[1]冯泽洲,王学锋,朱昀,等.紫绀型先心病患儿心肌组织SOCS3基因启动子甲基化分析及机制研究[J].第三军医大学学报,2014,36(10):1012-1015.
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紫绀型先心病患儿心肌组织SOCS3基因启动子甲基

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Title: DNA methylation of SOCS3 promoter and the possible mechonism in myocardia of infants with cyanotic congenital heart defects

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关键词: 紫绀型先心病; SOCS3; 甲基化; DNA甲基化转移酶; 慢性缺氧适应

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摘要: 目的 探讨紫绀型先心病患儿右心室心肌组织中细胞信号抑制因子3（suppressor of cell signaling 3，SOCS3）基因启动子甲基化水平及其可能机制。 方法 选取先心病患儿34例,其中紫绀组18例,非紫绀组16例。取手术中切除的右心室流出道心肌组织作为标本,采用甲基化特异性PCR(methylation specific PCR，MSP)及重亚硫酸盐测序(bisulfite sequencing PCR，BSP)检测心肌细胞中SOCS3启动子CpG岛甲基化程度。Western blot检测DNA甲基化转移酶3A（DNA methyltransferase，DNMT3A）、DNA甲基化转移酶3B（DNMT3B）蛋白在心肌细胞中的表达情况。 结果 与非紫绀组相比,紫绀组SOCS3启动子CpG岛甲基化程度较高，DNMT3A蛋白较高[(0.407±0.469) *vs* (0.160±0.034)，$P<0.05$]，DNMT3B 蛋白无明显差异[(0.054±0.012） *vs* （0.052±0.093），$P>0.05$]。 结论 紫绀型先心病患儿心肌组织中，SOCS3启动子CpG岛呈高甲基化。高表达的DNMT3A蛋白很可能参与了SOCS3启动子CpG岛高甲基化。高甲基化的SOCS3启动子CpG岛，可能不是调控慢性缺氧适应心肌中SOCS3转录的主要因素。高表达的SOCS3启动子CpG岛以及高表达的DNA甲基化转移酶3A蛋白可能是心肌慢性缺氧适应的一种体现。

Abstract: Objective To investigate the methylation level of suppressor of cell signaling 3

(SOCS3) promoter in the myocardia of infants with cyanotic congenital heart defects. Methods A total of 34 children with cyanotic (*n*=18) and acyanotic (*n*=16) congenital cardiac defects were investigated. Samples from the right ventricular myocardia were collected to detect the methylation level of SOCS3 promoter CpG island with methylation-specific PCR (MSP) and bisulfite sequencing PCR (BSP). Protein levels of DNA methyltransferase 3A (DNMT3A) and 3B (DNMT3B) were examined by Western blotting. Results Compared to the acyanotic group, the cyanotic group displayed the hypermethylation of SOCS3 promoter CpG island, higher protein level of DNMT3A (0.407±0.469 *vs* 0.160 ±0.034, *P*<0.05), and similar protein level of DNMT3B (0.054±0.012 *vs* 0.052± 0.093, *P*>0.05). Conclusion SOCS3 promoter CpG island presents hypermethylation in the infants with cyanotic congenital cardiac defects. Strony expression of DNMT3A might participate in the hypermethylation of SOCS3 promoter CpG island. The hypermethylated SOCS3 promoter CpG island might not be the major factor regulating SOCS3 transcription in the myocardial adaption to chronic hypoxia. The hypermethylated SOCS3 promoter and higher DNMT 3A expression are possibly the signs of adaptive myocardium to chronic hypoxia.

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