



动物营养学报

CHINESE JOURNAL OF ANIMAL NUTRITION

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动物营养学报 2014, Vol. 26 Issue (3) :651-658 DOI: 10.3969/j.issn.1006-267x.2014.03.014

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吡咯喹啉醌对脂肪肝蛋鸡肝损伤的保护作用机制

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Protective Mechanisms of Dietary Pyrroloquinoline Quinine on Fatty Liver Laying Hens

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摘要 本试验旨在研究吡咯喹啉醌(PQQ)防治蛋鸡脂肪肝综合征的作用机制。选用29周龄海兰褐蛋鸡288只,随机分为4组,每组6个重复,每个重复12只鸡。I组为对照组,饲喂基础饲料(代谢能11.03 MJ/kg,粗蛋白质16.2%);II组为病理模型组,饲喂高能低蛋白质饲料(代谢能12.75 MJ/kg,粗蛋白质13.0%);III和IV组分别在II组的高能低蛋白质饲料基础上添加0.08和0.16 mg/kg的PQQ。试验期4周。结果表明:1)与I组相比,II组高能低蛋白质饲料成功诱导了蛋鸡脂肪肝试验模型,表现为肝细胞弥散性脂肪变性,肝脏中甘油三酯、总胆固醇含量显著升高($P<0.05$),血浆谷丙转氨酶活性显著升高($P<0.05$);2)饲料PQQ可显著抑制高能低蛋白质饲料引起的肝脏中甘油三酯和总胆固醇含量及血浆中谷丙转氨酶活性的升高($P<0.05$),达到与对照组相当的水平;3)饲料PQQ可显著抑制高能低蛋白质饲料引起的肝脏中超氧化物歧化酶活性降低和丙二醛含量升高($P<0.05$);4)饲料PQQ可显著抑制高能低蛋白质饲料引起的蛋鸡肝脏线粒体相对含量减少($P<0.05$),抑制柠檬酸脱氢酶和细胞色素C氧化酶活性降低($P<0.05$);5)饲料PQQ可阻止采食高能低蛋白质饲料的蛋鸡发生脂肪肝,维持与正常对照组相当的水平,不同剂量PQQ之间未见显著差异($P>0.05$)。由此可见,饲料PQQ可通过改善蛋鸡肝脏线粒体功能、调节脂质代谢和抗氧化功能预防蛋鸡脂肪肝综合征。

关键词: 吡咯喹啉醌 蛋鸡脂肪肝综合征 高能低蛋白质饲料 脂质代谢 线粒体

Abstract: This experiment was conducted to investigate the protective mechanisms of dietary pyrroloquinoline quinine (PQQ) on laying hens with fatty liver syndrome. Two hundred and eighty eight Hy-Line brown laying hens aged 29 weeks were randomly divided into 4 groups with 6 replicates per group and 12 hens per replicate. Hens in group I (control group) were fed a basal diet (ME 11.03 MJ/kg; CP 16.2%), hens in group II (pathological model control group) were fed a high-energy low-protein diet (ME 12.75 MJ/kg; CP 13.0%), and hens in groups III and IV were fed the high-energy low-protein diet (the same as group II) supplemented with 0.08 or 0.16 mg/kg PQQ, respectively. The experiment lasted for 4 weeks. The results showed as follows: 1) compared with the control group, high-energy low-protein diet in group II successfully induced experimental fatty liver model; there were large quantity of fat degeneration in livers from pathological model control; the contents of triglyceride and total cholesterol in liver, and the plasma alanine aminotransferase activity in group II were significantly increased ($P<0.05$). 2) Dietary PQQ strikingly suppressed the elevation of contents of triglyceride and total cholesterol in liver, and the plasma alanine aminotransferase activity caused by high-energy low-protein diet ($P<0.05$), to the same levels as normal control. 3) Dietary PQQ significantly prevented the decrease of superoxide dismutase activity, and the increase of malondialdehyde content induced by high-energy low-protein diet ($P<0.05$). 4) Dietary PQQ significantly inhibited the decrease of mitochondrial relative content and the activities of citrate synthase and cytochrome C oxidase induced by high-energy low-protein diet ($P<0.05$). In conclusion, dietary PQQ can prevent the fatty liver syndrome induced by high-energy low-protein diets by improving mitochondrial function, and regulating the lipid metabolism and anti-oxidative activities in the liver.

Keywords: pyrroloquinoline quinine, laying hen fatty liver syndrome, lipid metabolism, anti-oxidation, mitochondria

收稿日期: 2013-10-28:

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引用本文:

赵芹, 张海军, 武书庚等. 吡咯喹啉醌对脂肪肝蛋鸡肝损伤的保护作用机制[J]. 动物营养学报, 2014, V26(3): 651-658

ZHAO Qin, ZHANG Haijun, WU Shugeng etc. Protective Mechanisms of Dietary Pyrroloquinoline Quinone on Fatty Liver Laying Hens[J]. Chinese Journal of Animal Nutrition, 2014, V26(3): 651-658.

链接本文:

http://118.145.16.228/Jweb_dwyy/CN/10.3969/j.issn.1006-267x.2014.03.014 或 http://118.145.16.228/Jweb_dwyy/CN/Y2014/V26/I3/651

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