DiaMedica THERAPEUTICS

DM199 for the Treatment of Acute Ischemic Stroke

KOL Webinar
March 19 2021

Disclaimer

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Agenda

Friday March 19th at noon Eastern

Introduction

5 minutes

Rick Pauls
President and CEO

DM199 MOA for Stroke and Stroke Recurrence

15 minutes

Paolo Madeddu, M.D.
University of Bristol

Stroke Overview & Review of DM199 Clinical Program

15 minutes

Scott Kasner, MD
University of Pennsylvania

Review of DM199 Phase 2/3 Program

10 minutes

Harry Alcorn
CMO

Commercial Landscape/Q&A

15 minutes

Q&A

All

DiaMedica Introduction: DM199 for Stroke and Vascular Diseases

Developer of novel recombinant proteins, with initial focus on stroke and kidney disease

Lead candidate DM199 is a potential future front-line therapy for all stroke patients
- Initial treatment option for the >500,000 patients in US with no treatment option

De-risked clinical program with extensive 3rd party validation
- >600,000 AIS patients in China treated with Kailikang® human urine derived KLK1 product

Clear regulatory pathway to approval; opportunity to provide first new stroke therapy since 1996

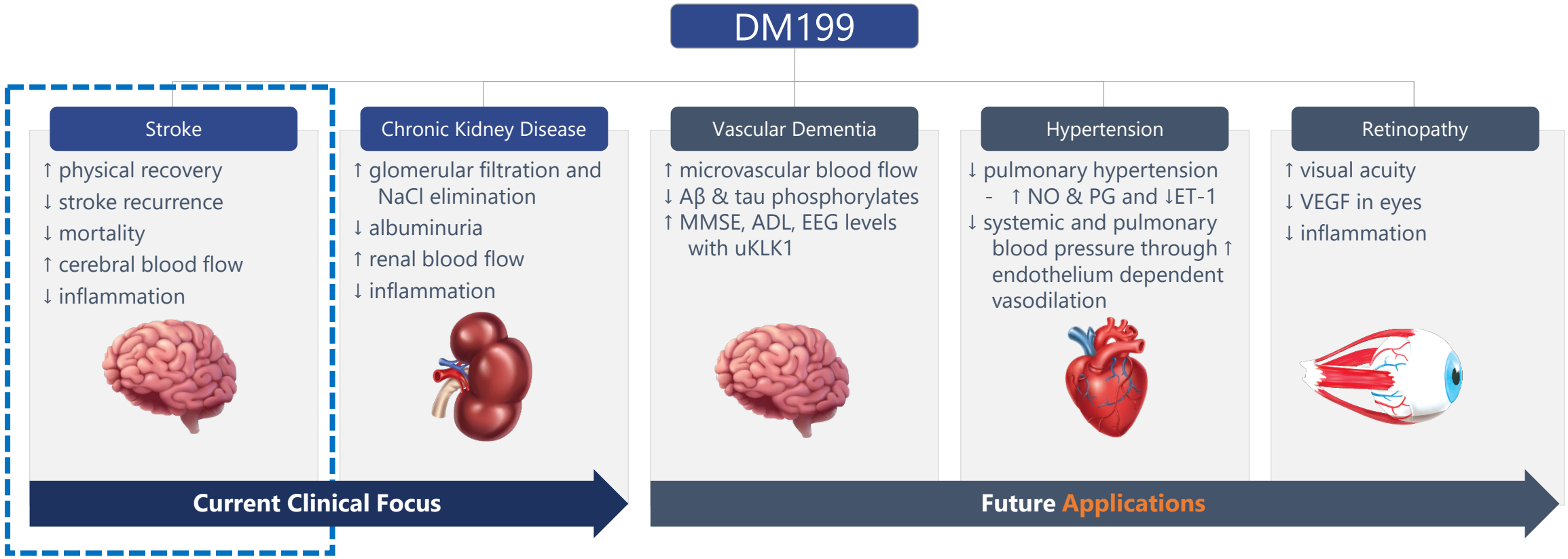
Designed for more efficient and consistent delivery of a key protein for millions of patients suffering from AIS and other diseases associated with low KLK1 levels

Multi-billion dollar commercial opportunity where few new products are in development

DM199: First Recombinant KLK1 for Clinical Use

Promoting homeostasis: Improving blood flow and reducing inflammation throughout the body

- Treatment options for small vessel diseases
- Reduced risk profile to human urinary and porcine KLK1 including impurities and immunogenicity



DiaMedica Pipeline

	Program	Product	Preclinical	Phase I	Phase 2	Phase 3	Milestones
Neuro	Acute Ischemic Stroke	DM199 IV/SC	ReMEDy2 Phase 2/3 (n=~350) Planned Recurrence sub-study in ReMEDy2				IND Submission Q1 2021 FDA Meeting/Discussions
Kidney Diseases	IgA Nephropathy	DM199 SC	REDUX Phase 2 (n=30)				Enrolling
	Diabetic Kidney Disease	DM199 SC	REDUX Phase 2 (n=30)				Topline Results Q2 2021
	African Americans, Hypertensive with CKD	DM199 SC	REDUX Phase 2 (n=30)				Enrolling
Other	Additional recombinant protein	DM300	Preclinical				Ongoing development

DM199 AIS MOA for Stroke and Stroke Recurrence

Dr. Paolo Madeddu

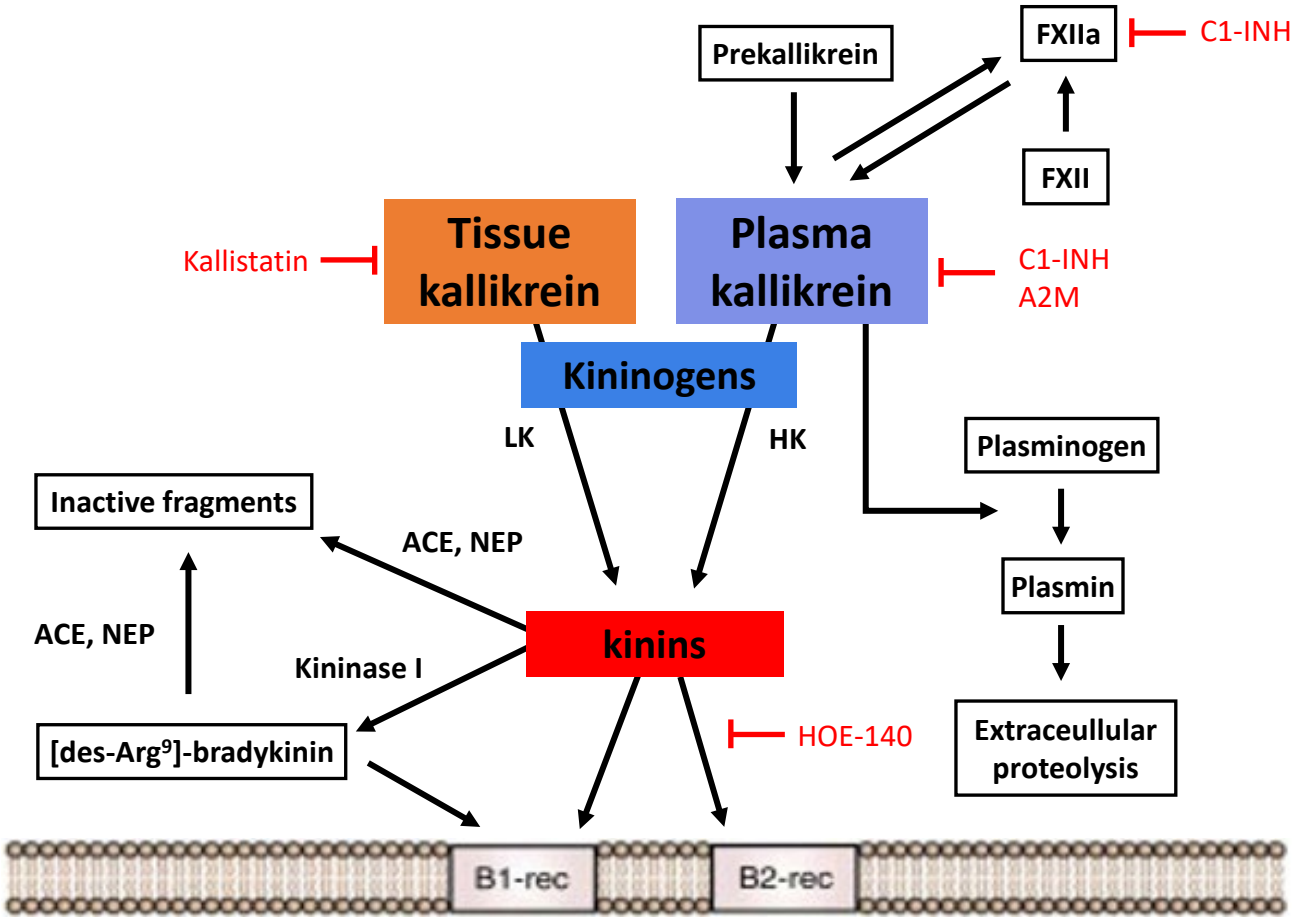
DiaMedica THERAPEUTICS

Dr. Paolo Madeddu – Kallikrein-Kinin system/KLK1 Authorship

58 pubMed papers

- KLK1-generated kinins participate in the anti-hypertensive action of ACE inhibitors (J Hypertens. 1987)
- Inhibition of KLK1 (aprotinin) and kinins (receptor antagonists) revealed a role of KKS in blood pressure and renal function regulation (Clin Sci. 1990, Hypertension. 1993, Kidney Int. 1996, Am J Physiol. 1996, Hypertension. 1997)
- Anatomical and functional independence of brain and vessel KKS (Hypertension. 1994, Hypertension. 1996)
- Low KKS phenotype in renal disease, hypertension, psoriasis, and eclampsia (Am J Nephrol. 1991; Kidney Int. 1996, Hypertension. 1997)
- Low kallikrein rats and kinin receptor knockout mice show altered cardiovascular phenotype (Circulation. 1997, Kidney International. 2001)
- Kallikrein gene therapy induces physiological healing (inhibition of restenosis, induction of reparative angiogenesis in ischemia, and prevention of diabetic microangiopathy) via the kinin/NO/Akt pathway (ATVB 2000, Circulation. 2001, Circulation. 2002, Circulation 2004, Diabetes. 2004, ATVB 2009).
- Circulating tissue kallikrein as a biomarker of peripheral and carotid artery stenosis/revascularization (Circulation. 2002, ATVB 2004)
- Role of the KKS in the recruitment of circulating progenitor cells with neovascularization potential (Circ Res. 2008, Circ Res. 2011)

Components of the Kallikrein-Kinin System (KKS)



Early divergence of plasma and tissue kallikreins (KLK1)

- Phylogenetic analysis indicates an early divergence of plasma kallikrein, which groups closely with coagulation factors plasminogen, chymotrypsin, and complement factor, from trypsin and tissue kallikrein

Expression of tissue kallikrein

- The protein is expressed in kidney, pancreas, salivary glands, brain, leukocytes, and biological fluids (blood and urine)

Action of kallikreins

- Cleavage of kininogens in biologically active kinins, acting on G-coupled receptors, with degradation by peptidases

Modulation possible through

- Supplementation of kinin generating enzyme or inhibition of kinin degrading enzymes (ACEi and NEPi)
- Inhibitors of the system, gene silencing - genetically modified animals, studies of gene polymorphisms

General Concepts/Rationale of KLK1 Supplementation

- Low KLK1 levels are associated with vascular diseases, such as arterial hypertension (salt dependent phenotype)
- Evidence that the low KLK1 phenotype associates with a higher risk of primary and recurrent stroke

Plasma Tissue Kallikrein Level Is Negatively Associated with Incident and Recurrent Stroke: A Multicenter Case–Control Study in China

Qin Zhang, PhD,[1,2] Hu Ding, PhD,[1] Jiangtao Yan, PhD,[1] Wei Wang, PhD,[3] Aiqun Ma, PhD,[4] Zhiming Zhu, PhD,[5] Katherine Cianflone, PhD,[6] Frank B. Hu, MD, PhD,[7] Rutai Hui, PhD,[8] and Dao Wen Wang, MD, PhD[1]

Objective: Tissue kallikrein (TK) cleaves kininogen to produce the potent bioactive compounds kinin and bradykinin, which lower blood pressure and protect the heart, kidneys, and blood vessels. Reduction in TK levels is associated with cardiovascular disease and diabetes in animal models. In this study, we investigated the association of TK levels with event-free survival over 5 years in Chinese first-ever stroke patients.

Methods: We conducted a case–control study with 1,268 stroke patients (941 cerebral infarction, 327 cerebral hemorrhage) and 1,210 controls. Plasma TK levels were measured with an enzyme-linked immunosorbent assay. We used logistic regression and Cox proportional hazards models to assess the relationship between TK levels and risk of first-time or recurrent stroke.

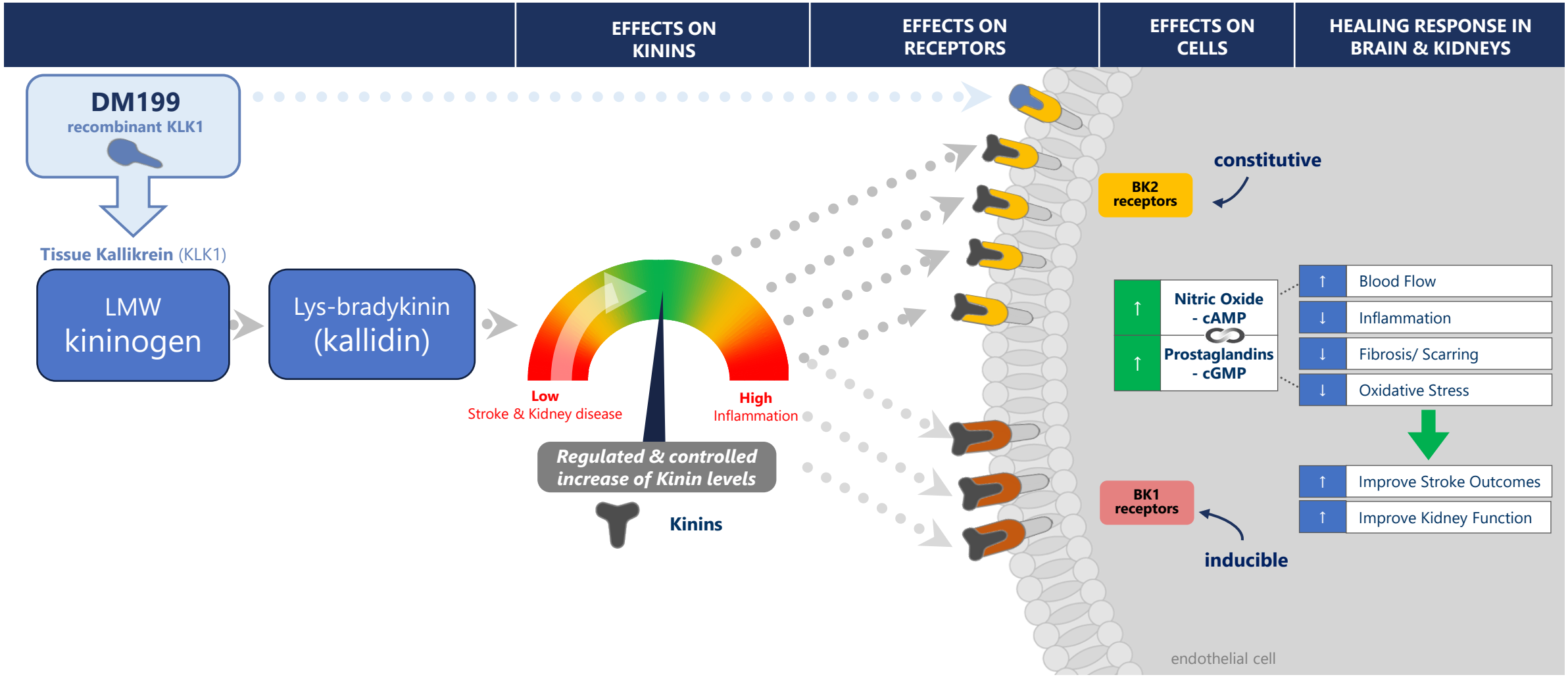
Results: Plasma TK levels were significantly lower in stroke patients (0.163 ± 0.064mg/l vs 0.252 ± 0.093mg/l, $p <$ 0.001), especially those with ischemic stroke. After adjustment for traditional risk factors, plasma TK levels were negatively associated with the risk of first-ever stroke (odds ratio [OR], 0.344; 95% confidence interval [CI], 0.30–0.389; $p < 0.001$) and stroke recurrence and positively associated with event-free survival during 5 years of follow-up (relative risk, 0.82; 95% CI, 0.74–0.90; $p < 0.001$). Compared with the first quartile of plasma TK levels, the ORs for first-ever stroke patients were as follows: second quartile, 0.77 (95% CI, 0.56–1.07); third quartile, 0.23 (95% CI, 0.17–0.32); fourth quartile, 0.04 (95% CI, 0.03–0.06).

Interpretation: Lower plasma TK levels are independently associated with first-ever stroke and are an independent predictor of recurrence after an initial stroke.

ANN NEUROL 2011;70:265–273

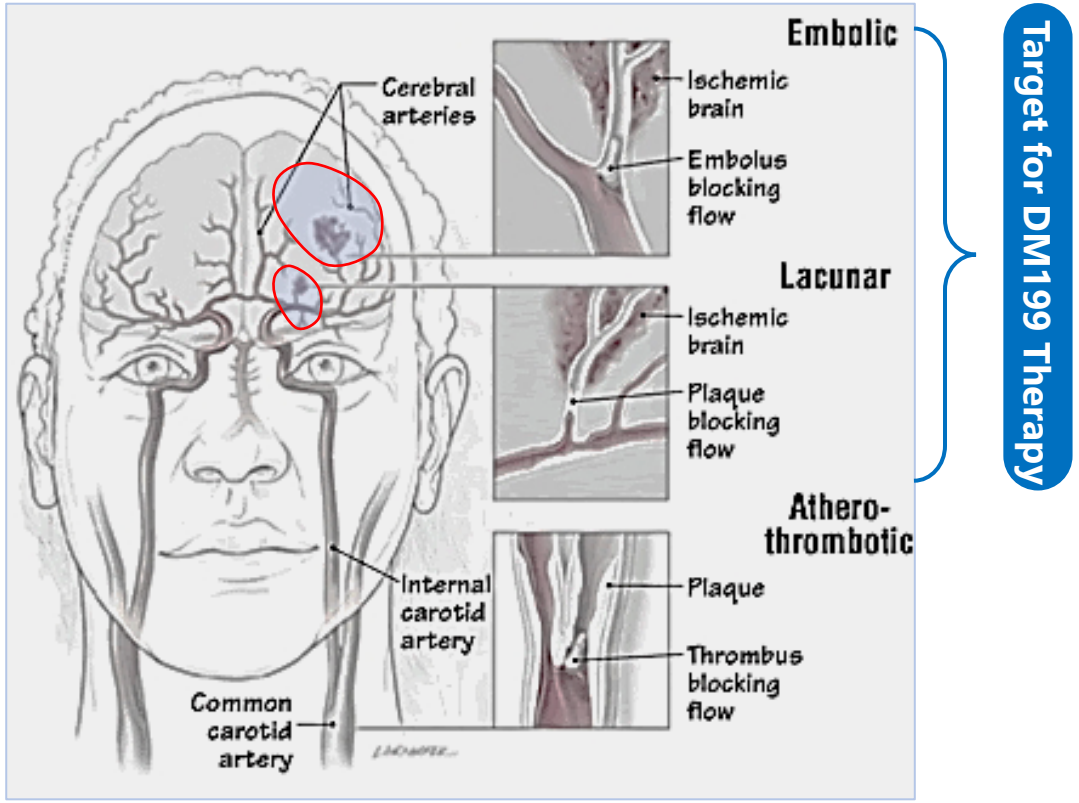
DM199 Reactivating the Kallikrein System Function

Rationale of DM199 replacement therapy



Rationale for KLK1 Therapy According to Stroke Subtypes

Types of ischemic stroke



Pathophysiological Rationale

- **Large vessel occlusion (LVO)/Cortical strokes:** are occlusions of the internal carotid artery or of the proximal segments (M1, M2) of the middle cerebral artery. Causes: a local acute thrombotic event or an embolism from a ruptured plaque in an extracranial artery, or from the heart (for instance, atrial fibrillation or congenital shunts).
- **Small vessel occlusion (SVO) and medium vessel occlusion (MVO) strokes** are areas of subcortical injury often caused by a clot, whose formation is adversely affected by the high blood pressure from the feeding artery upstream of the site of occlusion.
- **DM199** may improve the outcomes by actions at the level of the plaque (stimulating the coverage of the ruptured plaque by local endothelial cells and progenitor cells from the circulation), and at the level of damaged brain (inhibiting inflammation and propagation of ischemia to the surrounding viable tissue).

Initial Clinical Evidence[1,2]

- Treatment with urinary KLK1 significantly improved the excellent outcomes of SVO & MVO strokes but not LVO strokes; reason being the difficulty to mount an effective collateral circulation.

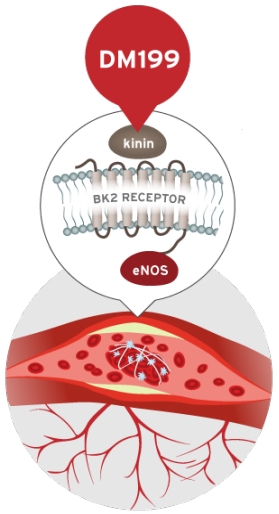
DM199 ReMEDy Phase 2 Stroke Study

- DM199 appeared most effective in patients with SVO and MVO strokes.

1. Brain and Behavior. 2020;10:e01461. https://pubmed.ncbi.nlm.nih.gov/31793238/
2. Pak. J. Pharm. Sci., Vol.28, No.4(Suppl), July 2015, pp.1505-1510. http://www.pjps.pk/wp-content/uploads/pdfs/28/4/Supplementary/12-SUP-292.pdf

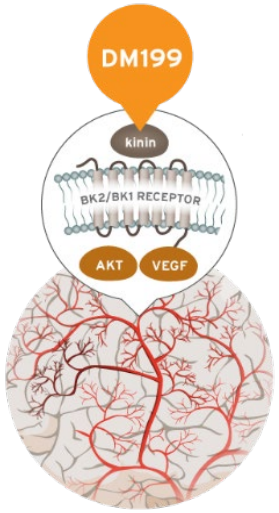
www.health.harvard.edu/a_to_z/lacunar-stroke-a-to-z

DM199 Replacement Therapy: Short- and Long-Term Protection



THERAPY INITIATION
Within 24 hours

PROMOTES
Microcirculation & anti-inflammation
(short-term blood flow)
+
IMPROVES
Neurovascular Coupling
(brain activity-blood flow balance)



THERAPY CONTINUATION
For 21 days

INHIBIT
Neuronal Apoptosis & Inflammation in area at risk
(protects neuro cell)
+
PROMOTE
Accelerates Network Reconstitution
(improving neuron plasticity)

ONSET | DAY 2 | DAY 7 | DAY 21

DM199 Resets autoregulatory mechanisms of BP control → control hypertensive crisis and subsequent hypotension

DM199 can modulate both innate and adaptive immune response.

DM199 promotes survival by inducing anti-apoptotic pathways and increasing ATP availability

DM199 may improve neuronal plasticity through the IGF pathway and activation of neurotrophic GFs

DM199 improves endothelial function and has direct vasodilating action on collaterals.

DM199 stimulates angiogenesis and arteriogenesis

DM199 promotes plaque stabilization, thereby preventing stroke recurrence

KLK1 Therapy to Reduce Stroke Recurrence by Stabilizing Plaque

General

Inhibit plaque inflammation, oxidative stress and apoptosis

- Polarization of macrophages toward the M2 phenotype.[6]
- Suppression of ROS through TLR4/NF-κB signalling pathway.[7]
- Inhibition of apoptosis via phosphorylation of Akt, glycogen synthase kinase-3beta, and Bad.[8]

Specific

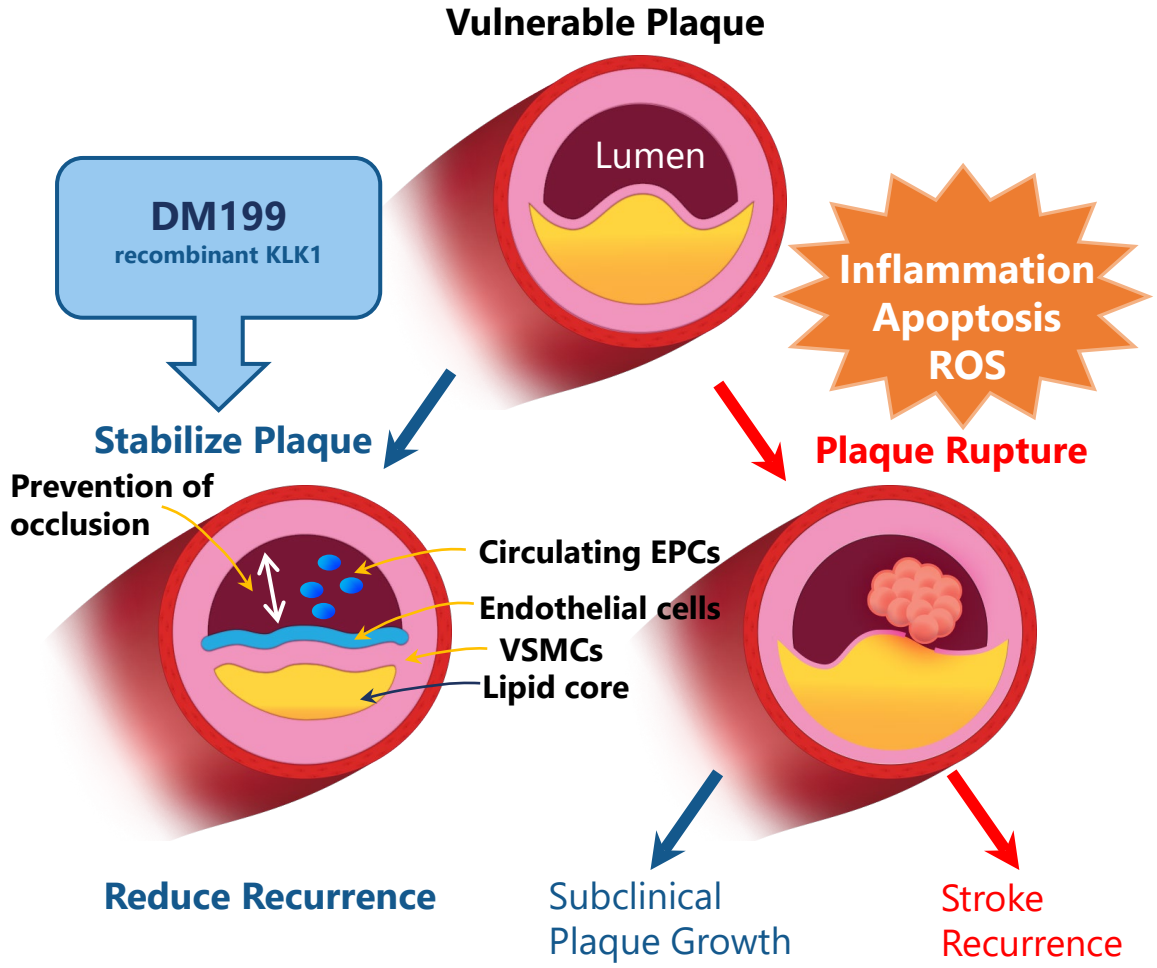
Small vessel/Lacunar strokes: Control of high blood pressure and plaque stabilization

- Prevention of additional occlusion of deep penetrating arterioles via inhibition of (i) mural cell constriction, (ii) vascular smooth muscle cell growth, and (iii) hypertension-induced thrombosis of nearby arterioles: kinin B2 receptor, cGMP, and MAPK mediated.[3,4,5]

Large vessel occlusion/Cortical strokes: Prevention of new embolisms

- Healing of the culprit (embolic) plaque and evolving plaques via (i) coverage of the rupture site by endothelial cells and (ii) recruitment of endothelial progenitor cells (EPCs) from the circulation: kinin B2 receptor - nitric oxide mediated.[1,2]

Plaque Instability Post Stroke



1. Kraenkel et al. Circulation, 2013;127:594–603; 2. Spinetti et al. Circulation Research. 2011;108:284–93; 3. Yemisci et al. Nat Med. 2009 Sep;15(9):1031-7; 4. Lan et al. Int J Stroke 2014 Jun;9(4):533-5; 5. Emanueli et al. Arterioscler Thromb Vasc Biol 2000, Nov;20(11):2379-85; 6. Hu et al. FASEB J 2019 Jul;33(7):8436-52; 7. Wei et a. Front. Neurol., Front Neurol 2018 Jun 5;9:403; 8. Jin et al. J Biol Chem 2005 Mar 4;280(9):8022-30.

KLK1 MOA Summary for Treatment of Stroke and Recurrent Stroke

- **Protein replacement therapy restores homeostasis for stroke patients**
  - Provides natural regenerative protein to reverse disease-associated deficit of KLK1
- **Acute Ischemic Stroke**
  - Limiting extension of damage plus aiding natural mechanisms of repair beyond the acute stage
- **Stroke recurrence**
  - Aiding plaque stabilization and thereby preventing embolism and occlusion
  - Neurovascular coupling

Stroke Overview & Review of DM199 Clinical Program

Dr. Kasner

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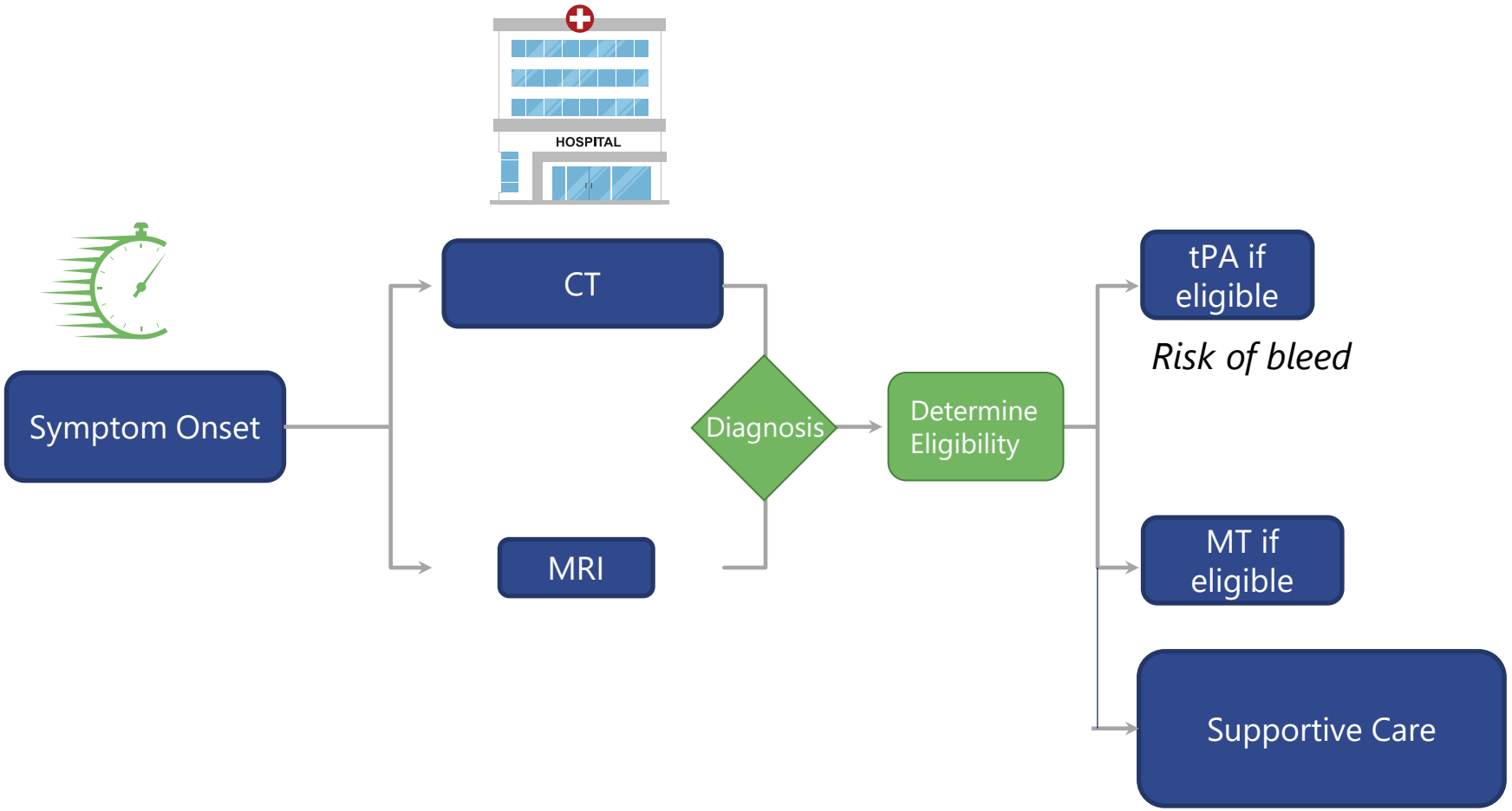
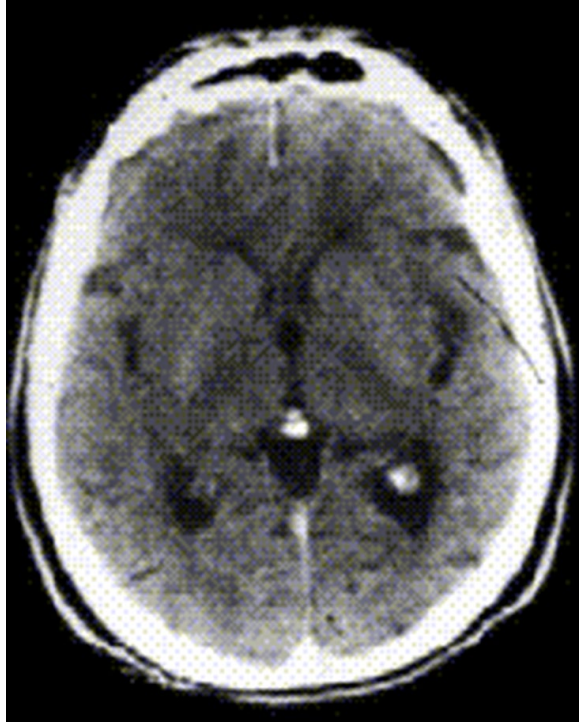
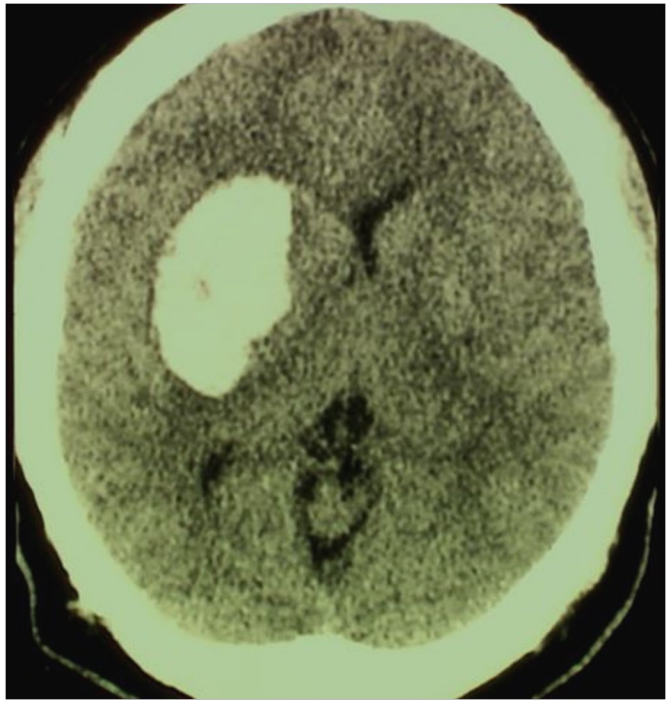
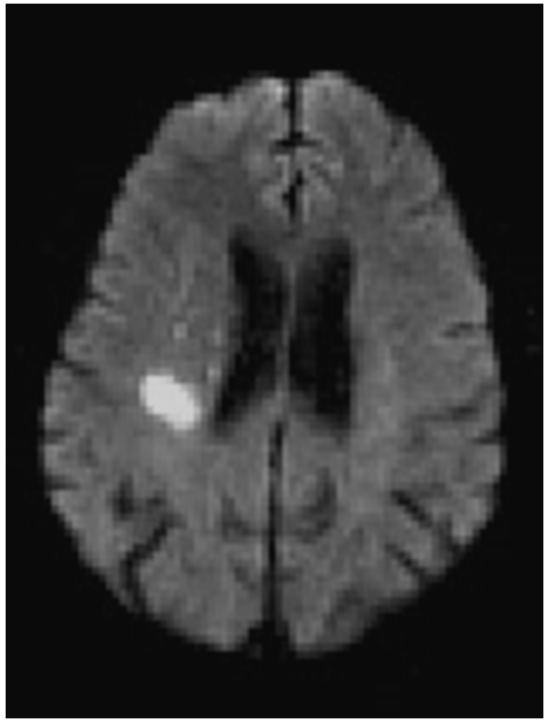
AIS Treatment Options Today

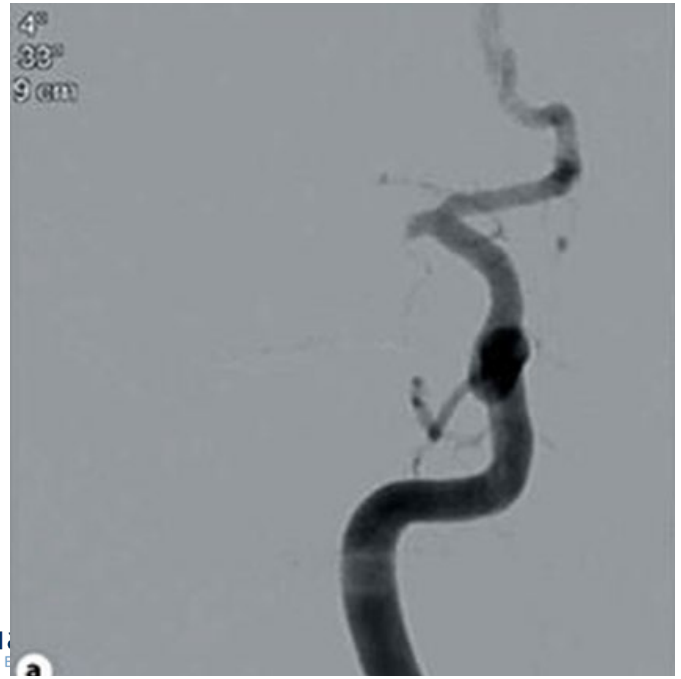
Origination | Diagnosis | Treatment Decision | Monitoring







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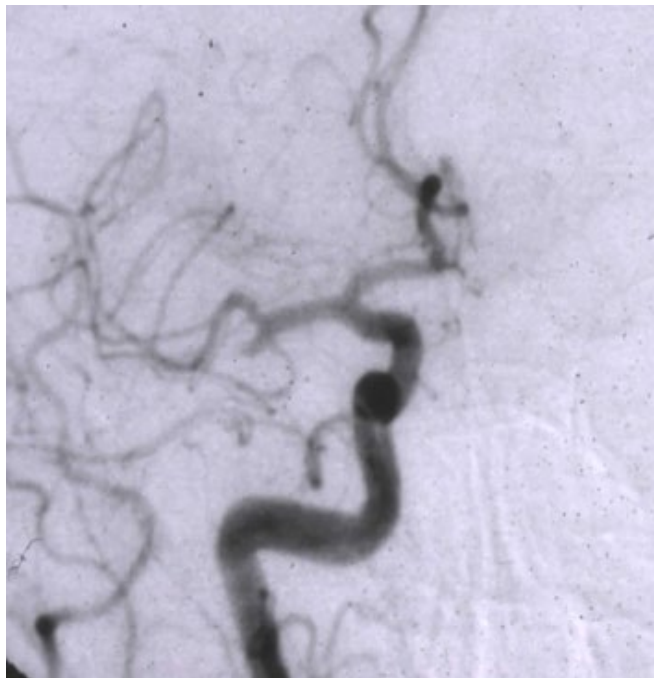
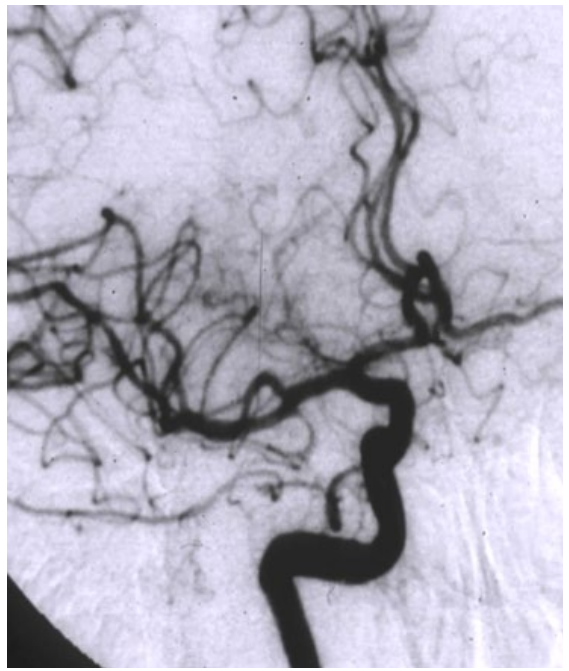
33°
9 cm





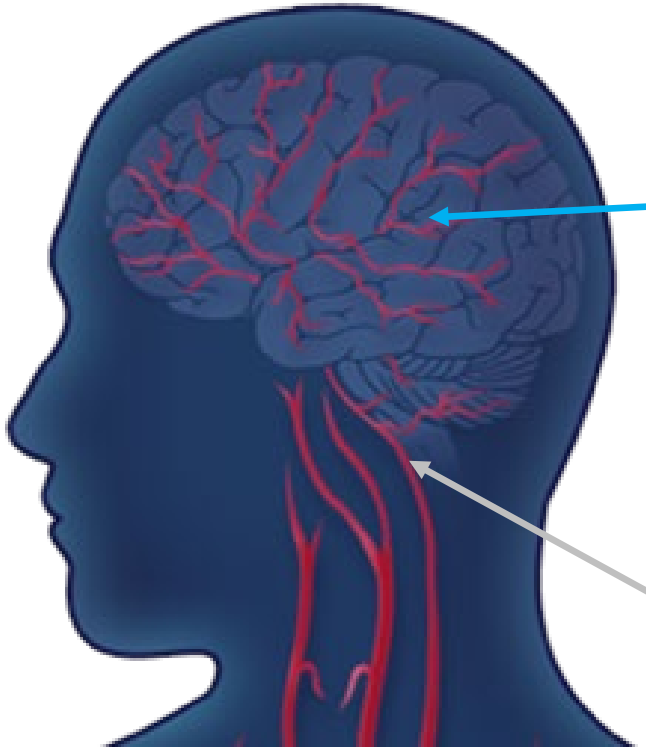
Current Treatment Landscape for AIS

IV Alteplase (tPA)	• Alteplase (recombinant tissue plasminogen activator) is the only FDA-approved medical therapy indicated for AIS • Utilization limited to patients presenting within 4.5 hours of onset of stroke with neurological deficit (utilized in ~5% of all AIS patients) • Untargeted mechanism of action acts throughout circulatory system • Carries risk of bleeding including intracranial hemorrhage - up to 6%

Mechanical Thrombectomy (MT)	• Endovascular surgical procedure to physically remove a clot
	  ○ For large vessel occlusion (LVO) stroke
	  ○ High risk procedure
	• Guidelines recommend treatment window within 6 hours and up to 24 hours of onset of stroke in selected patients
	• Limited to comprehensive stroke centers with specialists

DM199 Treatment Option for AIS

Potential treatment for up to 80% of AIS patients with no therapeutic options



tPA & MT Together Treat Less Than 20% of AIS Patients

Stroke Type	0-4.5 hr	4.5-9 hr	9-12 hr	12-24 hr	>24 hr
Small / Medium Vessel Occlusion (SVO/MVO)	**tPA**[1]	**DM199**[2]			
Large Vessel Occlusion (LVO)	**Mechanical Thrombectomy (MT)**				

tPA (Activase®)
Limited to 4.5 hrs

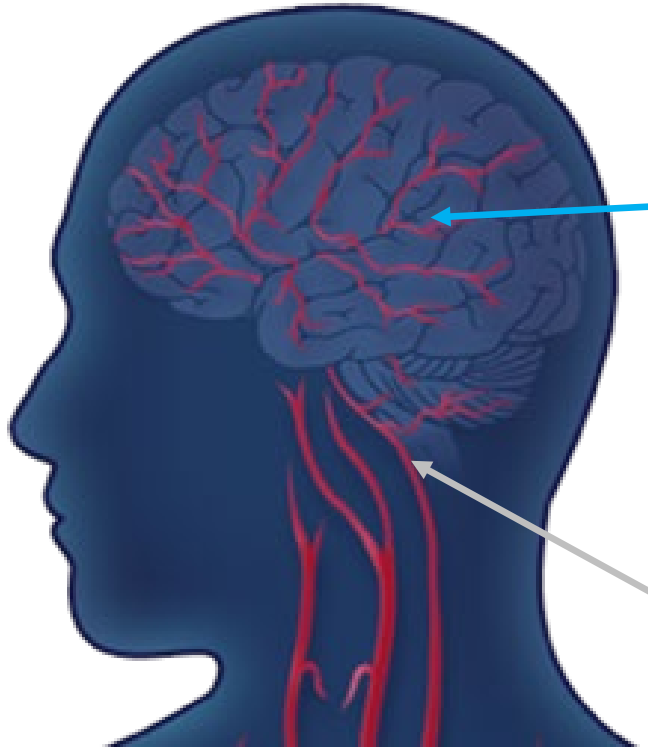
MT
Limited to LVO strokes
within 6 hours & up to 24hrs

1. tPA (Tissue Plasminogen Activator),
2. DM199 P2 improved excellent outcomes and reduced deaths in non-MT treated (non-LVO strokes) with limited effect in MT pretreated. Urinary KLK1 improved SVO & LA strokes, no effect in LVO strokes in 2 clinical studies (PMID: 31793238 and 26431650)

DM199 Future Frontline Therapy Opportunity

Potential safe treatment option for all stroke patients (ischemic & hemorrhagic)[3]

tPA & MT Together Treat Less Than 20% of AIS Patients



Stroke Type	0-4.5 hr	4.5-9 hr	9-12 hr	12-24 hr	>24 hr
Small / Medium Vessel Occlusion (SVO / MVO)	**tPA[1]**	**DM199[2]**			
Large Vessel Occlusion (LVO)	**Mechanical Thrombectomy (MT)**				

tPA (Activase®)
Limited to 4.5 hrs

MT
Limited to LVO strokes
within 6 hrs & up to 24hrs

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3. In ruptured intracranial aneurysm patients, Urinary KLK1 reduced morbidity and mortality https://onlinelibrary.wiley.com/doi/full/10.1002/brb3.1060. Also, reduced vasospasm in preclinical study https://www.tandfonline.com/doi/full/10.1080/01616412.2015.1110305

DM199 ReMEDy Phase 2 AIS Study

Design built upon on approved Kailikang®, human urinary KLK1 studies

Design	Randomized, double blind, placebo controlled
Sample Size & Treatment	N=92 (Evaluable N=91) First dose IV (1 µg/kg) **within 24 hours of stroke** symptoms onset followed by 3 weeks SC (3 µg/kg) dosing every 3rd day
Treatment Period	21 days treatment, primary endpoint at 90 days
Inclusion Criteria	Moderate stroke severity: NIHSS score 6 to 24; mRS<4 Age 18+, confirmed acute ischemic stroke via CT scan or MRI Allowed pretreatment with MT (for large vessel occlusion strokes) and/or tPA - 44 participants received MT prior to enrollment
Exclusion Criteria	Hemorrhagic stroke mRS>4
Primary Endpoint	Safety and tolerability
Secondary & Exploratory Endpoints	NIHSS at day 90 Modified Rankin Score (mRS) and Barthel Index (BI) Stroke recurrence Kidney function (eGFR)
Biomarkers	KLK1 levels, nitric oxide, prostaglandins, C-reactive protein

DM199 ReMEDy Phase 2 AIS Baseline Characteristics

	Placebo (N=45)	DM199 (N=46)
Age (Years)		
Mean	71.7	69.9
Range	31-95	38-95
Sex n (%)		
Male	28 (62%)	25 (54%)
Female	17 (38%)	21 (46%)
NIHSS Baseline Score		
Mean	12.2	11.3
Median	10	10
Administered		
tPA + MT	12 (27%)	5 (11%)
MT	12 (18%)	16 (35%)
tPA	8 (18%)	13 (28%)

MT- mechanical thrombectomy
tPA – tissue plasminogen activator

DM199 ReMEDy Phase 2 AIS Safety Profile

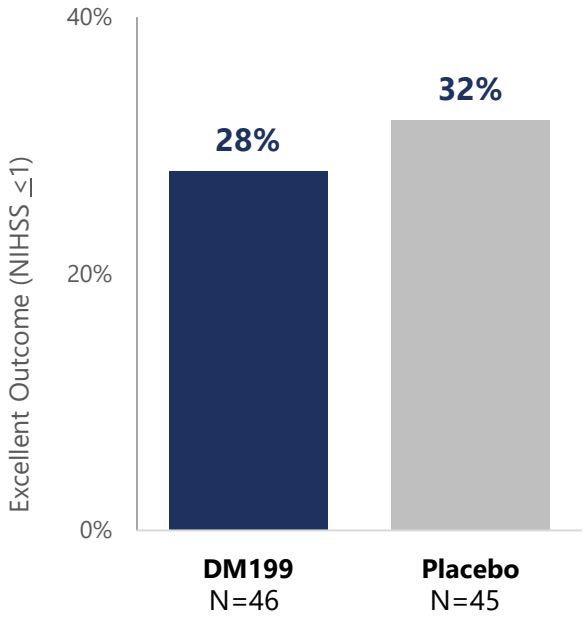
- No serious adverse events related to DM199 (2 possible/ 1 probable)
- No discontinuations or dosing interruptions due to tolerability
- No hypotension difference compared to placebo
- Most common related adverse events observed, all of which self-resolved:
  - Constipation - DM199 28; Placebo 14
  - Nausea - DM199 9; Placebo 4
  - Headache - DM199 7; Placebo 5

ReMEDy Phase 2 AIS Excellent Outcomes (NIHSS≤1)

- DM199 target patient population - excluding MT participants, patients with large vessel occlusion

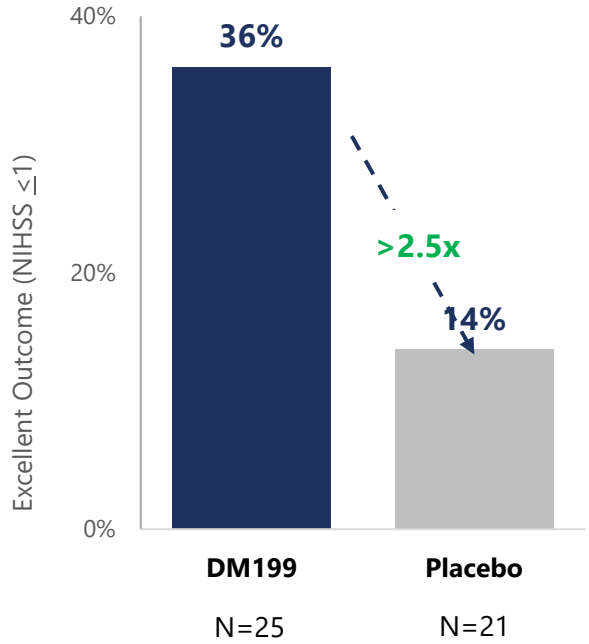
DM199 Excellent Outcomes (NIHSS ≤1) **at 90 Days**

Overall



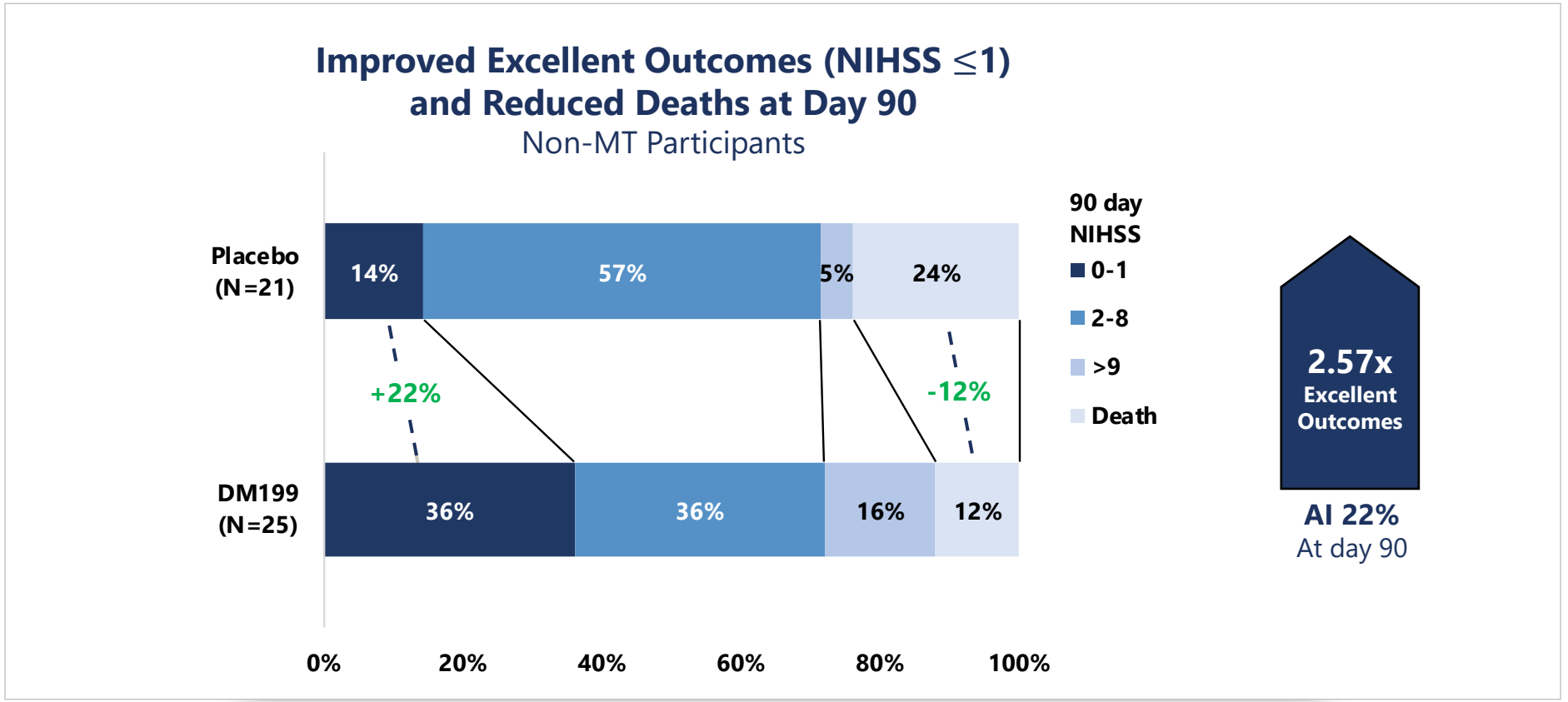
Excludes Mechanical Thrombectomy (MT)

Target Patient Population



DM199 >2.5x Improvement in Excellent Outcomes in ReMEDy Study

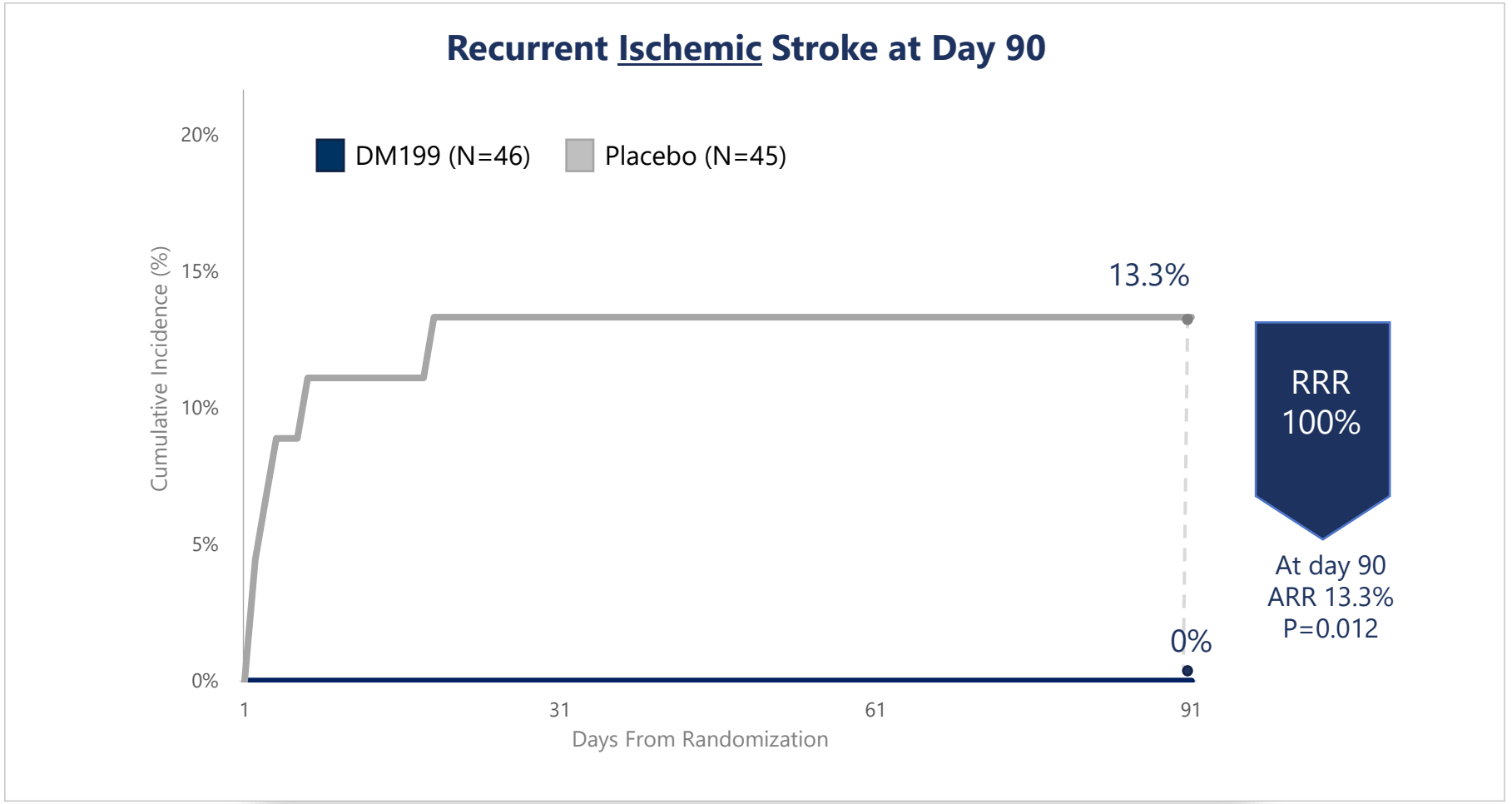
Consistent Signals for Clinical Benefit Observed



22% Absolute Increase in Excellent Outcome (NIHSS ≤1) and 12% Absolute Decrease in Deaths vs. Placebo With 24 Hour Treatment Window

AI – Absolute increase

DM199 Significant Reductions in Recurrent Ischemic Stroke in ReMEDy Study



RRR - Relative risk reduction;
ARR - Absolute risk reduction

Need for New AIS Treatment Options for Stroke and Stroke Recurrence

- DM199 may address the lack of innovation in stroke and stroke recurrence research
  - Novel approach to improving blood flow to the brain
- Expand treatment window to 24 hours for the majority of stroke AIS
- Encouraging signals in Phase II study vs placebo with excellent safety profile
  - Excellent outcomes improvements – 22% increase
  - Stroke recurrence reduction – 13% absolute reduction
- Low clinical bar for meaningful clinical efficacy
  - tPA (Activase®) - 11% excellent outcomes with 3-hour treatment window
  - Brillinta® - 1.1% absolute stroke recurrence reduction - label expansion November 2020

Review of DM199 Phase 2/3 Program

Dr. Alcorn

DiaMedica THERAPEUTICS

DM199 ReMEDy2 Phase 2/3 AIS Trial

Preparing to Commence Enrollment Summer 2021

Design	Randomized, double blind, placebo controlled, parallel groups
Sample Size	~350 participants First dose IV (1 µg/kg) **within 24 hours of stroke** symptoms onset followed by 3 weeks SC (3 µg/kg) dosing (2x weeks,)
Treatment Period	Up to 22 days treatment, primary endpoint at 90 days
Adaptive Study Design	Interim analysis planned with potential to adjust study sample size, if necessary
Inclusion Criteria	Moderate stroke severity: NIHSS score 5 to 20 Age 18+, stroke confirmed via CT scan or MRI
Exclusion Criteria	Large vessel occlusion (excludes MT) tPA (alteplase®) Hemorrhagic stroke
Primary Endpoint	Excellent Outcomes, mRS=0-1 at day 90
Secondary Endpoints	Stroke recurrence mRS shift, NIHSS and Barthel index Deaths
Biomarkers	KLK1 levels, nitric oxide, prostaglandins, C-reactive protein

DM199 ReMEDy2 Phase 2/3 Builds Upon ReMEDy Phase 2 Findings

Targeting the ~80% of patients with no treatment option

Items	ReMEDy Phase 2/3	ReMEDy Phase 2
Patients	~350	91
Sites	~75	12
SC Dosing frequency	2x week	Every 3rd day
NIHSS Inclusion	5-20	6-24
Large Vessel Occlusion / MT	No	Yes
tPA (Activase®)	No	Yes
Average time to first dose	Anticipate 12 hours	~18 hours

DM199 ReMEDy2 Phase 2/3 Interim Analysis Plan Proposed

Potential Outcomes

Stats Plan	• ~350 participants • 90% power at 15% absolute improvement in excellent outcomes
Interim Analysis	• Comparison of DM199 to placebo when 40% patients complete 90 days follow-up • DiaMedica will remain blinded to data, DSMB will provide one of three outcomes below
Potential Outcomes from DMC	1. Continue as planned 2. Increase sample size 3. Futility – inadequate improvements in excellent outcomes and stroke recurrence
Regulatory	• Preparing to file application for fast track designation • Plan to request FDA meeting to discuss path and powering for stroke recurrence
Upcoming Milestones	• IND Filing – Q1 2021 • First Patient Dosed – Summer 2021

Commercial Landscape/Q&A

DiaMedica THERAPEUTICS

Stroke: Major Unmet Need for Treatment Options

Up to 80% of AIS Patients Have no Treatment Options

Acute Ischemic Stroke (AIS)

Blockage of blood flow in brain

- Leading cause of adult disability
- 2nd leading cause of death worldwide
- Recurrent strokes - 25% of strokes
  - More disabling, costly & fatal[1]

15 million strokes per year globally

800,000 strokes per year in US

87% acute ischemic strokes

~550,000: DM199 US initial target market

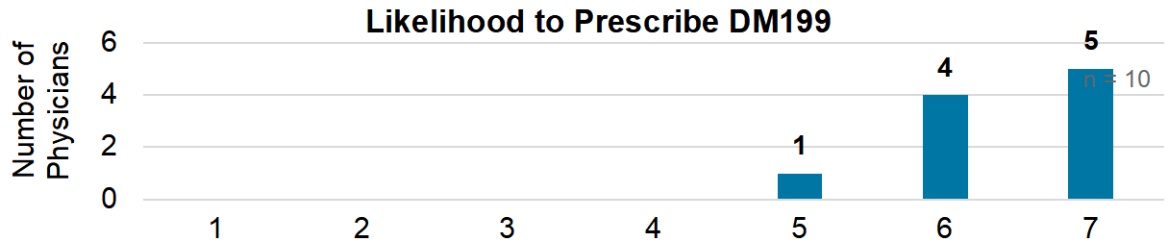
>$10B estimated annual U.S. revenue opportunity

Stroke and chronic kidney disease closely linked: 34% of Stroke Patients have CKD

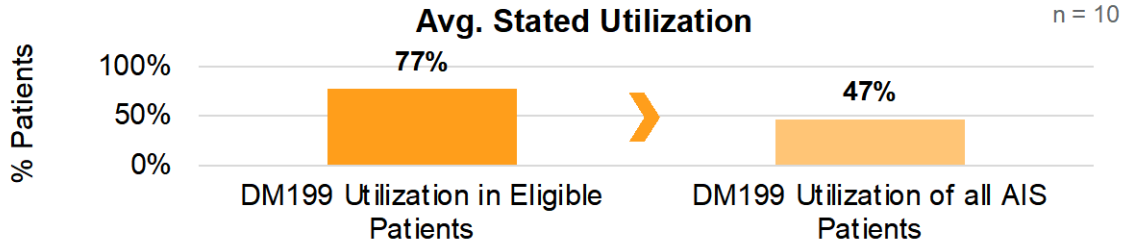
AIS Physician Primary Research Readout

Reaction to DM199—Physician Adoption

Physicians are excited about DM199 and expect to use it in most eligible patients, especially if DM199 is recommended by guidelines and is approved by their hospital Pharmacy and Therapeutics committee.



Q. How likely are you to prescribe Product X? (On a scale of 1 to 7, where 7: extremely likely and 0: extremely unlikely.)



Q. If the product met endpoints and was approved by the FDA, what percent (%) of your AIS patients would get this product?

Interviews with Physicians and Payers, conducted July-August 2020.

Reactions of market stakeholders to AIS target product profile

Neurologist and Payer views on the DM199 profile in AIS

Neurologist reactions to DM199 AIS product profile

- ✓ 24-hour window for treatment eligibility
- ✓ Compelling efficacy and safety
- ✓ Novel MOA
- ✓ Provides treatment for patients without other options
- ✓ No drug-to-drug interactions with SOC
- ✓ Does not require specialized training or patient monitoring

Payer reactions to DM199 AIS product profile

- ✓ Payers believe DM199 meets a significant unmet need for patients who are ineligible for tPA and thrombectomy
- ✓ Payers would be required to cover DM199 with no restrictions and regardless of cost if initial treatment is in the hospital

*Market research undertaken 2020, US HCPs and payers, conducted by Syneos Health. Research involved a survey of 40 neurologists, interviews with 10 neurologists and 12 payer interviews.

Q&A

DiaMedica THERAPEUTICS