7-2015-3167 | "DIBAIT": a Novel Small Peptide For Type 2 Diabetes Treatment

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Our Innovation

Based on a provocative working hypothesis, we most probably identified the molecular origin of insulin resistance (IR). Systemic IR is induced via post-translational modification (PTM) of liver key regulatory proteins. We have discovered that hepatic futile utilization of the PTM-inducing residue, resulted in the abrogation of IR and correction of the glycemic-state, in type-2-diabetes (T2D) mouse model. We further found a way to achieve this glycemic control via the utilization of novel small molecules that operate through well-defined MoA. These novel small molecules are orally bioavailable and has nice safety profile.

Application

- Providing a cure to T2D, as opposed to symptomatic treatment, by nullifying the vicious circle that starts with peripheral IR, as a result of fatty liver, and ends up as full-blown type-2-diabetes (T2D).

Pre-Clinical Results

- Significant, persistent reduction in blood glucose level demonstrated in T2D animal model (db/db mice), following once a day injection of the peptide, observed already after 1 day.
- Significant, persistent reduction in blood glucose level demonstrated in T2D animal model, following administration of DiBait therapy, observed already after 1 day.
- This reduction in blood glucose occurs in diabetic but not in normal mice.
- Significant reduction in plasma triglycerides in treated T2D mice with no effect on terminal plasma insulin or glucagon.

Opportunity

We are finishing now the process of drug-candidate selection and be ready in a few months to move to drug-development phase. A collaboration with major Pharma Company can help accelerating this process in terms of CMC, covering regulatory aspect etc. in order to bring an effective therapy to hundreds of millions of T2D patients around the world

Patent Status

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