Joint Event on
European Heart Congress & Traditional Medicine Congress
October 23-24, 2019 | Prague, Czech Republic

Potential target of insulin release from pancreatic islets by *Orthosiphon stamineus*

Ezarul Faradianna Lokman[1], Fatin Saparuddin[1], Hussin Muhammad[2, 3], Maizatul Hasyima Omar[2] and **Azlina Zulkapli[1, 3]**

[1]Institute for Medical Research, Kuala Lumpur, Malaysia
[2]Herbal Medicine Research Centre (HMRC), Malaysia
[3]Medical Research Resource Centre (MRRC), Malaysia

The antidiabetic effects of a traditional Malaysia plant known as Orthosiphon stamineus (OS) have been previously reported in rats and found to be associated with glucose stimulated insulin release. However, the mechanisms involved have yet to be identified. In this study, we explored the important target of insulin release by OS from pancreatic islets. Pancreatic islets from Sprague Dawley rat (mean body weight: 294.48±40.3 g) were isolated using collagenase digestion method. The islets were cultured overnight and incubated at 16.7 mM glucose. Several modulators and inhibitors were used; 0.25 mM diazoxide only (to open the K-ATP channel, 50 mM of KCl (for depolarization of beta cells), Nifedipine (to block the L-type Ca2+ channels), 10 uM H89, 1.5 uM of Calphostin-C (to block PKA-and PKC respectively) and 100 ng/ml pertussis toxin (to inhibit the GE protein). The insulin release from islets was measured using enzyme-linked immunosorbent assay (ELISA). Incubation of islets with diazoxide and Nifedipine significantly decreased insulin secretion by OS as compared to control islets by 2 fold and 1.8 fold respectively indicating the possibilities that OS acts partly on both K-ATP and L-type Ca2+ channels. However, no significant differences on insulin release with the addition of H89 and Calphostin C suggesting that insulin release by OS were not affected by the these pathways. Furthermore, the addition of pertussis toxin in the islets treated with OS significantly suppressed insulin secretion by 2.5 fold which indicates the importance of GE protein pathway on OS action. When exploring the mechanism of actions from islets, we have discovered that OS exerts its insulin effects partly via KATP Channel, L-type Ca2+ channels and on the exocytosis. Further studies need to be done to confirm the main insulin targets..