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Procyanidin B2 Protects Aged Oocytes Against Meiotic Defects Through Cortical Tension Modulation

Qingrui Zhuan[1†], Jun Li[2†], Guizhen Zhou[1], Xingzhu Du[1], Hongyu Liu[1], Yunpeng Hou[3], Pengcheng Wan[4] and Xiangwei Fu[1,4]*

[1] Key Laboratory of Animal Genetics, Breeding and Reproduction of the Ministry of Agriculture and Rural Affairs, National Engineering Laboratory for Animal Breeding, Beijing Key Laboratory for Animal Genetic Improvement, College of Animal Science and Technology, China Agricultural University, Beijing, China, [2] Department of Reproductive Medicine, Reproductive Medical Center, The First Hospital of Hebei Medical University, Shijiazhuang, China, [3] State Key Laboratories of Agrobiotechnology, College of Biological Sciences, China Agricultural University, Beijing, China, [4] State Key Laboratory of Sheep Genetic Improvement and Healthy Breeding, Institute of Animal Husbandry and Veterinary Sciences, Xinjiang Academy of Agricultural and Reclamation Sciences, Shihhotze, China

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****Correspondence:***
Xiangwei Fu
xiangweifu@126.com

[†] These authors have contributed equally to this work and share first authorship

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Defects in meiotic process are the main factors responsible for the decreased developmental competence in aged oocytes. Our recent research indicated that natural antioxidant procyanidin B2 (PCB2) promoted maturation progress in oocytes from diabetic mice. However, the effect of PCB2 on aging-induced chromosome abnormalities and the underlying mechanism have not been explored. Here, we found that PCB2 recovered aging-caused developmental arrest during meiotic maturation, germinal vesicle breakdown (GVBD) rate was significantly higher in aged oocytes treated with PCB2 ($P < 0.05$). Furthermore, we discovered that cortical mechanics were altered during aging process, cortical tension-related proteins were aberrantly expressed in aged oocytes ($P < 0.001$). PCB2 supplementation efficaciously antagonized aging-induced decreased cortical tension ($P < 0.001$). Moreover, PCB2 restored spindle morphology ($P < 0.01$), maintained proper chromosome alignment ($P < 0.05$), and dramatically reduced reactive oxygen species (ROS) level ($P < 0.05$) in aged oocytes. Collectively, our results reveal that PCB2 supplementation is a feasible approach to protect oocytes from reproductive aging, contributing to the improvement of oocytes quality.

Keywords: PCB2, reproductive aging, oocyte, cortical tension, meiotic maturation

INTRODUCTION

There is a global tendency that women delay conception until late 30's, by which time the chance of pregnancy is compromised as the reproductive capacity in women declines beyond their mid-30's (1). Reproductive aging deteriorates oocyte quality (2). It is known that maternal aging is associated with meiotic defects, and in addition to this, increased vulnerability of aged oocytes to reactive oxygen species (3) leads to mitochondrial dysfunction, since mitochondria are the most significant targets of oxidative stress (1, 4).

Cortical tension and stiffness experience dynamic changes through meiotic maturation and fertilization progression to facilitate and/or direct cellular remodeling in the mammalian oocyte (5). This cortex remodeling is part of the creation of cellular asymmetry and mediates the progression of the prophase I, germinal vesicle-intact (GVI) oocyte to the MII stage (6).