feces unchanged (10), while a large proportion of saccharin is absorbed and eliminated through urine; the acceptable daily intake of sucralose is 15 mg/kg, but only 5 mg/kg for saccharin. As I suggested a decade ago, regarding the possible risk of saccharin on IBD (7), sucralose may have a similar but stronger impact on gut bacteria, digestive protease inactivation and gut barrier function. This may provide a possible explanation for the more pronounced high incidence of IBD observed in Canada. The use of sucralose is soaring, and is now being used in thousands of food products (10). Therefore, it would be worthwhile to investigate whether possible links between sucralose intake and IBD exist, before it is too late.

Full text with 10 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3202359/

> “... animal studies have convincingly proven that artificial sweeteners cause weight gain, brain tumors, bladder cancer and many other health hazards. Some kind of health related side effects including carcinogenicity are also noted in humans.”

Journal Of Pharmacology And Pharmacotherapy • October 2011

Sugar substitutes: Health controversy over perceived benefits

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Abstract

Sugar is an inseparable part of the food we consume. But too much sugar is not ideal for our teeth and waistline. There have been some controversial suggestions that excessive sugar may play an important role in certain degenerative diseases. So artificial sweeteners or artificially sweetened products continue to attract consumers. A sugar substitute (artificial sweetener) is a food additive that duplicates the effect of sugar in taste, but usually has less food energy. Besides its benefits, animal studies have convincingly proven that artificial sweeteners cause weight gain, brain tumors, bladder cancer and many other health hazards. Some kind of health related side effects including carcinogenicity are also noted in humans. A large number of studies have been carried out on these substances with conclusions ranging from “safe under all conditions” to “unsafe at any dose”. Scientists are divided in their views on the issue of artificial sweetener safety. In scientific as well as in lay publications, supporting studies are often widely referenced while the opposing results are de-emphasized or dismissed. So this review aims to explore the health controversy over perceived benefits of sugar substitutes.

Full text, graphs, charts and 54 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3198517/

“Our data suggest that acesulfame-K can be ingested by the prenatal or postnatal mice through their mother’s amniotic fluid or breast milk, producing a long-dated function on the adult’s sweet preference.”

Oxford Journal • Chemical Senses • November 2011

Effects of mother’s dietary exposure to acesulfame-K in Pregnancy or lactation on the adult offspring’s sweet preference

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Abstract

This study investigates whether mother’s exposure to the artificial sweetener acesulfame-K (AK) during pregnancy or lactation affected her adult offspring’s sweet preference. It was found that mother’s dietary exposure to AK in pregnancy or lactation decreased the preference thresholds for AK and sucrose solutions in the adult offspring, whereas the preference pattern and the most preferred concentration for AK or sucrose solution were unchanged. Furthermore, the preference scores in the exposure groups were increased significantly when compared with the control group at a range of concentrations for AK or sucrose solution. The existence of AK and its dynamic changes within 24 h in amniotic fluid during pregnancy or in mother’s milk during lactation after a single oral infusion of AK solution were revealed by the methods of reversed-phase high-performance liquid chromatography and mass spectrometry. Our data suggest that AK can be ingested by the prenatal or postnatal mice through their mother’s amniotic fluid or breast milk, producing a long-dated function on the adult’s sweet preference.

Full Text, Graphs, Charts and 34 References

http://chemse.oxfordjournals.org/content/36/9/763.long

Food Chemical Toxicology • November 2011

Aspartame-fed zebrafish exhibit acute deaths with swimming defects and saccharin-fed zebrafish have elevation of cholesteryl ester transfer protein activity in hypercholesterolemia

Author information
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Abstract

Although many artificial sweeteners (AS) have safety issues, the AS have been widely used in industry. To determine the physiologic effect of AS in the presence of hyperlipidemia, zebrafish were fed aspartame or saccharin with a high-cholesterol diet (HCD). After 12 days, 30% of zebrafish, which consumed aspartame and HCD, died with exhibiting swimming defects. The aspartame group had 65% survivability, while the control and saccharin groups had 100% survivability. Under HCD, the saccharin-fed groups had the highest increase in the serum cholesterol level (599 mg/dL). Aspartame-fed group showed a remarkable increase in serum glucose (up to 125 mg/dL), which was 58% greater than the increase in the HCD alone group. The saccharin and HCD groups had the highest cholesteryl ester transfer protein (CETP) activity (52% CE-transfer), while the HCD alone group had 42% CE-transfer. Histologic analysis revealed that the aspartame and HCD groups showed more infiltration of inflammatory cells in the brain and liver sections. Conclusively, under presence of hyperlipidemia, aspartame-fed zebrafish exhibited acute swimming defects with an increase in brain inflammation. Saccharin-fed zebrafish had an increased atherogenic serum lipid profile with elevation of CETP activity.

http://www.ncbi.nlm.nih.gov/pubmed/21855599

> “After 12 days, 30% of zebrafish, which consumed aspartame and high cholesterol diet, died with exhibiting swimming defects ... aspartame-fed zebrafish exhibited acute swimming defects with an increase in brain inflammation.”

International Journal Of Occupational And Environmental Health • January 2010

Inadequate toxicity tests of food additive acesulfame

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Abstract

Despite poor-quality toxicity tests, acesulfame potassium was approved by the US Food and Drug Administration (FDA) for use as an artificial sweetener. At present, acesulfame is very widely used, most frequently in blends with the most popular artificial sweetener in the US, sucralose (Splenda). Acesulfame was nominated twice (in 1996 and again in 2006) for testing in the National Toxicology Program (NTP) bioassay program. Both nominations were rejected by NTP. Rather than carry out bioassays, NTP subjected acesulfame to tests in genetically modified mice (GMM). Those GMM tests yielded results that provided no insight into potential carcinogenicity of acesulfame. It is possible that FDA discouraged NTP from conducting bioassays of acesulfame. Acesulfame should be tested in the bioassay program as soon as possible, and steps should be taken to ensure the objectivity of the bioassay nomination process.

http://www.ncbi.nlm.nih.gov/pubmed/20166324

Yale Journal Of Biology And Medicine • June 2010

Gain weight by “going diet?” Artificial sweeteners and the neurobiology of sugar cravings: Neuroscience 2010

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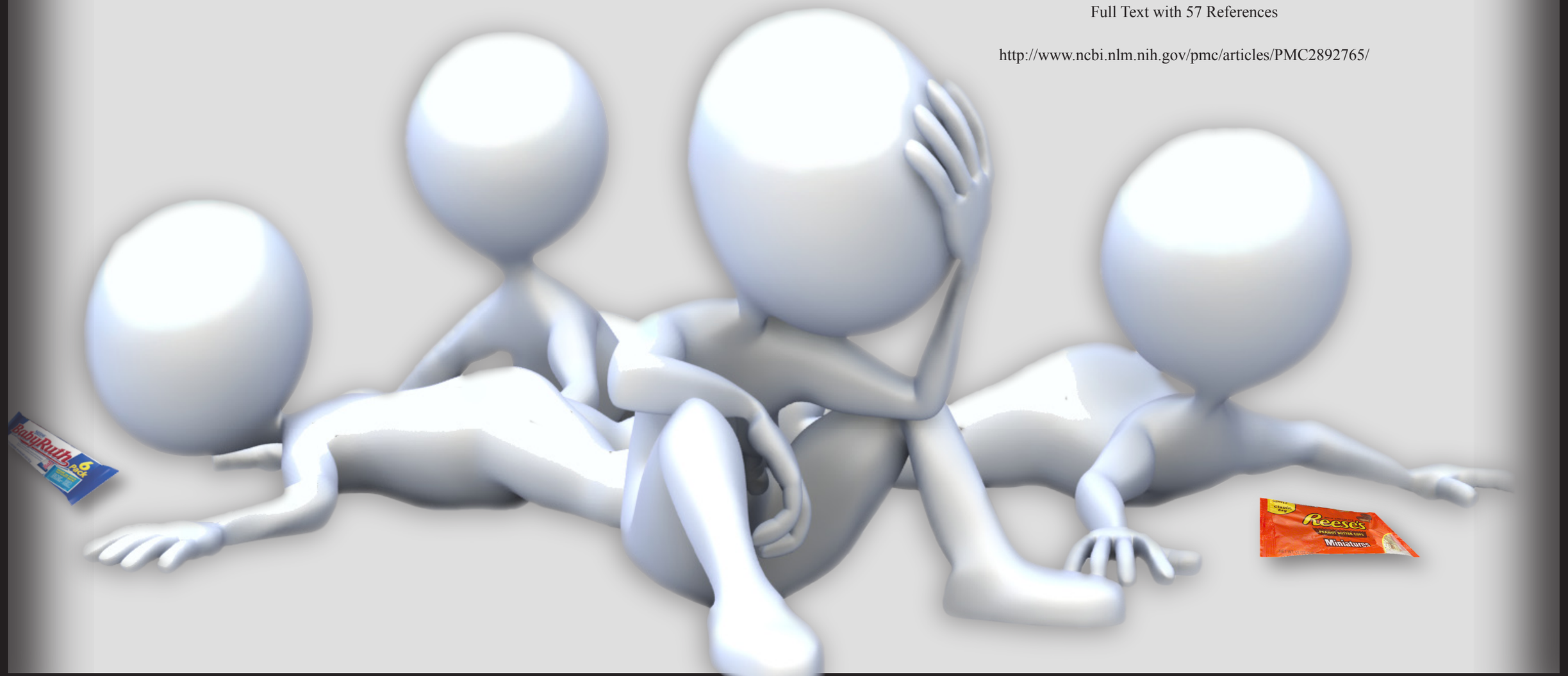
Abstract

America’s obesity epidemic has gathered much media attention recently. A rise in the percent of the population who are obese coincides with an increase in the widespread use of non-caloric artificial sweeteners, such as aspartame (e.g., Diet Coke) and sucralose (e.g., Pepsi One), in food products (Figure 1). Both forward and reverse causalities have been proposed. While people often choose “diet” or “light” products to lose weight, research studies suggest that artificial sweeteners may contribute to weight gain. In this mini-review, inspired by a discussion with Dr. Dana Small at Yale’s Neuroscience 2010 conference in April, I first examine the development of artificial sweeteners in a historic context. I then summarize the epidemiological and experimental evidence concerning their effects on weight. Finally, I attempt to explain those effects in light of the neurobiology of food reward.

Full Text with 57 References

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2892765/



“frequent consumption of infusions ... [of] potatoes, alcohol, sweets, and processed meat resulted in a high risk for urinary tract tumors”

European Journal Of Cancer Prevention • November 2010

Dietary patterns and food groups are linked to the risk of urinary tract tumors in Argentina

Author information

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Abstract

Epidemiological and laboratory research has shown that dietary components are associated with the risk of developing urinary tract tumors (UTT). The purpose of this case-control study, carried out between 2004 and 2008 in Córdoba, a Mediterranean city in Argentina, was to describe the role of dietary patterns and to investigate any association with the risk of developing UTT. One hundred and sixty-eight patients with histologically confirmed transitional UTT and 334 controls with acute, nonneoplastic, and nonurinary tract diseases from the same hospitals were studied. All patients were interviewed about their food habits and their exposure to a number of known or suspected risk factors for UTT. Multiple correspondence analysis was used to explore dietary patterns and data analyses were carried out by calculating odds ratios and their 95% confidence intervals by using multiple logistic regression. Two main dietary patterns identified were a ‘prudent’ pattern that was linked to controls and a ‘western’ pattern that was associated with cases. A frequent intake of vegetable oils, lean meats, grains, and fruits, the moderate use of alcohol (mainly red wine) together with potato and sweet consumption, and the habit of taking at least four meals per day, were associated with a reduced risk for UTT. In contrast, frequent consumption of infusions (mainly maté), potatoes, alcohol, sweets, and processed meat resulted in a high risk for UTT. The dietary patterns of our population have a role in the development of UTT, thus implying that appropriate nutritional education may decrease this risk.

http://www.ncbi.nlm.nih.gov/pubmed/20736839

“Consumption of sweetened beverages is however clearly associated with excess calorie intake, and an increased risk of diabetes and cardiovascular diseases through an increase in body weight.”

Nutrition • November 2010

Fructose and metabolic diseases: new findings, new questions

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Abstract

There has been much concern regarding the role of dietary fructose in the development of metabolic diseases. This concern arises from the continuous increase in fructose (and total added caloric sweeteners consumption) in recent decades, and from the increased use of high-fructose corn syrup (HFCS) as a sweetener. A large body of evidence shows that a high-fructose diet leads to the development of obesity, diabetes, and dyslipidemia in rodents. In humans, fructose has long been known to increase plasma triglyceride concentrations. In addition, when ingested in large amounts as part of a hypercaloric diet, it can cause hepatic insulin resistance, increased total and visceral fat mass, and accumulation of ectopic fat in the liver and skeletal muscle. These early effects may be instrumental in causing, in the long run, the development of the metabolic syndrome. There is however only limited evidence that fructose per se, when consumed in moderate amounts, has deleterious effects. Several effects of a high-fructose diet in humans can be observed with high-fat or high-glucose diets as well, suggesting that an excess caloric intake may be the main factor involved in the development of the metabolic syndrome. The major source of fructose in our diet is with sweetened beverages (and with other products in which caloric sweeteners have been added). The progressive replacement of sucrose by HFCS is however unlikely to be directly involved in the epidemy of metabolic disease, because HFCS appears to have basically the same metabolic effects as sucrose. Consumption of sweetened beverages is however clearly associated with excess calorie intake, and an increased risk of diabetes and cardiovascular diseases through an increase in body weight. This has led to the recommendation to limit the daily intake of sugar calories.

http://www.ncbi.nlm.nih.gov/pubmed/20471804

Asia Pacific Journal Of Clinical Nutrition • December 2010

Dietary habits and overweight/obesity in adolescents in Xi'an City, China

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Abstract

This study explored the association between dietary habits and overweight and obesity in adolescents from Xi'an City, China. A cross-sectional sample of 1804 adolescents was recruited in 2004 from 30 junior high schools in six districts of Xi'an City, northwest China. Weight and height was measured and eating habits assessed using a self-administered questionnaire. Logistic regression was used to identify dietary patterns associated with overweight and obesity and adjusted for socio-demographic factors. Consumption of foods and beverages outside three main meals, and potato chips was more popular in boys than in girls, while girls consumed more fried food and soft drinks than boys. In boys, an increased consumption of soft drinks was associated with increased risk of overweight and obesity (1100 mL/day, OR: 1.9, 95% CI: 1.1-3.8), while consuming preserved fruit was associated with decreased risk (OR: 0.6, 95% CI: 0.5-0.9). In girls, having breakfast outside the home (OR: 1.7, 95% CI: 1.1-2.3) and an increased consumption of energy-dense foods (OR: 1.7, 95% CI: 1.04-2.9), was associated with increased risk of overweight and obesity, while frequently having foods and beverages outside the three main meals (OR: 0.6, 95% CI: 0.4-0.9) was associated with decreased risk. The consumption of breakfast outside the home, soft drinks and energy-dense fast foods were positively associated with overweight and obesity in adolescents. Future health education programs to prevent excess weight gain should target such unhealthy eating habits.

Full text in both English and Chinese versions, graphs and 23 references

http://apjcn.nhri.org.tw/server/APJCN/19/1/76.pdf

"The consumption of breakfast outside the home, soft drinks and energy-dense fast foods were positively associated with overweight and obesity in adolescents."

> “Consumption of sweeteners [saccharin, cyclamate based, acesulfame-K based, and aspartame] resulted in significantly increased body weight; however, the food intake did not change. These results question the effect of non-caloric artificial sweeteners on weight-maintenance or body weight decrease.”

Acta Physiologica Hungarica • December 2010

Effects of artificial sweeteners on body weight, food and drink intake

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Abstract

Artificial sweeteners are widely used all over the world. They may assist in weight management, prevention of dental caries, control of blood glucose of diabetics, and also can be used to replace sugar in foods. In the animal experimentation mice were given oral doses of water solutions of table top artificial sweeteners (saccharin, cyclamate based, acesulfame-K based, and aspartame) the amount of maximum Acceptable Daily Intake (ADI) ad libitum. The controls received only tap water with the same drinking conditions as the treated groups. The mice were fed chow ad libitum. We measured food intake and body weight once a week, water and solutions of artificial sweeteners intake twice a week. The data were analysed by statistical methods (T-probe, regression analysis). Consumption of sweeteners resulted in significantly increased body weight; however, the food intake did not change. These results question the effect of non-caloric artificial sweeteners on weight-maintenance or body weight decrease.

http://www.ncbi.nlm.nih.gov/pubmed/21138816

American Journal Of Clinical Nutrition • January 2009

Non-nutritive sweetener consumption in humans: effects on appetite and food intake and their putative mechanisms

Author information

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Abstract

Non-nutritive sweeteners (NNS) are ecologically novel chemosensory signaling compounds that influence ingestive processes and behavior. Only about 15% of the US population aged >2 y ingest NNS, but the incidence is increasing. These sweeteners have the potential to moderate sugar and energy intakes while maintaining diet palatability, but their use has increased in concert with BMI in the population. This association may be coincidental or causal, and either mode of directionality is plausible. A critical review of the literature suggests that the addition of NNS to non-energy-yielding products may heighten appetite, but this is not observed under the more common condition in which NNS is ingested in conjunction with other energy sources. Substitution of NNS for a nutritive sweetener generally elicits incomplete energy compensation, but evidence of long-term efficacy for weight management is not available. The addition of NNS to diets poses no benefit for weight loss or reduced weight gain without energy restriction. There are long-standing and recent concerns that inclusion of NNS in the diet promotes energy intake and contributes to obesity. Most of the purported mechanisms by which this occurs are not supported by the available evidence, although some warrant further consideration. Resolution of this important issue will require long-term randomized controlled trials.

Full text, graphs, charts and 224 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2650084/

“The addition of Non-nutritive sweeteners to diets poses no benefit for weight loss or reduced weight gain without energy restriction. There are long-standing and recent concerns that inclusion of Non-nutritive sweeteners in the diet promotes energy intake and contributes to obesity.”

Public Health Nutrition • August 2009

Fast-food and sweetened beverage consumption: association with overweight and high waist circumference in adolescents

Author information

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Abstract

OBJECTIVE

Overweight and obesity have increased to epidemic proportions among adolescents and are associated with chronic non-communicable diseases and excess mortality in adulthood. The association of overweight/obesity with poor dietary habits has not been studied in adolescents in middle-income developing countries. The present study aimed to estimate the prevalence of overweight, obesity and high waist circumference (WC) in 15-19-year-old Jamaican adolescents and to investigate the association with fast-food and sweetened beverage consumption.

DESIGN

The study enrolled 1317 (598 male, 719 female) adolescents aged 15-19 years using multistage, nationally representative sampling. Age-specific prevalence calculation used internal Z-score lines connecting with the WHO adult cut-off points. Logistic regression was used to examine the association of overweight or high WC with fast-food and sweetened beverage consumption, adjusting for potential confounders.

RESULTS

The overall prevalence of overweight, obesity and high WC was approximately 15 %, 6 % and 10 %, respectively. Prevalence estimated using internal Z-scores was similar to that using the International Obesity Taskforce cut-off points. Obesity (8.0 % in females, 3.3 % in males) and high WC (16.2 % in females, 1.7 % in males) were significantly more prevalent in females when using internal Z-score cut-offs. High WC was associated with the absence of fruit consumption (P = 0.043) and overweight with high sweetened beverage consumption (P = 0.018).

CONCLUSION

Overweight occurs frequently among Jamaican 15-19-year-olds and is associated with increased consumption of sweetened beverages. High WC is more prevalent among females and is related to low consumption of fruits and vegetables. Measures to reduce the consumption of sweetened beverages and increase fruit intake may reduce the prevalence of excess body fat among adolescents.

http://www.ncbi.nlm.nih.gov/pubmed/19243675

“Overweight occurs frequently among Jamaican 15-19-year-olds and is associated with increased consumption of sweetened beverages. High Waist Circumference is more prevalent among females and is related to low consumption of fruits and vegetables.”

Cases Journal • September 2009

Vestibulocochlear toxicity in a pair of siblings 15 years apart secondary to aspartame: two case reports

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Abstract

INTRODUCTION

Aspartame may have idiosyncratic toxic effects for some people; however, there are few case reports published in the medical literature. We present two case reports in a pair of siblings, one with a vestibular and the other with a cochlear toxicity to aspartame. The cochlear toxicity is the first case to be reported, while the vestibular toxicity is the second case to be reported.

CASE PRESENTATION

A 29-year-old white female had a 20-month history of nausea and headache, progressively getting worse with time and eventually to also involve vomiting, vertigo, and ataxia. She was extensively evaluated and diagnosed with a vestibular neuronitis versus a chronic labyrinthitis and treated symptomatically with limited success. In response to a newspaper article, she stopped her aspartame consumption with total cessation of her symptoms. Fifteen years later, her then 47-year-old white brother had a 30-month history of an intermittent, initially 5-10 minute long episode of a mild sensorineural hearing loss in his right ear that progressed over time to several hour episodes of a moderately severe high-frequency sensorineural hearing loss to include tinnitus and a hypoesthetic area in front of his right tragus. After a negative magnetic resonance scan of the brain, he remembered his sister's experience with aspartame and stopped his consumption of aspartame with resolution of his symptoms, although the very high frequency hearing loss took at least 15 months to resolve. For both, subsequent intentional challenges with aspartame and unintentional exposures brought back each of their respective symptoms.

CONCLUSION

Aspartame had a vestibulocochlear toxicity in a pair of siblings, suggesting a genetic susceptibility to aspartame toxicity. Even though the yield may be low, asking patients with dizziness, vertigo, tinnitus, or high-frequency hearing loss about their aspartame consumption and suggesting cessation of its use, may prove helpful for some.

Full text with graphs, charts and references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2815650/

“Aspartame had a vestibulocochlear toxicity in a pair of siblings, suggesting a genetic susceptibility to aspartame toxicity. Even though the yield may be low, asking patients with dizziness, vertigo, tinnitus, or high-frequency hearing loss about their aspartame consumption and suggesting cessation of its use, may prove helpful for some.”

AMOXICILLIN INGREDIENTS

Chewable Tablets: Each cherry-banana-peppermint-flavored tablet contains 200 mg or 400 mg amoxicillin as the trihydrate. Each 200-mg chewable tablet contains 0.0005 mEq (0.0107 mg) of sodium; the 400-mg chewable tablet contains 0.0009 mEq (0.0215 mg) of sodium. The 200-mg and 400-mg pale pink round tablets are imprinted with the product name AMOXIL and 200 or 400 along the edge of 1 side. Inactive ingredients: **Aspartame**, **crospovidone NF**, **FD&C Red No. 40 aluminum lake, flavorings, magnesium stearate, and mannitol**.

Capsules: Each capsule of AMOXIL, with royal blue opaque cap and pink opaque body, contains 500 mg amoxicillin as the trihydrate. The cap and body of the 500-mg capsule are imprinted with AMOXIL and 500. Inactive ingredients: **D&C Red No. 28, FD&C Blue No. 1, FD&C Red No. 40, gelatin, magnesium stearate, and titanium dioxide**.

Tablets: Each tablet contains 500 mg or 875 mg amoxicillin as the trihydrate. Each film-coated, capsule-shaped, pink tablet is debossed with AMOXIL centered over 500 or 875, respectively. The 875-mg tablet is scored on the reverse side. Inactive ingredients: **Colloidal silicon dioxide, crospovidone, FD&C Red No. 30 aluminum lake**, **hypromellose, magnesium stearate, microcrystalline cellulose, polyethylene glycol**, **sodium starch glycolate,** and **titanium dioxide.**

Pediatric Drops for Oral Suspension: Each mL of reconstituted suspension contains 50 mg amoxicillin as the trihydrate and 0.03 mEq (0.69 mg) of sodium. Amoxicillin trihydrate for oral suspension 200 mg/5 mL, 250 mg/5 mL (or 50 mg/mL), and 400 mg/5 mL are bubble-gum-flavored pink suspensions. Inactive ingredients: **FD&C Red No. 3, flavorings, silica gel, sodium benzoate**, sodium citrate, **sucrose, and xanthan gum.**

Basic Clinical Pharmacology And Toxicology • February 2008

Consequences of exposure to carcinogens beginning during developmental life

Author information

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Abstract

The increased incidence of cancer over the last 50-60 years may be largely attributed to two factors: the ageing of the population and the diffusion of agents and situations presenting carcinogenic risks. Today, we have entered into a new era in which populations are ever-increasingly exposed to diffuse carcinogenic risks, present not only in the occupational, but also in the general environment. We must now also consider an additional factor in the carcinogenic process, that is, the age in which exposure to carcinogenic risks begins. Apart from the paradigmatic cases of diethylstilboestrol and ionizing radiation, the available epidemiological data concerning the adult consequences of developmental exposure to carcinogens is very limited. However, important data have been provided by long-term experimental carcinogenicity bioassays conducted using rodents. This paper reports a selection of studies conducted in the laboratories of the Cesare Maltoni Cancer Research Center of the European Ramazzini Foundation in which exposure to the chemical agents vinyl acetate monomer, ethyl alcohol and aspartame was started during developmental life and continued into adulthood. The results of these studies provide supporting evidence that lifespan exposure to carcinogenic agents beginning during developmental life produces an overall increase in the carcinogenic effects observed. Moreover, when comparing prenatal and postnatal exposure, the data demonstrate that the development of cancers may appear earlier in life.

Full text, graphs, charts and 14 references

http://onlinelibrary.wiley.com/doi/10.1111/j.1742-7843.2007.00200.x/epdf

Wrigley's
Juicy Fruit
INGREDIENTS

Sugar, Gum Base, **Corn Syrup**, **Dextrose**, Less than 2% of: Natural and **Artificial Flavors, Glycerol, Soy Lecithin, Aspartame, Acesulfame K, Hydroxylated Soy Lecithin, Color (Yellow 5 Lake), BHT** (to Maintain Freshness).

The ingredients listed in **bold** above for Wrigley's Juicy Fruit Gum may be hazardous to your health, especially with regular use.

"This paper reports a selection of studies conducted in the laboratories of the Cesare Maltoni Cancer Research Center of the European Ramazzini Foundation in which exposure to the chemical agents vinyl acetate monomer, ethyl alcohol and aspartame was started during developmental life and continued into adulthood. The results of these studies provide supporting evidence that lifespan exposure to carcinogenic agents beginning during developmental life produces an overall increase in the carcinogenic effects observed. Moreover, when comparing prenatal and postnatal exposure, the data demonstrate that the development of cancers may appear earlier in life."

The Journal Of School Nursing • February 2008

The role of sugar-sweetened beverage consumption in adolescent obesity: a review of the literature

By S. Harrington
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Abstract

Soft drink consumption has increased by 300% in the past 20 years, and 56-85% of children in school consume at least one soft drink daily. The odds ratio of becoming obese among children increases 1.6 times for each additional can or glass of sugar-sweetened drink consumed beyond their usual daily intake of the beverage. Soft drinks currently constitute the leading source of added sugars in the diet and exceed the U.S. Department of Agriculture's recommended total sugar consumption for adolescents. With the increase in adolescent obesity and the concurrent increase in consumption of sugar-sweetened beverages (SSB), the assumption infers a relationship between the two variables. SSB, classified as high-glycemic index (GI) liquids, increase postprandial blood glucose levels and decrease insulin sensitivity. Additionally, high-GI drinks submit to a decreased satiety level and subsequent overeating. Low-GI beverages stimulate a delayed return of hunger, thereby prompting an increased flexibility in amounts and frequencies of servings. Single intervention manipulation, elimination, or marked reduction of SSB consumption may serve to decrease caloric intake, increase satiety levels, decrease tendencies towards insulin resistance, and simplify the process of weight management in this population.

http://www.ncbi.nlm.nih.gov/pubmed/18220450

"The odds ratio of becoming obese among children increases 1.6 times for each additional can or glass of sugar-sweetened drink consumed beyond their usual daily intake of the beverage. Soft drinks currently constitute the leading source of added sugars in the diet and exceed the U.S. Department of Agriculture's recommended total sugar consumption for adolescents."

Corporate Crime

Capitalized Profit • Socialized Debt

The Coca Cola Corporation would be bankrupt if not for the socialization of debt and governmental interference establishing tax benefits and loop-holes for the corporate crimes that keep the 100s of companies like Coca Cola operating for shareholder profits.

~ They get the profits, we, the public, pay their debts ~

“The comet parameters of DNA were increased in the bone marrow cells due to the sweetener-induced DNA strand breaks, as revealed by increased comet-tail extent and percent DNA in the tail. Acesulfame K and saccharin were found to induce greater DNA damage than Aspartame.”

Drug Chemistry And Toxicology • April 2008

Genotoxicity testing of low-calorie sweeteners: aspartame, acesulfame-K, and saccharin

Author information
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Abstract

Low-calorie sweeteners are chemicals that offer the sweetness of sugar without the calories. Consumers are increasingly concerned about the quality and safety of many products present in the diet, in particular, the use of low-calorie sweeteners, flavorings, colorings, preservatives, and dietary supplements. In the present study, we evaluated the mutagenicity of the three low-calorie sweeteners in the Ames/Salmonella/microsome test and their genotoxic potential by comet assay in the bone marrow cells of mice. Swiss albino mice, Mus musculus, were orally administered with different concentrations of aspartame (ASP; 7, 14, 28, and 35 mg/kg body weight), acesulfame-K (ASK; 150, 300, and 600 mg/kg body weight), and saccharin (50, 100, and 200 mg/kg body weight) individually. Concurrently negative and positive control sets were maintained. The animals were sacrificed and the bone marrow cells were processed for comet assay. The standard plate-incorporation assay was carried with the three sweeteners in Salmonella typhimurium TA 97a and TA 100 strains both in the absence and presence of the S9 mix. The comet parameters of DNA were increased in the bone marrow cells due to the sweetener-induced DNA strand breaks, as revealed by increased comet-tail extent and percent DNA in the tail. ASK and saccharin were found to induce greater DNA damage than ASP. However, none could act as a potential mutagen in the Ames/Salmonella /microsome test. These findings are important, since they represent a potential health risk associated with the exposure to these agents.

http://www.ncbi.nlm.nih.gov/pubmed/18850355

“we propose that excessive aspartame ingestion might be involved in the pathogenesis of certain mental disorders (DSM-IV-TR 2000) and also in compromised learning and emotional functioning.”

European Journal Of Clinical Nutrition • April 2008

Direct and indirect cellular effects of aspartame on the brain

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Abstract

The use of the artificial sweetener, aspartame, has long been contemplated and studied by various researchers, and people are concerned about its negative effects. Aspartame is composed of phenylalanine (50%), aspartic acid (40%) and methanol (10%). Phenylalanine plays an important role in neurotransmitter regulation, whereas aspartic acid is also thought to play a role as an excitatory neurotransmitter in the central nervous system. Glutamate, asparagines and glutamine are formed from their precursor, aspartic acid. Methanol, which forms 10% of the broken down product, is converted in the body to formate, which can either be excreted or can give rise to formaldehyde, diketopiperazine (a carcinogen) and a number of other highly toxic derivatives. Previously, it has been reported that consumption of aspartame could cause neurological and behavioural disturbances in sensitive individuals. Headaches, insomnia and seizures are also some of the neurological effects that have been encountered, and these may be accredited to changes in regional brain concentrations of catecholamines, which include norepinephrine, epinephrine and dopamine. The aim of this study was to discuss the direct and indirect cellular effects of aspartame on the brain, and we propose that excessive aspartame ingestion might be involved in the pathogenesis of certain mental disorders (DSM-IV-TR 2000) and also in compromised learning and emotional functioning.

http://www.ncbi.nlm.nih.gov/pubmed/17684524

Cancer Epidemiology, Biomarkers & Prevention • May 2008

Aspartame and Incidence of Brain Malignancies

By Devra Lee Davis, Leanne Ganter and Jonathan Weinkle

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To the Editors:

Lim et al. (1) found no relationship between the "Consumption of aspartame-containing beverages and incidence of hematopoietic and brain malignancies" by surveying retired individuals about their consumption of diet sodas and determining whether they had an increase in brain tumors 5 years later. In fact, this study design was not able to test the hypothesized relationship because it did not assess all exposures to aspartame and because it only evaluated short-term exposures in older adults.

Lim et al. estimated average daily aspartame consumption values based solely on the consumption of beverages containing aspartame. Lim et al. states that 70% of consumed aspartame is obtained through diet beverages, citing the American Dietetic Association as the source of this information. The American Dietetic Association, in turn, cites Equal.com, a large supplier of aspartame-containing products, as the authority for their statement; when contacted for corroborating information, Equal.com could not provide this corroboration. According to the Aspartame Information Center, more dry products contain aspartame than do drink products. The Lim et al. study assumed that the average daily intake of aspartame was equivalent to approximately one 12-oz. can of diet soda and did not include products, such as table sugars, candies, yogurt, nutrition bars, and even chewing gum (2).

Table 1 , from the European Ramazzini Foundation, estimates the average daily amount of aspartame consumed from some of the most commonly used products out of the more than 6,000 in which it is present (3). If a woman ate these foods and weighed 60 kg (□132 lb), she would consume an aspartame daily dose of 15.1 mg/kg of body weight; a child weighing 30 kg (□66 lb) with a similar daily intake would have an aspartame daily dose twice as high—30.3 mg/kg of body weight (4). This estimated average daily level is well above the dose that was examined in Lim et al.'s study. See Table 1, Average daily intake of aspartame.

Table 1. Average daily intake of aspartame

Substance	Quantity per day	Concentration of aspartame consumed (mg)
Diet soda (200 mg/can)	2 cans	400
Yogurt (125 mg/yogurt)	2 yogurts	250
Diet custard/pudding (75 mg/mouse)	1 serving	75
Coffee with sweetener (40/mg packet)	4 cups	160
Candy/chewing gum (2.5/candy)	10 candies	25
Totals		910

For those who are dieting, daily consumption of aspartame can be considerably higher. For instance, the South Beach Diet attempts to eliminate sugar intake by endorsing the extensive use of sugar substitutes, such as aspartame (5). Despite its popularity in various diet programs, research does not indicate that aspartame promotes weight loss, and some report that it functions as an appetite stimulant (6).

The Lim et al. study included members of the AARP of ages 50 to 71 years. Aspartame was not approved by the Food and Drug Administration as a food additive until July 1981. The population of the Lim et al. study grew during a period when aspartame use was considerably less than it is now, and none would have consumed aspartame as children (7). Aspartame is now used in countless consumer products, some we may not even be aware of, and is consumed by about 200 million Americans every day. In America today, more than 10 million children regularly consume sodas and other foods containing aspartame. This study cannot evaluate the possibility that infants and children whose exposures can begin in utero and young persons who can drink several sodas a day will incur a greater risk of brain and other cancers when they reach the older ages.

The greater vulnerability of the young to carcinogens has consistently been shown experimentally for aspartame and a number of other compounds (8). Some experimental studies have not found a connection to brain malignancies and aspartame consumption in rats (9). But these studies ended after 2 years. In a rodent, 2 years corresponds to about 60 years in humans, and more and more of the population are living into their 80s. The Ramazzini Foundation relies on a lifespan protocol in which rodents live their entire life span and are examined for signs of tumors or other damage upon natural death or by the end of the third year. Soffritti's September 2007 publication (10) supported the 2005 (11) experiment that found an obvious association between aspartame and multiple types of cancers. Animals exposed prenatally to aspartame in this latest work have double the risk of multiple tumors compared with those exposed postnatally.

In light of these experimental findings, in evaluating public health effects of widely used materials, it is important to consider information on lifetime exposures, especially those that may begin prenatally. The possible effect of full lifetime and prenatal exposures on humans of aspartame cannot be evaluated in the Lim et al. study population, which only examined persons exposed as adults to aspartame.

More than half of all Americans are estimated to use aspartame every day, including many pregnant women and children. The negative findings in the Lim et al. study do not prove that there are no long-term effects of aspartame. The older study population analyzed and the limited ingestion of aspartame evaluated cannot reflect the long-term public-health effects of aspartame that may begin prenatally and run throughout a lifetime. In light of the limited nature of the study by Lim et al. and recent reports of Soffritti finding that aspartame significantly increases the risks of tumors in rodents, with a doubled risk in those in which exposures begin prenatally, the Food and Drug Administration should review its approval of aspartame.

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Full text and references: http://cebp.aacrjournals.org/content/17/5/1295.full.pdf

> “Regular use of artificial sweeteners for 10 years or more was positively associated with urinary tract tumors.”

Preventive Medicine • July 2008

Artificial sweetener consumption and urinary tract tumors in Cordoba, Argentina

Author information

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Abstract

OBJECTIVE

To determine the role of the habitual use of the most common artificial sweeteners (AS) in the development of urinary tract tumors (UTT) in Argentina.

METHODS

Case-control study of 197 patients with histologically confirmed UTT of transitional varieties, and 397 controls with acute, non-neoplastic, and non-urinary tract diseases, admitted to the same hospitals in Córdoba (Argentina) between 1999 and 2006. All subjects were interviewed about their use of AS and their exposure to other known or suspected risk factors for UTT.

RESULTS

Fifty-one UTT patients (26%) and 87 controls (22%) used AS. The risk of UTT was significantly increased in long-term (> or =10 years) AS users compared with none-AS users. The OR (95% CI) for long-term consumers was 2.18 (1.22-3.89) and for short-term users was 1.10 (0.61-2.00) after adjustment for age, gender, BMI, social status. and years of tobacco use.

CONCLUSION

Regular use of AS for 10 years or more was positively associated with UTT.

http://www.ncbi.nlm.nih.gov/pubmed/18495230

Archives In Internal Medicine • July 2008

Sugar-sweetened beverages and incidence of type 2 diabetes mellitus in African American women

Link: http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2708080/
By Palmer JR1, Boggs DA, Krishnan S, Hu FB, Singer M, Rosenberg L.

Abstract

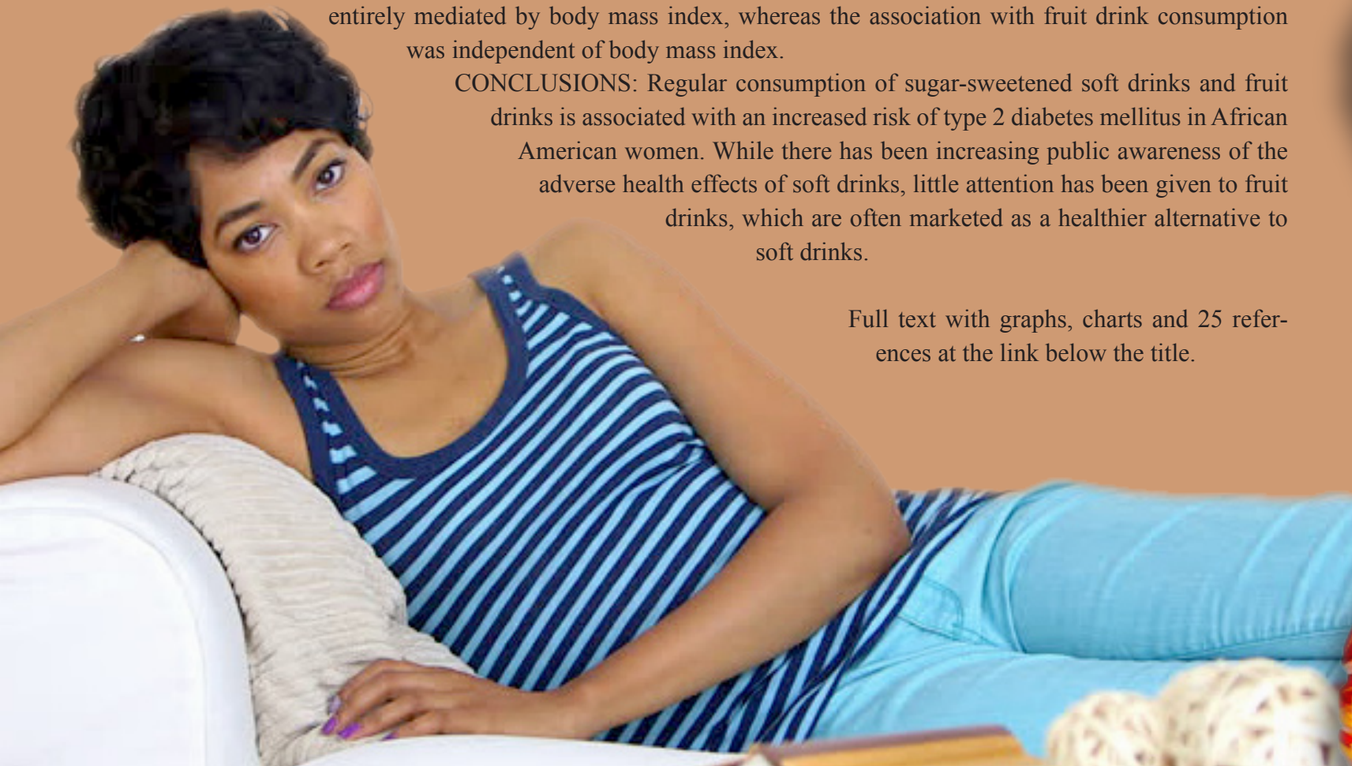
BACKGROUND: Type 2 diabetes mellitus is an increasingly serious health problem among African American women. Consumption of sugar-sweetened drinks was associated with an increased risk of diabetes in 2 studies but not in a third; however, to our knowledge, no data are available on African Americans regarding this issue. Our objective was to examine the association between consumption of sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes mellitus in African American women.

METHODS: A prospective follow-up study of 59,000 African American women has been in progress since 1995. Participants reported on food and beverage consumption in 1995 and 2001. Biennial follow-up questionnaires ascertained new diagnoses of type 2 diabetes. The present analyses included 43,960 women who gave complete dietary and weight information and were free from diabetes at baseline. We identified 2713 incident cases of type 2 diabetes mellitus during 338,884 person-years of follow-up. The main outcome measure was the incidence of type 2 diabetes mellitus.

RESULTS: The incidence of type 2 diabetes mellitus was higher with higher intake of both sugar-sweetened soft drinks and fruit drinks. After adjustment for confounding variables including other dietary factors, the incidence rate ratio for 2 or more soft drinks per day was 1.24 (95% confidence interval, 1.06-1.45). For fruit drinks, the comparable incidence rate ratio was 1.31 (95% confidence interval, 1.13-1.52). The association of diabetes with soft drink consumption was almost entirely mediated by body mass index, whereas the association with fruit drink consumption was independent of body mass index.

CONCLUSIONS: Regular consumption of sugar-sweetened soft drinks and fruit drinks is associated with an increased risk of type 2 diabetes mellitus in African American women. While there has been increasing public awareness of the adverse health effects of soft drinks, little attention has been given to fruit drinks, which are often marketed as a healthier alternative to soft drinks.

Full text with graphs, charts and 25 references at the link below the title.



“Regular consumption of sugar-sweetened soft drinks and fruit drinks is associated with an increased risk of type 2 diabetes mellitus in African American women.”

Food And Chemical Toxicology • September 2008

Reducing added sugar intake in Norway by replacing sugar sweetened beverages with beverages containing intense sweeteners - a risk benefit assessment

Author information

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Abstract

A risk benefit assessment in Norway on the intake of added sugar, intense sweeteners and benzoic acid from beverages, and the influence of changing from sugar sweetened to diet beverages was performed. National dietary surveys were used in the exposure assessment, and the content of added sugar and food additives were calculated based on actual contents used in beverages and sales volumes provided by the manufactures. The daily intake of sugar, intense sweeteners and benzoic acid were estimated for children (1- to 13-years-old) and adults according to the current intake level and a substitution scenario where it was assumed that all consumed beverages contained intense sweeteners. The change from sugar sweetened to diet beverages reduced the total intake of added sugar for all age groups but especially for adolescent. This change did not result in intake of intense sweeteners from beverages above the respective ADIs. However, the intake of acesulfame K approached ADI for small children and the total intake of benzoic acid was increased to above ADI for most age groups. The highest intake of benzoic acid was observed for 1- to 2-year-old children, and benzoic acid intake in Norwegian children is therefore considered to be of special concern.

http://www.ncbi.nlm.nih.gov/pubmed/18639604

“... the intake of acesulfame K approached the average daily intake for small children and the total intake of benzoic acid was increased to above the average daily intake for most age groups. The highest intake of benzoic acid [*see chapter on benzoic acid] was observed for 1- to 2-year-old children, and benzoic acid intake in Norwegian children is therefore considered to be of special concern.”

AAOHN Journal • June 2008

The potential toxicity of artificial sweeteners

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Abstract

Since their discovery, the safety of artificial sweeteners has been controversial. Artificial sweeteners provide the sweetness of sugar without the calories. As public health attention has turned to reversing the obesity epidemic in the United States, more individuals of all ages are choosing to use these products. These choices may be beneficial for those who cannot tolerate sugar in their diets (e.g., diabetics). However, scientists disagree about the relationships between sweeteners and lymphomas, leukemias, cancers of the bladder and brain, chronic fatigue syndrome, Parkinson's disease, Alzheimer's disease, multiple sclerosis, autism, and systemic lupus. Recently these substances have received increased attention due to their effects on glucose regulation. Occupational health nurses need accurate and timely information to counsel individuals regarding the use of these substances. This article provides an overview of types of artificial sweeteners, sweetener history, chemical structure, biological fate, physiological effects, published animal and human studies, and current standards and regulations.

http://www.ncbi.nlm.nih.gov/pubmed/18604921

“... scientists disagree about the relationships between sweeteners and lymphomas, leukemias, cancers of the bladder and brain, chronic fatigue syndrome, Parkinson's disease, Alzheimer's disease, multiple sclerosis, autism, and systemic lupus. Recently these substances have received increased attention due to their effects on glucose regulation. Occupational health nurses need accurate and timely information to counsel individuals regarding the use of these substances. This article provides an overview of types of artificial sweeteners, sweetener history, chemical structure, biological fate, physiological effects, published animal and human studies, and current standards and regulations.”

Drug And Chemical Toxicology • December 2008

Genotoxicity testing of low-calorie sweeteners: aspartame, acesulfame-K, and saccharin

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Abstract

Low-calorie sweeteners are chemicals that offer the sweetness of sugar without the calories. Consumers are increasingly concerned about the quality and safety of many products present in the diet, in particular, the use of low-calorie sweeteners, flavorings, colorings, preservatives, and dietary supplements. In the present study, we evaluated the mutagenicity of the three low-calorie sweeteners in the Ames/Salmonella/microsome test and their genotoxic potential by comet assay in the bone marrow cells of mice. Swiss albino mice, Mus musculus, were orally administered with different concentrations of aspartame (ASP; 7, 14, 28, and 35 mg/kg body weight), acesulfame-K (ASK; 150, 300, and 600 mg/kg body weight), and saccharin (50, 100, and 200 mg/kg body weight) individually. Concurrently negative and positive control sets were maintained. The animals were sacrificed and the bone marrow cells were processed for comet assay. The standard plate-incorporation assay was carried with the three sweeteners in Salmonella typhimurium TA 97a and TA 100 strains both in the absence and presence of the S9 mix. The comet parameters of DNA were increased in the bone marrow cells due to the sweetener-induced DNA strand breaks, as revealed by increased comet-tail extent and percent DNA in the tail. ASK and saccharin were found to induce greater DNA damage than ASP. However, none could act as a potential mutagen in the Ames/Salmonella /microsome test. These findings are important, since they represent a potential health risk associated with the exposure to these agents.

http://www.ncbi.nlm.nih.gov/pubmed/18850355

“acesulfame-K and saccharin were found to induce greater DNA damage than aspartame ... These findings are important, since they represent a potential health risk associated with the exposure to these agents.”

> “Evidence indicates that a 12-wk administration of Splenda exerted numerous adverse effects, including (1) reduction in beneficial fecal microflora, (2) increased fecal pH, and (3) enhanced expression levels of P-gp, CYP3A4, and CYP2D1, which are known to limit the bioavailability of orally administered drugs.”

The Journal Of Toxicology And Environmental Health
Part A • December 2008

Splenda alters gut microflora and increases intestinal p-glycoprotein and cytochrome p-450 in male rats

Author information
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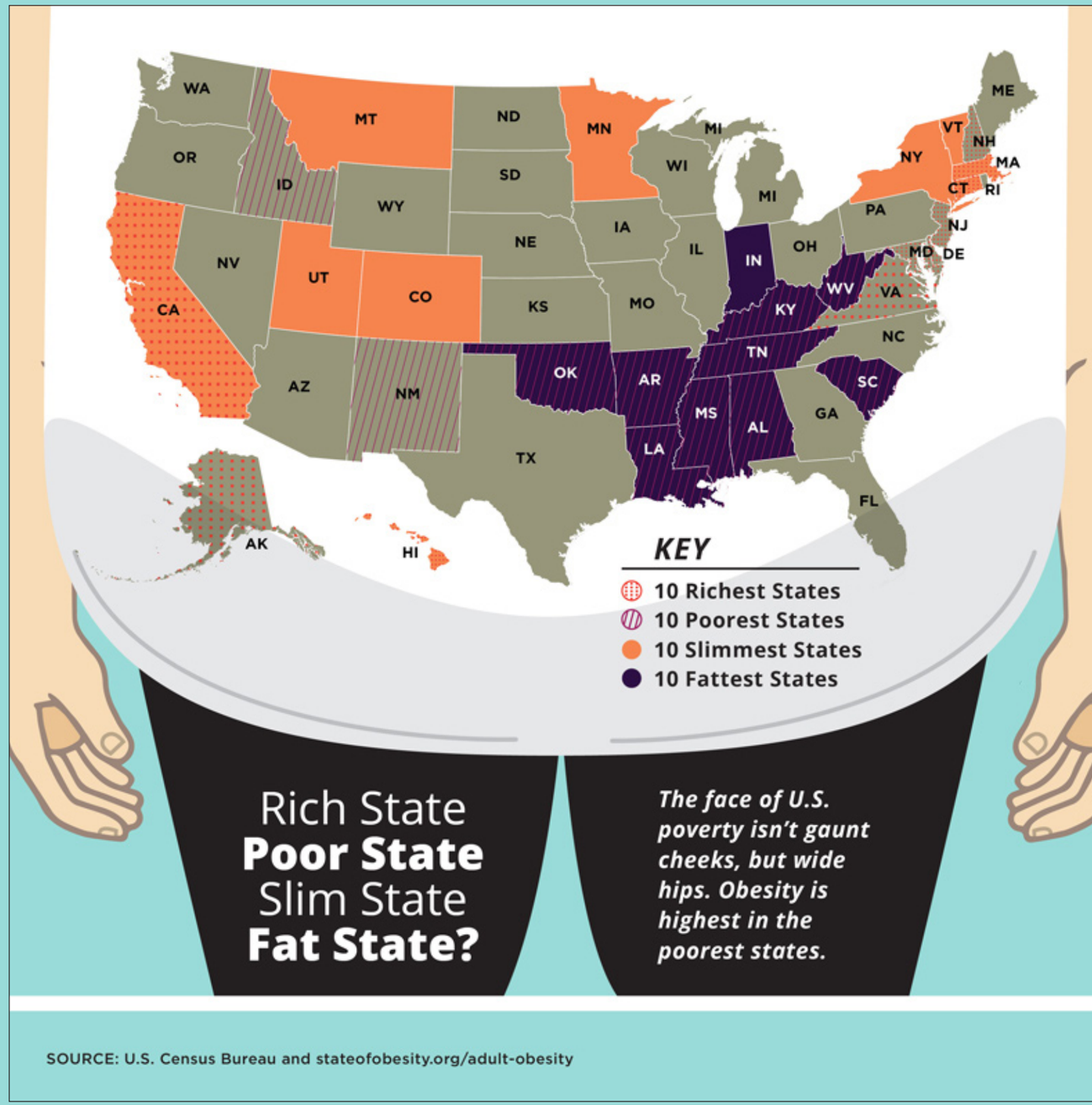
Abstract

Splenda is comprised of the high-potency artificial sweetener sucralose (1.1%) and the fillers maltodextrin and glucose. Splenda was administered by oral gavage at 100, 300, 500, or 1000 mg/kg to male Sprague-Dawley rats for 12-wk, during which fecal samples were collected weekly for bacterial analysis and measurement of fecal pH. After 12-wk, half of the animals from each treatment group were sacrificed to determine the intestinal expression of the membrane efflux transporter P-glycoprotein (P-gp) and the cytochrome P-450 (CYP) metabolism system by Western blot. The remaining animals were allowed to recover for an additional 12-wk, and further assessments of fecal microflora, fecal pH, and expression of P-gp and CYP were determined. At the end of the 12-wk treatment period, the numbers of total anaerobes, bifidobacteria, lactobacilli, Bacteroides, clostridia, and total aerobic bacteria were significantly decreased; however, there was no significant treatment effect on enterobacteria. Splenda also increased fecal pH and enhanced the expression of P-gp by 2.43-fold, CYP3A4 by 2.51-fold, and CYP2D1 by 3.49-fold. Following the 12-wk recovery period, only the total anaerobes and bifidobacteria remained significantly depressed, whereas pH values, P-gp, and CYP3A4 and CYP2D1 remained elevated. These changes occurred at Splenda dosages that contained sucralose at 1.1-11 mg/kg (the US FDA Acceptable Daily Intake for sucralose is 5 mg/kg). Evidence indicates that a 12-wk administration of Splenda exerted numerous adverse effects, including (1) reduction in beneficial fecal microflora, (2) increased fecal pH, and (3) enhanced expression levels of P-gp, CYP3A4, and CYP2D1, which are known to limit the bioavailability of orally administered drugs.

http://www.ncbi.nlm.nih.gov/pubmed/18800291

THE STATE OF OBESITY VERSUS POVERTY IN THE UNITED STATES OF AMERICA



SOURCE: U.S. Census Bureau and stateofobesity.org/adult-obesity

In Vivo • January 2007

The Effect of Aspartame Administration on Oncogene and Suppressor Gene Expressions

Author information

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5. National Medical Officer Service, County Institute of Békés Békéscsaba, Hungary

Abstract

BACKGROUND:
Aspartame (L-phenylalanine N-L-alpha-aspartyl-1-methyl ester) is an artificial sweetener with widespread applications. Previously published results have shown that among rats receiving aspartame a significant increase of lymphoreticular neoplasms, brain tumours and transitional cell tumours occurred. The aim of our short-term experiment was to investigate the biological effect of aspartame consumption by determining the expressions of key oncogenes and a tumour suppressor gene.

MATERIALS AND METHODS:
After one week per os administration of various doses of aspartame to CBA/CA female mice, p53, c-myc, Ha-ras gene expression alterations were determined in individual organs.

RESULTS:
The results showed an increase in gene expressions concerning all the investigated genes especially in organs with a high proliferation rate: lymphoreticular organs, bone-marrow and kidney.

CONCLUSION:
Aspartame has a biological effect
even at the recommended daily maximum dose.

Full Text PDF

http://iv.iiarjournals.org/content/21/1/89.long

“CONCLUSION:
Aspartame has a biological effect
even at the recommended daily maximum dose.”

Trident Layers Gum
INGREDIENTS

gum base, **maltitol, maltitol syrup, sorbitol, natural and artificial flavoring, mannitol,** less than 2% of: **acesulfame potassium, aspartame, blue 2 lake, gelatin, partially hydrogenated coconut oil, red 40, red 40 lake, soy lecithin, sucralose and yellow 5.**

Ingredients in **bold** are ingredients that have been proven to cause cancer, neurological and behavioral disorders and other debilitating diseases and disorders of the human species.

The Journal Of The American Dietetic Association • June 2007

Regular sugar-sweetened beverage consumption between meals increases risk of overweight among preschool-aged children

Author information

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Abstract

OBJECTIVE

To examine the relationship between consumption of sugar-sweetened beverages (eg, nondiet carbonated drinks and fruit drinks) and the prevalence of overweight among preschool-aged children living in Canada.

DESIGN

Data come from the Longitudinal Study of Child Development in Québec (1998-2002).

SUBJECTS/SETTING

A representative sample (n=2,103) of children born in 1998 in Québec, Canada. A total of 1,944 children (still representative of the same-age children in this population) remaining at 4 to 5 years in 2002 participated in the nutrition study.

STATISTICAL ANALYSES PERFORMED

Data were collected via 24-hour dietary recall interview. Frequency of sugar-sweetened beverage consumption between meals at age 2.5, 3.5, and 4.5 years was recorded and children's height and weight were measured. Multivariate regression analysis was done with Statistical Analysis System software. Weighted data were adjusted for within-child variability and significance level was set at 5%.

RESULTS

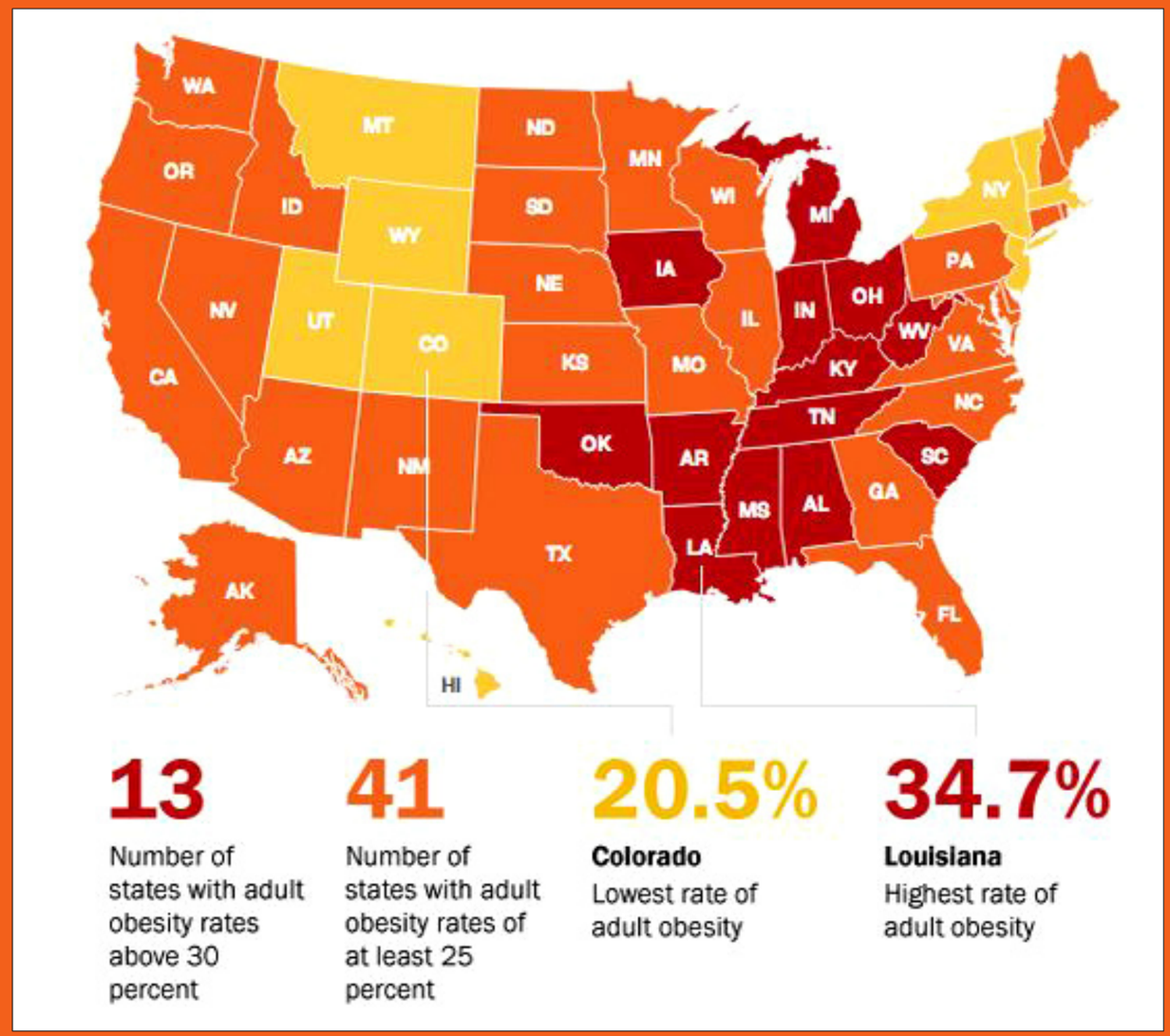
Overall, 6.9% of children who were nonconsumers of sugar-sweetened beverages between meals between the ages of 2.5 to 4.5 years were overweight at 4.5 years, compared to 15.4% of regular consumers (four to six times or more per week) at ages 2.5 years, 3.5 years, and 4.5 years. According to multivariate analysis, sugar-sweetened beverage consumption between meals more than doubles the odds of being overweight when other important factors are considered in multivariate analysis. Children from families with insufficient income who consume sugar-sweetened beverages regularly between the ages of 2.5 and 4.5 years are more than three times more likely to be overweight at age 4.5 years compared to non-consuming children from sufficient income households.

CONCLUSIONS

Regular sugar-sweetened beverage consumption between meals may put some young children at a greater risk for overweight. Parents should limit the quantity of sweetened beverages consumed during preschool years because it may increase propensity to gain weight.

http://www.ncbi.nlm.nih.gov/pubmed/17524711

"Children from families with insufficient income who consume sugar-sweetened beverages regularly between the ages of 2.5 and 4.5 years are more than three times more likely to be overweight at age 4.5 years compared to non-consuming children from sufficient income households."



BEST & WORST
drinks for thirsty kids

BEST

Water

You can't go wrong with plain water. It hydrates, helps regulate body temperature, and helps prevent constipation and urinary tract infections — all without adding calories or sugar to the diet. And it can be a good source of fluoride, which is important for healthy teeth.

RECOMMENDED
2-3 cups
(16-24oz)/day

Milk

Cow's milk is an excellent source of protein, calcium, vitamin D, and other nutrients. Offer your child 2 to 3 cups of milk a day starting at age 1. (Kids who drink more than 3 cups a day may not have room for other foods they need.) Whole milk is best for 1-year-olds; low-fat is fine for age 2 and older.

Juice

Juice is an acceptable way to get one serving of fruit or veggies each day. But it lacks fiber and is less nutritious than whole fruit. Plus, kids may fill up on juice instead of healthier foods. Choose 100 percent juice and limit it to ¾ cup (6 ounces) per day. (Children 7 and older can have a bit more, but it's best to keep juice to a minimum at any age.)

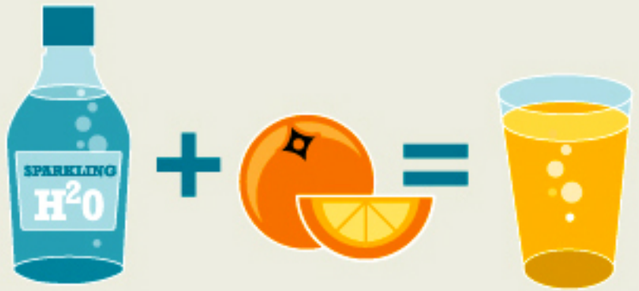
LIMIT
3/4 cup
(6oz)/day

WORST

Soda

Soda has no nutritional value. Most brands contain artificial color or flavor, as well as sugar or artificial sweeteners. A better alternative is to make your own "juice soda" with sparkling water and 100 percent fruit juice.

TRY THIS INSTEAD:



Sports & energy drinks

These have no nutritional value. They contain empty calories from added sugar and may contain artificial color or flavor. Some are made with herbs that may not be safe for children. Sports drinks have extra sodium, and energy drinks tend to have large amounts of caffeine.

Environmental Health Perspectives • September 2007

Life-span exposure to low doses of aspartame beginning during prenatal life increases cancer effects in rats

Author information

Soffritti M1, Belpoggi F, Tibaldi E, Esposti DD, Lauriola M.

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Abstract

BACKGROUND

In a previous study conducted at the Cesare Maltoni Cancer Research Center of the European Ramazzini Foundation (CMCRC/ERF), we demonstrated for the first time that aspartame (APM) is a multipotent carcinogenic agent when various doses are administered with feed to Sprague-Dawley rats from 8 weeks of age throughout the life span.

OBJECTIVE

The aim of this second study is to better quantify the carcinogenic risk of APM, beginning treatment during fetal life.

METHODS

We studied groups of 70-95 male and female Sprague-Dawley rats administered APM (2,000, 400, or 0 ppm) with feed from the 12th day of fetal life until natural death.

RESULTS

Our results show a) a significant dose-related increase of malignant tumor-bearing animals in males ($p < 0.01$), particularly in the group treated with 2,000 ppm APM ($p < 0.01$); b) a significant increase in incidence of lymphomas/leukemias in males treated with 2,000 ppm ($p < 0.05$) and a significant dose-related increase in incidence of lymphomas/leukemias in females ($p < 0.01$), particularly in the 2,000-ppm group ($p < 0.01$); and c) a significant dose-related increase in incidence of mammary cancer in females ($p < 0.05$), particularly in the 2,000-ppm group ($p < 0.05$).

CONCLUSIONS

The results of this carcinogenicity bioassay confirm and reinforce the first experimental demonstration of APM's multipotential carcinogenicity at a dose level close to the acceptable daily intake for humans. Furthermore, the study demonstrates that when life-span exposure to APM begins during fetal life, its carcinogenic effects are increased.

Full text, graphs, charts and 32 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1964906/

“The results of this mega-experiment indicate that Aspartame is a multipotential carcinogenic agent, even at a daily dose of 20 mg/kg body weight, much less than the current acceptable daily intake. On the basis of these results, a reevaluation of the present guidelines on the use and consumption of Aspartame is urgent and cannot be delayed.”

Environmental Health Perspectives • March 2006

First experimental demonstration of the multipotential carcinogenic effects of aspartame administered in the feed to Sprague-Dawley rats

Author information
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Abstract

The Cesare Maltoni Cancer Research Center of the European Ramazzini Foundation has conducted a long-term bioassay on aspartame (APM), a widely used artificial sweetener. APM was administered with feed to 8-week-old Sprague-Dawley rats (100-150/sex/group), at concentrations of 100,000, 50,000, 10,000, 2,000, 400, 80, or 0 ppm. The treatment lasted until natural death, at which time all deceased animals underwent complete necropsy. Histopathologic evaluation of all pathologic lesions and of all organs and tissues collected was routinely performed on each animal of all experimental groups. The results of the study show for the first time that APM, in our experimental conditions, causes a) an increased incidence of malignant-tumor-bearing animals with a positive significant trend in males ($p < or = 0.05$) and in females ($p < or = 0.01$), in particular those females treated at 50,000 ppm ($p < or = 0.01$); b) an increase in lymphomas and leukemias with a positive significant trend in both males ($p < or = 0.05$) and females ($p < or = 0.01$), in particular in females treated at doses of 100,000 ($p < or = 0.01$), 50,000 ($p < or = 0.01$), 10,000 ($p < or = 0.05$), 2,000 ($p < or = 0.05$), or 400 ppm ($p < or = 0.01$); c) a statistically significant increased incidence, with a positive significant trend ($p < or = 0.01$), of transitional cell carcinomas of the renal pelvis and ureter and their precursors (dysplasias) in females treated at 100,000 ($p < or = 0.01$), 50,000 ($p < or = 0.01$), 10,000 ($p < or = 0.01$), 2,000 ($p < or = 0.05$), or 400 ppm ($p < or = 0.05$); and d) an increased incidence of malignant schwannomas of peripheral nerves with a positive trend ($p < or = 0.05$) in males. The results of this mega-experiment indicate that APM is a multipotential carcinogenic agent, even at a daily dose of 20 mg/kg body weight, much less than the current acceptable daily intake. On the basis of these results, a reevaluation of the present guidelines on the use and consumption of APM is urgent and cannot be delayed.

Full text, graphs, charts and 47 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1392232/

PHARMACEUTICALS & ASPARTAME

Over 500 pharmaceutical drugs have been found to contain varying amounts of aspartame, a carcinogenic agent with only one benefit; profound sweetness. Yet it also causes cancer and tumors. What possible purpose could a pill manufacturer have for putting sweeteners in pills? Especially a sweetener that’s known to cause cancer. Do pills need to be sweet?

this is a highly recommend report with 45 references and useful for understanding drug synergy

Toxicology Science • March 2006

Synergistic interactions between commonly used food additives in a developmental neurotoxicity test

Author information
By K. Lau, WG. McLean, DP. Williams and CV. Howard

Abstract

Exposure to non-nutritional food additives during the critical development window has been implicated in the induction and severity of behavioral disorders such as attention deficit hyperactivity disorder (ADHD). Although the use of single food additives at their regulated concentrations is believed to be relatively safe in terms of neuronal development, their combined effects remain unclear. We therefore examined the neurotoxic effects of four common food additives in combinations of two (Brilliant Blue and L-glutamic acid, Quinoline Yellow and aspartame) to assess potential interactions. Mouse NB2a neuroblastoma cells were induced to differentiate and grow neurites in the presence of additives. After 24 h, cells were fixed and stained and neurite length measured by light microscopy with computerized image analysis. Neurotoxicity was measured as an inhibition of neurite outgrowth. Two independent models were used to analyze combination effects: effect additivity and dose additivity. Significant synergy was observed between combinations of Brilliant Blue with L-glutamic acid, and Quinoline Yellow with aspartame, in both models. Involvement of N-methyl-D-aspartate (NMDA) receptors in food additive-induced neurite inhibition was assessed with a NMDA antagonist, CNS-1102. L-glutamic acid- and aspartame-induced neurotoxicity was reduced in the presence of CNS-1102; however, the antagonist did not prevent food color-induced neurotoxicity. Theoretical exposure to additives was calculated based on analysis of content in foodstuff, and estimated percentage absorption from the gut. Inhibition of neurite outgrowth was found at concentrations of additives theoretically achievable in plasma by ingestion of a typical snack and drink. In addition, Trypan Blue dye exclusion was used to evaluate the cellular toxicity of food additives on cell viability of NB2a cells; both combinations had a straightforward additive effect on cytotoxicity. These data have implications for the cellular effects of common chemical entities ingested individually and in combination.

Full text, graphs, charts and 45 references

http://toxsci.oxfordjournals.org/content/90/1/178.full.pdf+html

SYNERGY

Synergy is the interaction or cooperation of two or more organizations, substances, or other agents to produce a combined effect greater than the sum of their separate effects. Until 2015-2016 the concept of synergy was overlooked by the medical research community. No one cared about synergy because it was far too complicated to investigate. We do know that when mercury and aluminum meet up at a cellular level that their negative neurological effects are substantially increased by as much as 100 times or more as opposed to either heavy metal alone. Synergy is of critical importance to the subject of food additives, preservatives and colors—food drugs—because a conventional diet includes a great number of these drugs every single day. Let's hope we get far more reports on food drug synergy.

Headache • March 2006

Migraine triggered by sucralose—a case report

Author information

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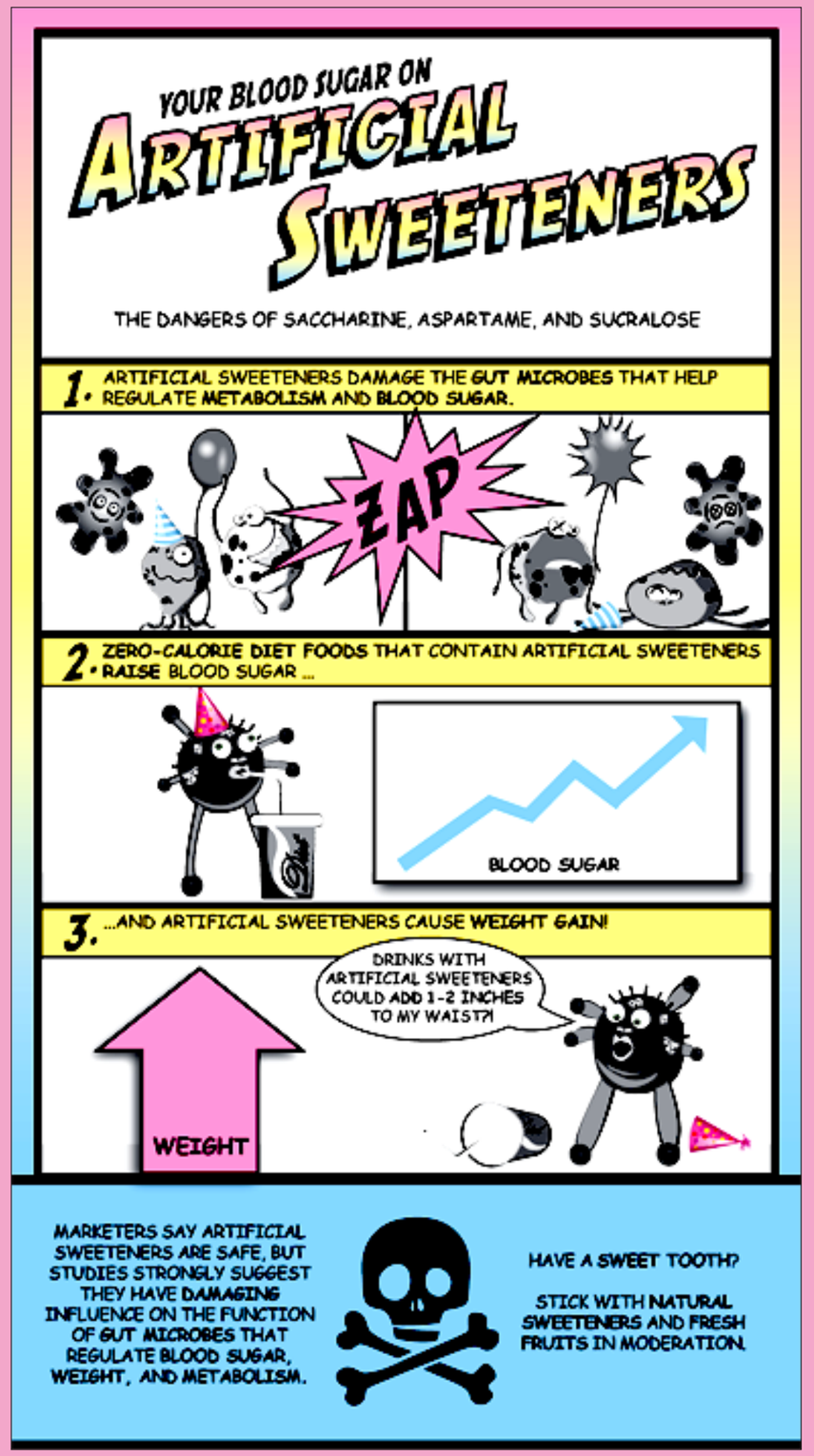
Bronx, NY 10461, USA

Abstract

Sucralose is the active compound of the most commonly sold sweetener in the United States. Different than aspartame, sucralose is not considered to be a migraine trigger. Herein we report a patient with attacks of migraine consistently triggered by sucralose. She also suffers from menstrually related migraine that had been well-controlled for several months since she switched her contraceptive from fixed estrogen to triphasic contraceptive pills. Some attacks triggered by sucralose were preceded by aura, and she had never experienced migraine with aura before. Withdrawal of the compound was associated with complete resolution of the attacks. Single-blind exposure (vs. sugar) triggered the attacks, after an attack-free period.

http://www.ncbi.nlm.nih.gov/pubmed/16618274



“Consumption of sugar-sweetened beverages (SSBs), particularly carbonated soft drinks, may be a key contributor to the epidemic of overweight and obesity, by virtue of these beverages’ high added sugar content, low satiety, and incomplete compensation for total energy. The weight of epidemiologic and experimental evidence indicates that a greater consumption of Sugar-Sweetened Beverages is associated with weight gain and obesity.”

American Journal Of Clinical Nutrition • August 2006

Intake of sugar-sweetened beverages and weight gain: a systematic review

Author information

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Harvard School of Public Health, Boston, MA, USA

Abstract

Consumption of sugar-sweetened beverages (SSBs), particularly carbonated soft drinks, may be a key contributor to the epidemic of overweight and obesity, by virtue of these beverages’ high added sugar content, low satiety, and incomplete compensation for total energy. Whether an association exists between SSB intake and weight gain is unclear. We searched English-language MEDLINE publications from 1966 through May 2005 for cross-sectional, prospective cohort, and experimental studies of the relation between SSBs and the risk of weight gain (ie, overweight, obesity, or both). Thirty publications (15 cross-sectional, 10 prospective, and 5 experimental) were selected on the basis of relevance and quality of design and methods. Findings from large cross-sectional studies, in conjunction with those from well-powered prospective cohort studies with long periods of follow-up, show a positive association between greater intakes of SSBs and weight gain and obesity in both children and adults. Findings from short-term feeding trials in adults also support an induction of positive energy balance and weight gain by intake of sugar-sweetened sodas, but these trials are few. A school-based intervention found significantly less soft-drink consumption and prevalence of obese and overweight children in the intervention group than in control subjects after 12 mo, and a recent 25-week randomized controlled trial in adolescents found further evidence linking SSB intake to body weight. The weight of epidemiologic and experimental evidence indicates that a greater consumption of SSBs is associated with weight gain and obesity. Although more research is needed, sufficient evidence exists for public health strategies to discourage consumption of sugary drinks as part of a healthy lifestyle.

Full text, graphs, charts and 113 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC3210834/

Environmental Health Perspective • September 2006

Testing Needed for Acesulfame Potassium, an Artificial Sweetener

by Myra L. Karstadt
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Drexel University School of Public Health
Philadelphia, Pennsylvania

The author declares that she has no competing financial interests
See the reply "Acesulfame Potassium: Soffritti Responds" on page A516
See the article "First Experimental Demonstration of the Multipotential Carcinogenic Effects of Aspartame Administered in the Feed to Sprague-Dawley Rats" in volume 114 on page 379.

Abstract

In their article "First Experimental Demonstration of the Multipotential Carcinogenic Effects of Aspartame Administered in the Feed of Sprague-Dawley Rats," Soffritti et al. (2006) present interesting data on the carcinogenic effects of long-term exposure to aspartame, an artificial sweetener, in experimental animals (rats). Recently, aspartame was supplanted as the leading artificial sweetener by sucralose, marketed in the United States under the trade name Splenda (McNeil Nutritionals, LLC, Ft. Washington, PA). As of 2005, Splenda was reported to have > 50% of the market for artificial sweeteners, while aspartame [Equal (Merisant Company, Chicago, IL); NutraSweet (NutraSweet Property Holdings Inc., Chicago, IL)] had < 20% (Associated Press 2005). Splenda is typically used in sweetener blends, most frequently with acesulfame potassium (CAS RN 55589-62-3) (Sunett; marketed in the United States by Nutrinova, Somerset, NJ).

The Food and Drug Administration's (FDA) multiple approvals of food additive petitions (FAPs) for acesulfame began in 1988 (FDA 1988), and culminated in 1998 with approval of the use of acesulfame in soft drinks (FDA 1998), historically the largest single use of artificial sweeteners. All of the FDA's approvals of FAPs for acesulfame were grounded on the conclusion that safety studies, including long-term animal tests of acesulfame carried out for Hoechst, the manufacturer of the chemical, in the Netherlands in the 1970s, were adequate and the test results indicated safety.

The 1970s tests of acesulfame—two tests carried out in rats and one in mice—are inadequate to establish lack of potential carcinogenicity. Here are a few reasons why the tests are inadequate [Center for Science in the Public Interest (CSPI) 1996]:

• Subchronic tests were not conducted for the rats and mice used in the tests on which the FAPs rested
• It is likely the minimum toxic dose/maximum tolerated dose (MTD) was not achieved in the rat and mouse studies
• Randomization of test groups was not carried out properly
• Mice were held on test for only 80 weeks, rather than the 104 weeks characteristic of National Toxicology Program (NTP) bioassays
• Animal husbandry and monitoring of animals on test were evidently poor, as indicated by high disease rates in the animals and extensive autolysis of tissues.

Even with the flaws in design and execution of the Hoechst tests, results indicated an association between treatment with acesulfame and carcinogenesis (CSPI 1996). The question remains whether these studies are sufficiently definitive or rigorous in light of the potential for widespread, [sic] high exposure to acesulfame K in all group [sic] in the population.

In 1996, the CSPI nominated acesulfame for testing in the NTP bioassay program (CSPI 1996), and provided the NTP with detailed information on the Hoechst tests and their flaws. Although an individual familiar with test design and implementation could have concluded with ease that the Hoechst tests were not consistent with the criteria established for NTP bioassays or the test guidelines set out in the FDA's Redbook (FDA 1982), and that it was likely that, at some point, many people would be exposed to acesulfame, the NTP rejected CSPI's nomination.

In 2003, the NTP announced the results of tests of both aspartame and acesulfame in genetically modified mice (GMM) (NTP 2005). Both chemicals gave negative results in the tests, carried out in two strains of GMM. The NTP's final report on those GMM studies (NTP 2005) noted that aspartame and acesulfame had been selected as "negative controls" for validation tests for the GMM models. The chemicals did indeed test negative, but that negative result did not advance our understanding of potential carcinogenicity of acesulfame. Regarding the GMM tests of aspartame and acesulfame, Martha Sandy of the California Environmental Protection Agency, stated that:

"[N]egative results [in the GMM tests] are not informative as to the test substance's carcinogenicity, and point to the need to conduct standard two-year carcinogenicity studies. At this time, transgenic models cannot replace the two-year bioassay and it would be unwise to list a chemical as safe for human exposure based upon negative results in not yet validated model systems."

(Sandy 2003) The findings of Soffritti et al. (2006) of multipotential carcinogenesis in rats fed aspartame over their lifetimes provide support for Sandy's (2003) statements. I have sent the NTP a new nomination of acesulfame for 2-year bioassay testing (Karstadt 2006). Full Text with References:

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1570055/

Headache • September 2006

Popular sweetener sucralose as a migraine trigger

Author information

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Mercer University School of Medicine, Macon, GA, USA

Abstract

Sucralose (trichlorogalactosucrose, or better known as Splenda) is an artificial sweetener from native sucrose that was approved by the FDA on April 1, 1998 (April Fool's Day). This observation of a potential causal relationship between sucralose and migraines may be important for physicians to remember this can be a possible trigger during dietary history taking. Identifying further triggers for migraine headaches, in this case sucralose, may help alleviate some of the cost burden (through expensive medical therapy or missed work opportunity) as well as provide relief to migraineurs.

http://www.ncbi.nlm.nih.gov/pubmed/16942478

Hansen's Diet Creamy Root Beer
INGREDIENTS

Pure Triple Filtered Carbonated Water, **Caramel Color, Potassium Citrate, Phosphoric Acid, Sucralose (Splenda Brand), Acesulfame Potassium, Natural Flavors (Licorice Root Extracts, Madagascan Vanilla, Wintergreen, Anise), Acacia.**

Hansen's Diet Sodas
Contain Caramel Coloring, Sucralose
and Phosphoric Acid

Phosphoric acid is an additive that gives soda its tangy flavor and makes it more acid than lemon juice or vinegar. A vast amount of sweetener is then used to mask and balance the acidity. Phosphoric acid has been linked to lower bone density in some epidemiological studies, including a discussion in the American Journal of Clinical Nutrition. The only good ingredient in Hansen's Diet Soda's is Triple Filtered Water and you certainly don't need Hansen's for that.

"This observation of a potential causal relationship between sucralose and migraines may be important for physicians to remember ..."

Regular consumption of Caramel Coloring can cause cancer

Journal Of Dental Research • October 2006

The role of sucrose in cariogenic dental biofilm formation—new insight

Author information

Paes Leme AF1, Koo H, Bellato CM, Bedi G, Cury JA.

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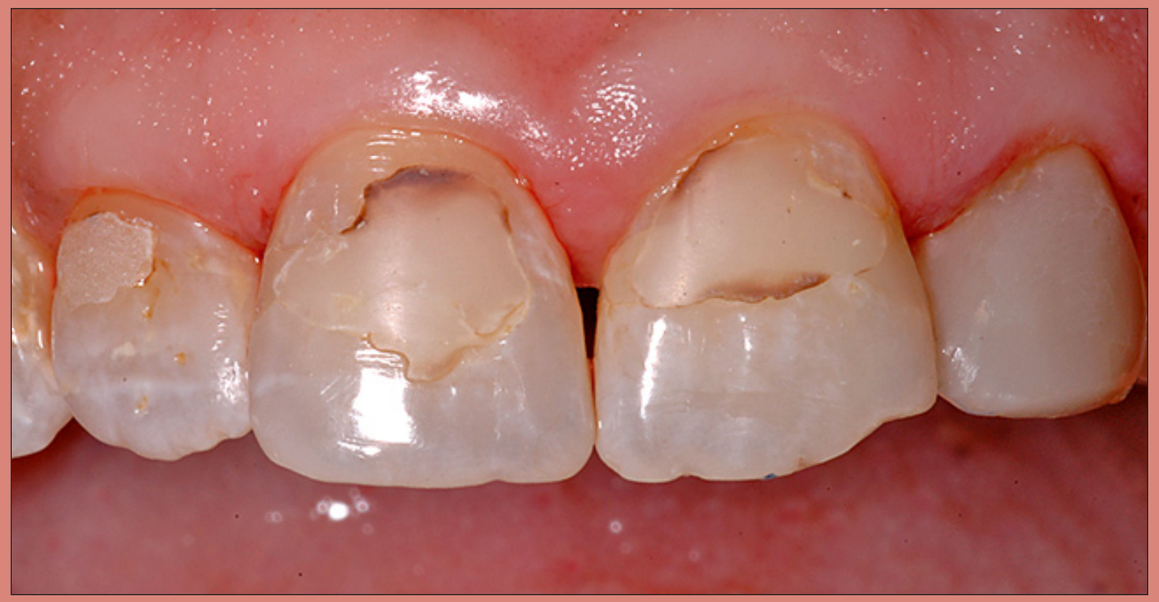
Abstract

Dental caries is a biofilm-dependent oral disease, and fermentable dietary carbohydrates are the key environmental factors involved in its initiation and development. However, among the carbohydrates, sucrose is considered the most cariogenic, because, in addition to being fermented by oral bacteria, it is a substrate for the synthesis of extracellular (EPS) and intracellular (IPS) polysaccharides. Therefore, while the low pH environment triggers the shift of the resident plaque microflora to a more cariogenic one, EPS promote changes in the composition of the biofilms' matrix. Furthermore, it has recently been shown that the biofilm formed in the presence of sucrose presents low concentrations of Ca, P(i), and F, which are critical ions involved in de- and remineralization of enamel and dentin in the oral environment. Thus, the aim of this review is to explore the broad role of sucrose in the cariogenicity of biofilms, and to present a new insight into its influence on the pathogenesis of dental caries.

Full text, graphs, charts and 90 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC2257872/



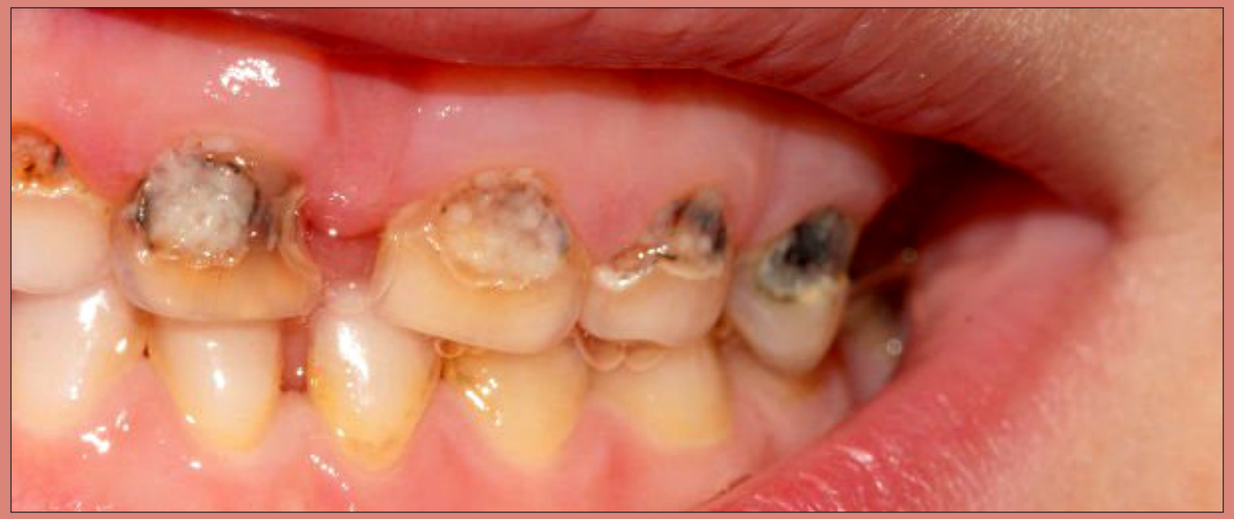
DENTAL CARIES

Dental caries, also known as tooth decay, cavities, or caries, is a breakdown of teeth due to the activities of bacteria. The cavities may be a number of different colors from yellow to black. Symptoms may include pain and difficulty with eating. Complications may include inflammation of the tissue around the tooth, tooth loss, and infection or abscess formation.

The cause of caries is bacterial breakdown of the hard tissues of the teeth (enamel, dentin and cementum). This occurs due to acid made from food debris or sugar on the tooth surface. Simple sugars in food are these bacteria's primary energy source and thus a diet high in simple sugar is a risk factor. If mineral breakdown is greater than build up from sources such as saliva, caries results. Risk factors include conditions that result in less saliva such as: diabetes mellitus, Sjogren's syndrome and some medications. Medications that decrease saliva production include antihistamines, antidepressants and cannabis among others. Caries is also associated with poverty, poor cleaning of the mouth, and receding gums resulting in exposure of the roots of the teeth.

Prevention includes: regular cleaning of the teeth and a diet low in sugar. Brushing the teeth two times per day and flossing between the teeth once a day is recommended by most doctors. Treating a mother's dental caries may decrease the risk in her children by decreasing the numbers of certain bacteria. Screening can result in earlier detection. Depending on the extent of destruction, various treatments can be used to restore the tooth to proper function or the tooth may be removed. The availability of treatment is often poor in the developing world.

Worldwide, approximately 2.43 billion people (36% of the population) have dental caries in their permanent teeth. The World Health Organizations estimates that nearly all adults have dental caries at some point in time. In baby teeth it affects about 620 million people or 9% of the population. They have become more common in both children and adults in recent years with the extraordinary increase in the use of sugar and sugar-like artificial substitutes. The disease is most common in the developed world and less common in the developing world due to greater simple sugar consumption. By the way, caries is Latin for "rottenness".



Biochemistry And Pharmacology • October 2004

The cytotoxic mechanism of glyoxal involves oxidative stress

Author information

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Abstract

Glyoxal is a reactive alpha-oxoaldehyde that is a physiological metabolite formed by lipid peroxidation, ascorbate autoxidation, oxidative degradation of glucose and degradation of glycated proteins. Glyoxal is capable of inducing cellular damage, like methylglyoxal (MG), but may also accelerate the rate of glycation leading to the formation of advanced glycation end-products (AGEs). However, the mechanism of glyoxal cytotoxicity has not been precisely defined. In this study we have focused on the cytotoxic effects of glyoxal and its ability to overcome cellular resistance to oxidative stress. Isolated rat hepatocytes were incubated with different concentrations of glyoxal. Glyoxal by itself was cytotoxic at 5mM, depleted GSH, formed reactive oxygen species (ROS) and collapsed the mitochondrial membrane potential. Glyoxal also induced lipid peroxidation and formaldehyde formation. Glycolytic substrates, e.g. fructose, sorbitol and xylitol inhibited glyoxal-induced cytotoxicity and prevented the decrease in mitochondrial membrane potential suggesting that mitochondrial toxicity contributed to the cytotoxic mechanism. Glyoxal cytotoxicity was prevented by the glyoxal traps d-penicillamine or aminoguanidine or ROS scavengers were also cytoprotective even when added some time after glyoxal suggesting that oxidative stress contributed to the glyoxal cytotoxic mechanism.

http://www.ncbi.nlm.nih.gov/pubmed/15345333

"Glyoxal is capable of inducing cellular damage, like methylglyoxal (MG), but may also accelerate the rate of glycation leading to the formation of advanced glycation end-products (AGEs)."

GLYOXAL

Glyoxal is an organic compound with the formula OCHCHO. It is a yellow-colored liquid that evaporates to give a green-colored gas. Glyoxal is the smallest dialdehyde (two aldehyde groups). Its structure is more complicated than typically represented because the molecule hydrates and oligomerizes. It is produced industrially as a precursor to many products.

Coated paper and textile finishes use large amounts of glyoxal as a crosslinker for starch-based formulations. It condenses with urea to afford 4,5-dihydroxy-2-imidazolidinone, which further reacts with formaldehyde to give the bis(hydroxymethyl) derivative used for wrinkle-resistant chemical treatments. It is used as a solubilizer and cross-linking agent in polymer chemistry:

- proteins (leather tanning process)
- collagen
- cellulose derivatives (textiles)
- hydrocolloids
- starch (paper coatings)

Glyoxal is a valuable building block in organic synthesis, especially in the synthesis of heterocycles such as imidazoles. A convenient form of the reagent for use in the laboratory is its bis-hemiacetal with ethylene glycol, 1,4-dioxane-2,3-diol. This compound is commercially available. Glyoxal solutions can also be used as a fixative for histology, that is, a method of preserving cells for examining them under a microscope.

Glyoxal In Food And The Environment

Glyoxal is an inflammatory compound formed when cooking oils and fats are heated to high temperatures. Glyoxal has been observed as a trace-gas in the atmosphere, e.g. as an oxidation product of hydrocarbons. Tropospheric concentrations of 0-200 pptv have been reported, in polluted regions up to 1 ppbv.

“Higher consumption of sugar-sweetened beverages is associated with a greater magnitude of weight gain and an increased risk for development of type 2 diabetes in women ...”

Journal Of The American Medical Association JAMA • August 2004

Sugar-sweetened beverages, weight gain, and incidence of type 2 diabetes in young and middle-aged women

Author information
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Abstract

CONTEXT

Sugar-sweetened beverages like soft drinks and fruit punches contain large amounts of readily absorbable sugars and may contribute to weight gain and an increased risk of type 2 diabetes, but these relationships have been minimally addressed in adults.

OBJECTIVE

To examine the association between consumption of sugar-sweetened beverages and weight change and risk of type 2 diabetes in women.

DESIGN, SETTING, AND PARTICIPANTS

Prospective cohort analyses conducted from 1991 to 1999 among women in the Nurses' Health Study II. The diabetes analysis included 91,249 women free of diabetes and other major chronic diseases at baseline in 1991. The weight change analysis included 51,603 women for whom complete dietary information and body weight were ascertained in 1991, 1995, and 1999. We identified 741 incident cases of confirmed type 2 diabetes during 716,300 person-years of follow-up.

MAIN OUTCOME MEASURES

Weight gain and incidence of type 2 diabetes.

RESULTS

Those with stable consumption patterns had no difference in weight gain, but weight gain over a 4-year period was highest among women who increased their sugar-sweetened soft drink consumption from 1 or fewer drinks per week to 1 or more drinks per day (multivariate-adjusted means, 4.69 kg for 1991 to 1995 and 4.20 kg for 1995 to 1999) and was smallest among women who decreased their intake (1.34 and 0.15 kg for the 2 periods, respectively) after adjusting for lifestyle and dietary confounders. Increased consumption of fruit punch was also associated with greater weight gain compared with decreased consumption. After adjustment for potential confounders, women consuming 1 or more sugar-sweetened soft drinks per day had a relative risk [RR] of type 2 diabetes of 1.83 (95% confidence interval [CI], 1.42-2.36; P<.001 for trend) compared with those who consumed less than 1 of these beverages per month. Similarly, consumption of fruit punch was associated with increased diabetes risk (RR for > or =1 drink per day compared with <1 drink per month, 2.00; 95% CI, 1.33-3.03; P =.001).

CONCLUSION

Higher consumption of sugar-sweetened beverages is associated with a greater magnitude of weight gain and an increased risk for development of type 2 diabetes in women, possibly by providing excessive calories and large amounts of rapidly absorbable sugars.

http://www.ncbi.nlm.nih.gov/pubmed/15328324/

Biochemical Society Transactions • December 2003

Toxicity of glyoxals—role of oxidative stress, metabolic detoxification and thiamine deficiency

Author information

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Abstract

Glyoxals are reactive alpha-oxoaldehydes that are formed endogenously from sugars, the levels of which are increased in various pathological conditions associated with hyperglycaemia and thiamine deficiency. However, the molecular cytotoxic mechanisms of glyoxal are not known. Results presented here and in the other studies cited provide a glimpse into the cytotoxicity mechanisms involved and their pathological implications. We found that glyoxal (10 microM) markedly increased the susceptibility of hepatocyte glutathione (GSH) to oxidation by hydrogen peroxide (H(2)O(2)) and markedly increased cytotoxicity by compromising the cellular antioxidant enzyme system. At higher concentrations, glyoxal was cytotoxic towards hepatocytes, which can be attributed to GSH depletion, oxidative stress and mitochondrial toxicity. Aminoguanidine or penicillamine protected the hepatocytes. Glyoxal cytotoxicity was prevented by increasing glyoxal metabolism with thiamine or NAD(P)H generators, and was increased in GSH- or thiamine-deficient hepatocytes. It was also found that feeding rats reduced thiamine levels in a diet high in simple sugars increased the number of aberrant crypt foci/colon in the absence of clinical evidence of beriberi. This was associated with decreased plasma thiamine and low erythrocyte transketolase activity. Western diets, which are frequently poor in thiamine and high in sugars, could result in increased levels of endogenous glyoxals, which in turn may lead to a predisposition to AGE (advanced glycation end-product)-related pathologies and neoplastic conditions.

http://www.ncbi.nlm.nih.gov/pubmed/14641070

THE INFAMOUS OREO COOKIE

High fructose corn syrup (HFCS) is a highly processed ingredient manufactured from surplus corn, and yielding a cheap replacement to table sugar. In the early 1980's many food manufacturers started using it instead of sugar as a cost cutting measure. That's about the same time obesity rates started to skyrocket in the US. Most scientists agree that HFCS is no better and no worse than plain sugar, though some newer studies seem to find the two affect the metabolism differently. Consumption of both should be drastically limited or eliminated completely.

OREO GRADING

Most people have an emotional attachment to certain sweets. For many Americans, this means Oreos. Are Oreos something to be avoided at all cost, or are they a safe occasional treat? Oreos are certainly not the healthiest cookies you could choose. They are made with high fructose corn syrup, vanillin (fake vanilla) and cheap oils. They are high in sugar, but low in fiber.

Unlike other industrial cookies, "Classic Oreo" cookies do not have artificial colors or partially hydrogenated oils. And while Oreos are high in sugar, one serving of Oreos (3 cookies) has less sugar than a single serving of most juices. Bottom line: If you can manage to keep this to a once-in-a-while treat and stick to the 3 cookies serving size, don't be so hard on yourself for breaking out of what is a normally healthy diet. Stick to classic Oreos to avoid artificial colors and avoid extra stuffing versions that pile on unneeded sugar. If you want to avoid HFCS or vanillin, try other branded -O- cookies. Vanillin is fake vanilla. Vanillin is chemically synthesized to taste like vanilla, but it's not the real deal. We are not recommending that commercial or industrial cookies are part of a healthy diet. It's best to avoid these food-like things completely.

Mutation Research • August 2002

The comet assay with 8 mouse organs: results with 39 currently used food additives

Author information

Sasaki YF1, Kawaguchi S, Kamaya A, Ohshita M, Kabasawa K, Iwama K, Taniguchi K, Tsuda S.

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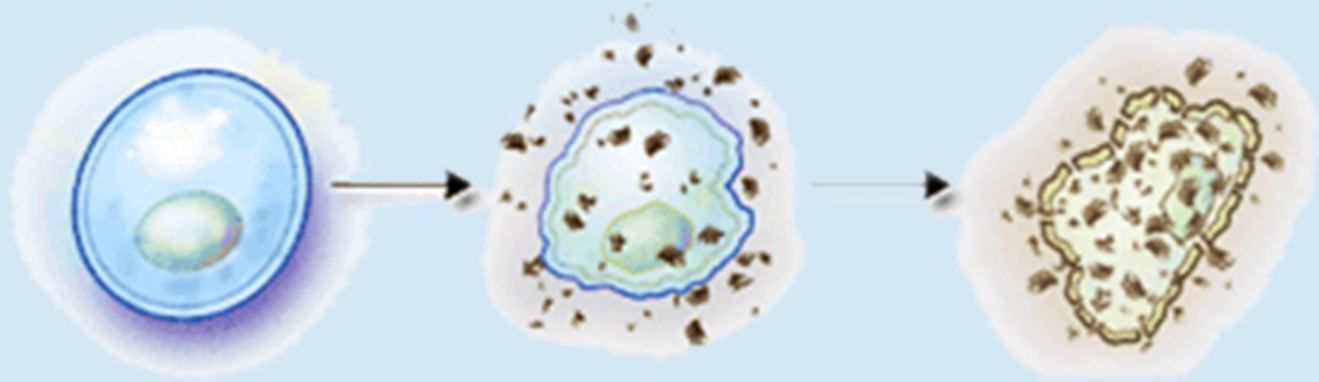
Abstract

We determined the genotoxicity of 39 chemicals currently in use as food additives. They fell into six categories-dyes, color fixatives and preservatives, preservatives, antioxidants, fungicides, and sweeteners. We tested groups of four male ddY mice once orally with each additive at up to 0.5xLD(50) or the limit dose (2000mg/kg) and performed the comet assay on the glandular stomach, colon, liver, kidney, urinary bladder, lung, brain, and bone marrow 3 and 24h after treatment. Of all the additives, dyes were the most genotoxic. Amaranth, Allura Red, New Coccine, Tartrazine, Erythrosine, Phloxine, and Rose Bengal induced dose-related DNA damage in the glandular stomach, colon, and/or urinary bladder. All seven dyes induced DNA damage in the gastrointestinal organs at a low dose (10 or 100mg/kg). Among them, Amaranth, Allura Red, New Coccine, and Tartrazine induced DNA damage in the colon at close to the acceptable daily intakes (ADIs). Two antioxidants (butylated hydroxyanisole (BHA) and butylated hydroxytoluene (BHT)), three fungicides (biphenyl, sodium o-phenylphenol, and thiabendazole), and four sweeteners (sodium cyclamate, saccharin, sodium saccharin, and sucralose) also induced DNA damage in gastrointestinal organs. Based on these results, we believe that more extensive assessment of food additives in current use is warranted.

http://www.ncbi.nlm.nih.gov/pubmed/12160896

“Of all the additives, dyes were the most genotoxic. Amaranth, Allura Red, New Coccine, Tartrazine, Erythrosine, Phloxine, and Rose Bengal induced dose-related DNA damage in the glandular stomach, colon, and/or urinary bladder. All seven dyes induced DNA damage in the gastrointestinal organs at a low dose (10 or 100mg/kg). Among them, Amaranth, Allura Red, New Coccine, and Tartrazine induced DNA damage in the colon at close to the acceptable daily intakes (ADIs). Two antioxidants (butylated hydroxyanisole (BHA) and butylated hydroxytoluene (BHT)), three fungicides (biphenyl, sodium o-phenylphenol, and thiabendazole), and four sweeteners (sodium cyclamate, saccharin, sodium saccharin, and sucralose) also induced DNA damage in gastrointestinal organs.”



Medical Hypotheses • August 2002

Impaired inactivation of digestive proteases by deconjugated bilirubin: the possible mechanism for inflammatory bowel disease

By X. F. Qin
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Abstract

Inflammatory bowel disease refers to ulcerative colitis and Crohn's disease, two gut diseases with unknown causes. The dramatic increase in the last half century and the big difference in incidence for people with the same ethnic background but living in different areas strongly suggested that environmental factors played the dominant role for these diseases. The similarity in many aspects for these two diseases suggested a common causative factor. Here I suggest the impaired inactivation of digestive proteases by deconjugated bilirubin, as the result of the inhibition of bilirubin deconjugation enzyme, beta-glucuronidase, originated from the luminal bacteria and mucosa of the gut, to be a possible mechanism for both ulcerative colitis and Crohn's diseases. I also provide evidence to suggest that saccharin could be the causative or one of the most important risk factors for inflammatory bowel disease as for its inhibition on beta-glucuronidase in the intestine.

http://www.ncbi.nlm.nih.gov/pubmed/12208202

“I also provide evidence to suggest that saccharin could be the causative or one of the most important risk factors for inflammatory bowel disease as for its inhibition on beta-glucuronidase in the intestine.”

“Any evidence of carcinogenesis —and there is ample such evidence— of such a widely used chemical should spur health officials to minimize human exposure to it.”

International Journal Of Occupational Environmental Health • October 2002

Carcinogenicity of saccharin in laboratory animals and humans: letter to Dr. Harry Conacher of Health Canada

Author information

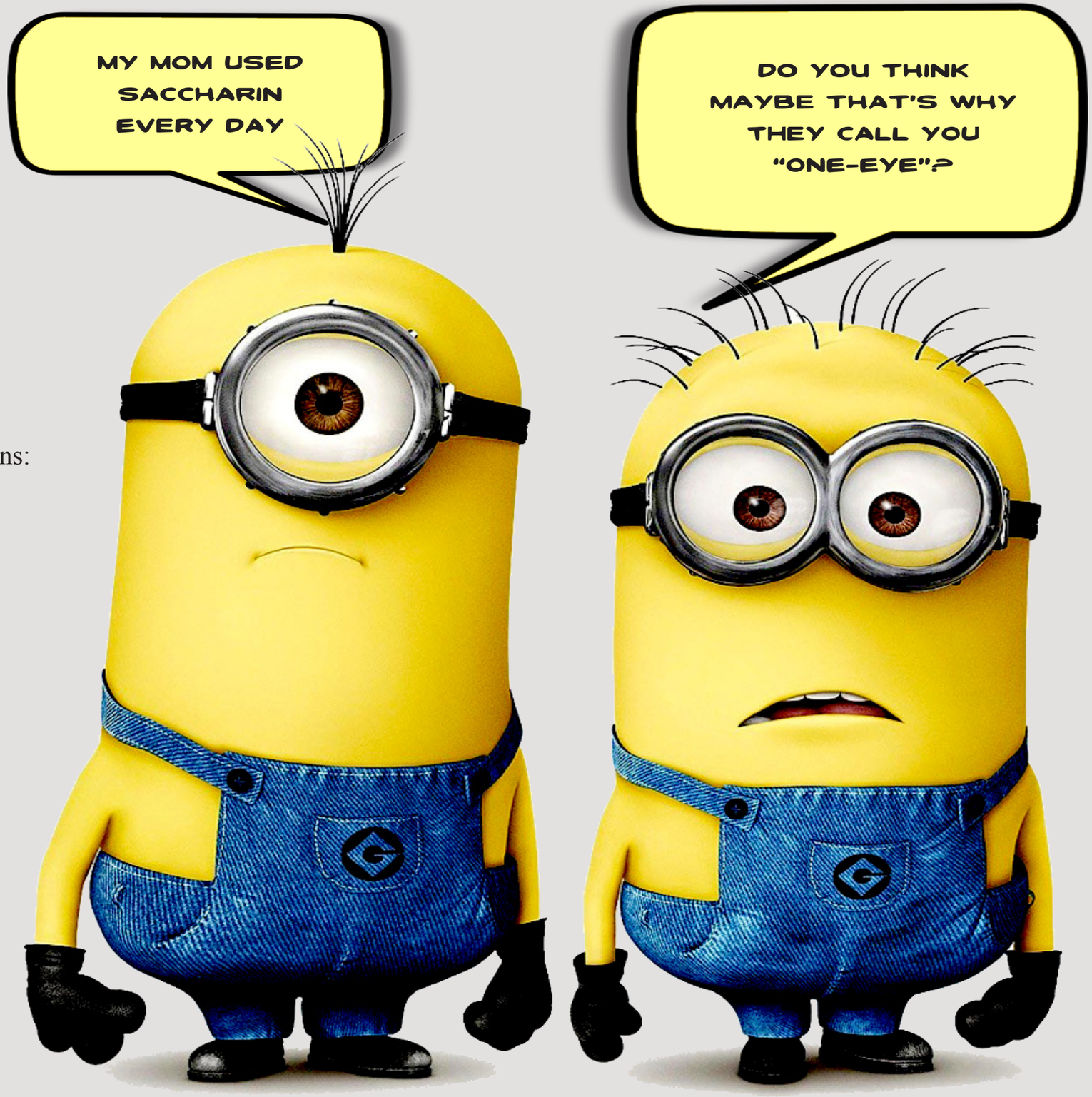
Bell W1, Clapp R, Davis D, Epstein S, Farber E, Fox DA, Holub B, Jacobson MF, Lijinsky W, Millstone E, Reuber MD, Suzuki D.

Canadian Association of Physicians for the Environment, Salmon Arm, BC

Abstract

We appreciate this opportunity to provide input to the Health Protection Branch’s (HPB’s) review of the artificial sweetener saccharin. Concerns with regard to the safety of saccharin are of great public health significance and of great interest to the public because saccharin is consumed by tens of millions of people, including children and fetuses. Any evidence of carcinogenesis—and there is ample such evidence—of such a widely used chemical should spur health officials to minimize human exposure to it. It is worth noting that on October 31, 1997, the Board of Scientific Counselors of the National Toxicology Program, a unit of the National Institute of Environmental Health Sciences (NIEHS), voted not to delist saccharin from its Report on Carcinogens.

http://www.ncbi.nlm.nih.gov/pubmed/12412858



> “Intakes of added sweeteners exceed levels compatible with meeting current dietary recommendations.”

Journal Of The American Dietetic Association • January 2000

Food sources of added sweeteners in the diets of Americans

By J.F. Guthrie[1] and J.F. Morton
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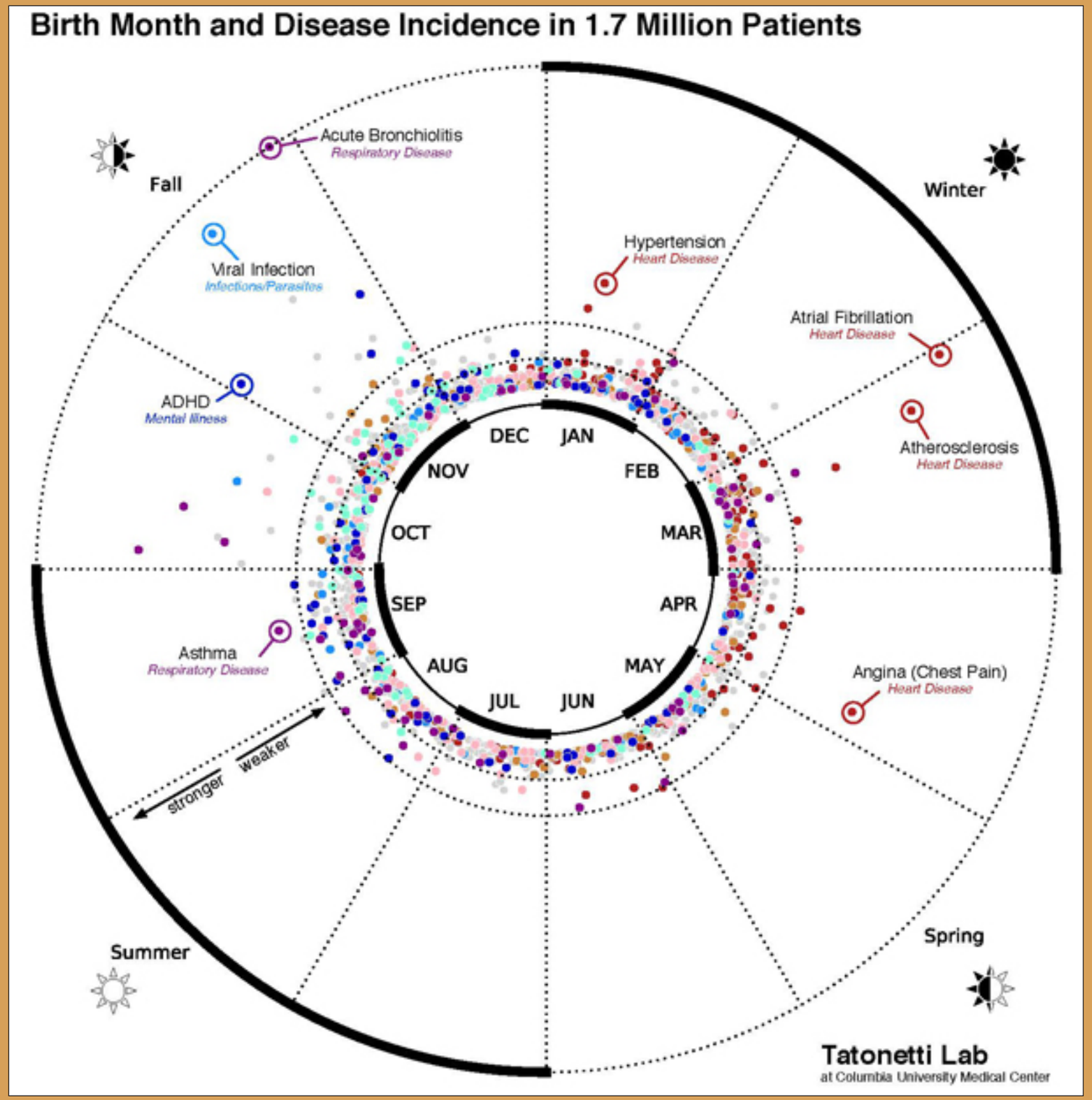
Abstract

OBJECTIVE
To identify food sources of added sweeteners in the US diet.
DESIGN
A descriptive study using data from the US Department of Agriculture (USDA) 1994-1996 Continuing Survey of Food Intakes by Individuals. Each subject provided one 24-hour dietary recall. Intake of added sweeteners was calculated using the USDA Food Guide Pyramid servings database.
SUBJECTS/SETTING
A national sample of noninstitutionalized persons aged 2 years and older (N = 15,010).
STATISTICAL ANALYSES
Mean intakes of added sweeteners from all food sources and from specific food categories; percentage contribution of added sweeteners to total energy intake; and percentage contribution of each food category to total intake of added sweeteners. All analyses were conducted for the total sample and for 12 age-gender groups.
RESULTS
During 1994 to 1996, Americans aged 2 years and older consumed the equivalent of 82 g carbohydrate per day from added sweeteners, which accounted for 16% of total energy intake. In absolute terms, adolescent males consumed the most; as a percentage of energy, male and female adolescents had the highest intakes (averaging 20% of total energy from added sweeteners). The largest source of added sweeteners was regular soft drinks, which accounted for one third of intake. Other sources were table sugars, syrups, and sweets; sweetened grains; regular fruitades/drinks; and milk products.
APPLICATIONS/CONCLUSIONS
Intakes of added sweeteners exceed levels compatible with meeting current dietary recommendations. Knowing food sources of added sweeteners for the overall population and for specific age-gender groups can help dietitians provide appropriate nutrition education.

http://www.ncbi.nlm.nih.gov/pubmed/10646004



Food Chemistry And Toxicology • December 2000

A combined chronic toxicity/carcinogenicity study of sucralose in Sprague-Dawley rats

Author information

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Abstract

The chronic toxicity and potential carcinogenicity of sucralose was evaluated by exposing Sprague-Dawley rats to dietary concentrations of this low-calorie sweetener both in utero and for up to 104 weeks following parturition. The rats assigned to the toxicity phase of this investigation were administered diets containing either 0% (control), 0.3% (3000 ppm), 1.0% (10,000 ppm) or 3.0% (30,000 ppm) sucralose. Each treatment group comprised 30 male and 30 female rats, of which 15 males and 15 females were sacrificed after 52 weeks of treatment. The surviving rats were killed following 78 weeks of sucralose administration. In the carcinogenicity phase of this investigation, groups of 50 male and 50 female rats were administered dietary sucralose at concentrations of 0% (control 1), 0% (control 2), 0.3%, 1.0% or 3.0% for 104 weeks. Evaluation of the data obtained from the two phases of this study showed that sucralose was not carcinogenic. Sucralose did not adversely affect the survival or clinical condition of the rats, and there were no toxicologically significant findings. Group mean body weight gain and food consumption were significantly decreased in a dose-dependent manner in sucralose-treated rats throughout the treatment period as compared to the controls. The primary effect of sucralose on food consumption, and secondarily on body weight gain, was established in later studies to be due to the fact that diets containing high concentrations of sucralose are unpalatable to rats. These subsequent studies established that the reduction of body weight gain seen in previous rat studies using sucralose in the diet at concentrations of 1% and below resulted from reduced food intake as a direct consequence of the unpalatable nature of sucralose. Similarly, at concentrations of 3% in the diet, it was shown that approximately 95% of the effect on body weight gain could be attributed to the reduction in food intake due to the reduced palatability of the diet, the remainder apparently due to a physiologic response to the high concentrations of non-digestible sucralose in the rats' diet. Complete toxicological evaluations of gavage studies with histopathological evaluations demonstrated that even at the 3% dietary level, toxicity was not responsible for the small body weight gain decrement. Gross and histopathologic examinations revealed that the administration of sucralose affected neither the types nor incidence of the tumours observed. The incidences of some non-neoplastic findings were statistically significantly increased in the sucralose treated groups relative to the controls. These included: renal pelvic epithelial hyperplasia in all female treatment groups, renal pelvic mineralization in females administered the intermediate or highest dietary concentrations of sucralose, adrenal cortical haemorrhagic degeneration in high-dose group female rats, and the histopathologic incidence of cataracts at necropsy in high-dose group male rats. The non-neoplastic findings that occurred were of no toxicological significance since they were either spontaneous findings commonly observed in aged rats of this strain or the physiological response to high dietary levels of a poorly absorbed compound.

http://www.ncbi.nlm.nih.gov/pubmed/10882819

“The incidences of some non-neoplastic findings were statistically significantly increased in the sucralose treated groups relative to the controls.

These included:

renal pelvic epithelial hyperplasia
in all female treatment groups,

renal pelvic mineralization in females
administered the intermediate
or highest dietary concentrations of sucralose,

adrenal cortical haemorrhagic degeneration
in high-dose group female rats,

and the histopathologic incidence of cataracts
at necropsy in high-dose group male rats.”

“The rapid absorption of glucose after consumption of high-dietary glycemic index meals induces a sequence of hormonal and metabolic changes that promote excessive food intake in obese subjects.”

Pediatrics • March 1999

High glycemic index foods, overeating, and obesity

Author information

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Abstract

OBJECTIVE
The prevalence of obesity has increased dramatically in recent years. However, the role of dietary composition in body weight regulation remains unclear. The purpose of this work was to investigate the acute effects of dietary glycemic index (GI) on energy metabolism and voluntary food intake in obese subjects.

METHODS
Twelve obese teenage boys were evaluated on three separate occasions using a crossover study protocol. During each evaluation, subjects consumed identical test meals at breakfast and lunch that had a low, medium, or high GI. The high- and medium-GI meals were designed to have similar macronutrient composition, fiber content, and palatability, and all meals for each subject had equal energy content. After breakfast, plasma and serum concentrations of metabolic fuels and hormones were measured. Ad libitum food intake was determined in the 5-hour period after lunch.

RESULTS
Voluntary energy intake after the high-GI meal (5.8 megajoule [mJ]) was 53% greater than after the medium-GI meal (3.8 mJ), and 81% greater than after the low-GI meal (3.2 mJ). In addition, compared with the low-GI meal, the high-GI meal resulted in higher serum insulin levels, lower plasma glucagon levels, lower postabsorptive plasma glucose and serum fatty acids levels, and elevation in plasma epinephrine. The area under the glycemic response curve for each test meal accounted for 53% of the variance in food intake within subjects.

CONCLUSIONS
The rapid absorption of glucose after consumption of high-GI meals induces a sequence of hormonal and metabolic changes that promote excessive food intake in obese subjects. Additional studies are needed to examine the relationship between dietary GI and long-term body weight regulation.

http://www.ncbi.nlm.nih.gov/pubmed/10049982

Ocean Spray Cranberry Juice Cocktail

Glycemic Index (Glucose is 100) = 68

Serving Size = Approximately 8 Ounces

Glycemic Load Per Serving = 24

The only drink with a higher Glycemic Load than Ocean Spray Cranberry Juice Cocktail was Average unsweetened Apple Juice (30).

High Glycemic Index For Other Edibles

Baked Russet Potato = 111
Cornflakes = 93
White Rice = 89
Instant Mashed Potato = 87
Pretzels = 83
Gatorade = 78
Graham Crackers = 74
Honey (Average) = 61

Source: Harvard Health Publications, Harvard Medical School,
Glycemic Index and Glycemic Load for 100+ Foods:

http://www.health.harvard.edu/healthy-eating/glycemic_index_and_glycemic_load_for_100_foods

“It is concluded that aspartame consumption may constitute a hazard because of its contribution to the formation of formaldehyde adducts.”

Life Sciences • January 1998

Formaldehyde derived from dietary aspartame binds to tissue components in vivo

Author information

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Remesar X, Fernández-López JA, Alemany M.

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Abstract

Adult male rats were given an oral dose of 10 mg/kg aspartame 14C-labelled in the methanol carbon. At timed intervals of up to 6 hours, the radioactivity in plasma and several organs was investigated. Most of the radioactivity found (>98% in plasma, >75% in liver) was bound to protein. Label present in liver, plasma and kidney was in the range of 1-2% of total radioactivity administered per g or mL, changing little with time. Other organs (brown and white adipose tissues, muscle, brain, cornea and retina) contained levels of label in the range of 1/12 to 1/10th of that of liver. In all, the rat retained, 6 hours after administration about 5% of the label, half of it in the liver. The specific radioactivity of tissue protein, RNA and DNA was quite uniform. The protein label was concentrated in amino acids, different from methionine, and largely coincident with the result of protein exposure to labelled formaldehyde. DNA radioactivity was essentially in a single different adduct base, different from the normal bases present in DNA. The nature of the tissue label accumulated was, thus, a direct consequence of formaldehyde binding to tissue structures. The administration of labelled aspartame to a group of cirrhotic rats resulted in comparable label retention by tissue components, which suggests that liver function (or its defect) has little effect on formaldehyde formation from aspartame and binding to biological components. The chronic treatment of a series of rats with 200 mg/kg of non-labelled aspartame during 10 days resulted in the accumulation of even more label when given the radioactive bolus, suggesting that the amount of formaldehyde adducts coming from aspartame in tissue proteins and nucleic acids may be cumulative. It is concluded that aspartame consumption may constitute a hazard because of its contribution to the formation of formaldehyde adducts.

http://www.ncbi.nlm.nih.gov/pubmed/9714421

Environmental Health Perspectives • February 1997

Reproductive toxicology—Sodium Saccharin—CAS#128-44-9 0

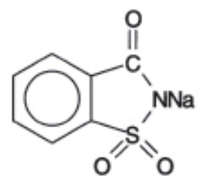
Swiss CD-1 mice, at 0.0, 1.25, 2.5, and 5.0% in water

James Lamb IV, NTP/NIEHS, Project Officer
Dushyant Gulati, Valerie Russell, Leta Hommel, K. Poonacha and P. Sabharwal,
Environmental Health Research and Testing Started 7/1/82 and Completed 2/20/85

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1470282/pdf/envhper00326-0340.pdf

Sodium Saccharin

CAS #128-44-9
Swiss CD-1 mice, at 0.0, 1.25, 2.5, and 5.0% in water
James Lamb IV, NTP/NIEHS Project Officer,
Dushyant Gulati, Valerie Russell, Leta Hommel, K. Poonacha, and
P. Sabharwal, Environmental Health Research and Testing
Started 7/1/82; Completed 2/20/85
NTIS #PB85188258/AS



Sodium saccharin (NaSac), used as an artificial sweetener since the late 1800s, was tested for potential effects on reproduction and fertility in Swiss CD-1 mice using the RACB protocol (Morrissey et al., Fundam Appl Toxicol 13:747–777 [1989]). Data on food and water consumptions, body weights, and clinical signs from the dose-range-finding study (Task 1) were used to set exposure concentrations for the continuous cohabitation phase (Task 2) at 1.25, 2.5, and 5% weight per volume in the drinking water. While water consumption was decreased at the high dose by approximately 10 to 20%, it was increased at the low and middle dose levels by approximately 40 and 20%, respectively. Consequently, the high dose animals gained slightly less weight during Task 2. Measures of body weights and water consumption allowed the calculation of daily exposure estimates: 3.5, 5.9, and 8.1 g/kg/day.

In Task 2, three, zero, one, and eight mice died in the control, low, middle, and high dose groups, respectively. The increased mortality at 5% NaSac was attributed to complications of dehydration. For the surviving pairs, there was no reduction in the mean number of litters per pair, although at the high dose, the number of live pups per litter decreased by 16% and the pup weight adjusted for litter size was decreased by 6%. There was no decrease in the viability of the offspring.

These effects were considered secondary to the decreased water intake seen at the high dose, and since the middle dose level had a relative increase in intake and showed no reproductive toxicity, Task 2 was judged to be essentially negative for reproductive toxicity, and the evaluation of the second generation was performed with only the controls and the middle dose level. Thus, the last litter from the control and middle dose groups was nursed, weaned, and reared to mating at approximately 70 to 80 days of age. There was no effect of exposure to NaSac on viability or growth to weaning, or on body weights at the start of the cohabitation week. Water consumption was increased by approx
mately 20% in the 2.5% NaSac gro
although this did not translate to a ch
in body weight. In the mating trial, t
were no differences due to NaSac co
sumption in the percent of F_1 pairs mating
or delivering a litter, or in the number,
weight, or viability of pups in that litter.

After the F_2 pups were evaluated and discarded, the F_1 controls and 2.5% NaSac-exposed mice were killed and necropsied. There were no differences between the groups in terminal body weights or organ weights. The concentration, percent motile, or percent morphologically abnormal sperm in the epididymis were unchanged by NaSac exposure, as was the length or characteristics of the estrous cycle.

In summary, NaSac reduced fertility only at a concentration that also significantly reduced water consumption and increased mortality. At concentrations that increased water consumption, there were no measurable effects on reproductive performance or necropsy end points.

> “Doses of 15, 30, 60, 450, 1500 and 2250 mg of acesulfame-K/kg [of] body weight induced a positive dose-dependent significant clastogenicity. These doses were within the no-toxic-effect levels reported by the Joint Expert Committee for Food Additives of the World Health Organization and the Food and Agriculture Organization of the United Nations.”

Food Chemistry And Toxicology • December 1997

In vivo cytogenetic studies on mice exposed to acesulfame-K—a non-nutritive sweetener

Author information

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Abstract

Acesulfame-K, a sweetening agent, was evaluated in vivo for its genotoxic and clastogenic potentials. Swiss albino male mice were exposed to the compound by gavage. Bone marrow cells isolated from femora were analysed for chromosome aberrations. Doses of 15, 30, 60, 450, 1500 and 2250 mg of acesulfame-K/kg body weight induced a positive dose-dependent significant clastogenicity (trend test alpha < 0.05). These doses were within the no-toxic-effect levels (1.5-3 g/kg body weight in rats) reported by the Joint Expert Committee for Food Additives of the World Health Organization and the Food and Agriculture Organization of the United Nations. In view of the present significant in vivo mammalian genotoxicity data, acesulfame-K should be used with caution.

http://www.ncbi.nlm.nih.gov/pubmed/9449223

~ This Peer Reviewed Report Is Almost 20 Years Old ~

Clastogenicity

In biology a clastogen is a mutagenic agent giving rise to inducing or causing the breakage or disruption of chromosmomes leading to sections of the chromosomes being deleted, added or rearranged. This process, a form of mutagenesis, can lead to carcinogenesis or cancer. Exposure increases the frequency of abnormal cells in paternal males which contributes to developmental effects in the fetus during fertilization and growth.

“Eleven percent of healthy preschoolers consumed > or = 12 fl oz/day of fruit juice, which is considered excessive. Excess fruit juice consumption has been reported as a contributing factor in some children with nonorganic failure to thrive and in some children with decreased stature. In other children, excessive fruit juice consumption has been associated with an increased caloric intake and obesity.”

Journal Of The American College Of Nutrition • October 1996

Fruit juice consumption by infants and children: a review

By B. A. Dennison
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Abstract

The pattern of fruit juice consumption has changed over time. Fifty years ago, orange juice was the major juice produced and it was consumed primarily to prevent scurvy. Now, apple juice is the juice of choice for the under 5 age group. While fruit juice is a healthy, low-fat, nutritious beverage, there have been some health concerns regarding juice consumption. Nursing bottle caries have long been recognized as a consequence of feeding juice in bottles, using the bottle as a pacifier, and prolonged bottle feeding. Non-specific chronic diarrhea or “toddler’s” diarrhea has been associated with juice consumption, especially juices high in sorbitol and those with a high fructose to glucose ratio. This relates to carbohydrate malabsorption, which varies by the type, concentration, and mixture of sugars present in different fruit juices. Fruit juice consumption by preschoolers has recently increased from 3.2 to about 5.5 fl oz/day. Consumption of fruit juice helps fulfill the recommendation to eat more fruits and vegetables, with fruit juice accounting for 50% of all fruit servings consumed by children, aged 2 through 18 years, and 1/3 of all fruits and vegetables consumed by preschoolers. Concomitant with the increase in fruit juice consumption has been a decline in milk intake. This is concerning as milk is the major source of calcium in the diet, and at present, only 50% of children, aged 1 through 5 years, meet the RDA for calcium. Studies of newborn infants and preschool-aged children have demonstrated a preference for sweet-tasting foods and beverages. Thus, it is not surprising that some children, if given the opportunity, might consume more fruit juice than is considered optimal. Eleven percent of healthy preschoolers consumed > or = 12 fl oz/day of fruit juice, which is considered excessive. Excess fruit juice consumption has been reported as a contributing factor in some children with nonorganic failure to thrive and in some children with decreased stature. In other children, excessive fruit juice consumption has been associated with an increased caloric intake and obesity. This paper reviews the role of fruit juice in the diets of infants and children and outlines areas for future research. Recommendations regarding fruit juice consumption based on current data are also given.

http://www.ncbi.nlm.nih.gov/pubmed/8892177

Regulatory Toxicology And Pharmacology • October 1996

Chronic toxicity and carcinogenicity study of erythritol in rats

Author information

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Abstract

The potential toxicity and carcinogenicity of erythritol, a low-calorie sugar substitute, were examined in Wistar Crl:(WI) WU BR rats. Groups of 50 rats of each sex consumed diets with 0, 2, 5, or 10% erythritol, or 10% mannitol, for a period of 104-107 weeks. To each of these main groups, two satellite groups of 20 males each were attached for interim kills after 52 and 78 weeks of treatment. At start of the study, the rats were 5-6 weeks old. The average intakes of erythritol in the 2, 5, and 10% groups were 0.9, 2.2, and 4.6 g/kg body wt/day for males and 1.0, 2.6, and 5.4 g/kg body wt/day for females, respectively. Mannitol intakes were 4.4 and 5.2 g/kg body wt/day in males and females, respectively. All treatments were well tolerated without diarrhea or other side effects. Body weights were significantly below control levels during most of the study in males of the 5% erythritol group and in males and females of the 10% erythritol and 10% mannitol groups. Survival of the animals was not adversely affected by the treatments. Hematological and clinicochemical examinations did not reveal noticeable changes which could be attributed to treatment. Analysis of urine samples collected during five 48-hr periods, from rats of the satellite groups in Weeks 26, 42, 50, and 78 and from rats of the main groups in Week 102, showed that about 60% of ingested erythritol was excreted unchanged. The urine volumes increased with increasing dietary erythritol levels. In line with previous observations on other polyols, erythritol and mannitol ingestion led to an increased excretion of urinary calcium and citrate. The urinary excretions of sodium, potassium, phosphate, N-acetylglucosaminidase (NAG), gamma-glutamyltransferase (GGT), low-molecular-weight protein (LMP), and total protein (TP) were slightly elevated in the 10% erythritol group. Increased GGT and NAG excretions also were seen occasionally at the 5% dose. Significantly increased relative cecum weights were seen in rats of either sex in the 10% mannitol and, somewhat less pronounced, 10% erythritol groups. Some cecal enlargement also was seen in the 5% erythritol group. The relative weight of the kidneys was highest in the 10% erythritol group, the difference from controls reaching statistical significance at interim kills (males) and termination (females). Except for more frequent pelvic nephrocalcinosis in female rats of all erythritol dose groups, the histopathological examinations did not reveal any nonneoplastic, preneoplastic, or neoplastic changes that could be attributed to the ingestion of erythritol. In male and female rats of the 10% mannitol group, pelvic nephrocalcinosis, which in females was associated occasionally with pelvic hyperplasia, was the only remarkable finding. The incidence and progression of nephrosis, which is commonly seen in aging rats of this strain, were not influenced by the treatments. In the absence of morphological alterations in the kidneys or other signs of nephrotoxicity, the increased excretions of NAG, GGT, LMP, and TP are regarded as innocuous, functional sequelae of the renal elimination of erythritol. In conclusion, the toxicological profile of erythritol in rats resembles that of other polyols in several respects. Except for nephrocalcinosis, which is commonly seen in polyol-fed rats, no other treatment-related, morphological changes were observed in the kidneys. Evidence for a tumor-inducing or tumor-promoting effect of erythritol was not seen.

http://www.ncbi.nlm.nih.gov/pubmed/8933643

ABOUT ERYTHRITOL

Erythritol ((2R,3S)-butane-1,2,3,4-tetraol) is a sugar alcohol (or polyol) that has been approved for use as a food additive in the United States and throughout much of the world. It was discovered in 1848 by Scottish chemist John Stenhouse. It occurs naturally in some fruit and fermented foods. At the industrial level, it is produced from glucose by fermentation with a yeast, Moniliella pollinis. Erythritol is 60–70% as sweet as sucrose (table sugar), yet it is almost noncaloric, does not affect blood sugar, does not cause tooth decay, and is partially absorbed by the body, excreted in urine and feces. Under U.S. Food and Drug Administration (FDA) labeling requirements, it has a caloric value of 0.2 kilocalories per gram (95% less than sugar and other carbohydrates), though nutritional labeling varies from country to country. Some countries, such as Japan and the United States, label it as zero-calorie, while the European Union currently labels it at 0 kcal/gram.

Erythritol And Human Digestion

In the body, most erythritol is absorbed into the bloodstream in the small intestine, and then for the most part excreted unchanged in the urine. About 10% enters the colon. Because 90% of erythritol is absorbed before it enters the large intestine, it does not normally cause laxative effects, as are often experienced after consumption of other sugar alcohols (such as xylitol. mannitol and maltitol), although extremely large doses can cause nausea and borborygmi (stomach rumbling).

Side Effects

In general, erythritol can cause a significant increase in nausea and stomach rumbling (borborygmi). Rarely, erythritol can cause allergic hives (urticaria). When compared with other sugar alcohols, it is also much more difficult for intestinal bacteria to digest, so it is less likely to cause gas or bloating more than other polyols, such as maltitol, sorbitol, or lactitol.

Blending For Sugar-Like Properties With Stevia

Erythritol is commonly used as a medium in which to deliver high-intensity sweeteners, especially stevia derivatives, serving the dual function of providing both bulk and a flavor similar to that of table sugar. Diet beverages made with this blend, thus, contain erythritol in addition to the main sweetener. Beyond high-intensity sweeteners, erythritol is often paired with other bulky ingredients that exhibit sugar-like characteristics to better mimic the texture and mouthfeel of sucrose. The cooling effect of erythritol is rarely desired, hence other ingredients are chosen to dilute or negate that effect. Erythritol also has a propensity to crystallize and is not as soluble as sucrose, so ingredients may also be chosen to help negate this disadvantage. Furthermore, erythritol is not hygroscopic, meaning it does not attract moisture, which can lead to the drying out of products, in particular baked goods, if another hygroscopic ingredient is not used in the formulation. Inulin is often combined with erythritol because of inulin's offering a complementary negative heat of solution (exothermic, or warming effect when dissolved, which helps cancel erythritol's cooling effect) and noncrystallizing properties. However, inulin has a propensity to cause gas and bloating in those having consumed it in moderate to large quantities, in particular in individuals unaccustomed to it. Other sugar alcohols are sometimes used with erythritol, in particular isomalt, because of its minimally positive heat of solution, and glycerin, which has a negative heat of solution, moderate hygroscopicity, and noncrystallizing liquid form. Erythritol is tooth-friendly; it cannot be metabolized by oral bacteria, so it does not contribute to tooth decay. We suggest avoiding this sweetener.

Journal Of Neuropathology And Experimental Neurology • November 1996

Increasing brain tumor rates: is there a link to aspartame?

Author information

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Abstract

In the past two decades brain tumor rates have risen in several industrialized countries, including the United States. During this time, brain tumor data have been gathered by the National Cancer Institute from catchment areas representing 10% of the United States population. In the present study, we analyzed these data from 1975 to 1992 and found that the brain tumor increases in the United States occurred in two distinct phases, an early modest increase that may primarily reflect improved diagnostic technology, and a more recent sustained increase in the incidence and shift toward greater malignancy that must be explained by some other factor(s). Compared to other environmental factors putatively linked to brain tumors, the artificial sweetener aspartame is a promising candidate to explain the recent increase in incidence and degree of malignancy of brain tumors. Evidence potentially implicating aspartame includes an early animal study revealing an exceedingly high incidence of brain tumors in aspartame-fed rats compared to no brain tumors in concurrent controls, the recent finding that the aspartame molecule has mutagenic potential, and the close temporal association (aspartame was introduced into US food and beverage markets several years prior to the sharp increase in brain tumor incidence and malignancy). We conclude that there is need for reassessing the carcinogenic potential of aspartame.

Full text, PDF, graphs, charts and 25 references

http://jnen.oxfordjournals.org/content/jnen/55/11/1115.full.pdf

“an exceedingly high incidence of brain tumors in aspartame-fed rats“

Carcinogenesis • November 1995

Effects of sodium ascorbate, sodium saccharin and ammonium chloride on the male rat urinary bladder

Author information

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Abstract

Sodium saccharin administered at high doses to male rats beginning after 5 weeks of age produces mild urothelial hyperplasia but does not result in a significant increase in incidence of bladder cancer unless it is administered after an initiating agent. However, if it is administered in a two-generation bioassay, a significant incidence of bladder tumors is produced. The hyperplastic and tumorigenic effects are inhibited by co-administration with high doses of NH4Cl. The present experiment was designed to evaluate the effects of another sodium salt, sodium ascorbate, administered through the neonatal time period. Sodium saccharin administered as 5% of the diet produced urothelial hyperplasia and increased labeling index, and this was inhibited by co-administration with 1.23% NH4Cl. Four doses of sodium ascorbate was evaluated. The lowest dose, 0.91%, was without effect on the urinary tract. A slight effect (not statistically significant) was observed at a dose of 2.73%, and a significant proliferative response was detected at 4.56 and 6.84%. Recent studies suggest that a calcium phosphate-containing amorphous precipitate forms in the urine of rats fed high doses of sodium saccharin, producing cytotoxicity of the urothelium and consequent regenerative hyperplasia. This precipitate was observed in the present experiment in the rats administered the high dose of sodium saccharin or the higher doses of sodium ascorbate. Formation of this precipitate and induction of urothelial proliferation were inhibited by co-administration of NH4Cl, but somewhat higher doses of ammonium chloride were required for doses of sodium ascorbate compared to sodium saccharin. These results demonstrate that sodium ascorbate administered through the neonatal time period of the male rat produces urothelial hyperplasia in the dose responsive manner, with a no-effect level of 0.91% of the diet. The formation of the calcium phosphate-containing amorphous precipitate and urothelial proliferation were inhibited by co-administration with NH4Cl.

http://www.ncbi.nlm.nih.gov/pubmed/7586194

“Sodium saccharin administered at high doses to male rats beginning after 5 weeks of age produces mild urothelial hyperplasia but does not result in a significant increase in incidence of bladder cancer unless it is administered after an initiating agent. However, if it is administered in a two-generation bioassay, a significant incidence of bladder tumors is produced.”

“The increases in the levels of urinary organic acids in the polyol-fed rats were explained in terms of impaired mitochondrial oxidation of these acids and of impaired conversion of methylmalonic acid to succinic acid.”

Toxicology And Applied Pharmacology • September 1987

Organic aciduria in rats fed high amounts of xylitol or sorbitol

By M.M. Hämäläinen

Abstract

The acidification of urine during polyol feeding was investigated with 27 Long-Evans male rats (aged 12 weeks) which were fed a xylitol diet (X), a sorbitol diet (S), or a basal diet for 4 weeks. The amount of polyols in the diet was increased from 5% to the final 20% level within 3 weeks. The polyol-fed animals showed reduced weight gain, lowered urine pH (from 6.5 to 5.6), and a 4-fold increase in the titratable acid excretion. X and S increased the daily urine volumes by 49 and 63%, respectively, but did not affect the wet weight or the pH values of the feces. as chromatographic-mass spectrometric analyses of organic acids revealed highly increased amounts of methylmalonic acid (13- to 20-fold) and 2-oxoglutaric acid (4- to 5-fold) in the urine of polyol-fed rats. The urinary excretion of citric acid and malic acid was also increased significantly (2- to 4-fold). The acidity of urine was not reflected in the blood acid-base balance of the animals. The increases in the levels of urinary organic acids in the polyol-fed rats were explained in terms of impaired mitochondrial oxidation of these acids and of impaired conversion of methylmalonic acid to succinic acid.

INGREDIENTS

sorbitol, gum base, glycerol, less than 2% of: **hydrogenated starch hydrolysate, natural and artificial flavors, soy lecithin, malic acid**, citric acid, **mannitol, sucralose, acesulfame k, fumaric acid, color (red 40, red 40 lake), aspartame, bht (to maintain freshness).**

http://www.ncbi.nlm.nih.gov/pubmed/3629597

“Such observed alterations in brain neurotransmitter concentrations may be responsible for the reported clinical and behavioral effects associated with Aspartame ingestion.”

Toxicology And Applied Pharmacology • March 1986

Neurobiochemical alterations induced by the artificial sweetener aspartame (NutraSweet)

Coulombe RA Jr, Sharma RP.

Abstract

The dipeptide aspartame (NutraSweet) is a newly approved and widely used artificial sweetener in foods and beverages. Consumption of aspartame (ASM) has been reported to be responsible for neurologic and behavioral disturbances in sensitive individuals. Unfasted male CD-1 mice were dosed orally with 13, 130, or 650 mg/kg ASM in corn oil, while control animals received corn oil alone. Three hours after dosing, the animals were killed, and the concentrations of the catecholamines norepinephrine (NE) and dopamine (DA), catecholamine metabolites 3-methoxy-4-hydroxymandelic acid (VMA), homovanillic acid (HVA), and dihydroxyphenylacetic acid (DOPAC), the indoleamine serotonin (5-HT), and its metabolite 5-hydroxyindoleacetic acid (5-HIAA) were determined by electrochemical high-performance liquid chromatography in six brain regions. ASM exerted its primary effect on adrenergic neurotransmitters in various brain regions. In the hypothalamus, the region richest in NE, increases in NE concentrations of 12, 49, and 47% were found in the low, medium, and high dose groups, respectively, relative to control. Significant increases of NE in the medulla oblongata and corpus striatum were also observed. Increases of the catecholamine DA and catecholamine metabolites VMA, HVA, and DOPAC were seen in various regions. The indoleamine serotonin and its metabolite 5-HIAA were unaffected by ASM treatment. These findings are consistent with ASM-induced increases in the brain catecholamine precursor amino acids phenylalanine and tyrosine, as reported earlier. Such observed alterations in brain neurotransmitter concentrations may be responsible for the reported clinical and behavioral effects associated with ASM ingestion.

http://www.ncbi.nlm.nih.gov/pubmed/2420032

WRIGLEY'S EXTRA GUM FOR KIDS INGREDIENTS

Sorbitol
Mannitol
Maltitol
Aspartame
Acesulfame K
Gum base
Carnauba wax
Lecithin
Gum arabic
Glycerine (non-animal)
Peppermint
E171 Titanium Dioxide
BHA

Items in **bold** can be hazardous to your health.

> “The diuretic effect of the polyols was considered responsible for the changes in the monovalent ion metabolism. The alterations in the excretion of multivalent cations most likely resulted from their increased intestinal absorption facilitated by the general chelating action of these polyols.”

The Journal Nutrition • April 1986

Alterations in electrolyte and iron metabolism in the rat in relation to peroral administration of galactitol, mannitol and xylitol

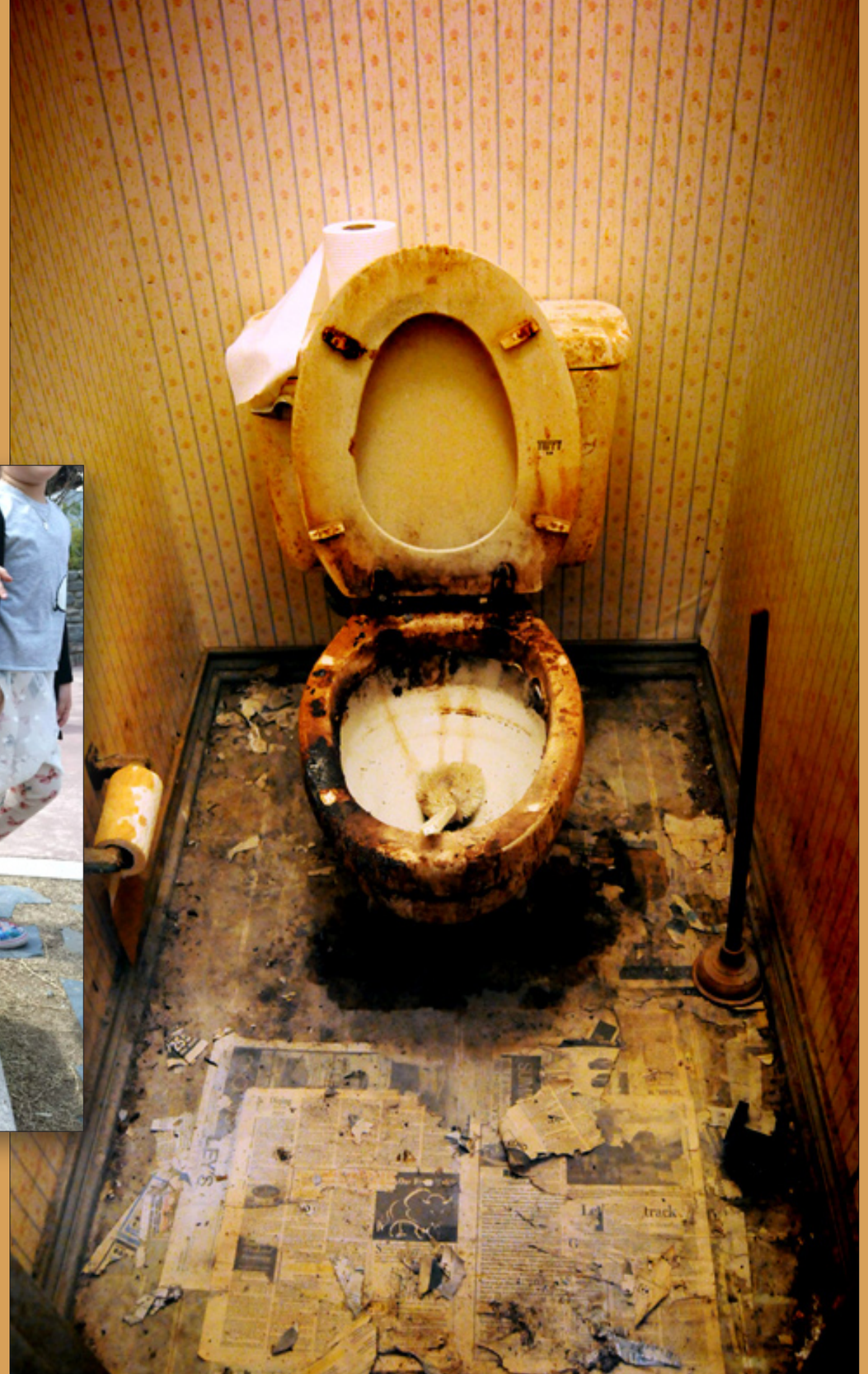
By M.M. Hämäläinen MM and K.K. Mäkinen

Abstract

Groups of 12 Long-Evans male rats were exposed to diets containing 20% galactitol (G), mannitol (M) or xylitol (X) for 5 wk. Serum electrolyte concentrations were within normal ranges for rats in all groups compared to control rats. All polyol-fed animals exhibited diuresis and a lower urinary pH (6.2-5.3) with a concomitant lower excretion of Na+, Cl- and protein (40% of controls). The excretion of K+ was lower in the X-fed rats than in any other group. Urinary Ca2+ excretion was sixfold higher and Mg2+ excretion, twofold higher in all polyol-fed rats than in controls. PO4 and NH4+ excretions were higher than controls in G- and M-fed animals only. Serum aldosterone concentrations in all polyol rats were 60% of those in controls. The serum corticosterone and parathyroid hormone levels were normal. Urinary citric acid was significantly higher in rats fed polyols but oxalic acid excretion was either normal (X) or lower (G,M) than in controls. Concentrations of serum and liver iron were higher in polyol-fed rats than in controls. Nevertheless, the normal serum creatinine and electrolyte concentrations and normal urinary creatinine levels established healthy kidney function. The diuretic effect of the polyols was considered responsible for the changes in the monovalent ion metabolism. The alterations in the excretion of multivalent cations most likely resulted from their increased intestinal absorption facilitated by the general chelating action of these polyols.

http://www.ncbi.nlm.nih.gov/pubmed/3083056

Above, The Chineses Toilet Musem, at right, your bathroom after over eating polyols like Mannitol and Xylitol.



“... all three polyols retarded the growth rate of the animals ... those fed galactitol or mannitol had lower blood glucose, and those fed mannitol had higher blood pyruvic acid ... Rats fed any of the polyols had lower serum total cholesterol and liver ascorbic acid than control rats.”

http://www.ncbi.nlm.nih.gov/pubmed/3925094

The Journal Of Nutrition • July 1985

Metabolic effects in rats of high oral doses of galactitol, mannitol and xylitol

By K.K. Mäkinen and M.M. Hämäläinen

Abstract

The effect of feeding high amounts of polyols on rat metabolism was studied. Adult male rats were fed the basal diet or the same diet to which had been added either galactitol, mannitol or xylitol for 8 wk (final polyol level 200 g/kg diet). Although all three polyols retarded the growth rate of the animals, the polyols were well tolerated. The four experimental groups did not differ significantly (P greater than 0.01) in the following analyses: blood lactic acid and serum transaminases, amylase, lactate dehydrogenase, triglycerides, insulin, glucagon and corticosterone. Compared to rats fed the basal diet, galactitol rats had higher blood hemoglobin levels (P less than 0.01); those fed galactitol or mannitol had lower blood glucose (P less than 0.001 and P less than 0.01, respectively), and those fed mannitol had higher blood pyruvic acid (P less than 0.01). Rats fed any of the polyols had lower serum total cholesterol and liver ascorbic acid (P less than 0.001) than control rats. Rats fed mannitol had higher liver glycogen levels (P less than 0.001) than control rats. Irrespective of the structural differences between the pentitol and the hexitols, a number of common metabolic effects were found. The proposed mechanisms of these effects include 1) the slow absorption and the rapid intraluminal metabolism of the polyols and 2) the similar handling of these polyols in the liver by a dehydrogenase.

Neurobehavior, Toxicology And Teratology • November 1984

Excitotoxic food additives—relevance of animal studies to human safety

By JW Olney

Abstract - Over 20 Years Old

“Evidence is reviewed supporting the view that excitotoxic food additives pose a significant hazard to the developing nervous system of young children. The following points are stressed: (1) although blood-brain barriers protect most central neurons from excitotoxins, certain brain regions lack such protection (a characteristic common to all vertebrate species); (2) regardless of species, it requires only a transient increase in blood excitotoxin levels for neurons in unprotected brain regions to be “silently” destroyed; (3) humans may be at particularly high risk for this kind of brain damage, since ingestion of a given amount of excitotoxin causes much higher blood excitotoxin levels in humans than in other species; (4) in addition to the heightened risk on a species basis, risk may be further increased for certain consumer sub-populations due to youth, disease or genetic factors; (5) despite these reasons for maintaining a wide margin of safety in the use of excitotoxins in foods, no safety margin is currently being observed, i.e., a comparative evaluation of animal (extensive) and human (limited) data supports the conclusion that excitotoxins, as used in foods today, may produce blood elevations high enough to cause damage to the nervous system of young children, damage which is not detectable at the time of occurrence but which may give rise to subtle disturbances in neuroendocrine function in adolescence and/or adulthood.”

http://www.ncbi.nlm.nih.gov/pubmed/6152304

> “Three two-generation studies using saccharin have since been conducted. The results from these studies clearly show that when rats were exposed to diets containing 5 or 7.5% sodium saccharin from the time of conception to death, an increased frequency of urinary bladder cancers was found, predominantly in the males.”

Environmental Health Perspectives • April 1983

Two-generation saccharin bioassays

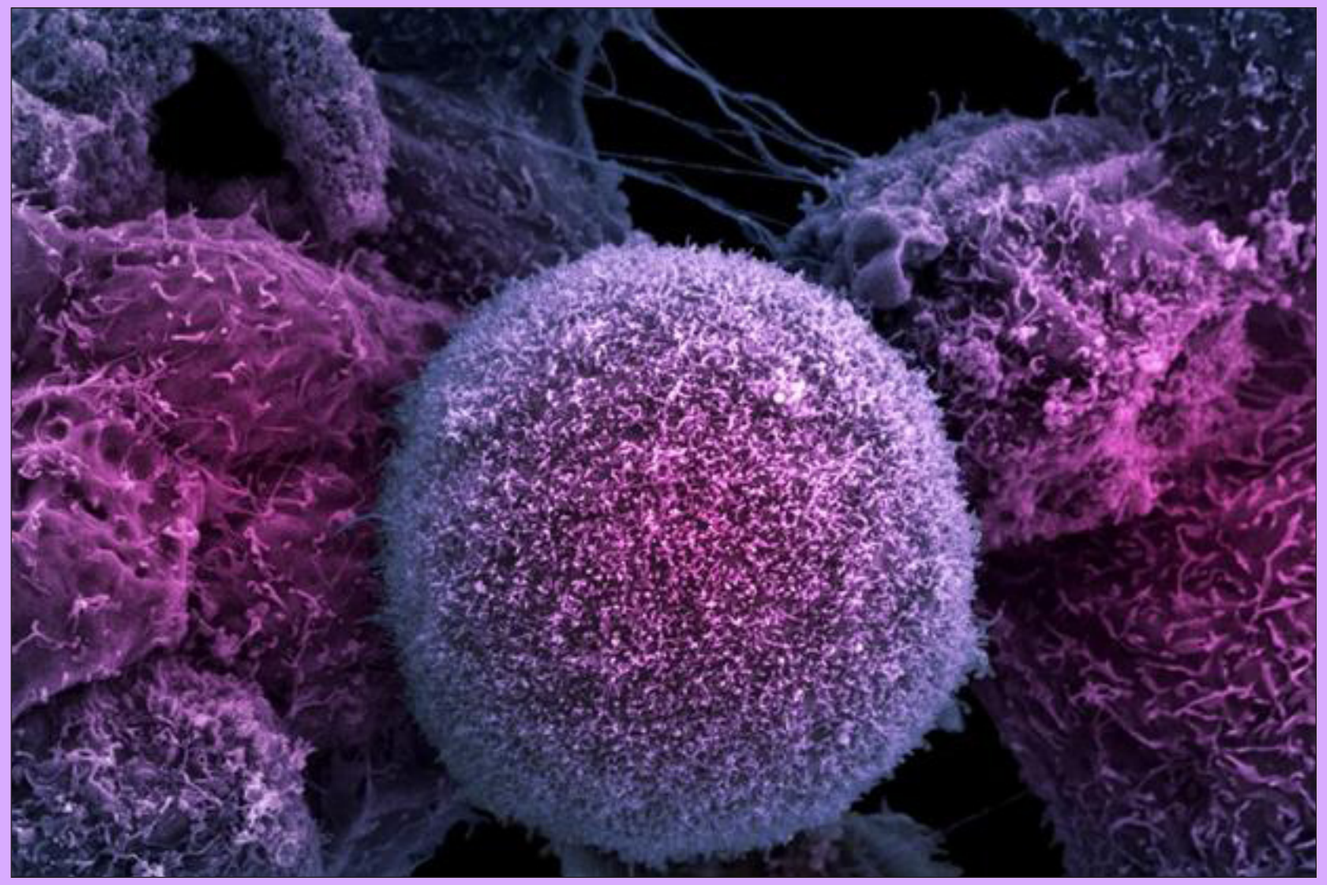
D L Arnold

Abstract

The controversy regarding the safety of saccharin for human consumption started shortly after its discovery over 100 years ago and has yet to subside appreciably. The consumption of saccharin, particularly in North America, began to escalate when the U.S. Food and Drug Administration set new standards of identity which allowed foods containing artificial sweeteners to be promoted as “nonnutritive” or “noncaloric” sweeteners for use by the general public. In 1969, when cyclamates were banned, at least 10 single-generation feeding studies were undertaken with saccharin to more accurately assess the potential toxicological consequences resulting from the anticipated increase in its consumption. None of these studies resulted in any overt regulatory action. Subsequently, the introduction of the two-generation chronic toxicity/carcinogenicity bioassay added a new tool to the toxicologist’s arsenal. Three two-generation studies using saccharin have since been conducted. The results from these studies clearly show that when rats were exposed to diets containing 5 or 7.5% sodium saccharin from the time of conception to death, an increased frequency of urinary bladder cancers was found, predominantly in the males. While some study results suggested that impurities in commercial saccharin or the presence of urinary tract calculi may have been responsible for the observed bladder tumors, it now appears that these possibilities are highly unlikely. The mechanism by which saccharin elicited the bladder tumors using the two-generation experiment has not been ascertained.

Full text with 37 references

http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1569221/



Enhanced scanning electron microscope image of a breast cancer cell

Annual Review Of Nutrition • July 1981

Nutritional significance of fructose and sugar alcohols

By Y.M. Wang YM and J. van Eys

Abstract

Human metabolism of D-fructose, D-sorbitol, D-mannitol, and xylitol has been documented. In humans, sorbitol and xylitol at a single oral dose of 20 g or less and fructose at 70 g or less most likely can be fully absorbed. These there sugars can maintain, either independently or nearly independently, the integrity or the carbohydrate requirement for the growth of cells and animals. The absorption of D-mannitol is no more than 80% and is more laxative. In general, there is no adverse effect other than osmotic diarrhea after oral administration of these sugars. Transient hyperuricemia was seen in some humans. The chronic toxicity of life-long usage of these sugars in humans or other primates is not known. However, a 2-year Turku sugar studies suggested the safety of fructose and xylitol. Two-year feeding experiments in mice and rats indicated possible carcinogenicity of a high-percentage xylitol diet. Abnormalities of cellular growth were also documented in animals fed high percentages of sorbitol and sucrose. Long-term mannitol feeding experiments also revealed an increased incidence of benign thymic tumors in rats. Intravenous feeding of fructose, xylitol, and sorbitol causes major concern. The toxicity is total-dose and infusion-rate dependent. The physical toxicity induced by hyperosmolar effect of the concentrated infusion solutions can be lethal. The primary metabolic toxicities, mainly lactic acidosis and hypruricemia, are reversible. The suggested safe infusion rate of these sugars is 0.25 g/kg/h; sporadic toxic observations have been reported at this or lower doses (0.125 g/kg/h). The combination of glucose, fructose, xylitol, and sorbitol mixture intravenously is in use in Europe due to the critical threshold of each element. There are positive findings from the use of the combination in human illness (114). The beneficial effect of xylitol, mannitol, sorbitol, and fructose in decreasing order has been well documented in the prevention of dental caries in animals and in humans. Oral organisms do not appear to metabolically adapt to xylitol even after 4 years of in vivo exposure. This was based on the quantitation of xylitol dehydrogenase activity in saliva and oral organisms. In addition, a therapeutic and preventive effect for xylitol in human and animal dental caries has been demonstrated. There appears to be at least a theoretical edge in the dietary use of fructose, xylitol, and sorbitol in diabetics.

http://www.ncbi.nlm.nih.gov/pubmed/6821187

“Two-year feeding experiments in mice and rats indicated possible carcinogenicity of a high-percentage xylitol diet. Abnormalities of cellular growth were also documented in animals fed high percentages of sorbitol and sucrose. Long-term mannitol feeding experiments also revealed an increased incidence of benign thymic tumors in rats. Intravenous feeding of fructose, xylitol, and sorbitol causes major concern. The toxicity is total-dose and infusion-rate dependent. The physical toxicity induced by hyperosmolar effect of the concentrated infusion solutions can be lethal.”

Internationale Zeitschrift Fur Vitamin Und Ernahrungsforschung Beiheft • July 1976

Sugar substitutes in the diabetic diet

By H. Mehnert

Abstract

The decreased glucsoe utilization in diabetes mellitus justifies the use of sugar substitutes (“diabetic sugar”) if two conditions are fulfilled: 1)The sugar substitute should be a carbohydrate which does not lead, or only to a slight degree, to hyperglycaemia and thus, in this respect, differs distinctly from sugars such as glucose and saccharose. 2) The sugar substitute must not cause undesired side-effects. The absorption, utilization and side-effects of the sugar substitutes fructose, sorbitol and xylitol were investigated. They were found to be more slowly absorbed than glucose and thus to offer the advantage of better utilization under conditions of limited insulin production. However, the particularly slow passive absorption of sorbitol and xylitol can sometimes be a disadvantage, since osmotic diarrhoea may occur after administration of high oral doses. The sugar substitutes enter the metabolism enzymatically and are utilized mainly in the liver. The peripheral state was investigated after intravenous, intraduodenal and oral administration of glucose and fructose to healthy subjects. Liver metabolism was examined (Dietze) by comparing hepatic venous and arterial concentrations after intravenous administration of the sugars. Also, diabetic patients received glucose and fructose orally. As previously demonstrated, the investigations using several techniques showed a smaller influence on blood glucose and serum insulin concentrations after administration of fructose, sorbitol and xylitol than after glucose. If no metabolic changes occur after intravenous administration of high doses, no such changes need be expected after oral administration of small doses. Nor did measurements in hepatic venous blood (Dietze) show any marked effect of fructose on the blood glucose level. The healthy subjects showed no significant changes in blood glucose or serum insulin concentration after either intraduodenal or oral administration of fructose, whereas they showed a considerable increase after glucose administration. Investigations in adult-type diabetics revealed a better utilization of fructose than glucose. With correct dosage, sugar substitutes are able to increase the carbohydrate tolerance and, under certain conditions, to achieve a relative stabilization of the metabolism of unstable diabetics. The antiketogenic activity of sugar substitutes is particularly pronounced. Side-effects such as high blood levels of urea, lactate, triglycerides and bilirubin or a decrease in hepatic adenin nucleotides do not occur after oral administration, nor are they of importance after intravenous administration with correct dosage. The osmotic diarrhoea occurring after intake of sorbitol or xylitol is caused by their slow absorption and limits the consumption of these sugar substitutes. In the often obese adult-type diabetics, the calorie intake inherent in the consumption of diabetic sugars may have an unfavourable influence on their weight...

http://www.ncbi.nlm.nih.gov/pubmed/783058

“... the particularly slow passive absorption of sorbitol and xylitol can sometimes be a disadvantage, since osmotic diarrhoea may occur after administration of high oral doses.”

BULLETPROOF

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Proving Monsanto's Crimes

The Serious GMO Dangers And
Related Pesticide Neural Disorders
And The Truly Unimaginable:
The Murders, the Millions
Murdered For Profit
And The Millions
Still To Be
Killed

Silent Partners

Over 7 Billion Humans Have Been Colonized By Bacteria - Not A Shot Fired

by Jeff Prager

Even the British Empire had nothing on gut bacteria. The successful
is a largely untold
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of us. But it can turn rogue, causing skin infections—or worse.
century has prompted the evolution of deadly superbug strains.
erium that is a member of the Firmicutes, and is frequently found
itive for catalase and nitrate reduction. Although S. aureus is not
ections (e.g. boils), respiratory disease (e.g. sinusitis), and food
poisoning. Disease-associated strains often promote infections by producing potent protein toxins, and expressing cell-surface proteins that bind and inactivate antibodies. The emergence of antibiotic-resistant forms of pathogenic S. aureus (e.g. MRSA) is a worldwide problem in clinical medicine.

The Benefits Of Probiotics

We don't even know if probiotics work. We don't know if they manufacture our needed B vitamins, including niacin, pyridoxine, folic acid, and biotin. We don't know if they enhance our immune system activity. We don't know if antibacterial substances that kill or deactivate hostile disease causing bacteria actually work at all. Friendly bacteria do this by changing the local levels of acidity, by depriving pathogenic bacteria of their nutrients, or by actually producing their own antibiotic substances, yet we don't know if drinking kefir, eating pickles and sauerkraut or ingesting probiotics even works. We don't know if the ingested bacteria builds a house on your intestinal wall, so to speak, and lives in your gut for a day, a week, a month or forever or whether they take the next gut-wrenching feces train to Sewer City, USA on the Toilet Express. Honestly, we're clueless. But we think it helps.

Gut Bacteria

by Jeff Prager

Gut microorganisms benefit the host by gleaning the energy from the fermentation of undigested carbohydrates and the subsequent absorption of short-chain fatty acids. The most important of these fatty acids are:

• Butyrates, metabolized by the colonic epithelium

• Propionates metabolized by the liver

• and acetates metabolized through the action of muscle tissue

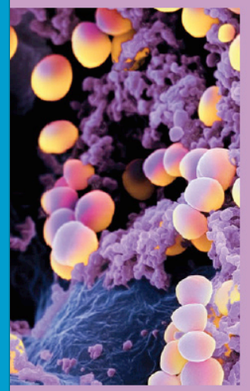
Intestinal bacteria also play a role in synthesizing vitamin B and vitamin K as well as metabolizing bile acids, sterols and xenobiotics. Xenobiotics are typically a synthetic chemical that is foreign to the body or in some cases to an entire ecological system. Xenobiotics are intestinal janitors.

The human body carries about 100 trillion microorganisms in its intestines, a number ten times greater than the total number of human cells in the body. The metabolic activities performed by these bacteria resemble those of an actual organ like a heart or a lung, leading some to liken gut bacteria to a "forgotten" organ. It's estimated that these gut flora have around a hundred times as many genes in aggregate as these are in the human genome.

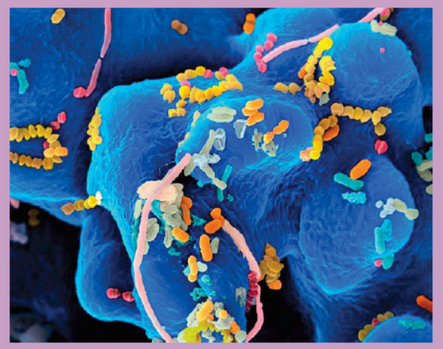
Our microbiota so very obviously play a large, significant and unquestionably necessary part in good human health and a part we still know very, very little about.

As a species we human beings are rapidly developing a new genera of immunerable and often times rare disorders manifesting with such intrusive and unimaginably odd neurological symptoms such that it's a wonder all approximately 305 million of us aren't already on the verge of a completely and swiftly disabling disorder.

Salmonella are bacteria that can live in the intestinal tracts of humans and other animals. Shown here is a color-enhanced and highly magnified view of Salmonella typhimurium (in red) invading cultured human cells in a great photo from Rocky Mountain Laboratories.

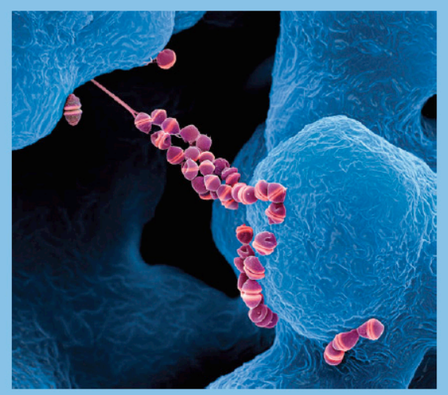


COCCUS AUREUS



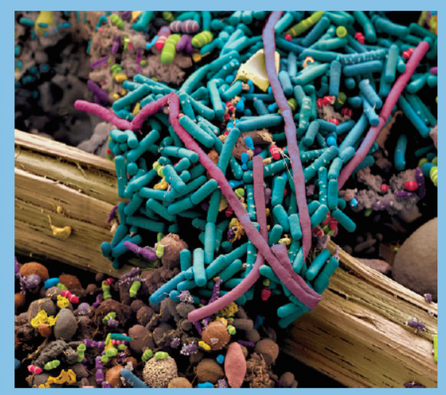
MOUTH MICROBES

The human mouth hosts a panoply of microbes, some taking up residence on the mouth lining (blue) within days after birth. Harmful species form biofilms, like the plaque that encourages tooth decay, or colonize the crevices between teeth and gums, causing periodontal disease. Oral probiotics designed to boost the population of species that outcompete pathogenic ones could help prevent or reverse dental disease. Researchers (very patient researchers) have painstakingly harvested all the plaque from every surface of every tooth. It weighs, on average, about 10 mg. But the teeth only comprise 1/20 of all the oral surfaces. You have to multiply the 10 mg from the teeth by 20 to get the total biomass including cheeks, tongue, etc. We also know that 1 mg of oral biomass typically contains about 100 million microbes. By multiplying the number of microbes in 1 mg by 20, we get the total number of microbes in the entire oral cavity. 100 million microbes x 20 mg biomass = 20 billion oral microbes living in your mouth right now. almost all of those billions of microbes that we swallow began their lives in an oral biofilm. Thus, despite only having (at any given time) 20 billion microbes in our mouths, we nevertheless swallow 100 billion! Five times more than we have. So, those 20 billion microbes in our mouths must be producing and shedding 100 billion additional microbes every day. That's five times their original number. Said another way, they are doubling their numbers five times every 24 hours. Dividing 24 hours by 5 = 4.8 hours, the amount of time it takes for the microbes in our mouths to double their number. There are 20 billion bacteria in your mouth and they reproduce every five hours. If you go 24 hours without brushing, those 20 billion become 100 billion!



STREPTOCOCCUS

A colorized electron microscope image captures delicate chains of streptococcus in a laboratory sample. Though some strep infections can be deadly, many strains are harmless—among the thousands of benign beings that make their home in our bodies. Most Streptococcus genomes are 1.8 to 2.3 Mb in size and encode 1,700 to 2,300 proteins. There are two types of Strep: group A and group B. Group A strep causes Strep throat - a sore, red throat, sometimes with white spots on the tonsils; Scarlet fever - an illness that follows strep throat. It causes a red rash on the body; Impetigo - a skin infection; Toxic shock syndrome; Cellulitis and necrotizing fasciitis (flesh-eating disease). Group B strep can cause blood infections, pneumonia and meningitis in newborns. A screening test during pregnancy can tell if you have it. If you do, I.V. antibiotics during labor can save your baby's life. Adults can also get group B strep infections, especially if they are elderly or already have health problems. Strep B bacteria can cause urinary tract infections, blood infections, skin infections and pneumonia in adults.



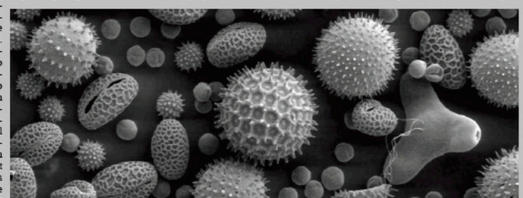
HELICOBACTER

Helicobacter pylori (yellow), a common bacterium that lives in the stomach lining, increases the risk of stomach cancer (brown cells) and peptic ulcers. But over time H. pylori can reduce stomach acid and acid reflux, which may help fend off esophageal cancer. The microbe also appears to help protect us from allergies and asthma. Some scientists suspect that the dramatic increase in those conditions in the industrialized world could be related to the decreasing frequency of H. pylori in our stomachs, which is partly due to high doses of antibiotics in childhood. Helicobacter pylori is a Gram-negative, microaerophilic bacterium found in the stomach, and may be present in other parts of the body, such as the eye. It was identified in 1982 by Australian scientists Barry Marshall and Robin Warren with further research led by British scientist Stewart Goodwin, who found that it was present in patients with chronic gastritis and gastric ulcers, conditions not previously believed to have a microbial cause. It is also linked to the development of duodenal ulcers and stomach cancer. However, over 80% of individuals infected with the bacterium are asymptomatic and it may play an important role in the natural stomach ecology.

metabolism is poorly understood but appears to involve transport to the liver by the portal circulation. It is believed that SCFAs also impact water absorption, local blood flow, and epithelial proliferation in the large intestine [9].

Genomic analysis of gut bacteria offers vivid examples of the role of microbes in nutrient utilization. For example, in 2003, Xu, et al. published the complete genome sequence of the gram-negative anaerobe Bacteroides thetaiotaomicron, a prominent member of the normal intestinal microbiota [10]. Annotation and analysis of the genome revealed a sophisticated apparatus for acquiring and digesting otherwise unusable dietary polysaccharides. This apparatus, including a complex, multi-component, multi-enzyme complex starch utilization system (SUS), consists of over 230 glycoside hydrolase and 15 polysaccharide lyase genes [15]. The genomic analysis demonstrated that B. thetaiotaomicron has evolved the remarkable capacity to sense the availability of carbohydrates in its microenvironment, and that it also has the ability to forage and utilize host-derived glycans (e.g., mucin and heparin). Mechanistic studies in gnotobiotic animals further demonstrated that, when B. thetaiotaomicron senses a scarcity of fucose in the intestinal lumen, it actually induces the gut epithelium to upregulate expression of fucosylated glycans that can be used by the bacteria as an energy source without harming the host [16]. This body of work illustrates how the remarkable host-microbe symbiosis can be teased apart by pairing genomic sequencing efforts with creative in vivo laboratory studies.

Microbiota and protein metabolism



amino acids. More work is needed to define these contributions in both healthy and undernourished humans.

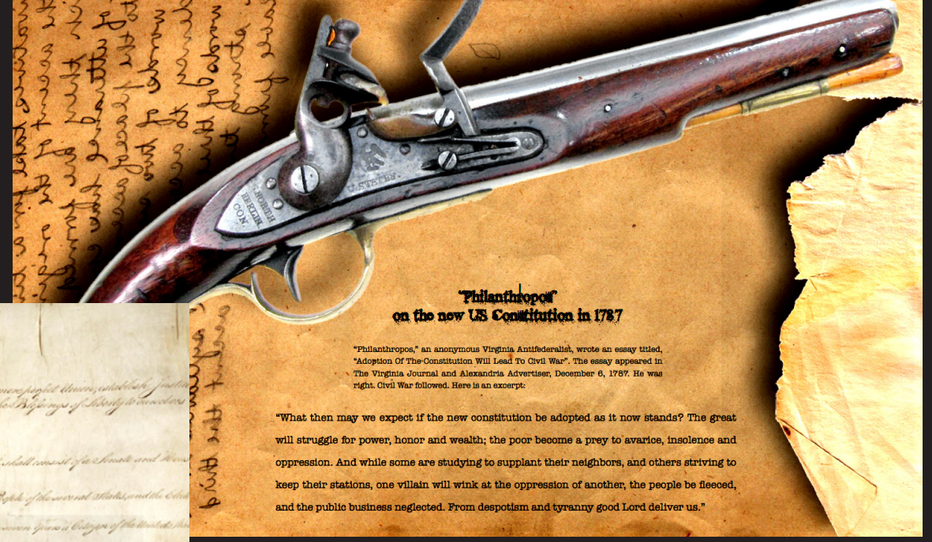
The intestinal microbiota also contributes to nitrogen balance by participating in urea nitrogen salvaging (UNS) [21, 22]. Elevated urease expression in gut microbes results in metabolism of urea in the GI tract into ammonia and carbon dioxide. Some of the ammonia can be utilized for microbial synthesis of amino acids. Perhaps more importantly, the nitrogen generated during this process (urea nitrogen) can re-enter the host circulation and serve as a substrate for synthetic processes [23]. Interestingly, reduced urea recycling has been reported in GF animals [24] and in humans receiving antibiotic therapy [25]. Furthermore, several reports indicate that regulation of UNS is important in settings of low N intake and high N demand (e.g., during pregnancy and during periods of rapid somatic growth in infancy) [26–28]. While still relatively preliminary, these studies underscore the relationship between gut microbes and protein metabolism that will likely be further described through on-going characterization of the human microbiome.

MICROBIOTA AND LIPID METABOLISM

Until recently, few studies of the association between lipid metabolism and the microbiome existed. However, important research by Jeffrey Gordon, Fredrick Backhed, and colleagues suggests that the body's supply of triglycerides, a prominent source of energy during critical illness [29], is tightly linked to the intestinal microbiota. These findings have enormous potential relevance for research in a wide range of disease states, including metabolic disorders such as obesity (see below) and cardiovascular disease.

Opinions of the US Constitution in 1787

A Back To Reality Feature Length Commercial Free Adventure Story by Jeff Prager



"Philanthropos" on the new US Constitution in 1787

"Philanthropos," an anonymous Virginia Antifederalist, wrote an essay titled, "Adoption Of The Constitution Will Lead To Civil War". The essay appeared in The Virginia Journal and Alexandria Advertiser, December 6, 1787. He was right. Civil War followed. Here is an excerpt:

"What then may we expect if the new constitution be adopted as it now stands? The great will struggle for power, honor and wealth; the poor become a prey to avarice, insolence and oppression. And while some are studying to supplant their neighbors, and others striving to keep their stations, one villain will wink at the oppression of another, the people be fleeced, and the public business neglected. From despotism and tyranny good Lord deliver us."

"Montezuma" on retraining the Lower Classes

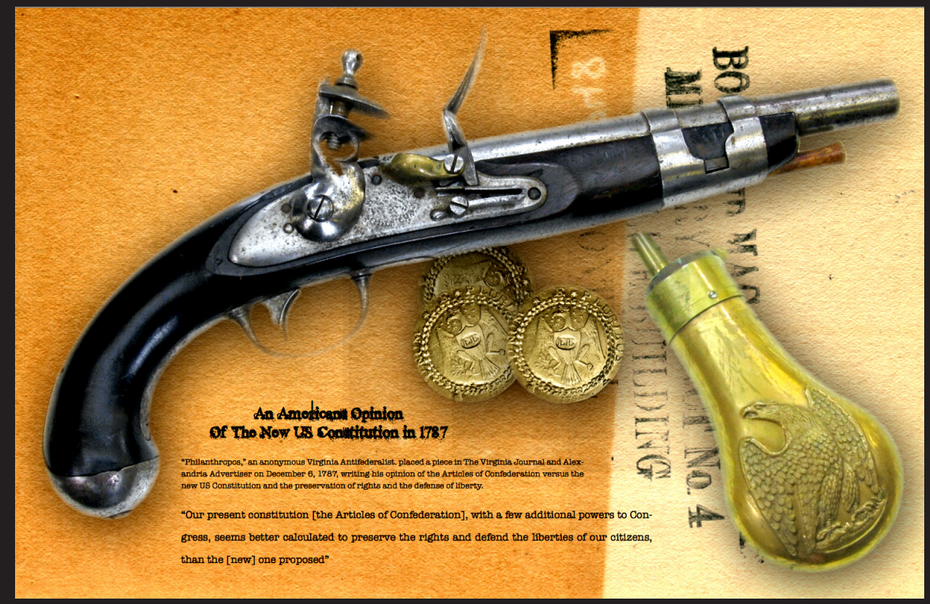
"Montezuma" was regarded as a Pennsylvanian and wrote this essay which showed up in the Independent Gazetteer on October 17, 1787. The essay then became Antifederalist Paper No. 9, titled, "A Consolidated Government Is A Tyranny".

Montezuma stated, "We the Aristocratic party of the United States" have a "monarchical, aristocratical democracy." He went on to say that we are "impressed with a conviction that this constitution is calculated to restrain the influence and power of the Lower Class."

An opinion from a speech by Patrick Henry in 1788

The following excerpt is from a speech by Patrick Henry on June 5th, 1788 at the Virginia Ratifying Convention. It was published as Anti-Federalist Paper No. 4 titled, "Foreign Wars, Civil Wars, and Indian Wars - Three Bugbears". This excerpt of the commentary by Henry refers to the new US CONstitution and was a warning, even as far back as 1788, that "the people" were in serious trouble.

"We are cautioned by the honorable gentleman, who presides, against faction and turbulence. I acknowledge that licentiousness is dangerous, and that it ought to be provided against: I acknowledge, also, the new form of government may effectually prevent it: yet there is another thing it will as effectually do — it will oppress and ruin the people."



An American Opinion Of The New US Constitution in 1787

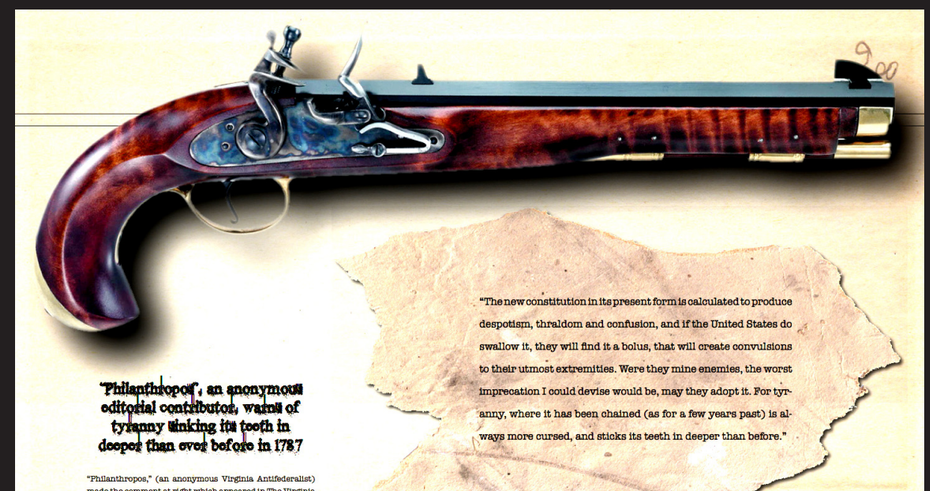
"Philanthropos," an anonymous Virginia Antifederalist, placed a piece in The Virginia Journal and Alexandria Advertiser on December 6, 1787, writing his opinion of the Articles of Confederation versus the new US Constitution and the preservation of rights and the defense of liberty.

"Our present constitution [the Articles of Confederation], with a few additional powers to Congress, seems better calculated to preserve the rights and defend the liberties of our citizens, than the [new] one proposed"

From a June 5th, 1788 speech by Patrick Henry

The following excerpt is from a speech by Patrick Henry on June 5th, 1788 at the Virginia Ratifying Convention. It was published as Anti-Federalist Paper No. 4 titled, "Foreign Wars, Civil Wars, and Indian Wars - Three Bugbears".

"This Constitution is said to have beautiful features; but when I come to examine these features, Sir, they appear to me horridly frightful: Among other deformities, it has an awful squinting; it squints to-



"Philanthropos", an anonymous editorial contributor, warns of tyranny sinking its teeth in deeper than ever before in 1787

"Philanthropos," (an anonymous Virginia Antifederalist)

"The new constitution in its present form is calculated to produce despotism, thraldom and confusion, and if the United States do swallow it, they will find it a bolus, that will create convulsions to their utmost extremities. Were they mine enemies, the worst imprecation I could devise would be, may they adopt it. For tyranny, where it has been chained (as for a few years past) is always more cursed, and sticks its teeth in deeper than before."

Autoimmune Syndromes and Inflammatory Syndromes

Caused By Adjuvants In Vaccines

Also Known As

ASIA

A Two Smoking Guns Publication
Produced by Jeff Prager

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Renegade Publishing


KRAFT
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Johnson&Johnson
GLACÉAU
Yum!
QUAKER
Splend a
Spin-off
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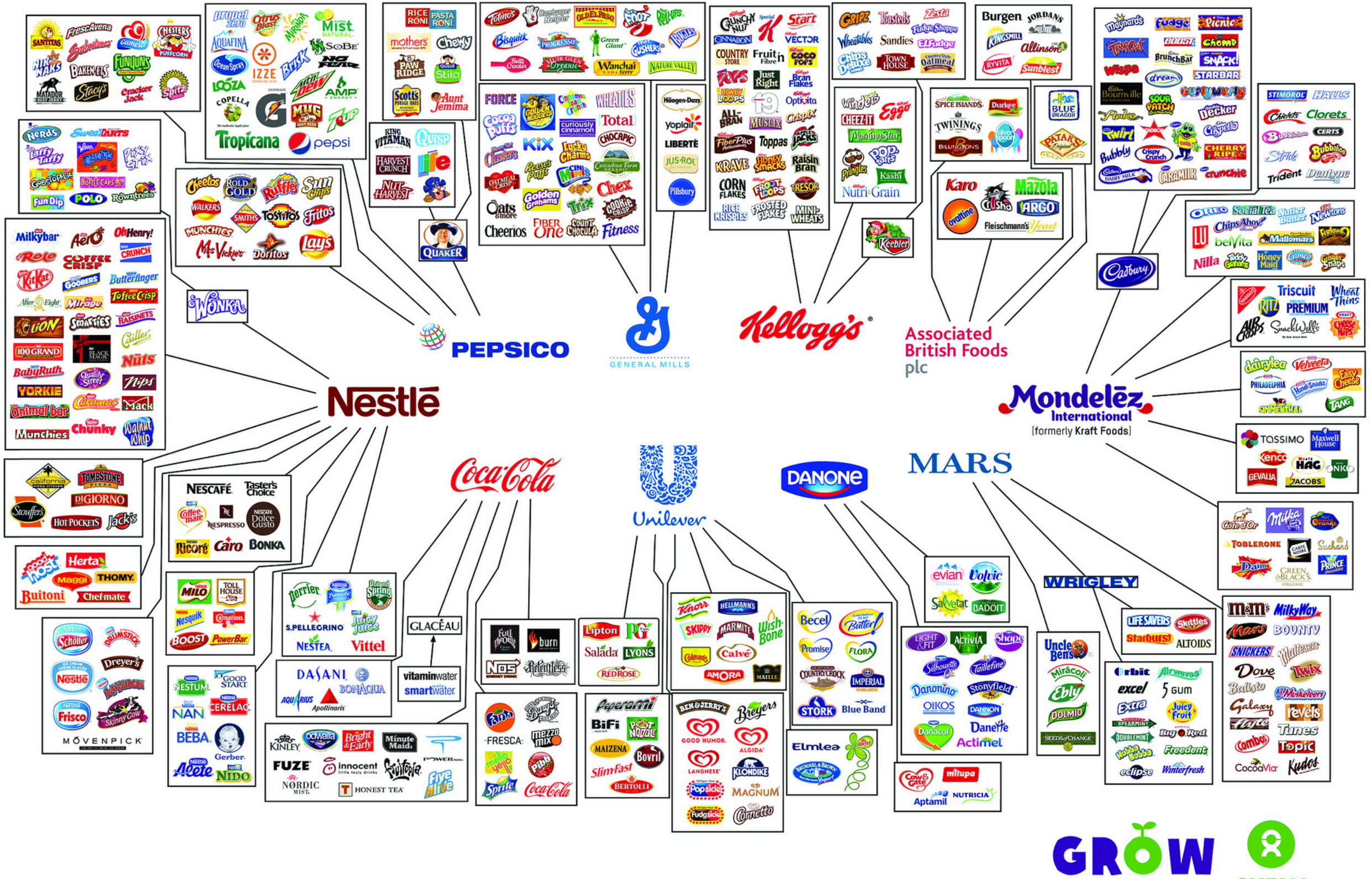
PEPSICO
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Until then, eat your
Organic spinach!