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Is Intraperitoneal Injection of Testosterone Propionate in Adult Animal Suitable to Study PCOS? [Letter]

Jiro Hasegawa Situmorang^{1,2}, Widoretno¹

¹Center for Biomedical Research, National Research and Innovation Agency (BRIN), Cibinong, Indonesia; ²Cardiovascular and Mitochondrial Related Disease Research Center, Buddhist Tzu Chi General Hospital, Buddhist Tzu Chi Medical Foundation, Hualien, Taiwan

Correspondence: Jiro Hasegawa Situmorang, Center for Biomedical Research, National Research and Innovation Agency (BRIN), Cibinong, Indonesia, Email jiro.hasegawa.s@gmail.com

Dear Editor

We read closely and eagerly the recent article by Siahaan et al.¹ The study used the leaves' ethanolic extract of *Moringa oleifera* to alleviate PCOS-related symptoms induced by Testosterone Propionate (TP). Even though we find the study interesting, we have some concerns worth discussing.

In the study, the authors administered the TP (100 mg/kg BW) for 21 days through the intraperitoneal (i.p.) route. The use and route of administration of a dose at such a high concentration seem to be questionable, since numerous previous studies have used relatively low doses (10–30 mg/kgBW) of TP subcutaneously (s.c.).^{2–4} In addition, it would have been informative if the authors explained the preparation of the TP in the article. Due to TP's low solubility in water-based solvents, oil-based solvents are commonly employed. The i.p. routes should not be used if the authors used an oil-based solvent since oil-based solvents can induce inflammation and apoptosis in the peritoneum.⁵ As a consequence, there could be potential bias in interpretation of the results. This is because it is unclear whether the solvent or TP itself is responsible for the increase in inflammatory indices. Thus, a vehicle solvent-only control group is needed.

PCOS induction by TP exposure is usually performed during the fetal or neonatal stage.^{4,6} The authors, however, administered TP to adult rats (3 months old). To our knowledge, the group is the first to expose adult female animals to a high dose of TP in order to model PCOS. The article lacks an explanation and discussion of using this method to induce PCOS in adult animal, fetus, and newborn.

To investigate the inflammatory response in the PCOS model, the authors assessed TNF- α expression in the ovary of the rats. However, TNF- α count was misrepresented in the article since the authors used Metabolite Insulin Receptor Substrate-1 or IRS-1 to represent TNF- α expression. In our opinion, this cannot be justified since TNF- α and IRS-1 have different properties and functions; thus, they cannot be used to represent each other. Further, protocols such as immunohistochemistry and ELISA assays to measure TNF- α and serum parameters are also absent. Due to the novelty of the method used to induce PCOS, reproducibility and repeatability are crucial.

Lastly, daily food consumption data is needed to ensure that the decrease in serum glucose and insulin level in the treatment group was not due to lower food consumption, hence corroborating the study's conclusion.

As a whole, this is an interesting study that deserves more discussion, particularly on the high dose of TP in adult rats and its potential translation to humans' PCOS.

Disclosure

The authors report no conflicts of interest in this communication.

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