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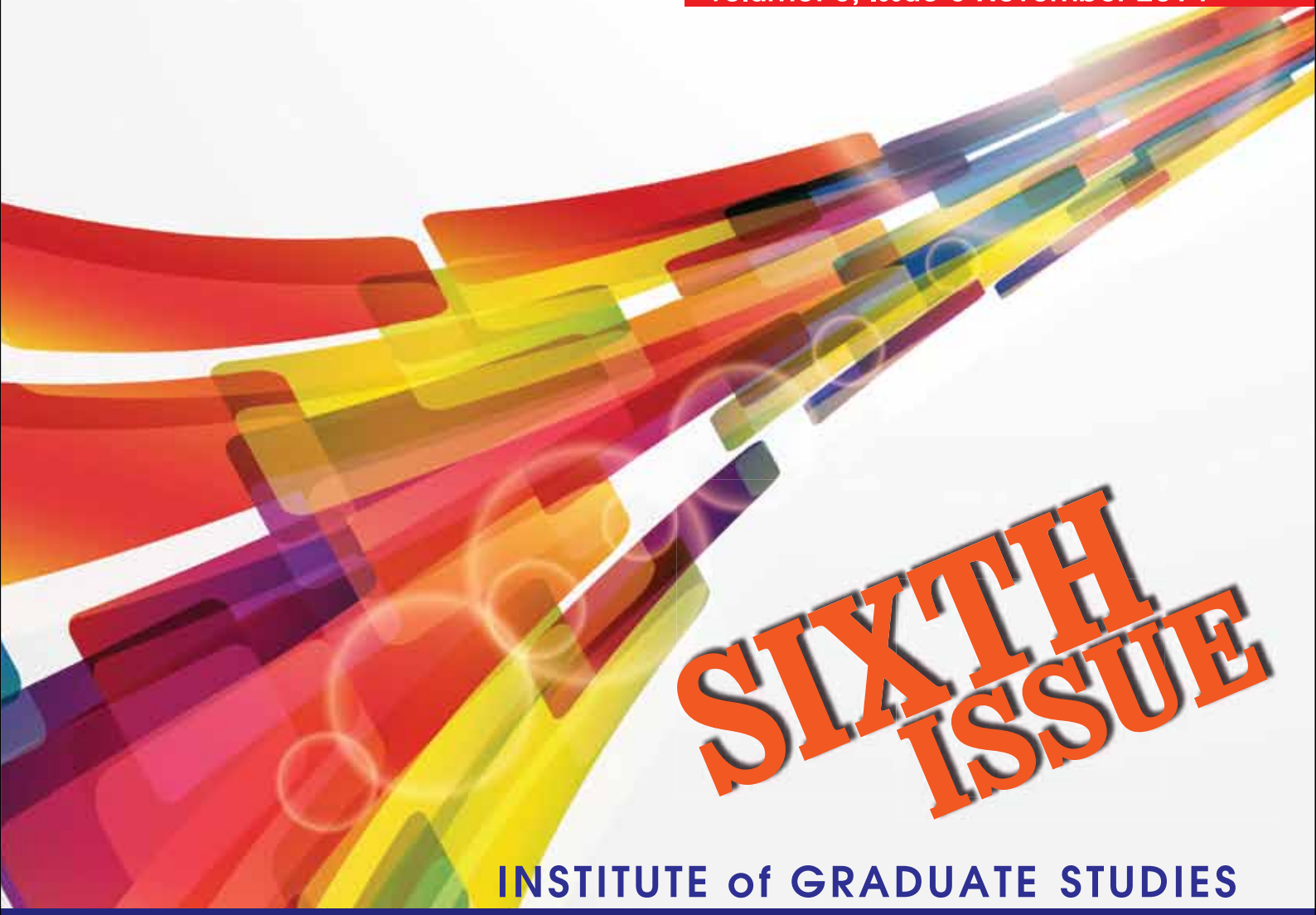

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Name :
Yuhaniza Shafinie Binti Kamsani

Title :
Gamma-Tocotrienol Reverses Nicotine-Induced Oxidatives Stress-Related *In Vitro* And *In Vivo* Embryonic Development And Pregnancy Outcome In Mice

Supervisor :
Prof. Dr. Mohd Hamim Rajikin (MS)

Prof. Dr. Amar Chatterjee (CS)

Assoc. Prof. Dr. Nor Ashikin Mohd Noor Khan (CS)

Assoc. Prof. Dr. Nuraliza Abd Satar (CS)

A study to evaluate the effects of nicotine and simultaneous supplementation of γ-tocotrienol (γ-TCT), one of the four isomers in tocotrienols (TCT), and nicotine, on *in vitro* and *in vivo* embryonic development in mice (*Mus musculus*) had been carried out. Several approaches were undertaken including an investigation on in vitro effects of various doses and durations of nicotine treatment on pre-implantation embryonic development. Results showed that nicotine treatment decreased the number of retrieved embryos, resulted embryo degeneration, delayed embryo cleavage, induced disproportionate size of blastomeres and degraded blastomeres ($p<0.05$). Moreover, the deleterious impact of nicotine on pre-implantation embryonic development was dose- and treatment duration-dependent with a corresponding increase in plasma malondialdehyde (MDA) concentrations ($p<0.05$). Based on this finding, the study was further elucidated in terms of examining the dose-related beneficial effects of γ–TCT in nicotine-induced cessation of pre-implantation embryonic development *in vitro*. Results showed that γ-TCT could prevent the duration- and dose-related

deterioration of pre-implantation embryo quality when supplemented simultaneously with nicotine. Moreover, γ–TCT of 60 mg/kg bw/day was found to be the optimal effective dose in lowering plasma levels of MDA during pre-implantation embryo development ($p<0.05$). Findings of *in vitro* study were applied in *in vivo* approach to evaluate the effect of simultaneous supplementation of γ-TCT with nicotine on embryo development, blastocyst implantation, foetal growth, length of gestation, foetal outcome and survival rate of the neonates. Results showed that nicotine impaired post-implantation embryo growth and development ($p<0.05$). Pregnancy outcome and survival of the neonates were also reduced ($p<0.05$). The rise in oxidant levels reduced the enzymatic antioxidant defense system ($p<0.05$). Pregnancy-related levels of progesterone (P_4) and oestrogen (E_2) were also significantly affected ($p<0.05$). Through combating nicotine-induced oxidative stress, γ-TCT was able to sustain the physiological/normal sequence of blastocyst implantation, foetal growth, pregnancy outcome and survival of the neonates. Moreover, maintenance of pregnancy until term possibly resulted from sustaining the levels of plasma P_4 and E_2 as evident in normal pregnancy. In conclusion, γ-TCT could be used to minimize nicotine-induced oxidative stress-related deterioration of pre- and post-implantation embryo development, intrauterine foetal growth, pregnancy outcome and survival of the neonates.