

## 萎胃康治疗慢性萎缩性胃炎的拆方研究

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**中文摘要:**目的: 探讨萎胃康及其拆方对慢性萎缩性胃炎(CAG)模型大鼠的治疗作用。方法: 将70只Wistar大鼠随机抽取12只为正常对照组(正常组), 其余58只采用多重刺激6周复制大鼠CAG模型。确定造模成功的50只随机分为5组, 即模型组、萎胃康全方组(全方组)、拆方I号组(补益组)、拆方II号组(祛邪组)、维霉素对照组(西药组), 分别ig 0.9%生理盐水(10 mL · kg<sup>-1</sup>)、萎胃康水煎液(8 g · kg<sup>-1</sup>)、拆方I号水煎液(5.5 g · kg<sup>-1</sup>)、拆方II号水煎液(2.5 g · kg<sup>-1</sup>)和维霉素混悬液(0.3 g · kg<sup>-1</sup>), 1次/日。给药30 d后, 观察对大鼠胃黏膜组织形态及血清超氧化物歧化酶(SOD)活力和丙二醛(MDA)含量的影响。结果: 全方组大鼠胃黏膜组织接近正常组。与正常组SOD(239.88±6.32)U · mL<sup>-1</sup>, MDA(3.17±0.02)μmol · L<sup>-1</sup>比较, 模型组SOD(174.59±12.81)U · mL<sup>-1</sup>明显降低(P<0.01), MDA(5.14±0.15)μmol · L<sup>-1</sup>明显升高(P<0.01)。与模型组比较, 各用药组大鼠血清SOD明显升高, MDA显著降低(P<0.01或P<0.05); 与全方组SOD(233.91±9.03)U · mL<sup>-1</sup>, MDA(3.11±0.19)μmol · L<sup>-1</sup>比较, 补益组、祛邪组大鼠血清SOD明显降低, MDA显著升高; 与补益组SOD(221.58±5.71)U · mL<sup>-1</sup>比较, 祛邪组大鼠血清SOD(209.89±5.27)U · mL<sup>-1</sup>活力明显降低(P<0.05)。结论: 萎胃康能改善和逆转实验性萎缩性胃炎大鼠胃黏膜萎缩。其中益气养阴药物起主要作用, 祛邪药物起协同作用, 其机制可能与抗自由基损伤有关。

**中文关键词:** [萎胃康](#) [拆方](#) [萎缩性胃炎](#) [病理形态学](#) [超氧化物歧化酶](#) [丙二醛](#)

## The Study of Weiweikang Treating Chronic Atrophic Gastritis and Analysis of the Prescription

**Abstract:** Objective: To study function of Weiweikang on treating chronic atrophic gastritis (CAG) model rats. Method: Twelve rats were randomly selected from 70 Wistar rats as normal control group (normal group). The remaining 58 rats were made CAG model with the multiple stimulations for 6 weeks. The remaining 50 rats which were modeled successfully were randomly divided into five groups such as model group, Weiweikang whole formula group (the whole formula group), split part I group (buyi group), split part II group (quxie group) and western medicine control group (western medicine group). The rats in every groups were ig 0.9% saline (0.09 g · kg<sup>-1</sup>), Weiweikang decoction (8 g · kg<sup>-1</sup>), split part I decoction (5.5 g · kg<sup>-1</sup>), split part II decoction (2.5 g · kg<sup>-1</sup>) and the neomycin-dimensional suspension (0.3 g · kg<sup>-1</sup>), once a day, respectively. After 90 days of treatment, the gastric mucosa pathological changes were observed, the changes of superoxide dismutase (SOD) and malonyl dialdehyde (MDA) content in blood serum were detected. Result: There was no marked difference in the differentiation of gastric mucosa pathematology and microanatomy between the whole formula group and the normal group. Compared with normal control group whose SOD is (239.88±6.32) U · mL<sup>-1</sup> and MDA is (3.17±0.02) μmol · L<sup>-1</sup>, SOD of model group with (174.59±12.81) U · mL<sup>-1</sup> decrease significantly (P<0.01) and MDA of model group with (5.14±0.15) μmol · L<sup>-1</sup> increase significantly (P<0.01). Compared with model group, the other medication groups made a significant increase of SOD and made the level of MDA decrease significantly (P<0.01 or P<0.05); Compared with model group with SOD (174.59±12.81) U · mL<sup>-1</sup>, MDA (5.14±0.15) μmol · L<sup>-1</sup>, the other medication groups made a significant increase of SOD and the level of MDA decrease significantly (P<0.01 or P<0.05); Compared with the whole formula group with SOD (233.91±9.03) U · mL<sup>-1</sup>, MDA: (3.11±0.19) μmol · L<sup>-1</sup>, the buyi group and quxie group made a

significant decrease of SOD and made the level of MDA increase significantly ( $P<0.01$  or  $P<0.05$ ); Compared with the buyi group with SOD ( $221.58\pm 5.71$ ) U · mL<sup>-1</sup>, the activity of SOD with ( $209.89\pm 5.27$ ) U · mL<sup>-1</sup> in serum was significantly lower in quxie group rats ( $P<0.05$ ). Conclusion: Weiweikang can improve and reverse the gastric atrophy of experimental atrophic gastritis of rats, among which ingredinets with benefiting QI and nourishing YIN founctios play a major role and ingredients with expelling pathogen plays synergy action. Its mechanism may be related to scavenging resisting the free radicals.

**keywords:** [Weiweikang](#) [analysis of the prescription](#) [chronic atrophic gastritis \(CAG\)](#) [pathological changes](#) [superoxide dismutas](#) [malonyl dialdehyd](#)

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