Did the EFSA Plagairize the Aspartame Manufacturer Review?

EFSA Draft Lines	EFSA Draft Text (Copied From Aspartame manufacturer-funded review) http://www.efsa.europa.eu/sites/default/files/consultation/130108.pdf	Aspartame Manufacturer Review Lines	Aspartame Manufacturer-Funded Review Text (Critical Reviews in Toxicology, 37:8, 629-727, 2007) (See: www.holisticmed.com/aspartame/burdock/)	Sentences Exactly the Same	Sentences Nearly the Same	Notes
				42	**37**	
3396 - 3405	Kruesi et al. (1987) evaluated the effect of sugar and aspartame on 'aggression and activity' in preschool boys (ages 2 to 6 years) who were identified as 'sensitive to sugar' by their parents. The study was a double-blind cross-over challenge with aspartame (30 mg/kg bw), sucrose (1.75 g/kg bw), saccharin (amount not specified) and glucose (1.75 g/kg bw) to sugar-responsive (children described as being sensitive to sugar; n = 14) and age-matched control boys (n = 10). The sweeteners were given in a lemon-flavoured drink each sweetener was given on two occasions; once in a laboratory setting, and once 4 days later in a home setting. Children were scored for 'aggression and activity' by researchers during the laboratory playroom challenge, and by their parents in the days following the challenge to detect any delayed reaction, and during the home challenge. Washout periods of 5–7 days occurred between challenges.	Page 694 Column 1	Kruesi et al. (1987) evaluated the effect of sugar and aspartame on aggression and activity in preschool boys (ages 2 to 6 years) who were identified as sensitive to sugar or “sugar responders”. The study was a double-blind cross-over challenge with aspartame (30 mg/kg bw), sucrose (1.75 g/kg bw), saccharin (amount not specified) and glucose (1.75 g/kg bw) to sugar-responsive (n = 14) and age-matched control boys (n = 10). The sweeteners were given in a lemon-flavored drink once in a laboratory setting, and once 4 days later in a home setting. Children were scored for activity and aggression by researchers during the laboratory playroom challenge and by their parents in the days following to detect any delayed reaction, and during the home challenge.Washout periods of 5–7 days occurred between challenges.	**3**	**2**	
3416 - 3426	In a randomised, double blind, and placebo-controlled crossover study Shaywitz et al. (1994) assessed the effect of aspartame on behaviour and cognitive function of children with attention deficit disorder. The dose of aspartame was 34 mg/kg bw/day. The children (n = 15, 11 males, 4 females, ages 5 to 13 years) were given capsules of either aspartame or placebo (microcrystalline cellulose) each morning for a 2-week period. Parents were instructed to provide an aspartame-free diet during the study. No effect was found on cognitive, attentive or behavioural testing or on urinary levels of neurotransmitters (noradrenaline, adrenaline, dopamine, homovanilic acid and 5-hydroxyindoleacetic acid), but plasma tyrosine and phenylalanine levels were higher 2 hours after the aspartame treatment. Plasma tyrosine level values were not provided. Plasma phenylalanine levels are only reported graphically, and increased from approximately 60 µM at baseline to approximately 85 µM two hours after aspartame dosing.	Page 694 Column 2	Shaywitz et al. (1994b) assessed the effect of aspartame on behavior and cognitive function of children with attention deficit disorder using a randomized, double blind, and placebo-controlled crossover study design. The dose of aspartame was 34 mg/kg bw/day. Children (n = 15, 11 males, 4 females, ages 5 to 13 years) were given capsules of either aspartame or placebo (microcrystalline cellulose) each morning for a 2-week period. Parents were instructed to provide an aspartame-free diet during the study. No effect was found on cognitive, attentive or behavioral testing or on urinary levels of neurotransmitters (norepinephrine, epinephrine, dopamine, HVA, and 5HIAA), although plasma tyrosine and phenylalanine levels were higher 2 h after the aspartame treatment. Plasma phenylalanine levels were reported graphically, and increased from approximately 6 µmol/dl at baseline to about 8.5 µmol/dl 2 h after aspartame dosing.	**2**	**3**	

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3432 - 3436	A double-blind randomised crossover trial (Lapierre et al., 1990) with 10 healthy adult volunteers (6 men, 4 women, ages 21–36 years) evaluated the effect of a single dose of aspartame (15 mg/kg bw) or placebo capsules on mood, cognitive function, and reaction time. No effect was observed on any of the parameters measured (i.e. hunger, headache, memory, reaction time, or cognition) during the study despite elevation of plasma phenylalanine levels following consumption (data values not reported).	Page 695 Column 1	A double-blind randomized crossover trial with 10 healthy volunteers (6 men, 4women, ages 21–36 years) evaluated the effect of a single dose of aspartame (15 mg/kg bw) or placebo capsules on mood, cognitive function, and reaction time. No effect was observed on hunger, headache, memory, reaction time, or cognition during the study despite elevation of plasma phenylalanine levels following consumption (data values not reported). The percentage of total LNAA that was phenylalanine increased from approximately 11% to a peak of about 18 at the 2-h time point after dosing, but dropped to normal after 8 h (Lapierre et al., 1990).	**1**	**1**	
3443 -3444	Pivonka and Grunewald (1990) compared the effect of water, and aspartame and sugar-containing beverages on mood in 120 young women and found no effect on self-reported surveys of mood.	Page 695 Column 1	Pivonka and Grunewald (1990) compared the effect of water, and aspartame- and sugar-containing beverages on mood in 120 young women and found no effect on self-reported surveys of mood.	**1**	**0**	
3446 -3457	The acute study (Stokes et al., 1991) involved 12 healthy certified pilots (four females and eight males). The study was double-blinded with each subject undertaking testing on 5 occasions, with at least 1 week between treatments that were given in random order among the 12 participants. Participants were tested for baseline values, then given placebo capsules, aspartame (50 mg/kg bw), or ethyl alcohol (positive control, estimated dose to raise plasma alcohol 0.1%), followed by a post-test with 3451 no treatment. For all treatments, participants consumed orange juice with either a trace or the test dose of alcohol, and capsules containing either placebo (dextrose) or aspartame, all participants consumed a small carbohydrate meal prior to treatments. Cognitive performance was tested using the SPARTANS cognitive test battery (a sensitive test to detect changes in performance of complex tasks required for aircraft operations). Cognitive impairment was detected in several tasks following consumption of the low dose of alcohol but not aspartame or placebo treatments.	Page 695 Column 1 and Column 2	The first study involved 12 healthy certified pilots (4 females and 8 males). The study was double-blinded with each subject being tested 5 times, with at least 1 week between treatments given in random order among the 12 participants. Participants were pretested for baseline values, then given placebo capsules, aspartame (50 mg/kg bw), or ethyl alcohol (positive control, estimated dose to raise blood alcohol 0.1%), followed by a posttest with no treatment. For all treatments, participants consumed orange juice with either a trace or the test dose of alcohol, and capsules with either placebo (dextrose) or aspartame. Cognitive performancewas tested using the SPARTANS cognitive test battery, which is a sensitive test to detect changes in performance of complex tasks required for aircraft operations. As has been discussed previously, concerns have been voiced regarding the possible potentiation of the effects of aspartame by consumption concurrently with carbohydrates. Therefore, all participants consumed a small carbohydrate meal prior to treatments. Consumption of other foods, aspartame, and alcohol was controlled prior to testing. Blood levels of amino acids were not measured. Cognitive impairment was detected in several tasks following consumption of the low dose of alcohol but not aspartame or placebo treatments (Stokes et al., 1991).	**3**	**4**	EFSA actually made several edits to make the Ajinomoto-sponsored review text more concise. Some sentences are still word-for-word (the exact same). It must be so much easier to draft a long review when you can start with an industry-funded review and make a few edits!

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3458 - 3467	The follow up study (Stokes et al., 1994) was undertaken in 12 subjects (college students, sex not reported) received placebo capsules or aspartame capsules (50 mg/kg bw/day) for 9 days, or an acute dose of ethyl alcohol to achieve 0.1% blood ethanol levels as described above. All participants received the placebo and ethanol treatments once and the aspartame treatment twice with a 7-day period between treatments. On the last day of treatment periods plasma phenylalanine levels averaged 59 µM following placebo treatments and 121.5 µM following aspartame consumption. Forty-seven task variables were measured and significant differences between pre- and post-test results and aspartame treatment were detected for three tasks. However, an improvement, rather than impairment, of function was observed in participants following the aspartame treatments, which the authors described as unexpected and attributed to chance.		Twelve subjects (college students, sex not defined) received placebo capsules or aspartame capsules (50 mg/kg bw/day) for 9 days, or an acute dose of ethyl alcohol to achieve 0.1% blood ethanol levels as described earlier. All participants received the placebo and ethanol treatments once and the aspartame treatment twice with a 7-day interval. Blood phenylalanine and breath alcohol levels were measured. On the last day of treatment periods, when subjects completed the cognitive testing, blood alcohol levels were 0.0% during all treatments except following the alcohol treatment when it averaged 0.09%. Plasma phenylalanine levels averaged 59.08 µmol following placebo treatments and 121.5 µmol following aspartame consumption. Forty-seven task variables were measured and significant differences between pre- and post-test results and aspartame treatment were detected for three tasks. However, unexpectedly, an improvement, rather than impairment, of function was observed in participants following the aspartame treatments.	**1**	**4**	When the EFSA was discussing these studies that do not measure the effects on pilots of aspartame use over months and years, they forgot to mention the Aspartame Pilot Hotline or the publications in aviation journals about aspartame: The Aviation Consumer (1988), Aviation Medical Bulliten (1988), Pacific Flyer (1988), CAA General Aviation (1989), Aviation Safety Digest (1989), General Aviation News (1989), Plane & Pilot (1990), Canadian General Aviation News (1990), National Business Aircraft Association Digest (NBAA Digest 1993), International Council of Air Shows (ICAS 1995), Pacific Flyer (1995). U.S. Air Force's magazine "Flying Safety" (1992) and the U.S. Navy's magazine, "Navy Physiology."
3468 - 3475	In a three-way crossover double-blind study a group of 48 healthy volunteers (24 men, 24 women, ages 18–34 years) received treatments consisting of aspartame, sucrose and placebo administered for 20 days (Spiers et al., 1998). Twenty-four participants received a high dose of aspartame (45 mg/kg bw/day) and the remaining received a low dose of aspartame (15 mg/kg bw/day). Acute effects were evaluated on day 10 of each treatment, with testing starting 90 min after consumption of test material; chronic effects were evaluated on day 20. Plasma phenylalanine levels increased dose dependently with aspartame consumption, but no cognitive, neurophysiologic and behavioural effects were observed	Page 695 Column 2	Cognitive, neurophysiologic and behavioral effects of consuming aspartame for 20 days were evaluated by Spiers et al. (1998) in a group of 48 healthy volunteers (24 men, 24 women, ages 18–34 years). This was a three-way crossover doubleblind study with treatments consisting of aspartame, sucrose and placebo. Twenty-four participants received a high dose of aspartame (45 mg/kg bw/day) and the remaining received 15 mg/kg bw/day. Acute effects were evaluated on day 10 of each treatment arm, with testing starting 90 min after consumption of test material. Chronic effects were evaluated on day 20. Plasma phenylalanine levels increased dose-dependently with aspartame consumption, but no other effects were observed.	**2**	**2**	I guess that EFSA was too busy copying and pasting to read the study and see that the authors picked subjects who had used aspartame without reacting. In the author's words: "In summary, we made a conscious effort to preselect individuals who we felt would be unlikely to experience any effect from chronic aspartame exposure." After months or years, even these subjects will likely experience chronic aspartame poisoning. Please see: http://www.holisticmed.com/aspartame/abuse/ and http://www.holisticmed.com/aspartame/aspfaq.html
3491 - 3492	Overall, the Panel concluded that the weight-of evidence suggested that aspartame ingestion had no effect on behaviour or cognitive function.	Page 696 Column 1	Overall, the weight of the evidence indicates that aspartame has no effect on behavior or cognitive function.	**0**	**1**	What a coincidence! The EFSA panel concluded, nearly word-for-word exactly what the manufacture-funded review concluded! Instant EFSA Classic!

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3494 - 3498	A double-blind study was undertaken in children recently diagnosed with generalised absence seizures or also called petit mal seizures to ascertain whether aspartame would exacerbate the occurrence of such seizures (Camfield et al., 1992). After eating breakfast of their own choice, children (n = 10) drank orange juice sweetened with either aspartame (40 mg/kg bw) or sucrose (1 g sucrose for every 25 mg aspartame) to achieve similar sweetness.	Page 696 Column 1	Children who had been recently diagnosed with generalized absence seizures were enrolled in a double-blind controlled study to ascertain whether aspartame would exacerbate occurrence of absence seizures (also called petite mal seizures) (Camfield et al., 1992). After eating their own choice of breakfast, children (n = 10) drank orange juice sweetened with either aspartame (40 mg/kg bw) or sucrose (1 g sucrose for every 25mg aspartame) to achieve similar sweetness.	**1**	**1**	
3498 - 3507	For six hours following consumption of the juice the number and length of spike-wave bursts, indicative of an absence seizure, were determined using EEG40 recordings. Each child was tested once with each substance, on two consecutive days, treatments were assigned in a random fashion. No information was provided regarding whether lunch or snacks were given. There were no significant differences in either the frequency or duration of spike-wave bursts; however, when the two factors were combined, the total time spent in spike-wave per hour of observation was significantly higher in children after consumption of aspartame compared with sucrose (Camfield et al., 1992). The Panel noted that combination of the two factors into a single measure was not adequately explained, and lack of control of food and drink intake before and after dosing may have affected the results.	Page 696 Column 2	The number and length of spike-wave bursts, indicative of an absence seizure, were determined using EEG40 recordings for 6 h following consumption of the juice. Each child was tested once with each substance, on two consecutive days, in random fashion. No information was provided regarding whether lunch or snacks were given. There were no significant differences in the frequency or duration of spike-wave bursts; however, when the two factors were combined, the total time spent in spike-wave per observation hour was significantly higher in children on the day aspartame was consumed as compared to when sucrose was consumed (Camfield et al., 1992). The major limitation of this study is the lack of control of food and beverage intake before and after dosing with aspartame or sucrose because fasting and dehydration can affect the susceptibility to seizures (Tollefson and Barnard, 1992).	**2**	**3**	Notice at the end how the EFSA Panel always has something negative to say about independent research that finds health problems caused by aspartame. Also, notice that their negative information is sourced from the aspartame manufacturer-funded review. For honest, scientific information on aspartame and seizures, please see: http://www.holisticmed.com/aspartame/abuse/seizures.html
3510 - 3513	Measurements prior to and following treatments included seizure incidence, overall activity and behaviour, EEG recordings, adverse experiences, liver function, urine analysis, and plasma levels of amino acid, methanol, formate, glucose, and monoamines and metabolites. Children ate their normal diet, but were asked to exclude a list of foods containing aspartame.	Page 696 Column 2	Measurements prior to and following treatments included seizure incidence, overall activity and behavior, EEG recordings, adverse experiences, liver function, urine analysis, and plasma levels of amino acid, methanol, formate, glucose, and monoamines and metabolites. Children were allowed to eat their normal diet, but excluding foods on a list of aspartame-containing products.	**1**	**1**	

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3518 - 3522	The subjects received placebo or 50 mg aspartame /kg bw in three doses throughout the day, on days 2 and 4. EEG recordings were preformed for five consecutive days. Meals were standardised throughout treatment. No clinical seizures were observed in subjects during the study. Electrographic seizures were recorded in two subjects on days consuming the placebo. Sleep variables were also measured, but no effect of aspartame was observed.	Page 696 Column 2	In this study, subjects received 50 mg/kg bw aspartame or an identical placebo in three divided doses throughout the day, on days 2 and 4. EEG recordings were preformed for 5 consecutive days. All meals were uniformly standardized on treatment days. No clinical seizures were observed in subjects during the study. Electrographic seizures were recorded in 2 subjects on days consuming the placebo. Sleep variables were also measured, but no effect of aspartame was observed.	**4**	**2**	The EFSA discussed manufacturer-sponsored "research" related to aspartame and seizures, but neglected to mention that the subjects were on anti-seizure medication during the short studies! They appear to be too busy copying text to be bothered to actually read the research! For honest, scientific information on aspartame and seizures, please see: http://www.holisticmed.com/aspartame/abuse/seizures.html
3525 - 3528	The possible effect of aspartame on headaches has been investigated in various studies, which reported conflicting results. Some reported no effect and others suggested that a small subset of the population may be susceptible to aspartame-induced headaches. The number of existing studies was small, and several had high participant dropout rates, making interpretation of results difficult.	Page 694 Column 1	Studies designed to evaluate the possible effect of aspartame on headaches have reported conflicting results, with some reporting no effect and others suggesting a small subset of the population may be susceptible to aspartame-induced headaches. The number of studies is small and several have high participant dropout rates, making interpretation of results difficult.	**1**	**2**	
3529 - 3535	A double-blind crossover trial (Schiffman et al., 1987) with 40 individuals (12 males, 28 females; ages 19–69) who had previously reported suffering headaches when they consumed aspartame, was a well controlled study with patients being housed and monitored in an inpatient unit. Participants were monitored for 2 days, and then challenged with capsules of aspartame (30 mg/kg bw) or placebo (microcrystalline cellulose) on days 3 and 5, with day 4 being a washout day. Diet and extraneous variables were controlled. There was no evidence of an effect of aspartame, as incidence of headache after consumption of aspartame (35%) or after the placebo (45%) was similar (Schiffman et al., 1987).	Page 693 Column 1 and Column 2	A doubleblind crossover trial with 40 individuals (12 males, 28 females; ages 19–69) who had reported having headaches each time they consumed aspartame was a well-controlled study with patients being housed and monitored in an inpatient unit (Schiffman et al., 1987). Participants were monitored for 2 days, and then challenged with capsules of aspartame (30 mg/kg bw) or placebo (microcrystalline cellulose) on days 3 and 5, with day 4 being a washout day. Diet and extraneous variables were controlled. There was no evidence of an effect of aspartame, as incidence of headache after consumption of aspartame (35%) was similar to after the placebo (45%) (Schiffman et al., 1987).	**3**	**1**	The whole paragraph was plagiarized, even the phrase, "well-controlled study!" Ha Ha! This one day study of aspartame was designed in a way that no statistical difference would be seen For honest scientific information on aspartame and headaches, please see: http://www.holisticmed.com/aspartame/abuse/migraine.html

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3536 - 3547	In contrast, the study by Koehler and Glaros (1988) had volunteers who suffered with migraines and stayed in their normal environments during a double-blind crossover study with three phases: a 4-week baseline phase and two four-week experimental phases, with a 1-week washout phase between treatments. Participants (two males, eight females; ages 18 to 47 years) consumed capsules of aspartame (300 mg) or placebo (microcrystalline cellulose) and self-recorded incidence of headaches and dietary information. The incidence of headaches did not differ from baseline during the placebo phase. Five of the eleven participants reported a higher number of migraines during the aspartame phase compared to during the baseline or placebo phases. The mean number of headaches reported was 1.72, 1.55, and 3.55 during the baseline, placebo, and aspartame phases, respectively. No differences were reported in the intensity or duration of migraine headaches. Dietary records did not show any substantial changes in diet among phases. The high dropout rate, from 25 to 11 participants in this study makes interpretation of the results difficult.	Page 693 Column 2	In contrast, the study by Koehler and Glaros (1988) had volunteers who suffered with migraines stay in their normal environments during a double-blind crossover study with three phases: a 4-week baseline phase, two 4-week experimental phases, and a 1-week washout phase between treatments. Participants (n =11; 2 males, 8 females; ages 18 to 47 years) consumed capsules of aspartame (300 mg) or placebo (microcrystalline cellulose) and self-recorded headaches and diets. The incidence of headaches did not differ from baseline during the placebo phase. Five of the 11 participants reported a higher number of migraines during the aspartame phase as compared to during the baseline or placebo phases. The mean number of headaches reported was 1.72, 1.55, and 3.55 during the baseline, placebo, and aspartame phases, respectively. No differences were reported in the intensity or duration of migraine headaches. Dietary records did not show any substantial changes in diet among phases. One concern is the small number of individuals and high dropout rate from 25 down to 11 participants in this study.	**5**	**3**	Independent study finds that aspartame can cause migraines. EFSA lifts and rewords the criticism from the aspartame manufacturer review. For honest scientific information on aspartame and headaches, please see: http://www.holisticmed.com/aspartame/abuse/migraine.html
3548 - 3554	Patients at a headache unit were asked to complete a survey reflecting whether they felt that alcohol, aspartame or carbohydrate intake were triggers of headaches (Lipton et al., 1989). Of the 171 patients who completed the survey, 8.3% reported aspartame as a trigger of headaches, and often a migraine headache. As this was significantly higher than the response to carbohydrates (2.3%), the authors concluded that aspartame might be a migraine headache trigger for some individuals. The Panel considered 'the power of suggestion' of having aspartame listed as a possible trigger of headaches to be a major limitation of this study.	Page 693 Column 2	Patients at a headache unit (n = 190) were asked to complete a survey regarding whether they felt that alcohol, aspartame or carbohydrate intake were triggers of headaches (Lipton et al., 1989). The limitation of this study is the power of suggestion by having aspartame listed as a possible trigger and then asking for a response. Of the 171 patients who completed the survey, 8.3% reported aspartame as a trigger of headaches, and often a migraine headache. As this was significantly higher than the response to carbohydrates (2.3%), the authors concluded that aspartame might be a migraine headache trigger for some individuals.	**3**	**1**	The EFSA copies the paragraph and then rewords (and places in a different location) the criticism from the aspartame manufacturer review. The researchers had a control in the study to address the "power of suggestion" criticism. For honest, scientific information about headaches, please see: http://www.holisticmed.com/aspartame/abuse/migraine.html

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3555 - 3567	Van den Eeden et al. (1994) conducted a double-blind randomised crossover trial with 32 subjects self-diagnosed as sensitive to aspartame. Only 18 participants completed the full protocol, as other subjects withdrew for various reasons including adverse effects. Subjects took capsules containing either aspartame or placebo (microcrystalline cellulose) three times a day to achieve a dose of 30 mg/kg bw/day for seven days. A significantly higher (p = 0.04) occurrence of self-reported headaches was recorded following exposure to aspartame (33% of days) compared to placebo (24% of days). The subjects who had excess headaches following aspartame dosing were those who had, at the beginning of the study, indicated they were 'very sure' that they were susceptible to aspartame-induced headaches. In contrast, those subjects who classified themselves as 'somewhat or not sure' reported similar headache incidence during aspartame and placebo exposure periods. The authors conclude that these results indicated that a small subset of the population was susceptible to aspartame-induced headaches (Van den Eeden et al., 1994). The Panel consider that with such a low number of participants it was not possible to draw a conclusion.	Page 693 Column 2 - 694 Column 1	In the most recent study to assess whether the consumption of aspartame is associated with headaches, Van den Eeden et al. (1994) conducted a double-blind randomized crossover trial with 32 subjects who self-reported sensitivity to aspartame. Only 18 participants completed the full protocol, as other subjects withdrew for various reasons including adverse effects. Subjects took capsules containing either aspartame or placebo (microcrystalline cellulose) 3 times a day to achieve a dose of 30 mg/kg bw/day for 7 days. A significantly higher (p = .04) occurrence of self-reported headaches was reported following exposure to aspartame (33% of days) as compared to placebo (24% of days). The subjects who had excess headaches following aspartame dosing were those who had, at the beginning of the study, indicated they were "very sure" that they were susceptible to aspartame-induced headaches. In contrast, those subjects who classified themselves as "somewhat or not sure" reported similar headache incidence during aspartame and placebo exposure periods. The authors conclude that these results indicate that a small subset of the population are susceptible to aspartame-induced headaches (Van den Eeden et al., 1994).	**6**	**1**	The study authors did not conclude that "a small subset of the population are susceptible to aspartame-induced headaches" as the aspartame manufacturer-funded review claimed. The authors did not use the term, "small." Amazingly, the EFSA criticized the study for the small number of participants even though it had more participants than aspartame manufacturer studies that they never criticize. For honest, scientific information about headaches, please see: http://www.holisticmed.com/aspartame/abuse/migraine.html
3570 - 3574	Drewnowski et al. (1994) fed 12 obese and 12 lean women one of four breakfast preloads sweetened with 50 g sucrose or 500 mg aspartame, or aspartame plus 50 g maltodextrin, in a cross-over design. As such all subjects were tested with all treatments. Subsequent food intake and calorie consumption during lunch, snack, and dinner was recorded and were reported not to be affected by the sweetener consumed in the preload (Drewnowski et al., 1994).	Page 697 Column 1	In one of the well-conducted studies, Drewnowski et al. (1994) fed 12 obese and 12 lean women one of four breakfast preloads sweetened with 50 g sucrose, 500 mg aspartame, or aspartame plus 50 g maltodextrin. All subjects were tested with all treatments. Subsequent food intake and calorie consumption during lunch, snack, and dinner were not affected by the sweetener consumed in the preload (Drewnowski et al., 1994).	**1**	**2**	At least the EFSA did not plagiarize "well-conducted studies" as they did in line 3529 (see above).

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3575-3580	A meta-analysis of 16 randomised controlled trials assessing the effect of aspartame consumption on energy intake with observations on weight loss and weight maintenance was undertaken by de la Hunty et al. (2006). The studies that have addressed the question of the effect of aspartame on appetite and body weight, that have actually measured food consumption, have shown that aspartame does not increase caloric intake. Significant reduction of energy intake with consumption of aspartame was observed, except when the control was a non-sucrose control such as water (de la Hunty et al., 2006).	Page 697 Column 2	Recently, a meta-analysis of 16 randomized controlled trials assessing the effect of aspartame consumption on weight loss, weight maintenance and energy intake was conducted (de la Hunty et al., 2006). The studies that have addressed the question of the effect of aspartame on appetite and body weight, that have actually measured food consumption, have shown that aspartame does not increase caloric intake. In contrast, significant reduction of energy intake with consumption of aspartame compared to other controls was observed, except when the control was a nonsucrose control such as water.	**1**	**2**	EFSA made a bit of an effort to rearrange a few words. Good work!
3586 - 3617	3.2.7.8. Allergenicity of aspartame …. (See Notes section to the right.)	See Notes section to the right.				This whole section appears to be lifted directly from the EFSA "Report of the Meetings on Aspartame With National Experts (Question Number: EFSA-Q-2009-00488) (2009) (Pages 26-27 and part from near the bottom of page 48.) The EFSA appears to be plagiarizing material written by other authors but published by EFSA. It might not be so bad except that on Pages 25-26 of their 2009 document they list the source material for their discussion of aspartame and "allergies." The sources include one long review written by the aspartame manufacturer (Butchko 2002), one long review funded by the aspartame manufacturer (Magnuson 2007) and only two case history reports. They did not include independent research and other published case history reports. See the 2009 EFSA document at: http://www.feingold.org/Research/PDFstudies/EFSAaspartame.pdf or http://www.efsa.europa.eu/en/supporting/doc/1641.pdf

Did the EFSA Plagairize the Aspartame Manufacturer Review?

EFSA Draft Lines	EFSA Draft Text (Copied From Aspartame manufacturer-funded review) http://www.efsa.europa.eu/sites/default/files/consultation/130108.pdf	Aspartame Manufacturer Review Lines	Aspartame Manufacturer-Funded Review Text (Critical Reviews in Toxicology, 37:8, 629-727, 2007) (See: www.holisticmed.com/aspartame/burdock/)	Sentences Exactly the Same	Sentences Nearly the Same	Notes
4987 - 4990	Olney and co-workers (Olney and Sharpe, 1969; Olney et al., 1972) also reported neuronal necrosis in neonatal nonhuman primates administered large bolus doses of glutamate (1000-4000 mg/kg bw subcutaneously or orally. This observation, however, could not be reproduced by a number of other scientists with either glutamate or aspartame at high dosages (reviewed by Butchko et al., 2002).	See Notes section to the right.	Olney and co-workers (Olney and Sharpe, 1969; Olney et al., 1972) also reported this phenomenon in neonatal nonhuman primates administered large bolus doses of glutamate. This observation, however, could not be reproduced by a number of other scientists with either glutamate or aspartame at high dosages	**1**	**1**	This section was copied and pasted from page S26 of the aspartame manufacturer review by Butchko (Reg Tox & Pharm, 35:S1-S93, 2002). If the EFSA had done event the slightest bit of reading about aspartame, they would be aware that the industry research they are citing involved giving brain-protecting drugs to the test animals in a study, recropping a image from one species of monkey and putting it in another study to represent another species, etc.