ABSTRACT TEMPLATE

OBJECTIVE: Write one sentence to summarize the "problem" (why this research needs to be conducted-- why it's important). Write one sentence of background information to give your study context. Then, in one sentence, write the objective or the hypothesis of the study.

RESEARCH DESIGN & METHODS: In one or two sentences, briefly explain how the research was conducted.

RESULTS: In two to three sentences, describe the most important results of the research.

CONCLUSION: In one or two sentences, write what can be concluded from this study. OPTIONAL: In one sentence, write what implications this research may have or what future studies need to be done.

Other tips:

- Maximum 250 words; avoid verbosity-- get to the point
- avoid using first person too much (once or twice is okay)
- Abstract can be written as one paragraph-- subheadings are provided to help with organization
- Additional instructions and examples found on UMVSD website: www.ohioumvsd.com or http://www.ohioumvsd.com/Students/Results_Conclusions_Abstract/RCA.php

EXAMPLE:

Glycerol-3-phosphate acyltransferase 1 deficiency in ob/ob mice diminishes hepatic steatosis but does not protect against insulin resistance or obesity.

Wendel AA, Li LO, Li Y, Cline GW, Shulman GI, Coleman RA.
Diabetes. 2010 Jun;59(6):1321-9. doi: 10.2337/db09-1380. Epub 2010 Mar 3.

(OBJECTIVE:) Hepatic steatosis is strongly associated with insulin resistance, but a causal role has not been established. In ob/ob mice, sterol regulatory element binding protein 1 (SREBP1) mediates the induction of steatosis by upregulating target genes, including glycerol-3-phosphate acyltransferase-1 (*Gpat1*), which catalyzes the first and committed step in the pathway of glycerolipid synthesis. We asked whether ob/ob mice lacking *Gpat1* would have reduced hepatic steatosis and improved insulin sensitivity. (RESEARCH DESIGN AND METHODS:) Hepatic lipids, insulin sensitivity, and hepatic insulin signaling were compared in lean (*Lep(+/?)*), lean-*Gpat1(-/-)*, *ob/ob (Lep(ob/ob)*), and *ob/ob-Gpat1(-/-*) mice. (RESULTS:) Compared with ob/ob mice, the lack of *Gpat1* in ob/ob mice reduced hepatic triacylglycerol (TAG) and diacylglycerol (DAG) content 59 and 74%, respectively, but increased acyl-CoA levels. Despite the reduction in hepatic lipids, fasting glucose and insulin concentrations did not improve, and insulin tolerance remained impaired. In both *ob/ob* and *ob/ob-Gpat1(-/-)* mice, insulin resistance was accompanied by elevated hepatic protein kinase C-epsilon activation and blunted insulin-stimulated Akt activation. (CONCLUSIONS:) These results suggest that decreasing hepatic steatosis alone does not improve insulin resistance, and that factors other than increased hepatic DAG and TAG contribute to hepatic insulin resistance in this genetically obese model. They also show that the SREBP1-mediated induction of hepatic steatosis in *ob/ob* mice requires *Gpat1*.