

论文

体外诱导肺炎支原体对喹诺酮类药物耐药机制的研究

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摘要:

**目的** 探讨在亚抑菌浓度喹诺酮类药物多代培养后, 肺炎支原体(MP)标准株FH对喹诺酮类药物敏感性的变化及其机制。**方法** 将标准株FH在含有亚抑菌浓度环丙沙星、左氧氟沙星和加替沙星的液体培养基中传代培养后, 检测其对3种药物的MIC值。提取标准株及诱导耐药株的DNA, PCR扩增喹诺酮耐药决定区的gyrA、gyrB、parC和parE基因。测序分析耐药株的基因突变情况。**结果** 经亚抑菌浓度喹诺酮类药物多代培养诱导后, FH出现对诱导药物的耐药及交叉耐药。6株药物诱导耐药株, 其中在左氧氟沙星诱导的耐药株gyrA基因编码的95位蛋氨酸转变为异亮氨酸, parC基因编码的87位天门冬氨酸转变为酪氨酸。在加替沙星诱导的耐药株中gyrB基因编码的464位精氨酸转变为赖氨酸。parE基因未检出错义突变。**结论** 亚抑菌浓度喹诺酮类药物可诱导肺炎支原体出现耐药及交叉耐药。其产生可能与喹诺酮耐药决定区的基因突变有关。

**关键词:** 肺炎, 支原体; 喹诺酮类; 抗药性, 微生物; 点突变

Induction and mechanisms of resistance to quinolones in *Mycoplasma pneumoniae*

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Abstract:

**Objective** To investigate the effect of the subinhibitory concentration of quinolones on resistance to quinolones in *Mycoplasma pneumoniae* (MP) and its mechanisms. **Methods** To induce resistance to quinolones, the reference strain FH was cultured for several passages with three different quinolones at sub-inhibitory concentrations. DNA was extracted from the FH strain and in vitro drug-induced quinolone-resistant strains. The quinolone resistance determining regions(QRDR) of gyrA, gyrB, parC and parE in each strain were amplified with the corresponding primers by PCR and then sequenced. **Results** The FH strain exhibited resistance or cross resistance to the three quinolones after induction. In 6 drug-induced quinolone-resistant strains, the strains induced by levofloxacin had a substitute of Met95→Ile in the gyrA gene and a substitute of Asp87→Tyr in the parC gene; and the strains induced by Gatifloxacin had a substitute of Arg→Lys in the gyrB gene. No mis-sense mutation was detected in the parC gene. **Conclusion** Long term use of quinolones at sub-inhibitory concentration can contribute to resistance and cross-resistance of MP. The mutation of QRDR is closely associated with quinolone-resistance.

**Keywords:** Pneumonia, mycoplasma; Quinolones; Drug resistance, microbial; Point mutation

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