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Ahmed T Alahmar, Zahraa Ali, Zahraa Muhsin and Hadeel Qasim
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Aim: Data on the e ect of obesity on seminal uid and men fertility are inconsistent. e aim of this study was to evaluate the impact of Body Mass Index (BMI) on semen characteristics.

Method: A cross-sectional study was conducted on 74 infertile men. Semen sample were collected and sperm concentration, progressive motility, total motility and normal sperm morphology were assessed in accordance with WHO 2010 criteria. For each patient weight and height were measure and patients were divided by BMI into normal weight (BMI: 18.5-24.9 kg/m^2, n=30), overweight (BMI: 25-29.9 kg/m^2, n=30) and obese (BMI: ≥30 kg/m^2, n=14). Seminal uid parameters were compared among the three groups.

Result: Although sperm concentration was lower in obese men, sperm concentration, progressive and total motility and normal sperm morphology did not signi cantly di er among normal weight, overweight and obese groups (P>0.05).

Conclusion: Our ndings suggest that BMI may have no in uence on sperm concentration, motility and normal morphology in infertile men.

ahmed.t.alahmar@gmail.com

The therapeutic effects of matrine for MCD-induced NASH are associated with upregulation of HSP72 and suppression of mTOR

Ali Mahzari, Songpei Li, Xiu Zhou, Xiaoyi Zeng, Sherouk Fouda, Stephen Robinson and Jiming Ye
RMIT University, Australia

Non-Alcoholic Steatohepatitis (NASH) is an advanced stage of the metabolic syndrome in liver with serious consequences largely because of hepatic injury, in ammation and brosis. Matrine (MW: 248) is used as a prescribed hepatoprotective drug in humans and it has been shown by us to decrease hepatosteatosis and glucose intolerance in high fat-fed mice. Here, we investigated whether matrine exerts therapeutic e cacy for NASH by attenuating hepatic injury, in ammation and brosis. e study was performed in Methionine Choline-De cient (MCD) diet-fed mice for 6 weeks with or without the treatment with matrine (100 mg/kg/d). Compared with untreated MCD-fed mice, matrine markedly reduced hepatic injury (indicated by ALT level, p<0.05), in ammation (indicated by TNFα, CD68 and in ammasome NLRP3, all p<0.05). Along with these e ects, matrine inhibited MCD-induced increases in brogenesis (as indicated by the expression levels of TGFβ, Smad3 and type-I collagen (all p<0.05). Further examination revealed that matrine resecured MCD-suppressed Heat Shock Protein 72 (HSP72, a protective chaperon protein against cell toxicity) and inhibited MCD-activated mTOR (a key master regulator triggering pathogenic pathways leading to NASH). Our ndings indicate that matrine attenuated MCD-induced NASH by a new mechanism involving the upregulation of HSP72 and inhibition of mTOR. is hepatoprotective drug may be repurposed for the treatment of NASH.

s3192279@student.rmit.edu.au