# INTRODUCTION

Diabetes mellitus (DM) is a chronic illness affecting nearly 285 million people worldwide. This is projected to increase by 6.5% per year, with 439 million inflicted by year 2030 ***(American Diabetes Association, 2008).***

The treatment algorithm updated in 2008 by American Diabetes Association and the European Association for the Study of Diabetes currently recommends the traditional medications of metformin, either as monotherapy or in combination with sulfonylurea and/or insulin, as the preferred choice in the level 1 option. The algorithm only suggests addition of alternative medications such as pioglitazone and incretin-based drugs as second-line agents in the level 2 ‘‘less well-validated’’ option ***(Nathan et al., 2009).***

However, these traditional medications have not proven to delay the progressive course of diabetes as evidence of increasing need over time for multiple drug therapy to maintain sufficient glycemic control. Because current diabetes medications have limited efficacy and unwanted side effects, the development of DM drugs with newer mechanisms of action continues ***(Lo et al., 2010).***

One of the newer diabetes medications are agents that target the incretin system. Incretins are intestinally derived hormones secreted in response to food. The 2 main intestinal incretins are glucagon-like peptide-1 (GLP-1) and glucose-dependent insulinotropic peptide (GIP). Both are released rapidly within minutes after food ingestion under neuroendocrine control. Patients with type-2 diabetes mellitus (T2DM)

have been found to be partially deficient or totally absent in GLP secretion in response to meals ***(Toft-Nielsen et al., 2001; Lugari et al., 2002).***

Incretin-based medications include either as an agonist to the natural endocrine hormone GLP-1 (ie, exenatide, liraglutide) or as an inhibitor of the dipeptidyl peptidase-4 (DDP-4) enzyme that degrades the incretin hormones, thereby prolonging their effects. (ie, Sitagliptin, Vildagliptin) ***(Lo et al., 2010).***

The GLP-1 agonists and DPP-4 inhibitors lower both fasting and postprandial glucose levels. The effects of GLP-1 agonists tend to be greater, probably because they produce pharmacologic levels of GLP-1 compared to physiologic levels with the DPP-4 inhibitors. Another difference is that unlike the DPP-4 inhibitors, the GLP-1 agonists also slow gastric emptying and promote satiety ***(Campbell et al., 2010).***