GENERVON

Pioneering Neuroprotection and Regeneration for Neurodegenerative Diseases and Disorders

Company Overview

December 2020

Executive Summary

GENERVON

To discover and develop a drug to treat
the high unmet need of central nervous system (CNS) diseases

Genervon is bringing GM6, a new clinical-stage drug asset, to regulatory approval and commercialization in China for CNS diseases including:

- Alzheimer's disease (AD)
- Parkinson's disease (PD)
- amyotrophic lateral sclerosis (ALS)
- Multiple Sclerosis (MS)
- other neurodegenerative diseases

Executive Summary

There is a high unmet need for central nervous system (CNS) diseases in the growing aging population.

Most clinical trials for drugs developed through the traditional single-target drug approach have failed to treat the complex neurological disorders that involve multiple interrelated pathways.

GM6 is neither an antibody nor a single-target agonist or antagonist. It is a pleiotropic regulator which simultaneously regulates multiple pathological pathways.

Genervon has already de-risked many factors of drug development concerns for GM6.

Genervon is interested in partnering through licensing or merger and acquisition.

GM6 Advantages: A New Clinically Advanced Pleiotropic Therapeutic for Neurodegenerative Diseases and CNS Disorders

- **Addresses high unmet medical needs. Worldwide cost of dementia is $1 trillion (USD, 2018 estimate). In China, there are nearly 3 million people affected by Parkinson's disease and will reach 5 million in 2030**
- **Discovered an endogenous developmental-stage master regulator called MNTF unlocking a pleiotropic therapeutic approach to treating complex neurological disorders**
- **Developed GM6, a 6-amino-acid active site of MNTF, to treat neurodegenerative diseases and disorders**
  - **GM6 rapidly crosses the blood-brain barrier**
  - **GM6 enables neuron survival through developmental-stage pathways, strengthening cell adhesion and extracellular matrix scaffolds, increasing synaptic transmission, and decreasing oxidative stress and apoptosis in the central and peripheral nervous systems**
- **Phase 2B/3-ready program in ALS following strong results in Phase 2A trial. GM6 received orphan drug designation from the FDA and the EMA**
- **Phase 2-ready programs for PD, AD and MS**
- **GM6 has good drug-like properties. GMP grade GM6 drug substance and drug products were manufactured. Complete CMC package ready for tech transfer. Ready for quick regulatory filings. High potential platform.**

Pipeline Includes Several Neurodegenerative Indications Including a Phase 3-Ready ALS Program

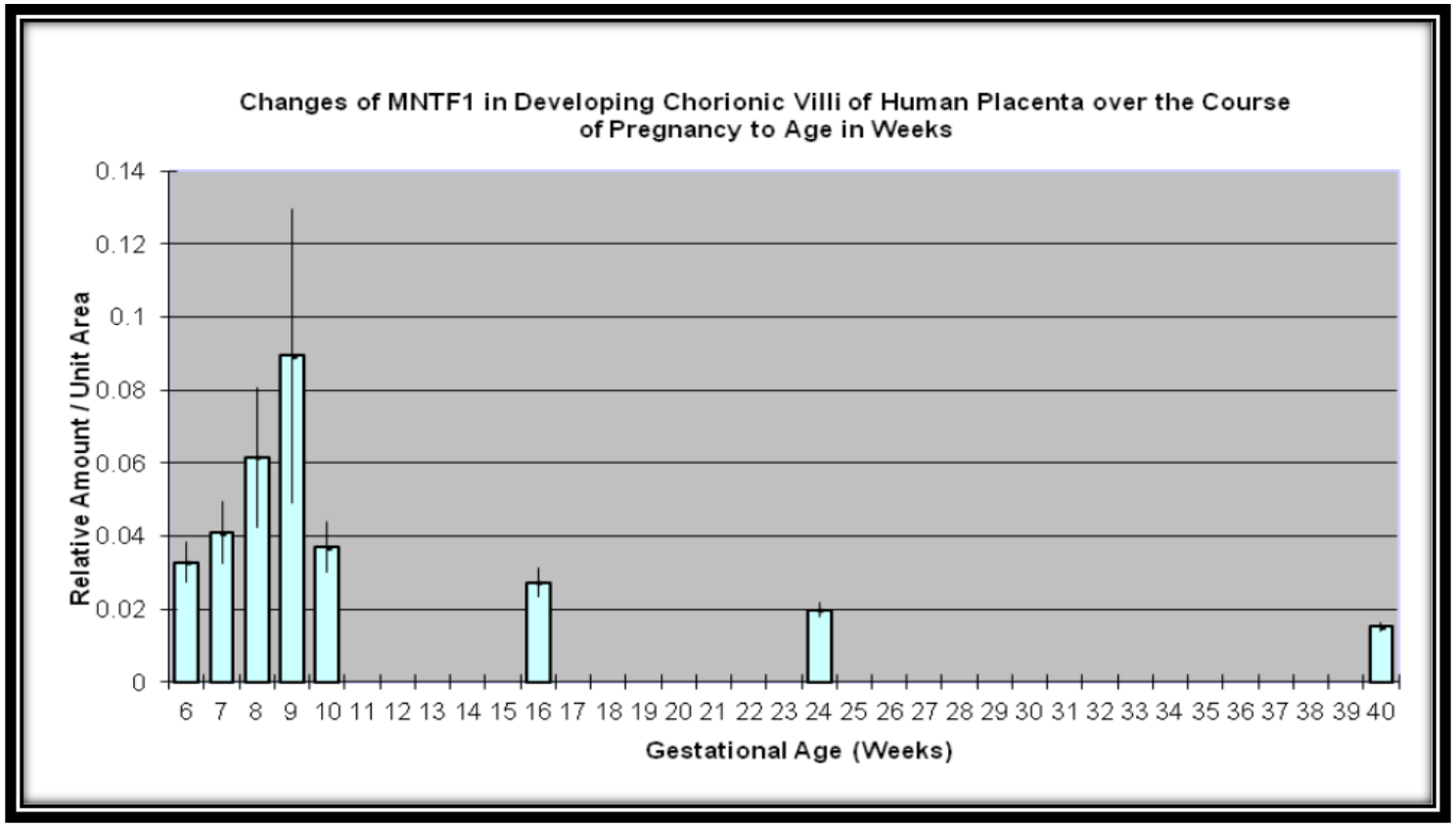
PRODUCT	INDICATION	DEVELOPMENT STAGE
		Discovery / Preclinical / Phase 1 / Phase 2 / Phase 3
GM604	ALS*	Phase 1 – Phase 2
GM608	Parkinson's disease (PD)	Phase 1 – Phase 2
GM605	Alzheimer's disease (AD)	Phase 1
GM607	Multiple Sclerosis (MS)	Phase 1

*Orphan Drug Designation granted by the US FDA and the European Medicines Agency

Note: Pipeline portfolio also includes several Phase 1 trials in oncology and other undisclosed therapeutic areas.

GM6 is a Fragment of Motoneuronotrophic Factor (MNTF) Which is Present in Human Embryonic Development

MNTF's peak expression is at 9 weeks gestation in stem-cell-rich chorionic villi

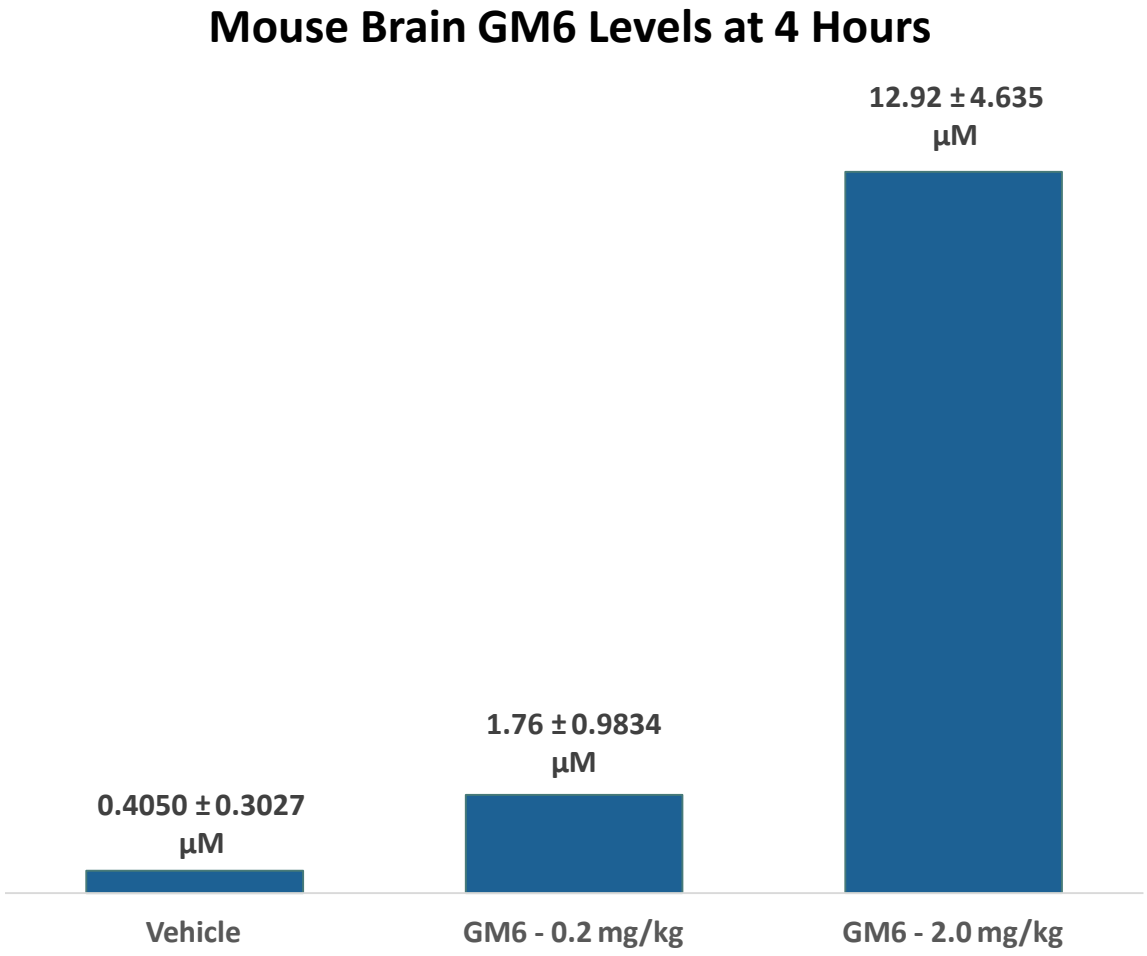


Genervon hypothesized that the answer to curing central nervous system diseases may lie within the human body itself. Genervon discovered an endogenous regulator Motoneuronotrophic factor (MNTF).

This chart shows the changes of MNTF expression in human placenta over the course of gestation in weeks.

MNTF expression rose rapidly during the 6th to 9th weeks of gestation in placenta with increasing week age, peaked at the 9th week, and then declined progressively.

GM6 Rapidly Transits the Blood-Brain Barrier

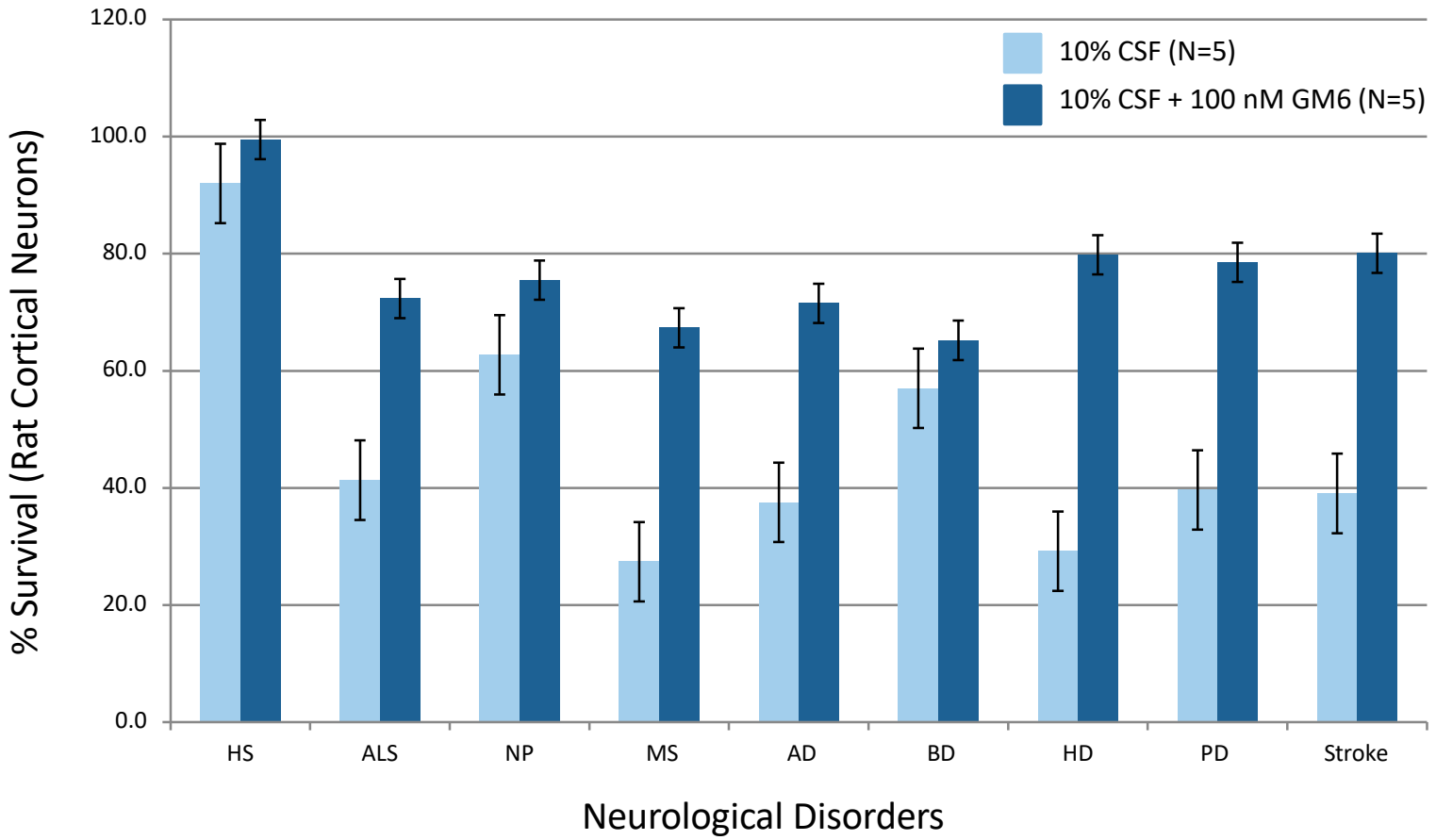


- C57BL6 mice were injected with a single bolus intravenous tail vein injection of GM6 at 0.2 and 2.0 mg/kg.
- At four hours, the animals were sacrificed, and half of the brain was frozen for ELISA analysis.
- ELISA assay with the supernatant from the brain homogenate detected GM6 at statistically significant levels, at all doses, compared to control (p=0.0001).

Despite a short half-life, GM6 binds rapidly and remains bound *in vivo*

- GM6 is a positive allosteric modulator for insulin receptor.
- GM6 rapidly transits the blood-brain barrier enabling neuron survival through developmental-stage pathways, strengthening cell adhesion and extracellular matrix scaffolds, increasing synaptic transmission, and decreasing oxidative stress and apoptosis in the central and peripheral nervous system.
- GM6 has a good brain to plasma ratio (1.65) in biomimetics measurement.

GM6 Demonstrates Neuroprotection in Preclinical Models of Neurodegenerative Diseases



- Neurons die when exposed to toxicity from postmortem CNS patient's Cerebrospinal Fluid ("CSF")
- GM6 demonstrated neuroprotection with a statistically significant increase in neuron survival ($p < 0.0001$) in:
  - ALS (175%)
  - MS (246%)
  - AD (191%)
  - HD (273%)
  - PD (198%)
  - Stroke (205%)

Note: Post mortem patients' tissues and CSF were provided by the National Neurological Research Specimen Bank at UCLA VA Hospital.

GM6 Was Safe and Well Tolerated in a Phase 1 Clinical Trial

In Phase I, 32 healthy subjects were randomized to GM6 or placebo by intravenous (IV) administration

- No SAEs or withdrawals due to drug
- No clinically significant lab results, ECG, or Qt prolongation concerns
- The most commonly reported AEs were dizziness, headache, and abdominal pain
- No laboratory abnormalities of potential clinical concerns

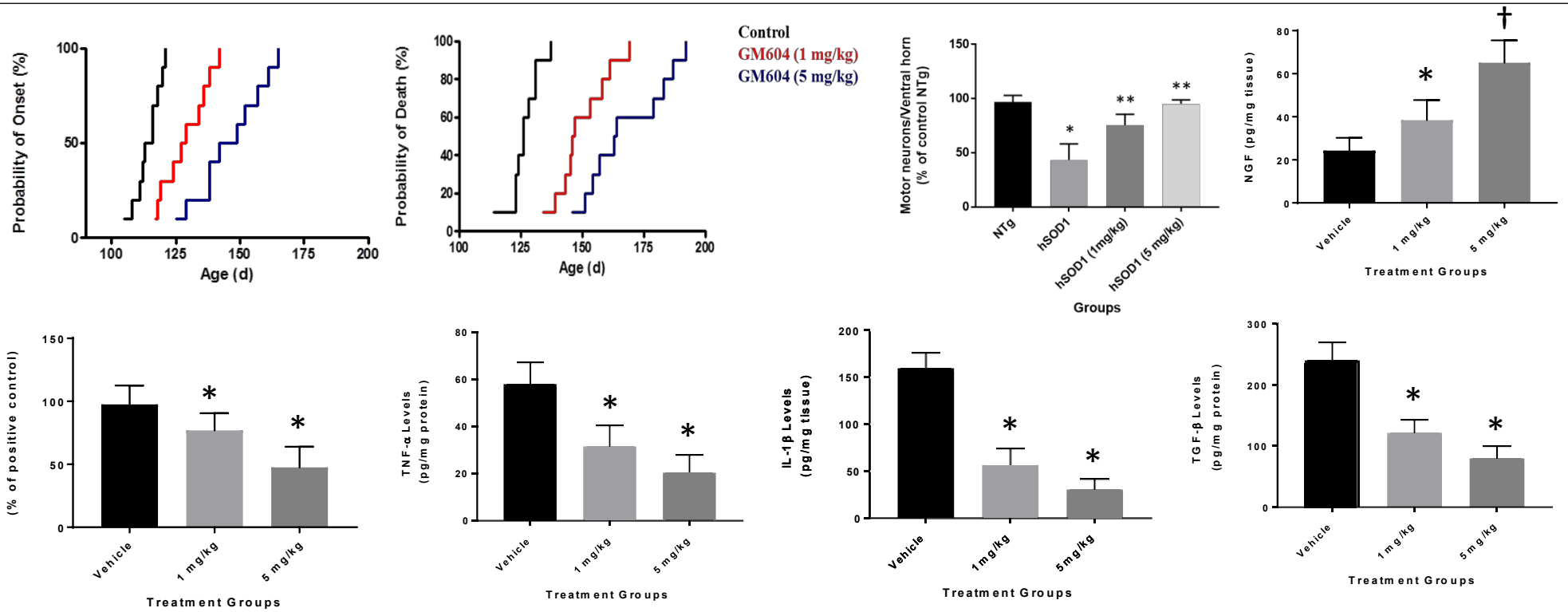
GM6 was safe and well tolerated at all doses.

Multiple INDs were filed and approved for multiple indications.

The Phase 1 safety data are applicable to multiple Phase 2 clinical trials following a similar dosing regimen.

GM6 and ALS

GM6 Increases Neurogenesis and Lowers SOD1 and Inflammation to Ameliorate ALS Disease in ALS SOD1 Animal Model



- In an ALS model SOD1 mice, in a dose dependent fashion, GM6 improve behavior, survival rate, strength, and clinical score
- GM6 reduced motor neuron loss, reduced hSOD1 protein level, and reduced inflammation biomarkers such as TNFα, IL-1β, and TGF-β, and increased NGF
- GM6 may modulate ALS disease through regulating inflammation response

GM6 Phase 2A ALS Multi-Center Trial: Clinical Data in Safety, FVC, and ALSFRS-R and Biomarkers

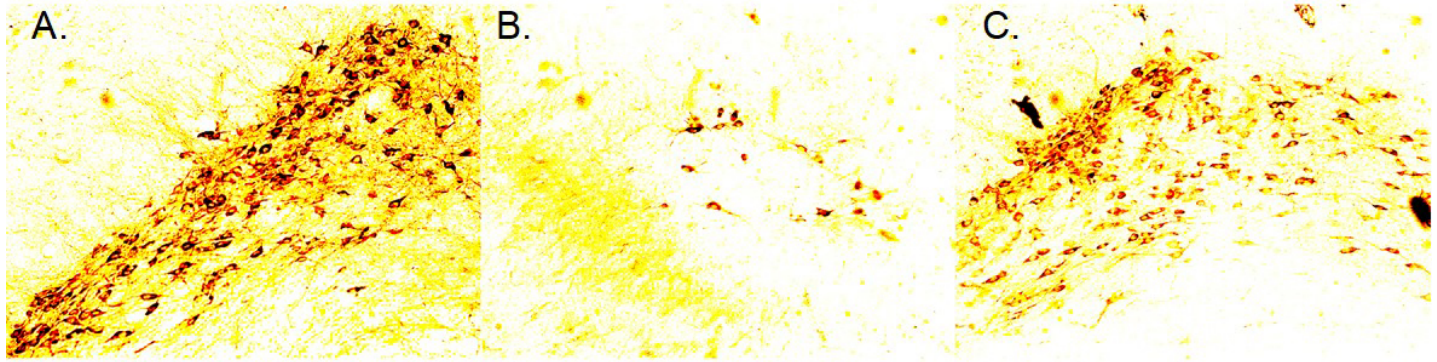
This trial recruits fast-progressing definite ALS patients within 2-year onset.
The endpoints of the clinical trial measurements are:

Safety	**No clinically significant drug-related serious adverse event.** Adverse events were similar between treated and placebo groups. The same safety data was found in our Phase 1 safety trial.
FVC	**GM6 slowed FVC decline:** Forced vital capacity (FVC) historical progression rate in ALS patients is -3% per month. For 12 weeks at all sites, the expected decrease was 9%. At week 12, the GM6 group decreased only 4.7% while the placebo group decreased 11.5%.
ALSFRS-R	**GM6 slowed functional decline** as measured by the ALSFRS-R rating scale In the GM6-treated group the ALSFRS-R progression from baseline to 12 weeks is slower when compared to a historical control for fast-progressing definite ALS patients within 2-year onset
Biomarkers	GM6 showed favorable shifts in the following plasma biomarkers with statistical significance: **SOD1, Tau, TDP-43**

GM6 and Parkinson’s Disease

GM6 in Two Animal Models Improved Functions and Motor Activities, Increased Dopamine and Neuron Protection

- The cause of Parkinson's disease is the loss of dopamine.
- Before the Phase 2A clinical trial, GM6 efficacy in PD was predicted in two animal models. GM6 showed statistically significant beneficial effects in improving motor functions and in increasing dopamine and Tyrosine Hydroxylase (TH) Positive Neurons in the (SNpc) region in both models.
- The figures below show that GM6 increased dopaminergic neurons in the Substantia Nigra Pars Compacta (SNpc) in PD 6-OHDA mouse model by Immunohistochemical staining
- Instead of being an exogenous source of dopamine, GM6 protects the dopaminergic neurons from dying and consequently increases dopamine.



GM6 protects the dopaminergic neurons in SNpc from the detrimental effects of PD induction

PD Phase 2A Clinical Trial Demonstrated GM6 is Safe Treatment Signals Slowing of the Rate of Clinical Worsening of PD

- In this Phase 2A PD clinical trial, GM6 treatment signals a slowing of the progression rate of PD by UPDRS rating scale in this trial.
- The level of neuroprotective biomarker BDNF in the GM6-treated group was higher than in the placebo group with statistical significance. GM6 increased the expression of BDNF to increase neuroprotection.
- No serious adverse events were recorded. No clinically significant lab test results difference.
- GM6 is ready for a larger Phase 2 clinical trial for PD in China.

GM6 and Alzheimer's Disease

Alzheimer's Disease (AD): a Worldwide Health Crisis

The total global annual cost of dementia is $1 trillion USD (2018 estimate)
The number of people living with dementia in Asia is 22.9 million (2015)

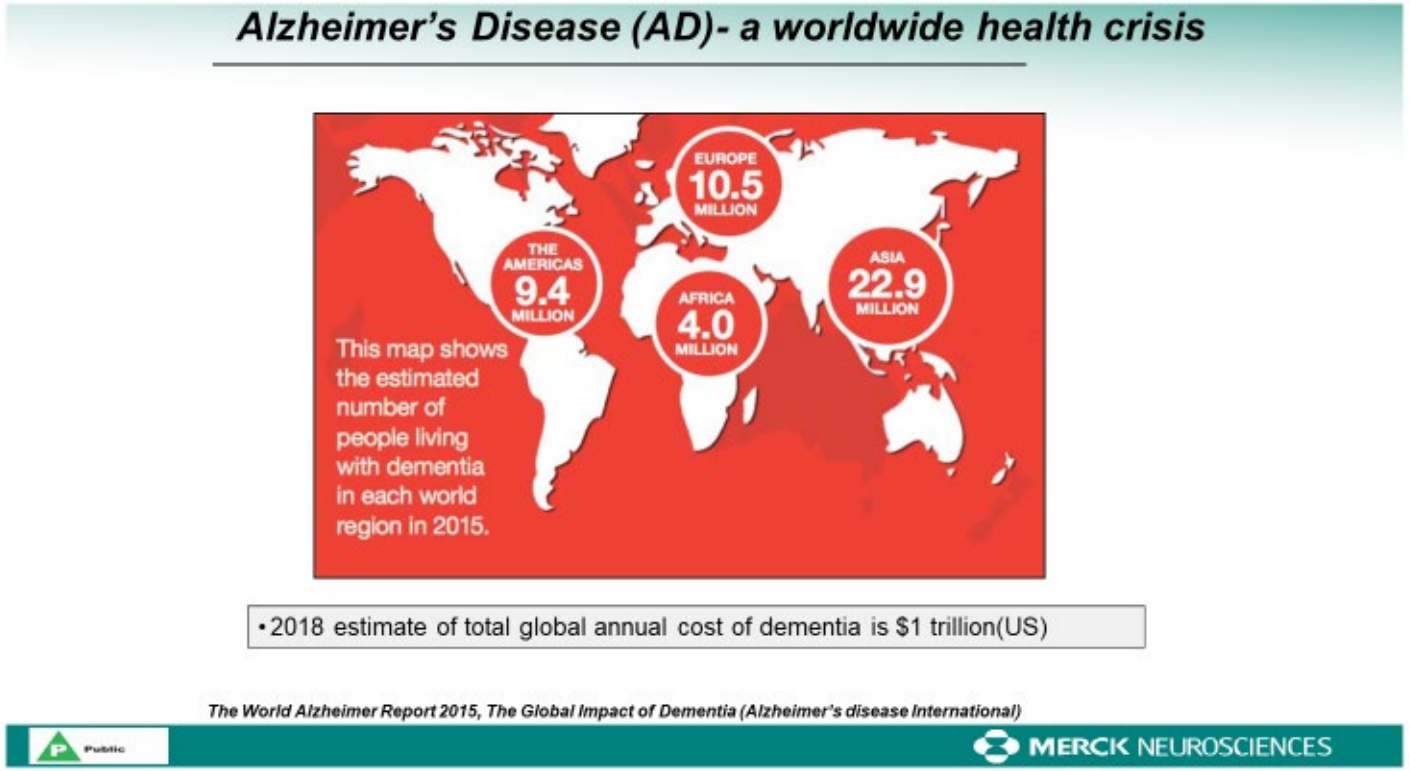
Alzheimer's: the disease that could bankrupt US Medicare Healthcare Systen
https://www.cnn.com/2017/03/07/health/alzheimers-report-2017/index.html

"What is driving these numbers is that there is no disease modifying treatment, no prevention and no cure"

- Alzheimer's Association

"The Alzheimer's Drug Discovery Foundation is convinced that the answer to treating AD will lie in using multiple drugs in combination, or ***drugs with multiple effects in one molecule****, reflecting the fact that it is an enormously complex disease with multiple causes and pathologies."*

-Alzheimer's Drug Discovery Foundation



Genervon proposes that GM6 is such a molecule with multiple effects.

GM6 Attenuates AD Pathology in APP Mice in Multiple Pathways

GM6 Reduces Inflammation and Modulates Key Biomarkers

GM6 treatment demonstrated a dose dependent statistically significant ($P < 0.05$) favorable outcome in APP mice after 4 months of treatment

1. Reduced Inflammation
2. Reduced accumulation of Aβ peptide
3. Reduced amyloid load
4. Decreased cathepsin B expression
5. Tempered the memory impairment
6. Improved spatial orientation by faster travel:
7. Increased NGF levels in brain

GM6 modulates various pathways early in the disease process including upregulating APP catabolism to reduce Aβ deposit and effectively alter the disease process in AD

GM6 Attenuates AD Pathology by Lowering Hyperphosrylated Tau

GM6 Reduces Inflammation, Lowers Tau Hyperphosphorylation, and Attenuates Behavioral Changes

In healthy neurons, tau normally binds to and stabilizes microtubules. Abnormal chemical changes cause tau to hyper phosphorylate and form tangles blocking the neurons' transport system and cause tauopathy and clinical manifestations of AD.

In vitro study GM6 treatment reduced hyperphosphorylated tau.

In a mouse tau model (h-Tau) which showed hyperphosphorylation of h-Tau and behavioral deficits, GM6 treatment demonstrated reduction of hyperphosphorylated tau and attenuated the behavioral

Cytokines were extracted from h-Tau mouse brains and measured by ELISA. Quantitative analysis of cytokine levels by ELISA for vehicle control group compared to GM6-treated group showed significant reduction of **TNF-α, IL-1β, and IL-6**

GM6 Ameliorates AD Through Lowering β Amyloid Protein Aggregates, P-tau, TDP-43, and Inflammation

- **GM6 may be used to treat AD by simultaneously lowering β amyloid protein aggregates, p-tau, TDP-43, and inflammation.**
- **The similar regulatory effects of GM6 in ALS and AD pathogenesis suggest that GM6 may show positive effects in an AD Phase 2 clinical trial as it had in an ALS Phase 2A clinical trial.**

Summary:
GM6 is a Pleiotropic Approach to Combatting Multiple Diseases

GM6	
Endogenous	Regeneration
Safe	Reinnervation
Crosses the blood-brain barrier	Repair
In ALS: Down regulates key biomarkers, Improves clinical outcomes	
In AD: Reduces protein aggregates and inflammation, Upregulates NGF for neurogenesis	
In PD: Protect dopaminergic neurons. Increase dopamine and BDNF	
In MS: Attenuates MS in clinical and histological analysis	

PROGRAM & PLATFORM DEVELOPMENTAL POTENTIAL	
NEURODEGENERATIVE	CNS
ALS	Stroke
Parkinson's disease	Orphan CNS
Alzheimer's disease	Spinal cord injury
Multiple Sclerosis	Traumatic brain injury
Huntington's disease	
Epilepsy	

Genervon is ready to partner to advance GM6 development to Phase 3 for ALS and Phase 2 for AD, PD and MS

GENERVON Biopharmaceuticals

健能万生化*制*药企业

神经的保护与再生

針对多种神经退行性疾病适应症治疗领域的先驱者

企业*介紹*

12. 2020

摘要

GENERVON

发现并开发药物

治疗对中枢神经系统疾病的高度未满足需求

Genervon正在将新的临床阶段药物资产GM6带入中国的中枢神经系统疾病监管机构批准和商业化阶段，其中包括：

- 阿尔茨海默氏病（AD)
- 帕金森病（PD)
- 肌萎缩侧索硬化症（ALS)
- 多发性硬化症（MS)
- 其他神经系统疾病

摘要

在日益老龄化的人口中，对中枢神经系统（CNS）疾病的需求尚未得到满足。

通过传统的单靶标药物方法开发的大多数药物临床试验都未能治疗涉及多种相互关联途径的复杂神经系统疾病。

GM6既不是抗体，也不是单靶激动剂或拮抗剂。它是一种多效性调节剂，可同时调节多种病理途径.

Genervon已经降低了许多与GM6有关的药物开发问题的风险。

Genervon有兴趣通过许可或合并和收购成为合作伙伴。

GM6优点:一款新的临床先进针对神经退行性疾病和中枢神经系统疾病的多效治疗药物

- 全球痴呆症的年度总费用为1万亿美元（美元，2018年估计）。在中国，目前有近300万人患有帕金森氏症，到2030年将达到500万人。
- 发现了一种名为MNTF的人类神经系统发育的主要调控因子, 开辟一种用于治疗复杂神经系统疾病的多效治疗方法
- 开发了GM6，一种6-氨基酸MNTF的短肽类似物，用于广泛的治疗神经退行性疾病：
  - GM6快速通过血脑屏障，
  - 通过发育阶段通路使神经元存活，增强细胞粘附和细胞外基质骨架，增加突触传递，减少中枢和外周神经系统中的氧化应激和细胞凋亡。
- 脊索侧索硬化症（ALS）治疗项目3期临床试验准备就绪。GM6已经在2a期试验中取得了很好的成果。 并获得了美国FDA和欧洲EMA的孤儿药指定。
- 帕金森症（PD）阿尔茨海默氏症（AD）和多形性硬化症（MS） 2期临床试验准备就绪。
- GM6具有良好的类药物特性. 生产了GMP级GM6原料药和药品。有完整的CMC **化学制造管控及批文**;准备好进行技术转让;快速的监管备案. 平台技术应用于多种神经退行性适应症以及其他未公开的适应症.

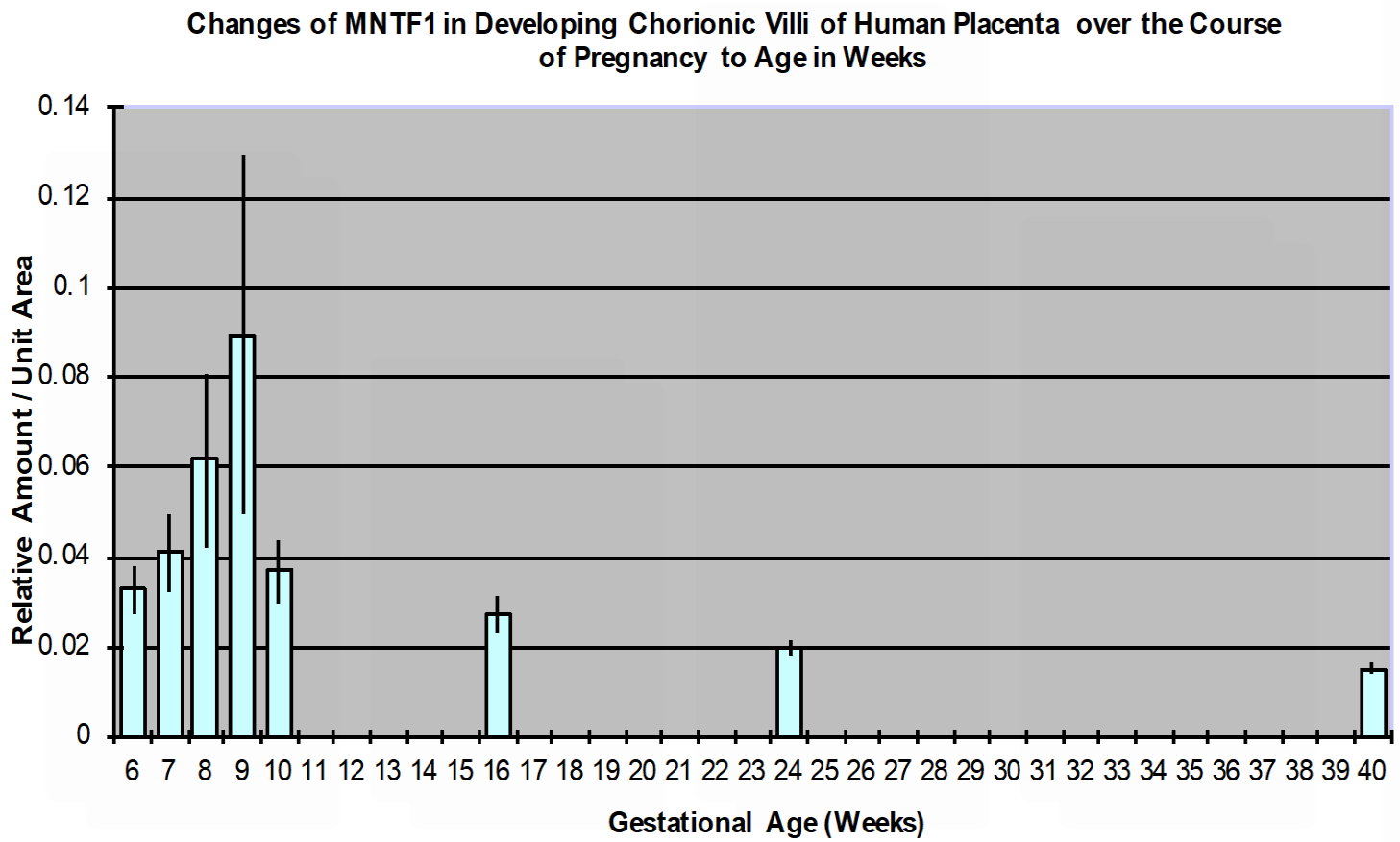
药品管道包括多个神经退行性适应症，包括3期就绪ALS计划

PRODUCT	INDICATION	DEVELOPMENT STAGE				
		Discovery	Preclinical	Phase 1	Phase 2	Phase 3
GM604	ALS*					
GM608	Parkinson's disease (PD)					
GM605	Alzheimer's disease (AD)					
GM607	Multiple Sclerosis (MS)					

*美国FDA和欧洲药品管理局授予孤儿药称号

GM6是内源性运动神经营养因子（MNTF的片段）在妊娠第9周在人胎盘中表达量达到高峰

在富含干细胞的绒毛膜绒毛中，MNTF的高峰表达是在妊娠9周时

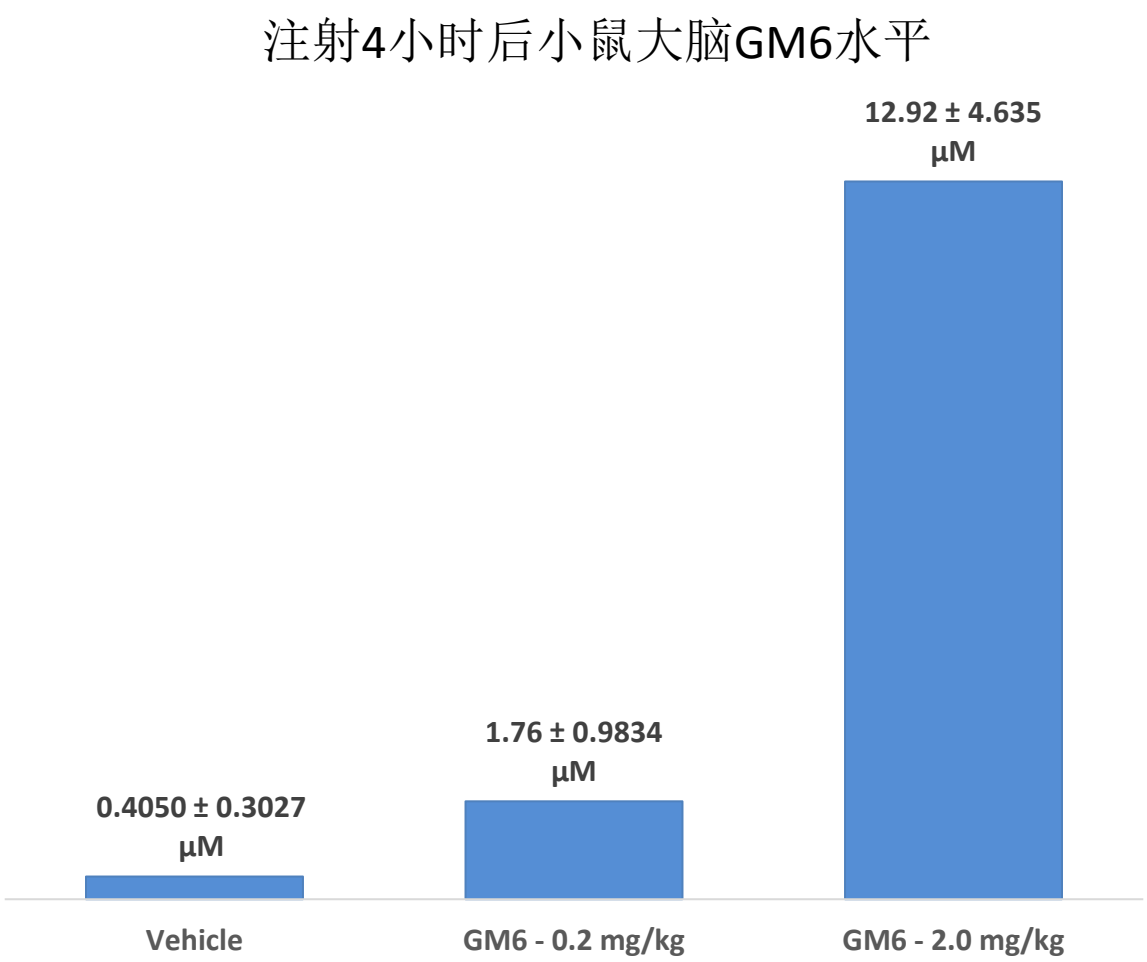


Genervon假设，治愈中枢神经系统疾病的答案可能在于人体本身。Genervon发现了一种内源性调节神经元营养因子（MNTF）。

该图显示了在数周内，人胎盘中MNTF表达的变化。

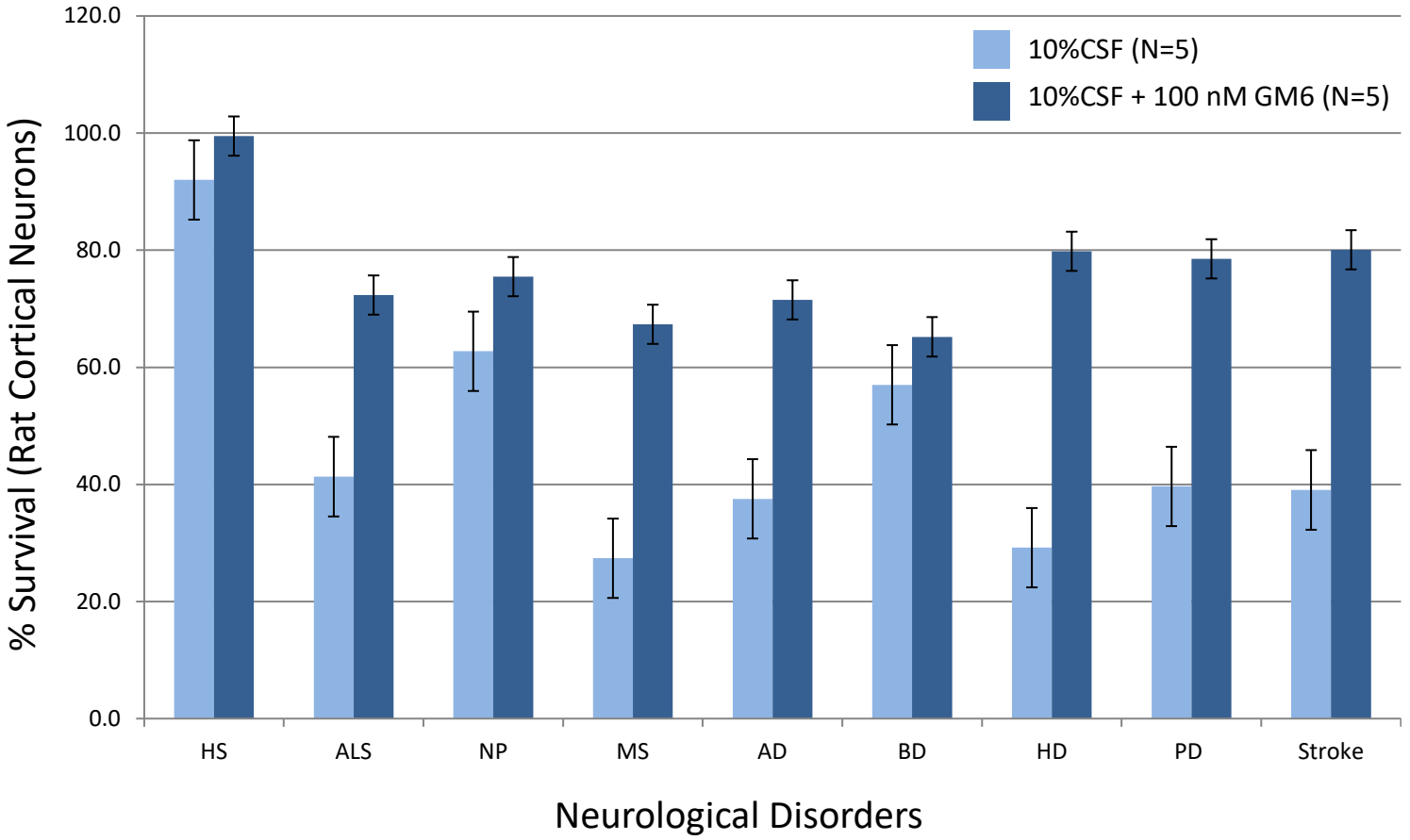
随着胎龄的增加，胎盘中 MNTF 表达在妊娠的第6至9周迅速上升，在第9周达到峰值，然后逐渐下降，出生后数值很低，几乎无法检测到。

GM6可以迅速跨越血脑屏障



- 单次给C57BL6小鼠尾静脉推注注射0.2和2.0mg / kg的GM6
- 在4小时时，处死动物并冷冻一半脑用于ELISA分析
- 使用来自脑匀浆的上清液通过ELISA检测GM6浓度，在所有剂量下与对照相比，GM6提高达到统计学显著水平（p = 0.0001）
- 尽管半衰期短，但GM6结合迅速并在体内持续结合
- GM6是胰岛素受体的正变构调节剂
- GM6快速通过血脑屏障，通过神经系统发育通路使神经元存活，增强细胞粘附和细胞外基质骨架，增加突触传递，减少中枢和外周神经系统中的氧化刺激和细胞凋亡
- GM6在仿生物测量中具有良好的脑浆比（1.65）。

GM6可以在神经退行性疾病临床前模型中的展现功效



- 当神经元接触具有毒性的已死亡的CNS患者的脑脊液(“CSF”) 时，神经元会死亡

- GM6明显提高了神经元培养在以下几种脑脊液(“CSF”)中时的存活率（p<0.0001）：
  - 肌萎缩侧索硬化症(175%)
  - 多发性硬化症 (246%)
  - 阿尔茨海默氏症(191%)
  - 亨廷顿氏症(273%)
  - 帕金森氏病(198%)
  - 中风 (205%)

注：死后患者的组织和CSF由UCLA VA医院的国家神经研究标本库提供。

GM6在1期临床试验中安全且耐受良好

GM6的所有1期和2期临床试验均为随机双盲安慰剂对照试验

在第一阶段，通过静脉内（IV）给药将32名健康受试者随机分配给GM6或安慰剂

- 没有因药物引起的SAE或停药
- 没有临床上重要的实验室异常结果，ECG或Qt延长问题
- 最常见的不良事件是头晕，头痛和腹痛
- 没有可能引起临床关注的实验室异常结果

GM6在所有剂量下均安全且耐受性良好

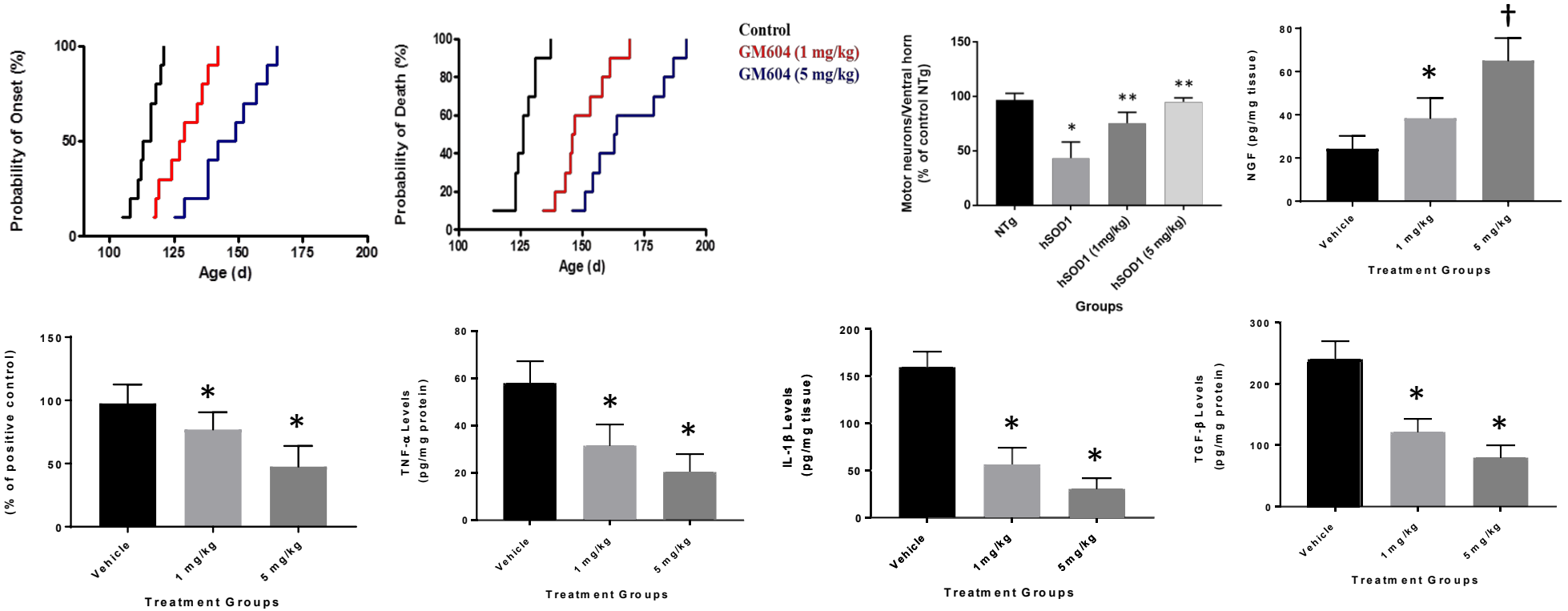
提交了多个IND，并获得了多种适应症的批准。

1期安全性数据适用于类似给药方案的2期临床试验

GM6 and ALS

GM6 和 肌萎缩侧索硬化症 (ALS)

GM6在ALS SOD1动物模型中
通过增加神经, 降低SOD1和炎症以改善疾病



- 在ALS模型SOD1小鼠中，GM6以剂量依赖的方式改善小鼠行为，存活率，强度以及临床评分。
- 此外，GM6减少运动神经元损失，降低hSOD1蛋白水平，减少炎症生物标志物如TNFα，IL-1β和TGF-β
- GM6可能通过调节炎症反应来调节ALS疾病。

GM6 2A期(ALS): 安全性, FVC, ALSFRS-R和生物标志物的临床数据

该临床试验招募了在发病2年内进展迅速的ALS患者。
临床试验测量的终点是：

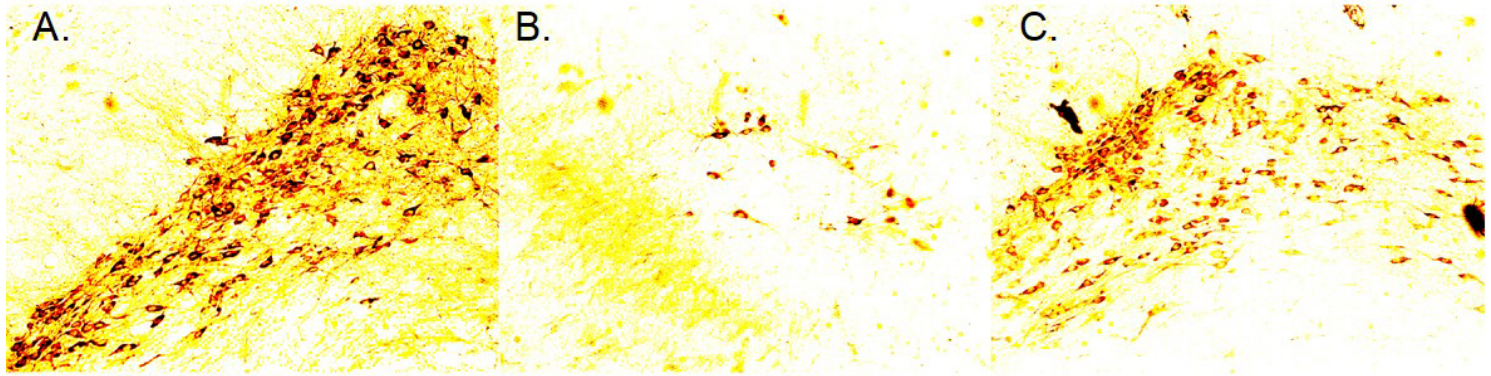
安全 (Safety)	无临床显着与药物相关的严重不良事件。治疗组和安慰剂组之间的不良事件相似。我们的第1阶段安全试验中发现了相同的安全数据。
呼吸功能（FVC)	FVC （呼吸功能 ）一般退化速度为每月减3%，12周应减9%。从基线到第12周：治疗组减了4.7%, 比较安慰剂组减了11.5%的病程退化减慢了
ALS功能等级量表-修订 (ALSFRS-R)	GM6减缓患者ALS功能等级量表的功能下降: 在GM6治疗组中，与2年内快速进展的明确ALS患者的历史对照相比，从基线到12周的GM6治疗组ALSFRS-R功能下降较慢
生物标志物	GM6在下列血浆生物标志物中显示出有利的转变，具有统计学意义： SOD1，Tau，TDP-43

GM6 and Parkinson's Disease

GM6 和 帕金森病 (PD)

GM6在两个PD动物模型中改善了功能和运动活动，增加了多巴胺和神经元保护

- 帕金森氏病(PD)的原因是多巴胺的流失。
- 在2A期临床试验之前，已在两个动物模型中预测了GM6在PD中的功效。在两个模型中，GM6在改善运动功能以及增加（SNpc）区域多巴胺和酪氨酸羟化酶（TH）阳性神经元方面均显示出统计学上显着的有益作用。下图显示，通过免疫组织化学染色，在PD 6-OHDA小鼠模型中，GM6增强了黑质致密实体（SNpc）黑多巴胺能神经元
- GM6不是多巴胺的外源来源，而是保护多巴胺能神经元免于死亡，因此增加了多巴胺。



GM6 protects the dopaminergic neurons in SNpc from the detrimental effects of PD induction.

PD阶段2A临床试验证明GM6是安全的，治疗信号减慢PD的临床恶化速度

- 在该2A期 PD临床试验中，GM6治疗通过该试验中的UPDRS评分量表表明PD的进展速度减慢。
- GM6治疗组神经保护性生物标志物BDNF的水平高于安慰剂组，具有统计学意义。GM6增加BDNF的表达以增加神经保护作用。
- 没有记录到严重的不良事件。没有临床上显着的实验室测试结果差异。
- GM6已准备好在中国进行更大的2期PD临床试验。

GM6 and Alzheimer's Disease

GM6 和 阿尔茨海默氏病 (AD)

阿尔茨海默氏病(AD)：全球性健康危机

全球痴呆症的年度总费用为1万亿美元（2018年估计）
亚洲患有痴呆症的人数为2290万（2015年）

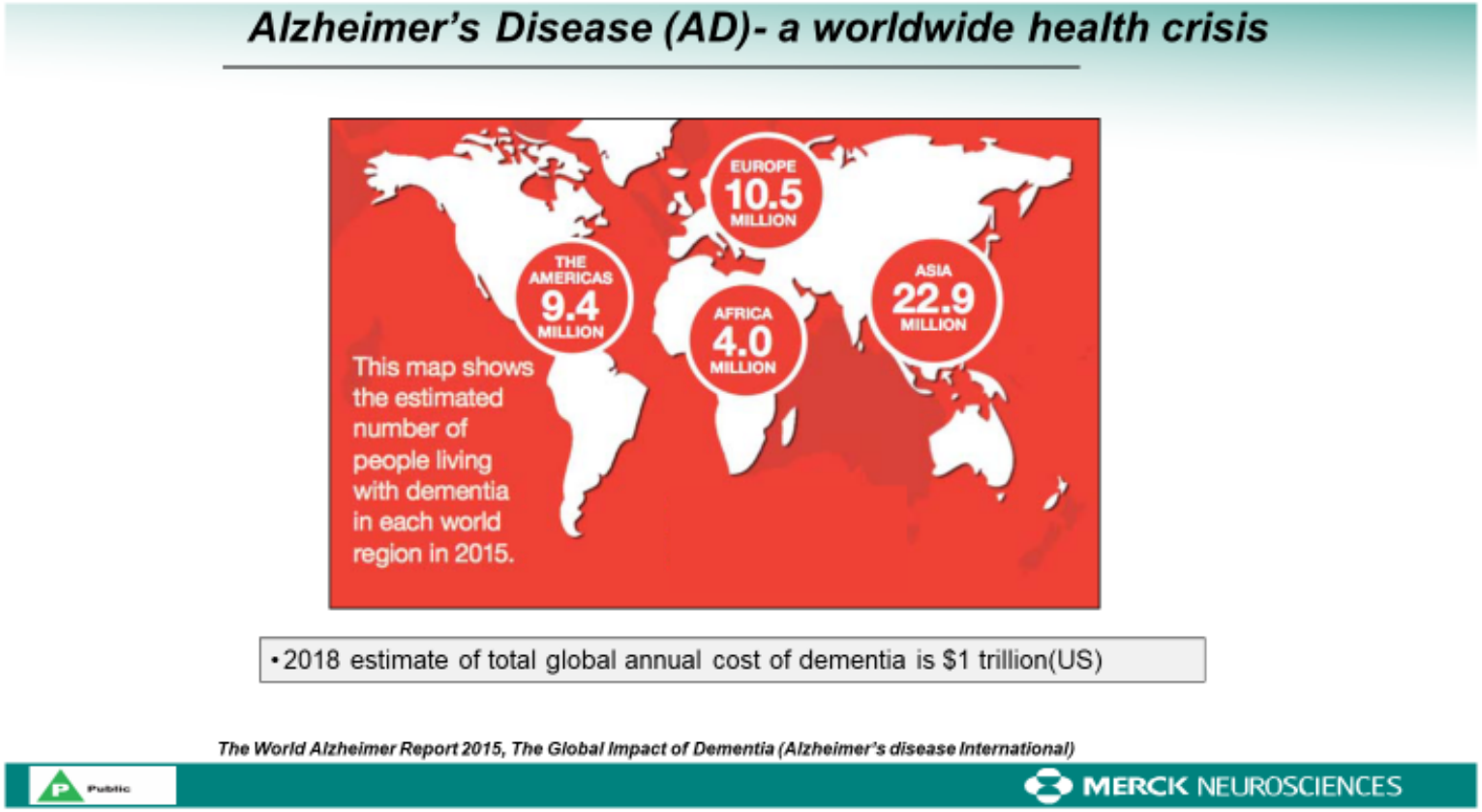
阿尔茨海默氏病：可能使美国Medicare Healthcare System破产的疾病
https://www.cnn.com/2017/03/07/health/alzheimers-report-2017/index.html

“推动这些数字增长的是没有疾病改良疗法，没有预防措施也没有治愈方法”

–老年痴呆症协会

“阿尔茨海默氏症药物发现基金会坚信，治疗AD的答案将在于联合使用多种药物或**在一个分子中具有多种作用的药物**，这反映出这是一种极为复杂的疾病，具有多种原因和病理”

–阿尔茨海默氏症药物发现基金会



Genervon提出GM6是这种具有多种作用的一个药物分子

GM6通过多种途径减轻APP小鼠的AD病理

GM6减少炎症并调节关键生物标志物

GM6治疗在治疗4个月后显示APP小鼠具有剂量依赖性的统计学显着性（P <0.05）有利结果

1. 减少炎症
2. 减少Aβ肽的积累
3. 减少淀粉样蛋白负荷
4. 组织蛋白酶B表达降低
5. 缓解记忆障碍
6. 通过更快的行程改善空间定位：
7. 脑中NGF水平升高

GM6在疾病过程的早期调节各种途径，包括上调APP分解代谢以减少Aβ沉积并有效改变AD的疾病过程

GM6通过降低过度磷酸化的tau减轻AD病理

GM6减少炎症，降低Tau过度磷酸化并减轻行为改变

在健康的神经元中，tau通常会结合并稳定微管。化学异常变化会导致tau过度磷酸化并形成缠结，阻塞神经元的运输系统，并引起tauopathy和AD的临床表现。Tau是GM6的下游目标，表现为tau过度磷酸化的降低。

体外研究GM6治疗可降低tau蛋白的过度磷酸化。

在显示出h-Tau过度磷酸化和行为缺陷的小鼠tau模型（h-Tau）中，GM6治疗证明了tau过度磷酸化的减少并减弱了行为

从h-Tau小鼠脑中提取细胞因子，并通过ELISA进行测量。与GM6治疗组相比，媒介物对照组通过ELISA进行的细胞因子水平定量分析显示：**TNF-α，IL-1β和IL-6 显着减少**

GM6通过降低β淀粉样蛋白聚集体，P-tau,TDP-43和炎症改善AD

- GM6可通过同时降低β淀粉样蛋白，p-tau，TDP-43和炎症来治疗AD 。
- GM6在ALS和AD发病机理中的类似调节作用表明，GM6可能在AD 2期临床试验中显示出积极作用，就像在ALS 2A期临床试验中一样。

总结：GM6以多因素方法，对抗多种目前无法治愈的疾病

- GM6是具有良好安全性的内源性化合物
- 以受剂量控制的促进神经元的再生
- 促进运动神经再支配，修复和存活，激活发育途径
- 快速穿过血脑屏障
- ALS: 在2a期ALS研究中下调关键生物标志物并改善临床结果
- AD: 减少蛋白质聚集和炎症, 上调NGF的神经发生GM6
- PD: 保护多巴胺能神经元。增加多巴胺和BDNF
- MS: 在临床和组织学分析中减弱MS
- 多目标作用机制可适用于其他神经系统疾病

项目和平台发展潜力	
神经退行性疾病	中枢神经系统疾病
肌萎缩侧索硬化症	中风
帕金森病	及其它罕见中枢神经系统
阿尔茨海默氏病	脊髓损伤
多发性硬化症	创伤性脑损伤
亨廷顿氏病	
癫痫	

结论：
Genervon准备与合作伙伴合作，将GM6开发推进到ALS的第3阶段以及AD, PD和MS的第2阶段临床试验