

a2플래티넘 4단계 TVC
광고심의자료

2021.01.08

1. no팜유, no덱스트린, no유화제

한글표시사항

제품명	a2 플래티넘 4			
식품유형	음료베이스(분말)	내용량	900 g(3,942 kcal)	원산지 뉴질랜드
제조사	Synlait Milk Limited 1028 Heslerton Road, Rakaia, Canterbury, New Zealand Packed for A2 Infant Nutrition Limited Level 10, 51 Shortland Street, Auckland, 1010, New Zealand			
원재료명 및 함량	우유(a2 밀크™) 52%, 탈지유, 유당, 갈락토올리고당, 천연바닐라향, DHA분말, 레시틴, 제이인산마그네슘, 글루콘산아연, 피로인산제이철, 아스코브산나트륨, dl-α-토코페릴아세테이트, 나이아신아미드, 분말비타민A, 비타민D3, 비타민B1염산염, 비타민B6염산염, 엽산 우유, 대두 함유			
수입판매원	(주)유한건강생활 서울특별시 동작구 노랑진로 74			
유통기한	캔 밀면 표기일까지 (USE BY는 유통기한, MFD는 제조일자, 일자는 일/월/년 순으로 표기)			
포장재질	용기(철), 뚜껑(LDPE), 속뚜껑(알미늄), 스푼(폴리프로필렌)			
보존기준	실온보관			
살균방법	72°C 이상에서 15초 이상 살균			
섭취량 및 섭취방법	200 ml 물에 4스푼(36 g)을 넣고 잘 흔들어 섭취하십시오. * 스푼이 동봉되어 있습니다. 3세 이상 1일 1~2회 섭취하십시오.			
반품 및 교환처	구입처 및 수입판매원			
주의사항	<ul style="list-style-type: none"> • 3세 이상 어린이에게 적합한 제품입니다. • 알루미늄 포장지가 손상되거나 없는 경우에는 사용하지 마십시오. • 사용 전 캔 밀면에 있는 유통기한과 제품의 상태를 확인한 후 사용하십시오. • 속뚜껑 개봉 시 날카로운 절단부에 다치지 않도록 주의하시고, 개봉 후 즉시 버리십시오. • 개봉 후에는 4주 이내에 사용해 주시고, 습기, 벌레, 이물질 등 불순물이 들어가지 않게 뚜껑을 잘 닫아 직사광선을 피하여 서늘한 곳에 보관하십시오. 			
기타	※ 부정·불량식품 신고는 국번없이 1399 ※ 본 제품은 공정거래위원회 고시 소비자분쟁 해결기준에 의거 교환 또는 보상 받을 수 있습니다. ※ 파손된 제품은 교환 또는 환불 받을 수 있습니다. ※ 질소 및 이산화탄소 혼합충전			
고객상담센터	소비자상담실 1670-6527 / 00798-611-0038 www.neworigin.co.kr www.a2nutrition.co.kr			


영양정보		총 내용량 900g(4스푼(36g) x 25회분량)	
		4스푼(36 g)당	163 kcal
4스푼(36g)당	1일 영양성분 기준치에 대한 비율	100 g 당	
나트륨	79 mg 4%	219 mg	11%
탄수화물	16 g 5%	45 g	14%
당류	16 g 16%	44 g	44%
지방	7 g 13%	18 g	33%
트랜스지방	0.4 g	1.1 g	
포화지방	4.5 g 30%	12.5 g	83%
콜레스테롤	19 mg 6%	54 mg	18%
단백질	9 g 16%	24 g	44%
칼슘	277 mg 40%	769 mg	110%
철	3.1 mg 26%	8.6 mg	72%
아연	2.6 mg 31%	7.2 mg	85%
인	245 mg 35%	681 mg	97%
비타민A	130 µgRE 19%	361 µgRE	52%
비타민D	2.0 µg 20%	5.6 µg	56%
비타민E	1.7 mgα-TE 15%	4.7 mgα-TE	43%
비타민B1	0.3 mg 25%	0.8 mg	67%
비타민B2	0.4 mg 29%	1.2 mg	86%
나이아신	1.5 mgNE 10%	4.2 mgNE	28%
비타민B6	0.1 mg 7%	0.4 mg	27%
비타민B12	1.5 µg 63%	4.2 µg	175%
비타민C	16.2 mg 16%	45.0 mg	45%
엽산	50 µg 13%	139 µg	35%
DHA	25 mg	69 mg	
EPA	10 mg	28 mg	
갈락토올리고당	1.6 g	4.4 g	
1일 영양성분 기준치에 대한 비율(%)은 2,000kcal 기준이므로 개인의 필요 열량에 따라 다를 수 있습니다.			

영양정보		총 내용량 900g(4스푼(36g) x 25회분량)
		4스푼(36g)당 163 kcal
4스푼(36g)당	1일 영양성분 기준치에 대한 비율	100g 당
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콜레스테롤	19 mg 6%	54 mg 18%
단백질	9 g 16%	24 g 44%
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비타민A	130 µgRE 19%	361 µgRE 52%
비타민D	2.0 µg 20%	5.6 µg 56%
비타민E	1.7 mgα-TE 15%	4.7 mgα-TE 43%
비타민B1	0.3 mg 25%	0.8 mg 67%
비타민B2	0.4 mg 29%	1.2 mg 86%
나이아신	1.5 mgNE 10%	4.2 mgNE 28%
비타민B6	0.1 mg 7%	0.4 mg 27%
비타민B12	1.5 µg 63%	4.2 µg 175%
비타민C	16.2 mg 16%	45.0 mg 45%
엽산	50 µg 13%	139 µg 35%
DHA	25 mg	69 mg
EPA	10 mg	28 mg
갈락토올리고당	1.6 g	4.4 g



1일 영양성분 기준치에 대한 비율(%)은 2,000kcal 기준이므로 개인의 필요 열량에 따라 다를 수 있습니다.

제품 뒷면에 표기된
원재료명 및 함량 섹션에서
“팜유, 덱스트린, 유화제”가 포함되지 않음

2. no A1단백질



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Microbiology, FACTA & MAS Laboratory 5/352 Macaulay Road Kensington VIC 3031
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QLD Microbiology Laboratory 1-3, 148 Tennyson Memorial Ave Tennyson QLD 4105 P: +61 7 3426 9750
NSW Microbiology Laboratory 3 Gateway Business Park 63-79 Parramatta Rd Silverwater NSW 2128 P: +61 2 8007 7447
WA Microbiology Laboratory 2/26 Ilda Road Canning Vale WA 6155 P: +61 8 9455 9600



LABORATORY REPORT

on

LIQUID MILK

Date: 14/05/2018

Our Ref: DTS18044820

Report No: 2626907

Final

FOR: THE A2 MILK COMPANY (AUSTRALIA) PTY LTD KYABRAM VIC

28-34 Dunn Street
Smeaton Grange NSW 2567

Dianne Nardella

Date received: 14/05/2018

Origin:

Code/Ref: Samples Submitted by Kyvalley Dairy Group

Order Number:

Package Type:

Temperature on receipt: 4°C

TEST	RESULTS	METHOD	TEST DATE
14MAY18/11186938			
Client ID: DTS1213955 - 1.0 A2 Export Reduced Fat 1L Tank 1 UB 4 JUN; P27			
A1 beta-casein level	Not Detected	A2MK 01 04.14	14/05/2018
14MAY18/11186940			
Client ID: DTS1213956 - 1.0 A2 Export Full Cream 1L Tank 4 UB 4 JUN; P37			
A1 beta-casein level	Not Detected	A2MK 01 04.14	14/05/2018

호주 식품안전보장 기관인 DTS의 검사를 통해
A1단백질이 검출되지 않음이 확인되었음

3. 변형된 A1단백질

3. Beta-Caseins and BCM-7

Beta-casein proteins make up approximately 30% of the total protein of cows' milk [2] and may be present as one of two major genetic variants: A1 and A2 [3]. A2 beta-casein is recognized as the original beta-casein variant because it existed before a proline⁶⁷ to histidine⁶⁷ point mutation caused the appearance of A1 beta-casein in some European herds some 5000–10,000 years ago [4]. Once milk

젖소 우유의 베타카제인은 A1과 A2 2개의 단백질 타입이 있다. 5000년~1만년전 모든 소들은 본래 A2베타카제인 단백질을 갖고 태어나지만, **환경 변화로 인해 변형된 A1단백질이 생겨나게 됐다.**

4-1. 내 몸의 단백질과 같은 A2단백질 100%

Some studies have not found an association between early exposure to cows' milk and type 1 diabetes.^{31,35-37} This may be explained in part by the interaction between early exposure to cows' milk-based infant formula and other environmental influencing factors. For example, enterovirus infection is commonly cited as being involved in type 1 diabetes, but it may be the combination of enterovirus infection and early exposure to cows' milk that is important in determining progression to type 1 diabetes-associated autoimmunity.³⁸ Using regression analysis, Lempainen *et al.*³⁸ reported a combined effect of enterovirus infection before 12 months of age and early exposure to cows' milk infant formula (before 3 months) on type 1 diabetes-associated autoantibodies in the Finnish Diabetes Prediction and Prevention Study. Furthermore, differences in cows' milk proteins, and consequently infant formula protein composition, could influence the findings associated with cows' milk protein consumption and type 1 diabetes risk.⁴ The magnitude/amount of cows' milk protein exposure represents another influencing factor as demonstrated in a Finnish case-control study, where children with type 1 diabetes ($n=33$) had a greater likelihood of high milk consumption (>540 ml milk per day) (odds ratio 5.37, 95% confidence interval 1.6-18.4) compared with control children consuming <540 ml milk per day ($n=254$).²⁹

Introducing cereal foods before ~3 months of age is also associated with early β -cell autoimmunity,³¹ and the cereal protein gluten has diabetogenic effects in rodents.³⁹ Although the practical implications of cereals in infant feeding may be limited because infant feeding guidelines in developed countries do not recommend such early introduction to cereals, Norris *et al.*³¹ suggested that early (<4 months old) and late (≥ 7 months old) exposure to cereals was associated with increased risk of β -cell autoimmunity.³¹ This idea of an opportune 'window' for certain food introduction has received attention in terms of the best time to introduce allergens to minimise allergy development in at-risk infants.⁴⁰

Recent evidence from Lamb *et al.*⁴¹ indicates that cows' milk protein may influence the entire type 1 diabetes disease process. In this prospective Diabetes Autoimmune Study in the Young (DAISY) childhood cows' milk protein was associated with islet autoimmunity in children with low/moderate genetic risk of type 1 diabetes, but not with high genetic risk. However, once islet autoimmunity was established cows' milk protein was associated with an increased risk of progression to type 1 diabetes independent of the underlying genetic risk. The timing of introduction of cows' milk protein was only significant at a very early age for the development of islet autoimmunity. The authors' conclude that cows' milk may be diabetogenic when consumed throughout childhood, and may impact both early and later stages of T1D development.

Milk, beta-casein and type 1 diabetes
JSJ Chia *et al*

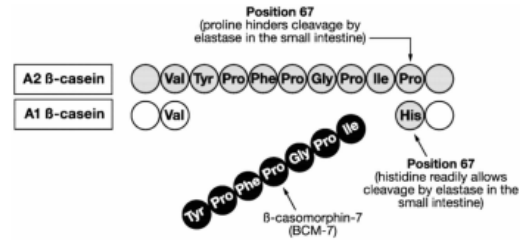


Figure 2. Structures of A1 and A2 β -casein. Adapted from Pal *et al.*⁴⁴

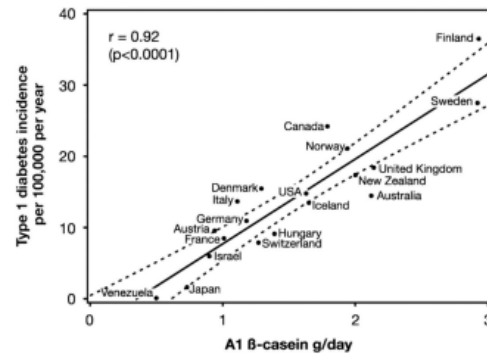


Figure 3. Correlation between A1 β -casein supply per capita in 1990 and type 1 diabetes incidence (1990-1994) in children aged 0-14 years in 19 countries. ($r=0.92$; 95% confidence interval: 0.72-0.97; $P<0.0001$). Dotted lines are the 95% confidence limits of the regression line. Reproduced with permission from R Elliott and The New Zealand Medical Journal (2003).⁴²

protein A1 β -casein have been implicated as dietary antigens in type 1 diabetes, it is of note that gluten also releases the 7-amino-acid opioid peptide gliadorphin-7.⁴⁸ Gluten has also been associated with increased T-cell reactivity in some patients with newly diagnosed type 1 diabetes.⁴⁹

The proline residue at position 67 in A2 β -casein minimises the likelihood of cleavage. Notably, human breast milk β -casein contains a proline in the homologous position as bovine A2 β -casein protein, so human β -casein is of the A2 type.⁵⁰ Thus breastfeeding during early infancy eliminates early exposure to A1 β -casein, although BCM-7 derived from dietary bovine A1

Notably, human breast milk β -casein contains a proline in the homologous position as bovine A2 β -casein protein, so human β -casein is of the A2 type.⁵

특히 모유의 베타카제인은 젖소의 A2 베타카제인과 상응하는 구조의 프롤린을 포함한다. 그러므로, 사람의 베타카제인은 A2 타입이다.

4-2. 내 몸의 단백질과 같은 A2단백질 100%

▪ a2플래티넘 4단계 성분 및 원료 배합비율표 최종

원재료명	함량 (%)
a2 밀크™	52.7817%
a2 밀크™ 탈지유	24.7920%
a2 밀크™ 유당	13.1752%
갈락토올리고당	7.2611%
천연바닐라향	0.7098%
DHA분말	0.5987%
레시틴	0.2954%
제이인산마그네슘	0.2461%
글루콘산아연	0.0384%
피로인산제이철	0.0354%
아스코브산나트륨	0.0354%
dl-α-토코페릴아세테이트	0.0166%
나이아신아미드	0.0046%
분말비타민A	0.0044%
비타민D3	0.0030%
비타민B1염산염	0.0015%
비타민B6염산염	0.0005%
엽산	0.0002%
Total	100.0000%

좌측 원료 배합비율표에 의하면
A1단백질을 포함하는 성분 및 원료 등이
없음을 확인할 수 있음

****A2단백질 90.7%:**
a2밀크™ 52.9%, a2밀크™ 탈지유 24.8%, a2밀크™ 유당13.1%

5. 두뇌발달성분까지 2배

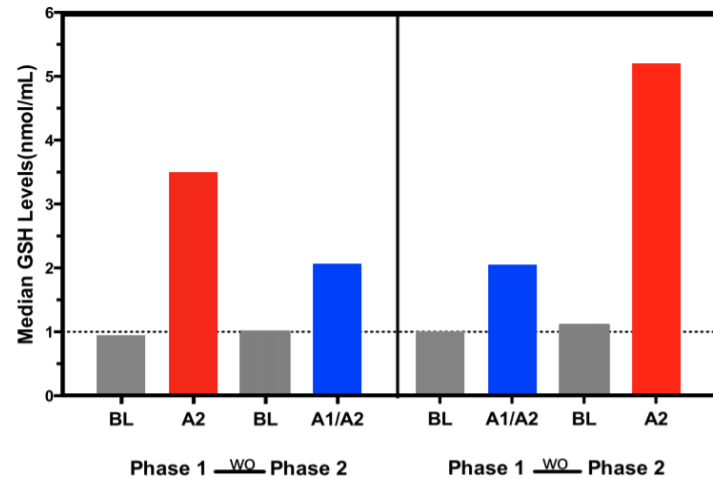


Fig. 2 Median glutathione (GSH) concentrations before and after consumption of milk. Participants consumed milk containing both the A1 and A2 types, or only the A2 type of β -casein. Data are shown for the first of two repeated measures in each participant; the results of both measures were similar. The phase difference was statistically significant ($P = 0.0059$) but the cross-over difference was not significant ($P = 0.7615$) (Wilcoxon two-sample test). A1 = milk containing A1 and A2 β -casein; A2 = milk containing only A2 β -casein; BL = baseline; WO = washout

A1단백질/A2단백질이 혼합된 우유 대비
A2단백질만 있는 분유가
두뇌발달에 영향을 끼치는 “글루타치온” 성분이
2배 가량 많음을 확인할 수 있음

6. 글루타치온-두뇌발달 사이의 상관관계

ABSTRACT

Background: A reduction in key antioxidants such as glutathione has been noted in brain tissue undergoing oxidative stress in aging and neurodegeneration. To date, no dietary factor has been linked to a higher glutathione concentration. However, in an earlier pilot study, we showed evidence of a positive association between cerebral glutathione and dairy intake.

Objective: We tested the hypothesis that dairy food consumption is associated with cerebral glutathione concentrations in older adults.

Design: In this observational study, we measured cerebral glutathione concentrations in 60 healthy subjects (mean \pm SD age: 68.7 \pm 6.2 y) whose routine dairy intakes varied. Glutathione concentrations were measured by using a unique, noninvasive magnetic resonance chemical shift imaging technique at 3 T and compared with dairy intakes reported in 7-d food records.

Results: Glutathione concentrations in the frontal [Spearman's rank-order correlation (r_s) = 0.39, P = 0.013], parietal (r_s = 0.50, P = 0.001), and frontoparietal regions (r_s = 0.47, P = 0.003) were correlated with average daily dairy servings. In particular, glutathione concentrations in all 3 regions were positively correlated with milk servings ($P \leq 0.013$), and those in the parietal region were also correlated with cheese servings (P = 0.015) and calcium intake (P = 0.039). Dairy intake was related to sex, fat-free mass, and daily intakes of energy, protein, and carbohydrates. However, when these factors were controlled through a partial correlation, correlations between glutathione concentrations and dairy and milk servings remained significant.

Conclusions: Higher cerebral glutathione concentrations were associated with greater dairy consumption in older adults. One possible explanation for this association is that dairy foods may serve as

tracellular production of ROS is greatly elevated in many neurodegenerative diseases in conjunction with inflammation and mitochondrial dysfunction (5–7). Accordingly, oxidative stress has been implicated in normal aging and many neurodegenerative diseases.

Glutathione is a powerful antioxidant that plays a key role in the brain's capacity for scavenging ROS and free radicals involved in oxidative stress. Protection against oxidative stress is directly afforded through the oxidation of glutathione in mitochondria (8, 9), and decreased concentrations of glutathione were reported in the aging rat brain (10–12). A significantly elevated ROS generation and cellular stress because of mitochondrial dysfunction and inflammation commonly accompany neurodegenerative disorders, and the resulting burden on the brain's antioxidant defenses would likewise be reflected by reductions in concentrations of glutathione (13–15). However, the measurement of glutathione in living tissue is technically challenging, particularly in the human brain, and until recently, the level of oxidative stress could be evaluated only indirectly by using in vitro measures obtained from blood, cerebrospinal fluid, or biopsy tissue samples. By contrast, the work reported here was based on our successful application of magnetic resonance (MR) chemical shift imaging (CSI) to provide regional mapping of in vivo glutathione concentrations in the living human brain (16–18).

Dietary factors may influence the antioxidant capacity of the brain because dietary supplementation was reported to modulate enzyme activities of antioxidants in animal brains (19, 20).

¹ From the Hoglund Brain Imaging Center (I-YC, PL, and JAL) and the

글루타치온은 강력한 산화 방지제로서,
산화적 스트레스에 관여하는 활성산소와
활성산소를 청소하는 **뇌의 능력에 중요한 역할을 한다.**