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STK25 – a new key regulator of metabolic profile and a possible target for anti-diabetic drug

Type 2 diabetes (T2D) affects at least 285 million people worldwide and its prevalence is rapidly increasing. Understanding the molecular mechanisms controlling ectopic lipid deposition and insulin response in metabolic tissues is essential for developing new pharmacological strategies to treat T2D. Obesity and overweigh are the main risk factors for developing T2D, but nonalcoholic fatty liver disease (NAFLD) also contributes to the pathogenesis of T2D. To date, no specific therapy exists for NAFLD.

In this thesis, we describe protein kinase STK25 as a new key regulator of ectopic lipid deposition in skeletal muscle, liver and pancreas as well as whole-body metabolism. We also show that treatment with *Stk25* antisense oligonucleotides in obese mice protects against high-fat diet-induced liver steatosis, glucose intolerance and insulin resistance. These findings demonstrate that inhibition of STK25 may provide new-in-class therapeutics for NAFLD, T2D and related metabolic complications.

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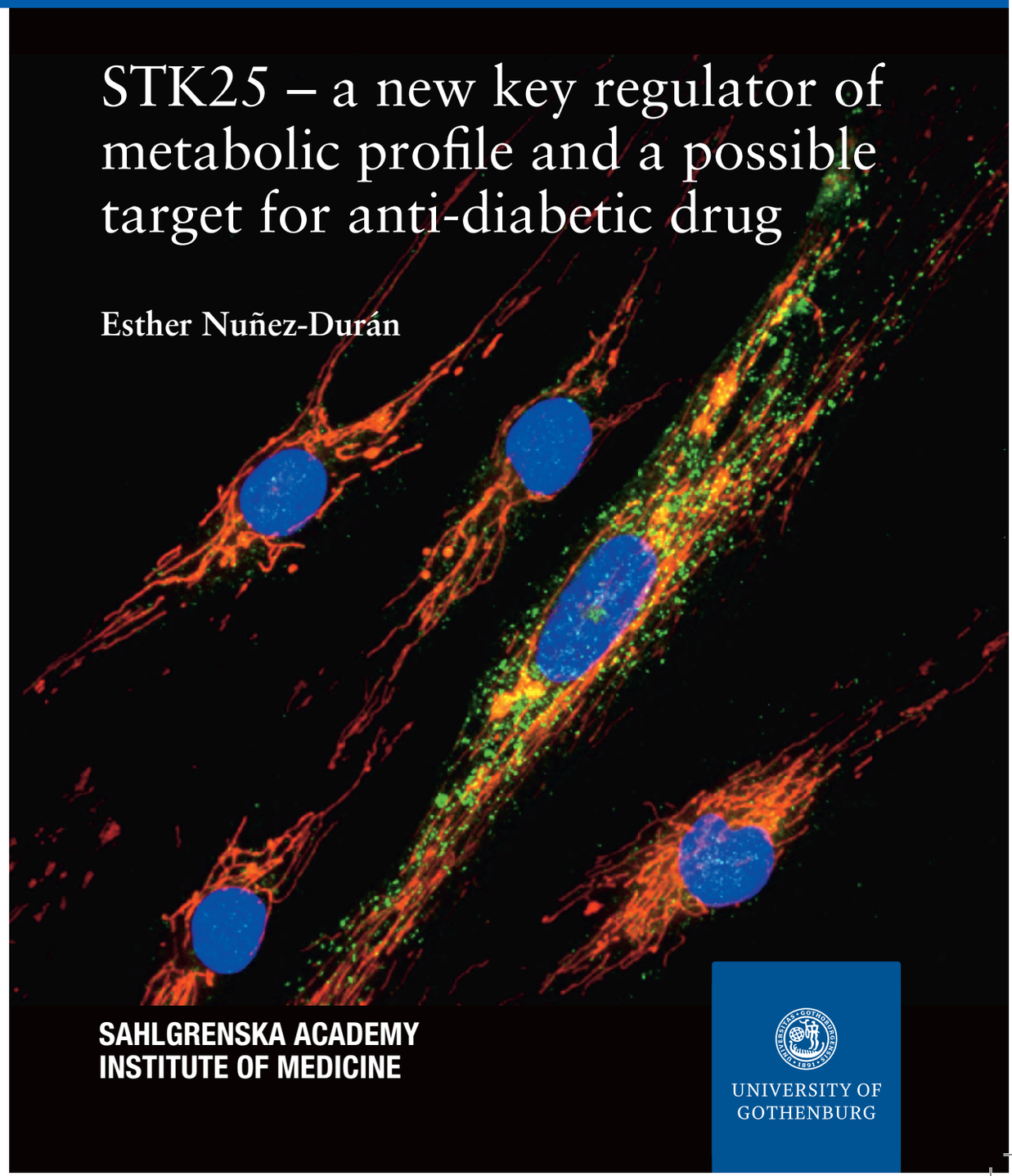

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