SUBMITTING A SCIENTIFIC ABSTRACT

Title

The title should emphasize the abstract’s relevance and main conclusions.

Format

The body of a Scientific Abstract should provide a clear, concise summary of the major aspects of your research, including:

- An introduction or background
- Your hypothesis and any underlying question(s) the research addresses
- An overview of your experimental design or methodology
- Major results
- An interpretation of the results and conclusions

Style

- Abstract submissions may only include text; **no figures or tables may be submitted.**
- Use **past tense** when writing a Scientific Abstract.

Abbreviations

Abbreviations that are familiar to endocrinologists (eg, PCR, GHRH, TSH, etc) may be used without explanation.

Laboratory Values

For laboratory parameters, the units of measurement and normal ranges must be provided.

Statements

Avoid making statements about ongoing studies or pending results.

References

References are not necessary; keep them to a minimum. If used, references will count towards the 450-word limit.

Authors and Disclosure

Disclosure(s) and source(s) of support, if applicable, should not be included in your abstract but must be provided in the submission process.

EXAMPLE OF AN OUTSTANDING SCIENTIFIC ABSTRACT

Brain Insulin Signaling Increases Hepatic Triglyceride Secretion In Vivo

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Abstract: Hepatosteatosis and dyslipidemia are hallmarks of the metabolic syndrome and plasma triglycerides (TG) tightly correlate with insulin resistance (IR). Since hepatic lipogenesis is increased in the IR state, TG secretion must not be too low to prevent steatosis. Insulin action comprises direct effects on peripheral organs e.g. liver and adipose, and indirect effects mediated via the central nervous system (1). Systemic insulin decreases very low-density lipoprotein (VLDL) production by the liver, yet it is unknown if brain insulin can independently regulate VLDL flux. To study the role of brain vs. systemic insulin signaling on hepatic VLDL secretion, we performed tyloxapol infusion studies in male Sprague Dawley rats during systemic or isolated brain hyperinsulinemia. The latter was accomplished by infusing insulin or vehicle for 4 hrs into the 3rd ventricle (ICV) or the mediobasal hypothalamus (MBH). ICV insulin infusion increased hepatic VLDL secretion compared to controls (2.59±0.28 vs. 1.80±0.2 μmol/kg/min; $P=0.039$; $n \geq 11$ per group). To the contrary, a hyperinsulinemic euglycemic clamp decreased TG flux (0.85±0.05 μmol/kg/min; $P=0.020$; $n=4$), which is in agreement with prior reports (2). Plasma lipid profiling in these rats demonstrated that ICV insulin increased the accumulation of TG associated fatty acids such as palmitate or oleate (+30%; $P<0.05$). Of note, insulin infusion into the MBH had no effect on VLDL flux vs. controls (1.85±0.32 μmol/kg/min vs. 1.71±0.32 μmol/kg/min; $P=0.773$; $n=5$ per group) indicating that another brain region integrates the central insulin-signal. Conversely, mice lacking insulin receptor in the whole brain had reduced hepatic TG flux compared to littermate controls, which was again assessed by tyloxapol studies (154±6 vs. 126±12 μmol/kg/h; $P = 0.038$; $n \geq 9$ per group). To begin to understand the molecular underpinnings that alter hepatic VLDL flux when ICV insulin is infused, we assessed hepatic microsomal TG transfer protein (MTTP) expression, the rate-limiting enzyme in VLDL assembly. Consistent with VLDL flux, ICV insulin increased MTTP expression compared to controls ($P=0.046$; $n \geq 5$). While systemic hyperinsulinemia and isolated loss of neuronal insulin signaling both suppress TG flux, ICV insulin infusion acutely increases VLDL secretion. We speculate that the elevated TG production in obesity and diabetes may be due to preserved central insulin effects in a presently unknown brain region.

Reference: (1) Scherer et al., Cell Metab. 2011 Feb 2;13(2):183-94. (2) Grefhorst et al., Am J Physiol Gastrointest Liver Physiol. 2005 Sep;289(3):G592-8.