O-Force

Catalytide

- An innovative peptide drug for neurodegenerative diseases

Non-Confidential Introduction

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O-Force Corporate Overview

O-Force is a biotechnology company developing catalytic peptide therapeutics (“Catalytides”) capable of directly degrading disease-associated proteins and pathological deposits.

2015 **Discovered catalytic peptides and named them “Catalytide”.**
2017 Founded O-Force, Ltd.
2018 Started pre-clinical studies based in a lab at Kochi University.

Publications and Patents
https://o-force.info/

*Patents covering the Catalytide platform and therapeutic applications
Details available under NDA

Toshifumi Akizawa, PhD.

CEO

PhD (Medicall Sciences)
Pharmacist

Inventor of Catalytides
30+ years in peptide pharmacology

Specially Professor of Kochi University





Rina Nakamura, PhD.

Director

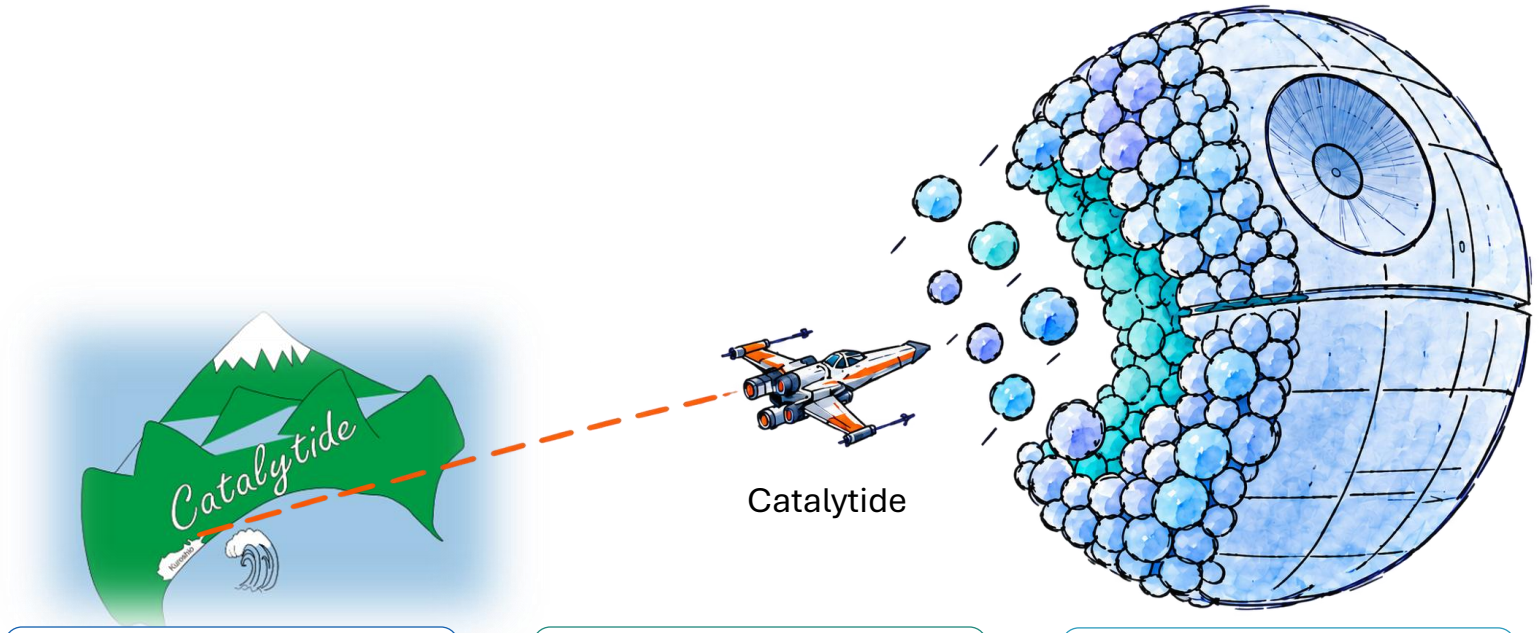
PhD. (Pharmaceutical Sciences)
Pharmacist

Lead scientist for Catalytide programs
Pharmacology and translational development

Specially Appointed Lecturer of Kochi University

Registered Office
3454 Irino Kuroshio-cho,
Hatata-gun, Kochi, Japan
https://o-force.info/
rina@OForce0824.onmicrosoft.com
Lab
At School of Medicine, Kochi University,

What is Catalytide?



O-Force locates at Kuroshio-cho, thesmall island of Japan

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Catalytide is the small hydrolase-peptide possessing serine protease-like activity

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Catalytides destroy the large pathogenic aggregated proteins

Catalytide enters small spaces in aggregated proteins / immune complexes.

The concept is similar to Star Wars. A small X-wing fighter attacks the giant Death Star at its weak point.

What is Catalytide?

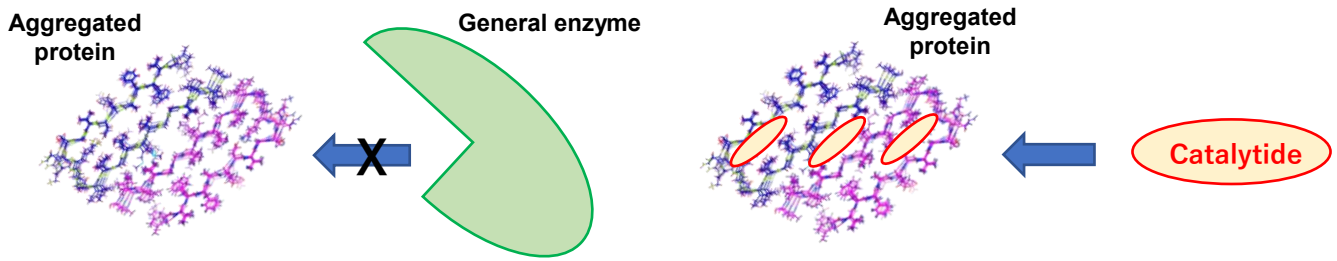
We were the first to discover that a synthetic short peptide can exhibit proteolytic activity and termed as “Catalytide”.

Key FeaturesFirst

- synthetic short peptides discovered to exhibit enzyme-like hydrolytic activity
- Serine protease-like mechanism with a catalytic triad
- Small size enables penetration into protein aggregates
- Chemically synthesized, allowing rapid optimization
- Stable and easy to manufacture
- Potentially safer than conventional enzymes

Unique Mechanism

- Catalytides preferentially degrade aggregated proteins.
- Because of their small size, Catalytides are thought to penetrate the internal spaces of pathogenic protein aggregates and degrade them from the inside.



Target proteins： α-Synuclein, Amyloid-β, TDP-43, SOD1, IgA immune complexes, Fibrotic collagen, etc.

Catalytide Pipeline

Target Proteins	Indications	Discovery	Pre-clinical	IND-enabling
α-Syn	PD			
Amyloid β	AD			
Tau	AD/Tauopathy			
TDP-43	ALS/FTD			
SOD-1	ALS			
IgA immune complex	IgAN			
Fibrosis of collagen	Dupuytren's Contracture			



Highlight

PD/AD

- Mouse efficacy
- Cynomolgus monkey CNS delivery

IgAN

- Human kidney tissue
- HIGA efficacy

Partnering Opportunities

Milestone Status

- Lab-scale development completed
- Ready for pre-clinical safety tests

Seeking a Pharmaceutical Partner

- We are looking for a partner that will take over the technology and lead subsequent clinical trials and CMC operations.

Our Offer

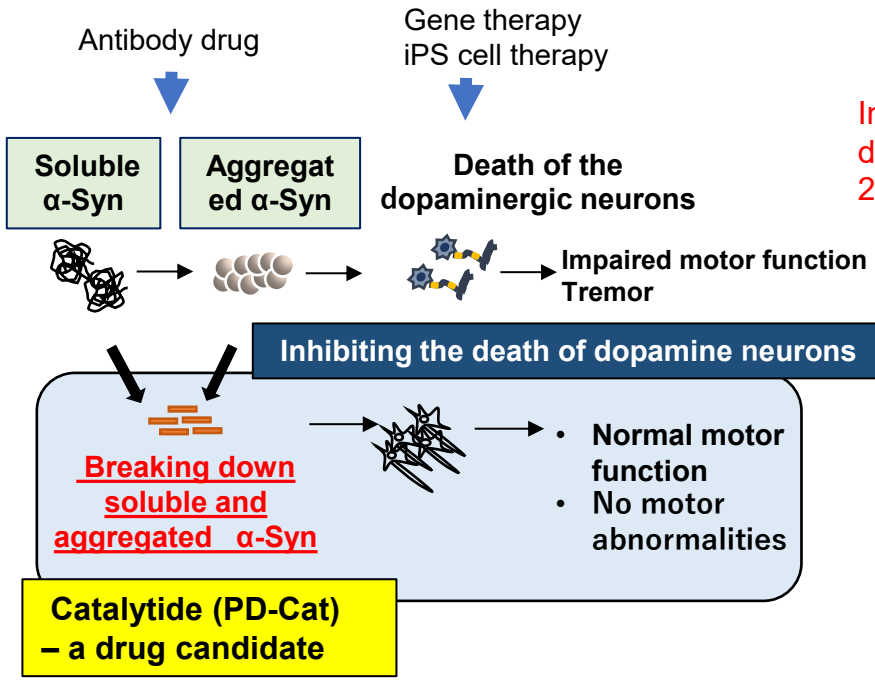
- Comprehensive unpublished data packages are available for review under a NDA.
- Flexible licensing terms
- Ongoing scientific expertise and advisory, while the pharmaceutical partner maintains full control over commercialisation.

[Development of Parkinson's Disease Treatment]

<Current Treatments>

- Parkinson's disease (PD) is a neurodegenerative disorder, causing motor impairments such as tremor, rigidity and akinesia.
- No cure to date. Existing treatments are limited to symptomatic therapy to alleviate symptoms.

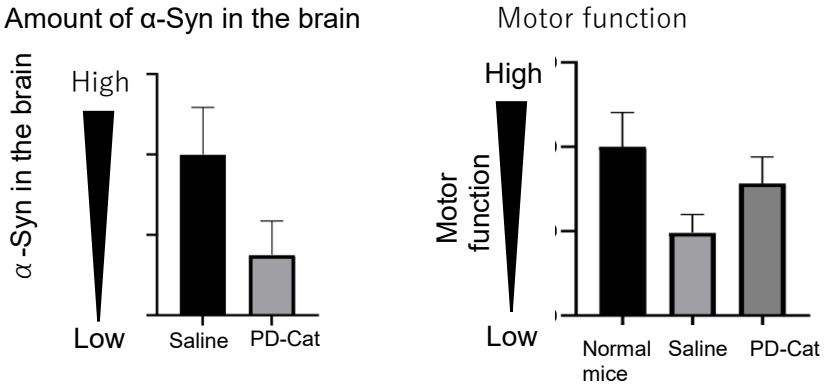
There is a need for a new approach to break down and remove α-synuclein accumulated in the brain.



<Research Goal>

To develop a cure for PD by using Catalytide which breaks down and removes α-Syn.

Results to Date



Intranasal administration of PD-Cat improves motor function by breaking down and removing α-Syn accumulated in the brain. (JP Patent App. No. 2026-067224, *α-Synuclein disaggregation agent and use thereof*)

R&D Plan

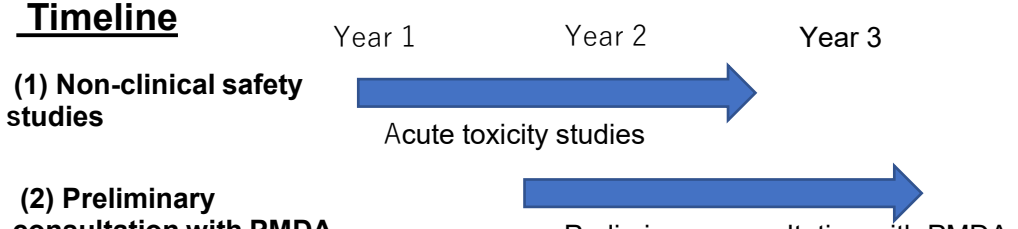
(1) Non-clinical safety studies

Conduct acute toxicity studies at an external organisation compliant with Good Laboratory Practice (GLP) standards.
Determine toxicity endpoints (immune response, organ toxicity) and obtain highly reliable data.

(2) PMDA preliminary consultation

Consult with PDMA regarding the non-clinical trial plan, evaluation parameters and observation period and formulate guidelines necessary to determine a dosage regimen for clinical trials.

Timeline

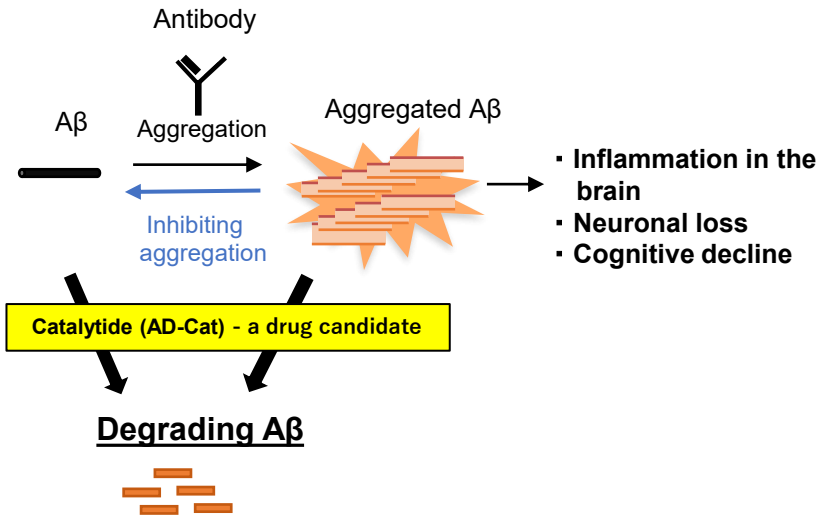


[Development of Alzheimer's Disease Treatment]

<Current Treatments>

- Alzheimer's disease (AD) is a progressive neurodegenerative disorder caused primarily by the accumulation of amyloid-β (Aβ) in the brain.
- No cure to date. Existing treatments are limited to symptomatic therapy.

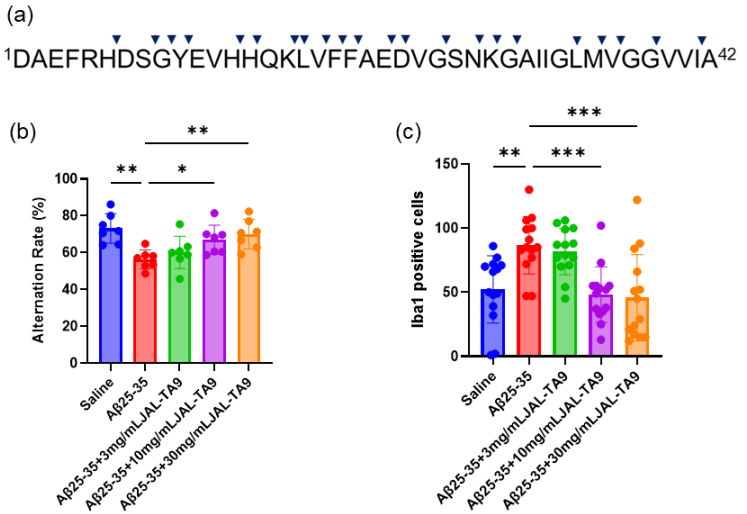
There is a need for new approaches to break down and remove amyloid-β (Aβ) accumulated in the brain.



- **Inflammation in the brain**
- **Neuronal loss**
- **Cognitive decline**

<Research Goal>
To develop a cure for AD by using Catalytide which breaks down and removes Aβ.

Results to Date



Intranasal administration of AD-Cat degrades Aβ in the brain, suppresses inflammation and improves cognitive function. (Patent No. WO2017119511, *Novel peptide exhibiting hydrolytic activity and use thereof*)

R&D Plan

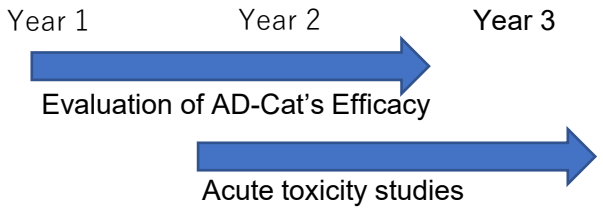
(1) Efficacy studies using genetically modified AD model mice

Evaluate efficacy in mouse models that closely mimic the human disease state. Based on acquisition of non-clinical proof of concept (POC) aimed at clinical trials, examine dosage and interval and determine method of administration and dosage.

(2) Non-clinical safety studies

Conduct acute toxicity studies at an external organisation compliant with Good Laboratory Practice (GLP) standards. Determine toxicity endpoints (immune response, organ toxicity).

Timeline

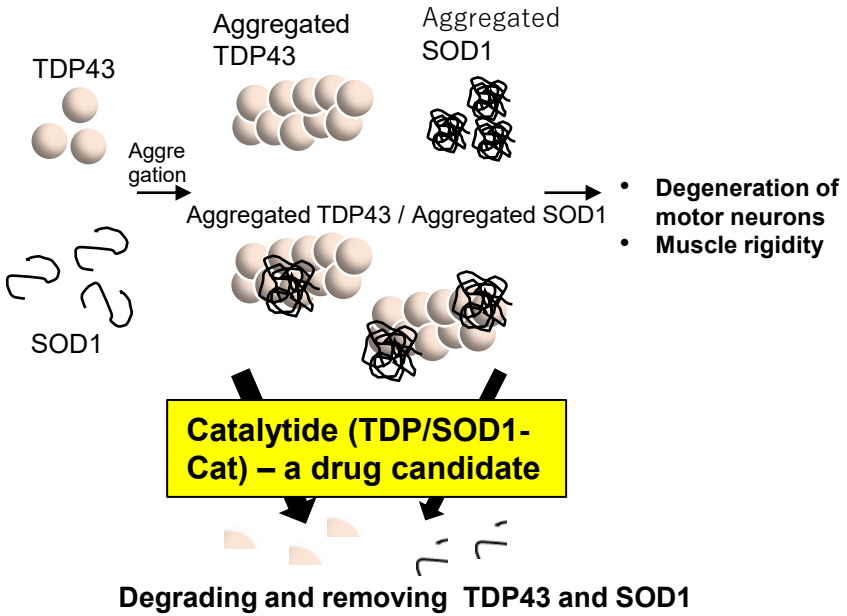


[Development of Amyotrophic Lateral Sclerosis Treatment]

<Current Treatments>

- Amyotrophic lateral sclerosis (ALS) is a progressive neurodegenerative disease caused by abnormal aggregation of TDP43 and SOD1; it is an extremely serious intractable disease that leads to death within a few years of onset due to respiratory muscle paralysis.
- No cure to date. Existing treatments are limited to symptomatic therapy.

There is a need for new approaches to break down and remove aggregated TDP43 or SOD1.



<Research Goal>
To develop a cure for ALS by using Catalytide which breaks down and removes abnormal aggregated proteins.

Results to Date

SOD1-related:

- Identification of nuclei of SOD1 aggregation
- Identification of Catalytides that degrade SOD1

TDP-43-related:

- Identification of nuclei of TDP aggregation

R&D Plan

SOD1-related:

(1) Structural optimisation of Catalytide for the degradation of Ag-SOD1

Structurally optimise the identified Catalytide and dtermine final compounds (SOD1-Cat).

(2) Evaluation of the pharmacological effects of SOD1-Cat

Evaluate pharmacological efficacy using SOD1 mutant model mice.

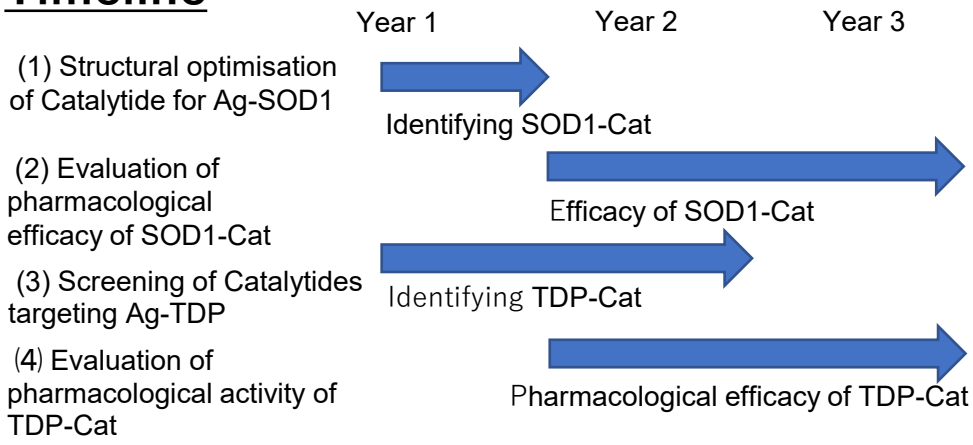
(3) Screening of Catalytides targeting Ag-TDP

Screen and structurally optimise Catalytides exhibiting Ag-TDP-degrading activity. Identify final compounds (TDP-Cat).

(4) Evaluation of pharmacological efficacy of TDP-Cat

Evaluate pharmacological efficacy using ALS model mice.

Timeline

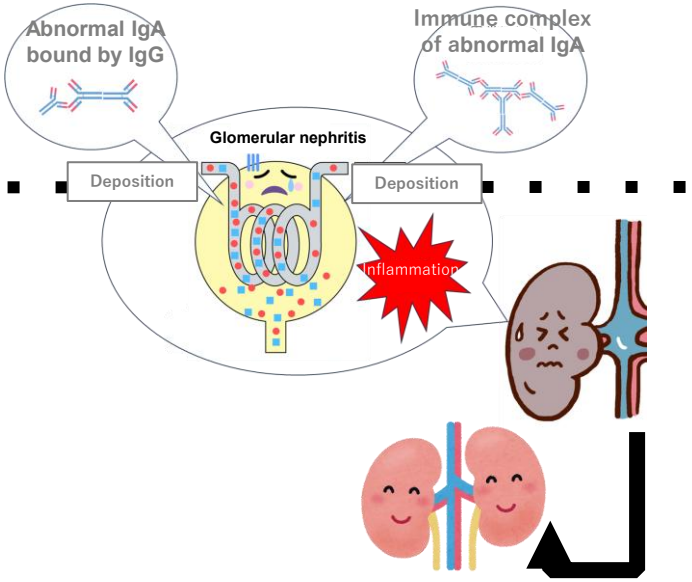


[Development of IgA Nephropathy Treatment]

<Current Treatments>

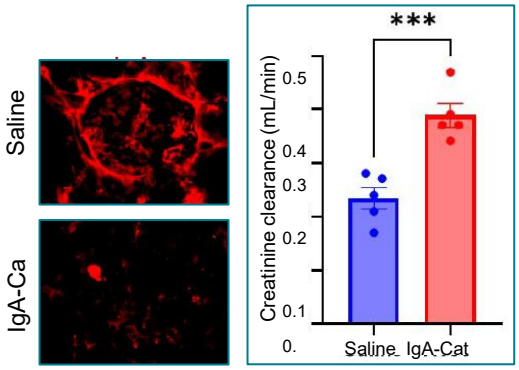
- IgA nephropathy is a type of chronic glomerulonephritis which, if left untreated, can progress to end-stage renal failure.
- No cure to date. Existing treatments are limited to symptomatic therapy (tonsillectomy and steroids).

There is a need for new approaches to break down and remove IgA immune complexes deposited in kidneys.



Results to Date

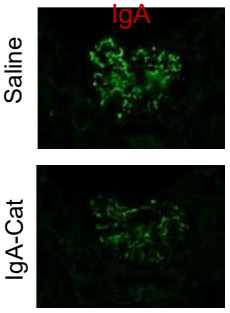
Murine *in vivo* (HIGA mouse)



IgA deposition

Renal function

Human *ex vivo* (IgAN kidney biopsy)



IgA-Cat breaks down and removes IgA immune complexes deposited in human tissue of IgA nephropathy

IgA-Cat improves renal function by breaking down and removing IgA immune complexes deposited in kidneys (JP Patent App.No. 2026-012909; *IgA complex disaggregation agent and use thereof*)

R&D Plan

(1) Non-clinical safety studies

Conduct acute toxicity studies at an external organisation compliant with Good Laboratory Practice (GLP) standards. Determine the toxicity assessment parameters (immune response, organ toxicity) and obtain highly reliable data.

<Research Goal>

To develop a cure for IgA nephropathy by using Catalytide which breaks down and removes IgA immune complexes.

Timeline

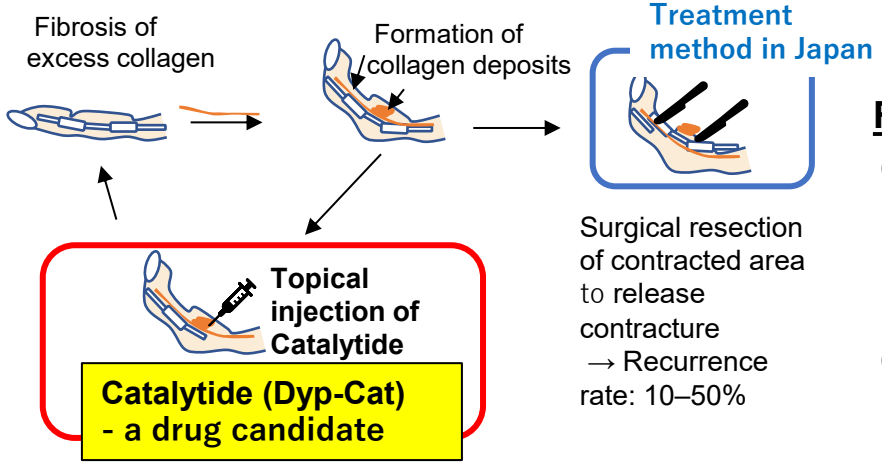
	Year 1	Year 2	Year 3
(1) Non-clinical safety studies	Acute toxicity studies	Selecting a licensee	

[Development of Dupuytren's Contracture Treatment]

<Current Treatments>

- Dupuytren's contracture is a condition in which collagen fibres in the palms proliferate excessively, leading to the formation of contracture cords.
- No cure to date. Existing treatments are limited to symptomatic therapy.

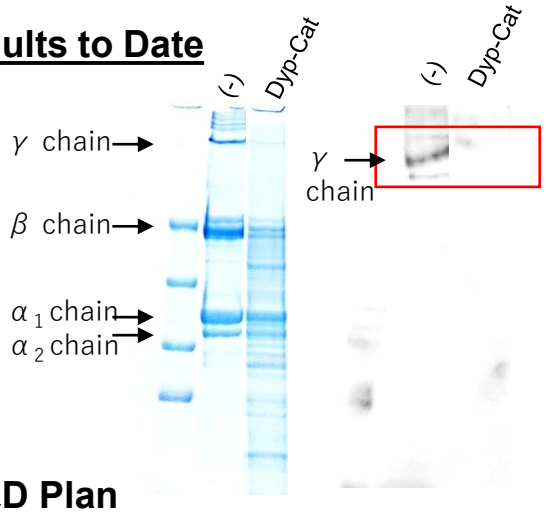
There is a need for new approaches to break down and remove fibrotic collagen.



Surgical resection of contracted area to release contracture
→ Recurrence rate: 10–50%

Breaking down and removing deposited and fibrotic collagen

Results to Date



Dyp-Cat degrades fibrotic collagen

R&D Plan

(1) Comparative studies and analysis of cleavage points

Calculate specific activity of Dyp-Cat relative to type I collagenase, an existing drug. Analyse degradation rate using fluorescently labelled collagen as a substrate. Identify specific sites of type I collagen targeted by Catalytide to demonstrate its degradative activity.

(2) Safety studies on local administration

Inject Catalytide locally around the femoral nerve in rats. Assess toxicity in nerves, muscles and subcutaneous tissues.

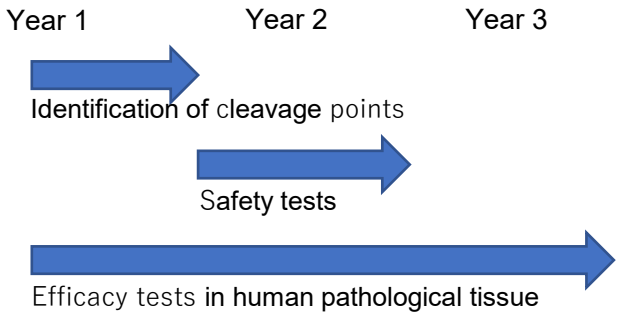
(3) Pharmacological evaluation using human pathological samples

Obtain data directly demonstrating efficacy in human pathological tissues.

<Research Goal>

To develop a cure for Dupuytren's contracture by using Catalytide which breaks down and removes fibrotic collagen.

Timeline



Contact for Business Development

Rina Nakamura, PhD.

E-mail: rina@OForce0824.onmicrosoft.com