

Statement on A2 Milk's Continued Use of Misleading Science Claims

The National Milk Producers Federation is disappointed, but hardly surprised, by the A2 Milk Company's reaction to the National Advertising Division's referral of our false-marketing claims complaint against them to the Federal Trade Commission. After refusing to cooperate with the NAD, the A2 Milk Company then trotted out the same thin science to justify their deceptive practices after the NAD announced its referral on Oct. 23. This is similar to their reactions when authorities in other countries also called into question the purported benefits of its product. Eventually, A2 backed down in those cases. That is also happening in the United States. Despite its bluster, A2 quietly removed some of its marketing claims after U.S. dairy cooperatives called them on those claims.

- The "Lactose Intolerance: Fact or Fiction" page on its website is gone.
- Its claim that regular milk can cause "painful bloating, wind, [and] cramps" is also gone
- So is its claim that with a2 milk, a consumer feels "no pain, no gas, no running to the restroom"

A2 is running scared in the wake of the NAD decision, [detailed here](#). NMPF urges the FTC to take on A2's misleading claims and compel them to end once and for all.

In the meantime, we want to set the record straight on A2's junk science, specifically three studies they continue to peddle as "proof" of their product's effects.

Three Frequently A2 Referenced Human Clinical Trials

Study 1: Comparative effects of A1 versus A2 beta-casein on gastrointestinal measures: a blinded randomized cross-over pilot study. European Journal of Nutrition (2014).

The study focused on the digestive symptoms of conventional milk versus A2 milk in human adults, with participants consuming approximately 3 cups of milk (750 ml) per day for two-weeks. The main outcomes examined in this study, which didn't control for what other foods participants were eating during their participation, were several subjective measures of gastrointestinal symptoms and stool consistency. No differences were observed between conditions, except for higher stool consistency (i.e., softer stool) with conventional milk. There were no significant mean differences in an indicator of gut inflammation between conventional and A2 milk.

Despite these largely neutral findings for inflammation in the A2-funded study, the authors made several attempts to explain a potential negative effect of conventional milk and a favorable effect of A2 milk by examining individual responses of some outliers; however, a close examination of these individual results reveals no consistent effect of higher or lower inflammation with either condition. In fact, there are some cases in which levels of inflammation are considerably higher in the A2 condition.

Study 2: Effects of milk containing only A2 beta casein versus milk containing both A1 and A2 beta casein proteins on gastrointestinal physiology, symptoms of discomfort, and cognitive behavior of people with self-reported intolerance to traditional cows' milk. Nutrition Journal (2016).

Like the first study, measurements included subjective measures of digestive symptoms and physiological markers of inflammation. Like the first study, dietary controls were lacking, and the period of study was short. Subjects consumed approximately 2 cups of milk (500 ml) per day after meals for

two weeks. The authors reported conventional milk was associated with worsening of digestive symptoms relative to baseline in lactose tolerant and lactose intolerant subjects and that consumption of A2 milk did not aggravate these symptoms relative to baseline in lactose tolerant and intolerant subjects. Overall, physiological markers of inflammation were generally lower with the A2 milk vs. conventional milk.

Other aspects of this publication raise questions. First, the data presentation is not consistent with standard clinical trials of the same design and is therefore very difficult to interpret. Also, the authors' main hypothesis is that conventional milk releases the peptide BCM-7 when it is digested, which is theorized to result in digestive symptoms. Curiously, there were no data for BCM-7 reported in the publication even though the study abstract indicates there are such data. A close examination of the paper reveals no data were presented in the results section or data tables. The authors would need to report these data to be evaluated to draw conclusions.

Study 3: Effects of cow's milk beta-casein variants on symptoms of milk intolerance in Chinese adults: a multi-center, randomized controlled study. Nutrition Journal (2017).

This study examined the acute effects of consuming conventional milk vs. A2 milk (300 ml or about 1 ¼ cups) on subjective feelings of gastrointestinal discomfort at 1, 3, and 12 hours after consumption. In this design, a meal was provided to subjects 1-hour after milk consumption, although the type of meal varied according to study site. The authors report that digestive symptoms were significantly lower after consuming A2 milk versus conventional milk in both lactose absorbers and lactose malabsorbers.

As with previous studies, there are several limitations in terms of the design that make drawing conclusions difficult. This new study doesn't remedy the significant criticism of the previous studies, which was that they were short and did not allow for adaptation to the intervention. Rather, this was simply a measurement of an acute response during a single day. Like the previous studies, problems around lack of dietary control, short duration and lack of dosing adaptation for lactose intolerant individuals all apply. Additionally, the 1-hour post-milk consumption test meal was inconsistent across the three study sites, and there was no nutritional detail of the meal reported (macronutrients, fiber content, etc.), nor information regarding whether the test meals were individualized based on caloric needs.

The three human studies examining effect of consuming A2 milk versus conventional milk on digestive symptoms do not provide sufficient scientific support for strong conclusions. Most importantly, all three suffer from a lack of dietary control, which is critically important for studies examining digestive symptoms. There were also no non-milk control conditions in any of the three studies. Furthermore, the studies were short duration (1 day or 14 days) and do not allow for potential adaptation with gradual increasing dose as has been demonstrated in studies on lactose intolerance.

Sufficient scientific support does not exist for the proposed mechanisms and beneficial effects of A2 milk versus conventional milk. The human studies available have significant design limitations and do not provide strong evidence for a benefit for A2 milk over conventional milk for symptoms of digestive discomfort. Given the current state of the science, consumers have no reason to suspect that A2 milk differs in any meaningful way from conventional milk, as both would provide the same health benefits.