



## DEPARTMENT OF HEALTH AND HUMAN SERVICES

### National Institutes of Health

#### Notice; Licensing and Collaboration Opportunity

**AGENCY:** National Institutes of Health, HHS.

**ACTION:** Notice.

**SUMMARY:** The invention listed below is directed to potential peptidyl therapeutics that counteract with amyloid forming IAPP and amyloid- $\beta$  in treatments of diabetes and Alzheimer's disease and serve as blood-based biomarkers for Alzheimer's disease. This technology was discovered and is being developed by the National Institute on Aging (NIA). The NIA is currently seeking a licensee and/or collaborator to further develop this technology.

**FOR FURTHER INFORMATION CONTACT:** Inquiries related to this licensing and collaboration opportunity should be directed to: Zarpheen Jinnah, Technology Transfer Manager, NCI Technology Transfer Center, 9609 Medical Center Drive, RM 1E530 MSC 9702, Bethesda, MD 20892-9702 (for business mail), Rockville, MD 20850-9702 Telephone: (240)-276-5530; Facsimile: (240)-276-5504 E-mail: [zarpheen.jinnah@nih.gov](mailto:zarpheen.jinnah@nih.gov). A signed Confidential Disclosure Agreement will be required to receive copies of unpublished information related to this invention.

**SUPPLEMENTARY INFORMATION:** The following patent application is available for licensing and/or collaboration under a Cooperative Research and Development Agreement (CRADA):

US Provisional Application No. 63/417,582.

Achieving expeditious commercialization of federally funded research and development is consistent with the goals of the Bayh-Dole Act, codified as 35 USC 200-212.

### *Background and Description of Technology:*

Over 34 million Americans are living with diabetes and an estimated 6.5 million Americans are living with Alzheimer's disease (AD). A hallmark feature of type 2 diabetes mellitus (T2DM) is the accumulation of islet amyloid polypeptide fibrils in pancreatic islets. Such accumulations form amyloid plaques, referred to as islet amyloidosis. Amyloidosis due to aggregation of amyloid- $\beta$  is key pathogenic event in AD, whereas aggregation of mature islet amyloid polypeptide (IAPP<sub>37</sub>) in human islet leads to  $\beta$ -cell dysfunction. Researchers at NIA used a bioinformatic approach to identify two novel islet amyloid polypeptide isoforms: IAPP $\beta$ , encoding an elongated propeptide and non-aggregating IAPP $\gamma$ , which is processed to mature IAPP<sub>25</sub> instead of IAPP<sub>37</sub>. They developed a quantitative selective reaction monitoring (SRM) proteomic assay to measure the isoform peptide levels in human clinical plasma and CSF from individuals with early AD and found that their levels were significantly reduced. Further, mature IAPP<sub>25</sub> derived from IAPP $\gamma$  isoform inhibits fibrillation of IAPP and amyloid- $\beta$  efficiently *in vitro*.

*Potential Commercial Applications:* The novel IAPP $\beta$  and IAPP $\gamma$  isoforms are potential peptidyl therapeutics to counteract with amyloid forming IAPP and amyloid- $\beta$  in treatments of diabetes and Alzheimer's disease and serve as blood-based biomarkers for Alzheimer's disease.

### *Competitive Advantages:*

- Peptide based anti-amyloid medicine.
- Potential market applications for neurodegenerative diseases

*Development Stage:* Pre-clinical (*in vivo* validation)

*Publications:* Liu, Q.-R., et al. Novel Hominid-Specific IAPP Isoforms: Potential Biomarkers of Early Alzheimer's Disease and Inhibitors of Amyloid Formation. (PMID 36671553) at <https://pubmed.ncbi.nlm.nih.gov/36671553/>.

Meng, Lanxia, et al. Islet amyloid polypeptide triggers  $\alpha$ -synuclein pathology in Parkinson's disease. (PMID 37150314)

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**Richard U. Rodriguez,**

*Associate Director,*

*Technology Transfer Center,*

*National Cancer Institute.*

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