景岳生物科技(股)公司

功能性益生菌領導品牌

GenMont Biotech Incorporation

The leading brand of functional probiotics

葉天佑 協理

Alvin Yeh Senior Manager

益生菌市場前景

Probiotics market outlook

- **全球益生菌市場正在快速成長**

  The global probiotics market is growing rapidly.

  其成長的主要原因是消費者對於預防保健的意識提升

  The major factor boosting the market growth is increasing concerns of consumers on preventive healthcare.

- **後疫情時代來臨，人們更積極尋求維持健康的方法**

  Beyond the pandemic, people have more conscious about their health and are taking proactive measures to help maintain their well-being.

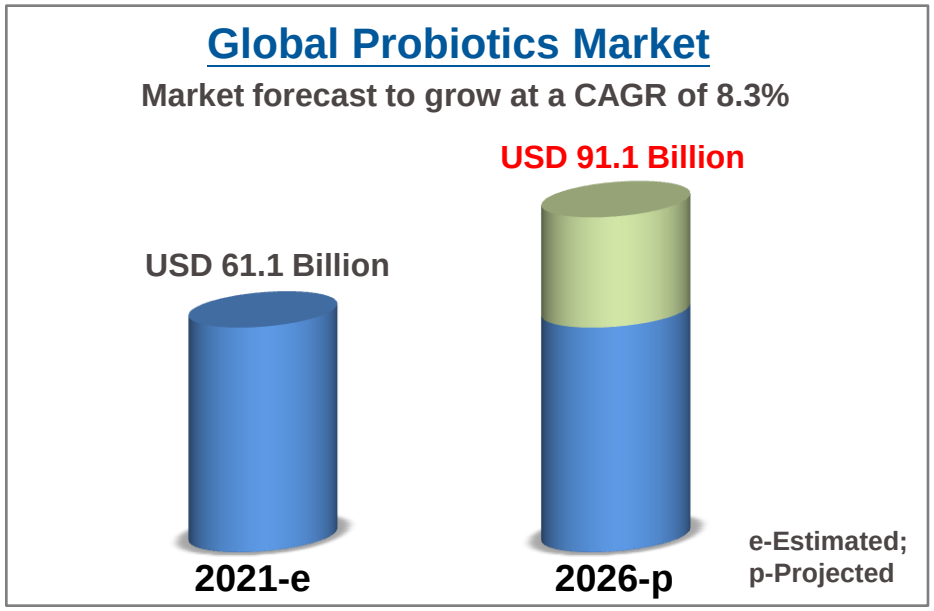
資料來源： Functional Food & Beverage 2020 Report Highlights from the Hartman Group

全球益生菌市場快速發展

Global probiotics market is growing rapidly

全球市場分析報告顯示，預計2026年全球益生菌市場約為911億美元。

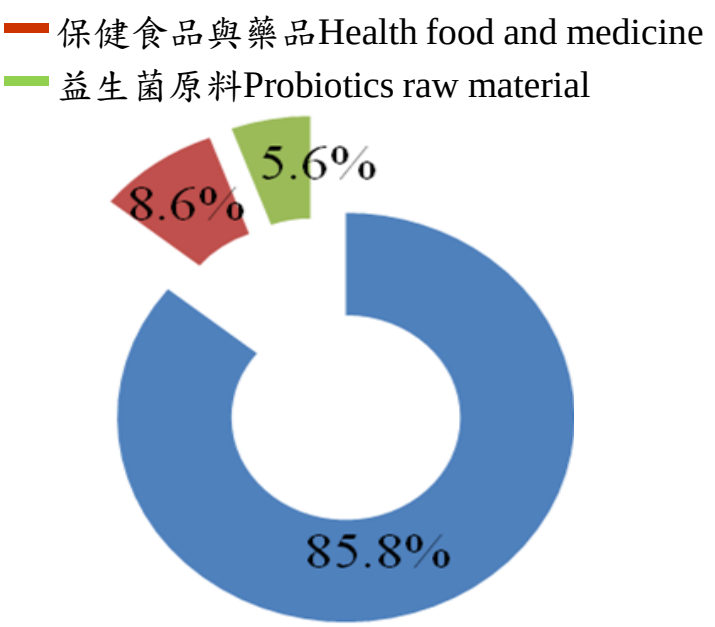
The global market analysis report shows that the global probiotic market is expected to be about 91.1 billion US dollars in 2026.



全球益生菌市場應用

Global probiotics market by product application

*市場分布 MARKET SHARE



資料來源：

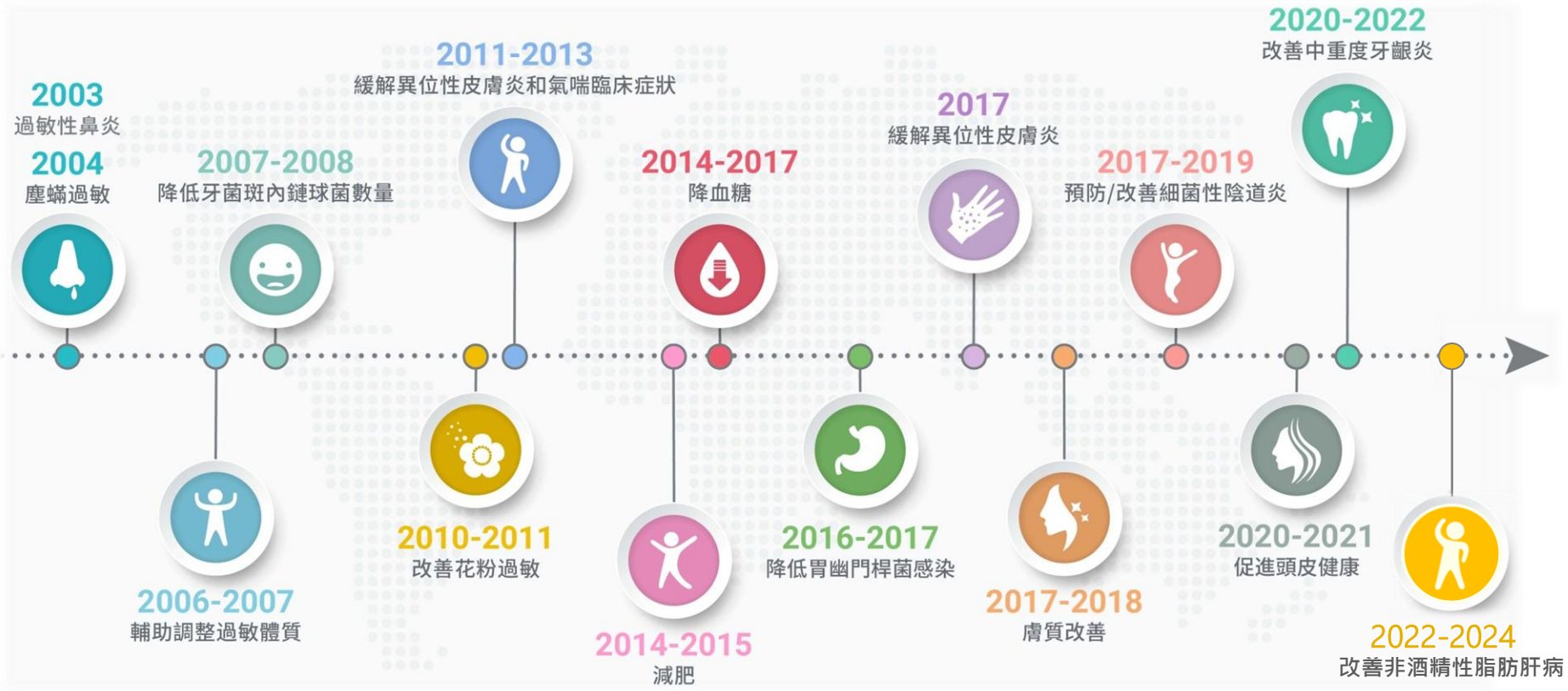
https://www.reportlinker.com/p05129364/Probiotics-Market-by-Application-Dietary-Supplements-Animal-Feed-Source-Form-End-User-And-Region-Forecast-to.html?utm_source=GNW;
https://www.researchandmarkets.com/reports/5370159/global-probiotics-market-by-application

2022年度研究成果介紹

The Annual Research Presentation 2022

景岳生技｜人體臨床試驗 Clinical trials

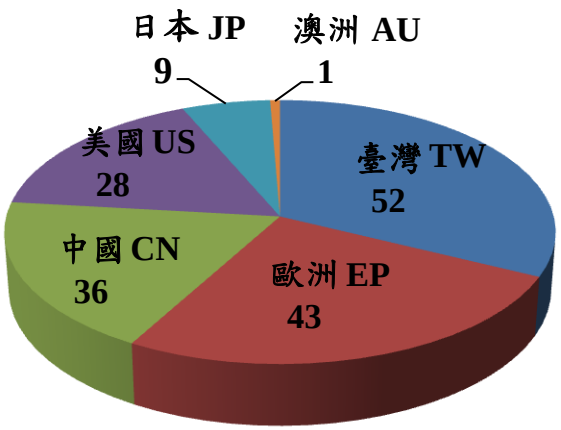
國內外大規模人體實驗累積近1600人數



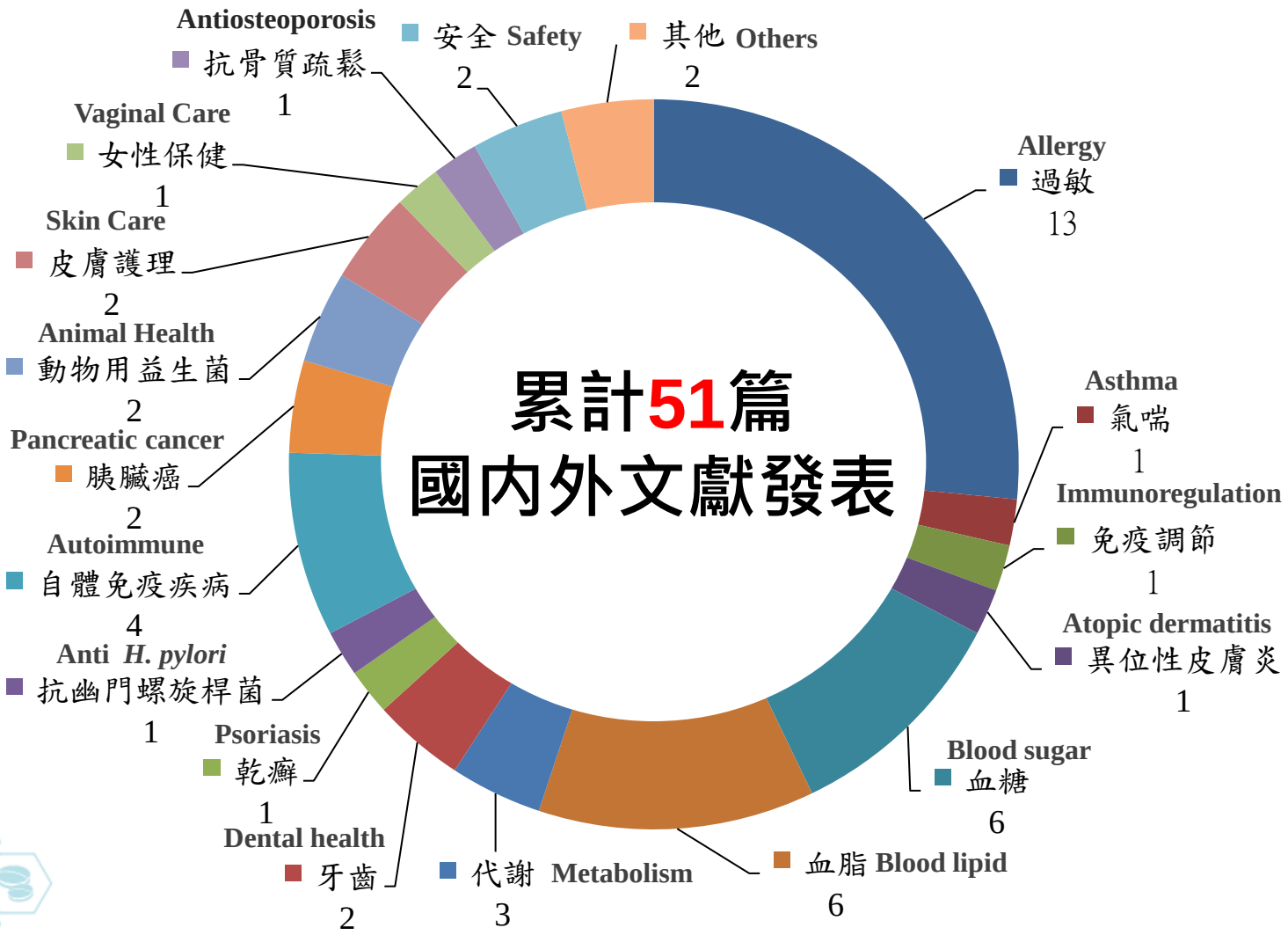
Accumulated nearly 1600 subjects

2003 Perennial allergic rhinitis
2004 Dust mite allergy
2006-2007 Assisted adjusting allergies
2007-2008 Dental health
2010-2011 Pollen allergy
2011-2013 Atopic Dermatitis & Asthma
2014-2015 Obesity
2014-2017 Type 2 diabetes
2016-2017 *H. pylori* infection
2017 Atopic Dermatitis
2017-2018 Skin Problems
2017-2019 Bacterial Vaginosis
2020-2021 Hair & Scalp problem
2020-2022 Gingivitis
2022-2024 Non-Alcoholic Fatty Liver Disease

景岳生技 | 研發專利 Patents



景岳生技｜國際文獻 Published Papers



乳酸桿菌功效研究

Efficacy studies of *Lactobacillus*

nutrients MDPI

Article

Effects of Maternal Gut Microbiota-Targeted Therapy on the Programming of Nonalcoholic Fatty Liver Disease in Dams and Fetuses, Related to a Prenatal High-Fat Diet

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† These authors contributed equally to this work.
‡ These authors contributed equally to this work.

Abstract: Metabolic disorders can start in utero. Maternal transmission of metabolic phenotypes may increase the risks of adverse metabolic outcomes, such as nonalcoholic fatty liver disease (NAFLD); effective intervention is essential to prevent this. The gut microbiome plays a crucial role in fat storage, energy metabolism, and NAFLD. We investigated the therapeutic use of probiotic *Lactobacillus reuteri* and postbiotic butyrate gestation in the prevention of perinatal high-fat diet-induced programmed hepatic steatosis in the offspring of pregnant Sprague–Dawley rats who received regular chow or a high-fat (HF) diet 8 weeks before mating. *L. reuteri* or sodium butyrate was administered via oral gavage to the gestated rats until their sacrifice on day 21 of gestation. Both treatments improved liver steatosis in pregnant dams; *L. reuteri* had a superior effect. *L. reuteri* ameliorated obesity and altered the metabolic profiles of obese gravid dams. Maternal *L. reuteri* therapy prevented maternal HF diet-induced fetal liver steatosis, and reformed placental remodeling and oxidative injury. Probiotic therapy can restore lipid dysmetabolism in the fetal liver, modulate nutrient-sensing molecules in the placenta, and mediate the short-chain fatty acid signaling cascade. The therapeutic effects of maternal *L. reuteri* on maternal NAFLD and NAFLD reprogramming in offspring should be validated for further clinical translation.

Keywords: prenatal; high-fat diet; DOHaD; nonalcoholic fatty liver disease; *Lactobacillus reuteri*; butyrate

check for updates

Citation: Yu, H.-R.; Sheen, J.-M.; Hou, C.-Y.; Lin, I.-C.; Huang, L.-T.; Tain, Y.-L.; Cheng, H.-H.; Lai, Y.-J.; Lin, Y.-J.; Tiao, M.-M.; et al. Effects of Maternal Gut Microbiota-Targeted Therapy on the Programming of Nonalcoholic Fatty Liver Disease in Dams and Fetuses, Related to a Prenatal High-Fat Diet. *Nutrients* **2022**, *14*, 4004. https://doi.org/10.3390/nu14194004

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1. Introduction

Evidence indicates that maternal obesity will be a major determinant of the "developmental origins of health and disease" in the next generation. Obesity before pregnancy and gestational weight gain, especially in early pregnancy, may increase the risk of obesity in the offspring [1–3]. Adverse cardiovascular risks in the offspring, such as coronary heart disease, type 2 diabetes, and stroke, also increase from childhood to adulthood [1,4].

Non-alcoholic fatty liver disease (NAFLD), an excessive accumulation of fat in the liver, is the most common form of chronic liver disease and is closely related to obesity. A meta-analysis conducted in Asia between 1999 and 2019 showed that the incidence of NAFLD was 50.9 cases per 1000 person-years [5]. Yunosi et al. estimated the global prevalence of NAFLD to be as high as 25% [6]. In an autopsy study conducted in San Diego, the authors enrolled 742 children and found that the prevalence of fatty liver was 9.6%, whereas the prevalence in obese children was as high as 38% [7]. NAFLD is strongly associated with

題目:母體腸道微生物標靶治療方式，對產前高脂飲食引起母體和胎兒非酒精性脂肪肝病的影響

Article: Effects of Maternal Gut Microbiota-Targeted Therapy on the programming of Nonalcoholic Fatty Liver Disease in Dams and Fetuses, Related to a Prenatal High-Fat Diet. *Nutrients*. 2022 Sep 27;14(19):4004.

摘要:在出生之前代謝失調問題可能就已經開始。來自母親的代謝表型可能為後代增加罹患代謝疾病的風險，像是非酒精性脂肪肝病。此實驗結果顯示 ，給予羅伊氏乳桿菌具有恢復胎兒肝臟的脂肪代謝紊亂問題，調節胎盤吸收營養能力和短鏈脂肪酸訊號傳遞。

Abstract: Metabolic disorders can start in utero. Maternal transmission of metabolic phenotypes may increase the risks of adverse metabolic outcomes, such as nonalcoholic fatty liver disease (NAFLD). The results show that *L. reuteri* can restore lipid dysmetabolism in the fetal liver, modulate nutrient-sensing molecules in the placenta, and mediate the short-chain fatty acid signaling cascade.

乳酸桿菌功效研究

Efficacy studies of *Lactobacillus*

Probiotics and Antimicrobial Proteins
https://doi.org/10.1007/s12602-022-09982-w

Neuroprotective Effects of Probiotic *Lactobacillus reuteri* GMNL-263 in the Hippocampus of Streptozotocin-Induced Diabetic Rats

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Abstract

Diabetes-related brain complications have been reported in clinical patients and experimental models. The objective of the present study was to investigate the neuroprotective mechanisms of *Lactobacillus reuteri* GMNL-263 in streptozotocin (STZ)-induced diabetic rats. In this study, three different groups, namely control group, STZ-induced (55 mg/kg streptozotocin intraperitoneally) diabetic rats (DM), and DM rats treated with *Lactobacillus reuteri* GMNL-263 (1×10^9 CFU/rat/day), were utilized to study the protective effect of GMNL-263 in the hippocampus of STZ-induced diabetic rats. The results demonstrated that GMNL-263 attenuated diabetes-induced hippocampal damage by enhancing the cell survival pathways and repressing both inflammatory and apoptotic pathways. Histopathological analysis revealed that GMNL-263 prevented structural changes in the hippocampus in the DM group and decreased the level of inflammation and apoptosis in the hippocampus of DM rats. The IGF1R cell survival signaling pathway also improved after GMNL-263 treatment. These results indicate that probiotic GMNL-263 exerts beneficial effects in the brain of diabetic rats and has potential ability for clinical application.

Keywords Diabetic mellitus · Streptozotocin · Lactic acid bacteria · *Lactobacillus reuteri* · Hyperglycemia

Introduction

Diabetes is the most common metabolic disorder, with increasing mortality rate worldwide. It is predicted that the number of diabetic patients may exceed 500 million by the middle of the twenty-first century in global population, if there is no effective prevention or treatment strategies [1, 2]. Diabetic encephalopathy, like impaired spatial learning and memory damage, has been previously described in animal models and diabetic patients [3]. Furthermore, diabetes induces oxidative stress or other metabolic abnormalities that affect the normal brain function, leading to several brain diseases, including depressive disorder, anxiety, and cognitive dysfunctions [4]. Diabetes can also alter the morphology and structure of the central nervous system, which causes cognitive deficits in clinical patients [5]. Similarly, the results of quantitative magnetic resonance imaging evaluation shows that the brain of diabetic mouse exhibits abnormal white matter and brain atrophy [6]. However, the pathophysiology of diabetic encephalopathy is poorly understood. Diabetes-induced brain tissue damage is likely associated with oxidative injury, neuronal loss, and deregulated glucose metabolism through hyperglycemia [4, 7]. Otherwise, diabetes presents with neuronal apoptosis in the hippocampus [5]. Recent studies have shown that diabetes-induced neuronal apoptosis results in diabetic encephalopathy and brain atrophy [7], and that diabetes-induced hippocampal neuronal damage can lead to cognitive impairment [5].

Previous studies have shown that inflammation is found in several neurodegenerative diseases, such as stroke, multiple sclerosis, and traumatic injury [8]. Apoptosis, a

Wei-Wen Kuo and Chih-Yang Huang contributed equally to this paper.

Highlights
- GMNL-263 suppresses diabetes-induced damage in the hippocampus.
- GMNL-263 reduces inflammation and apoptosis in the diabetic hippocampus.
- GMNL-263 improves IGF1R/AMPK/GLUT3 to prevent damage in diabetic hippocampus.
- GMNL-263 can potentially be used to prevent diabetic-induced hippocampus injury.

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Extended author information available on the last page of the article

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Springer

題目:熱滅活羅伊氏乳桿菌GMNL-263對STZ誘發之糖尿病大鼠的海馬迴神經保護作用

Article: Neuroprotective Effects of Probiotic *L. reuteri* GMNL-263 in the Hippocampus of Streptozotocin-Induced Diabetic Rats. Probiotics Antimicrob Proteins. 2022 Aug 31.

摘要:許多臨床病人和實驗模式的研究結果均指出糖尿病易造成腦部的併發症。本實驗結果證明GMNL-263能減輕糖尿病造成海馬迴損傷，表示GMNL-263對糖尿病鼠的大腦具有神經保護效益。

Abstract: Diabetes-related brain complications have been reported in clinical patients and experimental models. The results demonstrate that GMNL-263 attenuates diabetes-induced hippocampal damage and exerts beneficial neuroprotective effects on the brain of diabetic rats.

財務狀況 Financial status

※母公司財報-景岳生技 Separate Financial Statements-GenMont

單位：千元 In Thousands of New Taiwan Dollars	108年母公司財報 (Year 2019 Single)	109年母公司財報 (Year 2020 Single)	110年母公司財報 (Year 2021 Single)
營業收入NET REVENUE	346,244	325,191	427,929
營業毛利GROSS PROFIT	229,701	215,390	299,326
營業費用OPERATING EXPENSES	(148,838)	(137,605)	(150,140)
營業利益INCOME(LOSS) FROM OPERATIONS	80,863	77,785	149,186
營業外損益NON-OPERATING INCOME AND EXPENSES	(5,107)	(269)	(6,231)
稅前淨利INCOME(LOSS) BEFORE INCOME TAX	75,756	77,516	142,955
稅後淨利NET INCOME(LOSS)	57,451	60,884	113,132

財務狀況 Financial status

※合併財報 Consolidated Financial Statements

單位：千元 In Thousands of New Taiwan Dollars	109年合併 (Year 2020 Consolidated)	110年合併 (Year 2021 Consolidated)	111年前三季合併 (Year 2022 Q1~Q3 Consolidated)
營業收入NET REVENUE	325,191	427,929	262,529
營業毛利GROSS PROFIT	215,390	299,326	180,406
營業費用OPERATING EXPENSES	(144,376)	(174,287)	(140,818)
營業利益INCOME(LOSS) FROM OPERATIONS	71,014	125,039	39,588
營業外損益NON-OPERATING INCOME AND EXPENSES	5,833	14,385	9,946
稅前淨利INCOME(LOSS) BEFORE INCOME TAX	76,847	139,424	49,534
稅後淨利NET INCOME(LOSS)	60,215	109,601	36,290
停業單位損益Income (Loss)from Discountinued Operations	0	0	0
本期淨利NET INCOME(LOSS)	60,215	109,601	36,290

景岳(中國)天津廠

GenMont in Tianjing, China.

景岳在天津興建國際級規格的專業益生菌製造廠，建構全面性的乳酸菌專業生產平台。

GenMont has built an international standard probiotics manufacturing plant in Tianjing, China. We have established a comprehensive fermentation platform for lactic acid bacteria.

Thanks For Your Attention

Q & A