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Solving the controversy over the involvement of uncoupling protein-2 in palmitate-induced impairment of glucose-stimulated insulin secretion: Early stage and late stage impairment

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There is a debate on the function of mitochondrial uncoupling protein-2 in beta cells and its involvement in palmitate-induced impairment of glucose-stimulated insulin secretion. Some investigators suggested that uncoupling protein-2 is involved in this impairment while others denied its involvement. Based on the results of their studies, this controversy can be solved by hypothesizing that palmitate-induced impairment of glucose-stimulated insulin secretion occurs in two stages, early stage and late stage, depending on the integrity of electron supply and transport system through electron transport chain after palmitate treatment. Prolonged exposure of beta cells to palmitate can impair this system. Early stage impairment occurs due to uncoupling by uncoupling protein-2 when this system is still intact. When this system becomes impaired, late stage impairment occurs due to reduced ATP production independent of uncoupling by uncoupling protein-2. The change in glucose-stimulated oxygen uptake after palmitate treatment reflects the integrity of this system and can be used to differentiate between the two stages. Some beta-cell lines appear to be more resistant to palmitate-induced impairment of electron supply and transport system than others and therefore, early stage is prominent in the more resistant cell lines and less prominent in the less resistant cell lines.

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A clinical review of GLP-1 receptor agonsits: Efficacy and safety in diabetes and beyond

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The prevalence of type 2 diabetes is increasing at an astounding rate. Many of the agents used to treat type 2 diabetes have undesirable adverse effects of hypoglycemia and weight gain. Glucagon-like peptide-1 (GLP-1) receptor agonists represent a unique approach to the treatment of diabetes, with benefits extending outside glucose control, including positive effects on weight, blood pressure, cholesterol levels and beta-cell function. They mimic the effects of the incretin hormone GLP-1, which is released from the intestine in response to food intake. Their effects include increasing insulin secretion, decreasing glucagon release, increasing satiety and slowing gastric emptying. There are currently four approved GLP-1 receptor agonists in the United States: exenatide, liraglutide, albiglutide and dulaglutide. A fifth agent, lixisenatide, is available in Europe. There are important pharmacodynamic, pharmacokinetic and clinical differences of each agent. The most common adverse effects seen with GLP-1 therapy include nausea, vomiting and injection-site reactions. Other warnings and precautions include pancreatitis and thyroid cell carcinomas. GLP-1 receptor agonists are an innovative and effective option to improve blood glucose control, with other potential benefits of preserving beta-cell function, weight loss, and increasing insulin sensitivity. Once-weekly formulations may also improve patient adherence. Overall, these are effective agents for patients with type 2 diabetes, who are either uncontrolled on metformin or intolerant to metformin.

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