

# 慢性胃病脾虚证转归中血浆环核苷酸、<sup>3</sup>H-TdR 淋巴细胞转化的量变和作用

## ——兼论阴阳学说的物质基础

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**内容提要** 本文动态观察 51 例慢性胃病脾虚证患者血浆环核苷酸、<sup>3</sup>H-TdR 淋巴细胞转化和胃粘膜肠上皮化生的变化。cAMP 在脾虚证演化中起主导作用；cAMP 与 cGMP 并无互为消长的关系，阴阳学说的现代概念应建立在整体条件下神经体液调节的正负反馈基础上，而不能简单地把 cAMP 与 cGMP 作为这个学说的物质基础。

中医虚证与环核苷酸的关系，许多学者在实验和临床方面做了大量工作<sup>①~④</sup>。脾虚证比较集中地反映了机体消化吸收、代谢和免疫功能的低下，它多见于消化系统疾病。为探索脾虚证发生、发展和转归的规律，动态地观察 51 例慢性胃病脾虚证的血浆环磷酸腺苷 (cAMP pmol/ml)、环磷酸鸟苷 (cGMP pmol/ml)、氟胸腺嘧啶核苷掺入淋巴细胞转化试验 (<sup>3</sup>H-TdR LCT cpm/0.2ml 全血) 和胃粘膜肠上皮化生 (肠化) 的变化，发现它们的量变与脾虚证转归有关。现报道如下。

### 对象和方法

纤维胃镜或手术取胃粘膜作活组织病理学检查，明确诊断为慢性浅表性胃炎、萎缩性胃炎、胃溃疡和胃癌，并符合脾气虚证和脾虚气滞证标准者<sup>⑤</sup>，列为观察统计对象。51 例分为数个对照组，并与正常健康人组比较。每例均作四项指标检测：(1) 血浆 cAMP (放射免疫竞争性蛋白结合法)；(2) 血浆 cGMP (放射免疫膜片分离法)；(3) <sup>3</sup>H-TdR LCT；(4) 病理组织学胃粘膜肠化观察，病理切片中肠化程度按下列标准分轻、中、重三度。轻度：肠化在固有膜中呈灶性散在分布；中度：1/2 左右粘膜组织被灶性或斑片状肠化占据；重度：大部分粘膜组织被肠化占据。

### 临床资料

51 例中，慢性浅表性胃炎 16 例，慢性萎

缩性胃炎 9 例，胃溃疡 10 例，胃癌 16 例。分成二组：(1) 脾气虚证：27 例 (男 21、女 6)，年龄 23~71 岁，平均 43.8 岁；有肠化 16 例 (中度 6、轻度 10) 占 59.26%，无肠化 11 例占 40.74%；病变在胃窦部 20 例占 74.07%。(2) 脾虚气滞证：24 例 (男 17、女 7)，年龄 35~68 岁，平均 50.3 岁；有肠化 22 例 (重度 7、中度 12、轻度 3) 占 91.67%，无肠化 2 例占 8.33%；病变在胃窦部 20 例占 83.33%。二组的肠化率、肠化程度差异显著， $P < 0.01$ 。

### 结果和分析

几组对照结果，血浆 cAMP、<sup>3</sup>H-TdR LCT 量变比 cGMP 敏感。脾气虚证、脾虚气滞的 cAMP、<sup>3</sup>H-TdR LCT 比正常人低， $P < 0.01 \sim 0.001$ ；两证间差异显著， $P < 0.001$  (表 1)；脾气虚证肠化与无肠化间差异显著， $P < 0.01 \sim 0.001$  (表 2)；24 例脾虚气滞证仅 2 例无

表 1 脾虚证与正常人比较 (M±SD)

	正常人组	脾虚气滞证组 (n=24)	脾气虚证组 (n=27)	P*
cAMP	22.58±5.72 (n=39)	10.01 ±3.21*	18.61 ±4.93*	*
cGMP	6.15±1.68 (n=42)	5.72 ±2.83△	6.01 ±3.75△	△
cAMP/cGMP	3.67	2.54 ±1.76▲	4.55 ±3.08△	▲
<sup>3</sup> H-TdR LCT	38083±18296 (n=73)	7092.73 ±6887.26*	28578.76 ±15101.29▲	*

P\* 是脾虚气滞证与脾气虚证间比较；\* $P < 0.001$

▲ $P < 0.01$  △ $P > 0.05$  ○ $P < 0.05$ , 下同

表2 脾气虚证有肠化生与无肠化生比较

	有肠化生 (n=16)	无肠化生 (n=11)	P
cAMP	15.64±4.13	20.49±4.58	▲
cGMP	5.54±4.09	6.69±3.07	△
cAMP/cGMP	4.75±3.09	4.27±3.04	△
<sup>3</sup> H-TdR LCT	20501.84±8324.16	40327±15035.24	*

肠化, 故未作肠化间 t 检验。

肠化率、肠化程度和脾虚气滞证随慢性浅表性胃炎、萎缩性胃炎、胃溃疡和胃癌顺序相对递增, 而血浆 cAMP、<sup>3</sup>H-TdR LCT 和脾气虚证随此顺序递减(表3、4)。

5例脾虚气滞证(慢性浅表性胃炎2例, 萎缩性胃炎3例)经中西药治疗转化为脾气虚证, 复查胃粘膜病理组织, 3例中度肠化转化为轻度, 1例轻度肠化消失, 1例轻度肠化未变; 3例脾虚气滞证胃癌手术切除癌灶, 经中西药治疗转化为脾气虚证, 这8例cAMP、<sup>3</sup>H-TdR LCT在二证转化间差异显著,  $P<0.05\sim0.001$ (表5)。6例脾气虚证(慢性浅表性和萎缩性胃炎各3例)经中西药治疗消失, 复查胃粘膜病理组织, 3例轻度肠化消失, 1例中度肠化转为轻度, 2例肠化未变。这6例cAMP、<sup>3</sup>H-TdR LCT在脾气虚消失前后差异显著,  $P<0.05\sim0.01$ (表5)。2例脾气虚证萎缩性胃炎治疗未

表3 四种慢性胃病比较

	例数	肠化数	肠化程度 (例)	中肠 重化 度率 (%)	脾 虚 气 滞 证	脾 气 虚 证	cAMP→	cGMP	cAMP/cGMP	<sup>3</sup> H-TdR LCT
慢性浅表性胃炎	16	9	7 2	12.5	4	12	16.64±6.77	6.00±2.65	3.55±2.87	26023.41±14145.98
慢性萎缩性胃炎	9	8	2 5 1	66.7	4	5	14.47±6.38	5.52±3.29	3.98±2.83	15561.78±10421.67
胃 溃 疡	10	7	2 5	50.0	4	6	13.44±5.53	5.48±4.81	3.60±2.33	22627.70±23320.41
胃 癌	16	14	2 6 6	75.0	12	4	13.05±4.38	5.21±2.59	3.39±2.74	9946.29±10498.11

表4 肠化程度间比较

	脾 虚 气 滞 证 肠 化 程 度			脾 气 虚 证 肠 化 程 度		
	轻 度 (n=3)	中 度 (n=12)	重 度 (n=7)	轻 度 (n=10)	中 度 (n=6)	重 度 (n=0)
cAMP	10.61±1.47	9.22±3.52	8.84±3.45	15.96±3.53	14.08±3.63	
cGMP	5.02±2.59	4.28±2.25	4.38±1.65	4.43±1.32	5.58±6.29	
cAMP/cGMP	3.36±2.63	2.13±0.91	2.55±1.83	4.38±2.58	3.11±3.43	
<sup>3</sup> H-TdR LCT	13061.53±4352.86	6824.14±7726.37	3546.77±2469.78	20403.55±8651.99	16685.25±8285.59	

表5 脾虚证转化前后的比较

	8例脾虚气滞证转化为脾气虚证			6例脾 气 虚 证 消 失		
	转 化 前	转 化 后	P	消 失 前	消 失 后	P
cAMP	11.58±3.05	23.83±7.33	*	11.48±3.01	26.16±6.82	▲
cGMP	4.82±1.68	6.13±5.43	△	4.02±1.03	9.22±3.16	○
cAMP/cGMP	2.83±1.69	5.85±3.23	○	4.30±3.22	3.09±1.08	△
<sup>3</sup> H-TdR LCT	11458.25±9747.13	34515.60±24664.05	○	17550.10±7293.94	26285.67±7112.82	○

好转, 转化为脾虚气滞证, 转化前 cAMP 为  $14.12 \pm 2.83$ ,  $^3\text{H}$ -TdR LCT 为  $8538.20 \pm 7314.03$ ; 转化后 cAMP 为  $9.25 \pm 5.35$ ,  $^3\text{H}$ -TdR LCT 为  $4561.10 \pm 622.29$ , 中度肠化未变。

综上所述, 慢性胃病脾虚证患者血浆 cAMP 和细胞免疫功能水平低下, 脾虚证转化与环核苷酸、细胞免疫功能的水平有关。

## 讨 论

环核苷酸是调节机体生理功能、免疫能力、物质代谢和细胞增殖、分化的重要因子。在脾虚证转化的几个变量因子中, cAMP 是起主导作用的, 随其在血浆和组织中的浓度改变呈双向调节。cAMP 量过高或过低, 均能抑制组织细胞分化, 加速分裂, 也能抑制淋巴细胞转化。cAMP 低, 使一些非分裂周期的胃粘膜细胞进入分裂, 干扰了通过蛋白激酶对基因调节的过程。DNA 正常碱基组成或顺序(基因)发生变更, 引起细胞肠化、间变甚至癌变; cAMP 低, 抑制了淋巴细胞转化, 免疫机能降低, 故  $^3\text{H}$ -TdR LCT 下降。cAMP 愈低,  $^3\text{H}$ -TdR LCT 下降愈显著, 而肠化率和程度则随之升高, 以致脾气虚证向脾虚气滞证转化; 反之, 则脾虚气滞证向脾气虚证转化。临床上检测血浆环核苷酸和免疫机能, 对指导治疗、推测预后有一定的价值。

自 Goldberg 提出了环核苷酸可能是阴阳学说的物质基础以来, 国内外学者的研究初步

发现环核苷酸的变化与人体阴阳消长有一定的关系<sup>(1,2,6-8)</sup>。Koide 等研究肝部分切除后肝细胞的再生过程, 发现 cGMP 是通过在细胞内的移位来发挥调节作用的, 它与 cAMP 并无互为消长的关系<sup>(9)</sup>。本文资料亦不支持 Goldberg 观点。阴阳学说作为矛盾对立统一的哲学概念, 决非是一对物质所能包罗万象解释通的。这个古老学说的现代概念应建立在整体条件下的神经体液正负反馈基础上。

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## 中国中西医结合研究会河南分会成立虚证与老年病防治委员会并举办老年医学研究进展讲习班

中国中西医结合研究会河南分会于 1985 年 6 月 27 日在郑州成立虚证和老年病防治委员会, 由 15 名委员组成, 主任委员邱保国, 副主任委员宁选(兼)、李忠, 秘书周希桂。委员会拟在省内各基层医疗单位开展有关方面的研究, 以推动全省中西医结合虚证和老年病防治工作。同时, 并与中华医学会河南分会老年医学委员联合举办了第一届老年医学研究进展讲习班, 来自全省各地学员共 81 名, 由省内有关院校、研

究单位和医院的专家、教授担任讲课, 其内容有: “衰老的机制”、“衰老的测定”、“抗衰老药物”、“老年脑血管病”、“病态窦房结综合征”、“冠心病”、“高血压”、“肿瘤”的中西医结合防治进展等 16 个专题。通过学习, 大家表示要在本地区本部门积极开展老年医学方面的研究, 在中西医结合防治老年病方面争取于短期内取得成果。

(李 忠)



# **A Study of $^3\text{H}$ -TdR Lymphocyte Transfer Value and Plasma cAMP and cGMP Level in Patients with Chronic Gastric Diseases due to Spleen Deficiency and Its Clinical Significance**

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The relationship between the intestinal metaplastic changes and plasma cAMP and cGMP level and  $^3\text{H}$ -TdR lymphocyte transfer rate in 51 cases of chronic gastric diseases with spleen deficiency has been observed. The cAMP level and  $^3\text{H}$ -TdR lymphocyte transfer in patients with spleen deficiency were found lower than those in normal subjects ( $P < 0.001$ ). There was significant difference in these between patients with deficiency of spleen-energy and spleen deficiency with energy stagnation, between patients with or without metaplasia, between patients with severe or mild metaplasia, and between patients with spleen deficiency in a changing process ( $P < 0.05 \sim 0.001$ ). However, no marked alteration of plasma cGMP level was observed. All these findings suggest that cAMP might affect through its quantitative alteration the chronic gastric diseases with spleen deficiency changes and cell metabolism, atypical and cancerous changes in mucosa. The quantitative alteration of cAMP may serve as a means to regulate the cell immune capacity when cAMP is very low, cell immune capacity could be restricted; The regulating function of cGMP upon cell generation and cell immunity does not depend on its quantitative alteration. Therefore, cAMP and cGMP cannot be regarded as material basis of "Yin-Yang" theory.

(Original article on page 671)

## **On the Theory that Fat Persons Are Subject to Deficiency of Yang and Phlegm-Damp While Thin Persons to Deficiency of Yin and Vigorous Fire — An Investigation of Relationship Between Bodily Form and Health Condition Made among 1257 cases**

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Synchronous investigation was made of 1257 subjects in Yi Wu in Southeast China, Yan An in Northwest China and Shanghai, to study relationship between bodily form and health condition. It has been found that the proportion of cases with deficiency of Yang and phlegm-damp of fat persons is obviously higher than that of the other two types, while thin persons are more susceptible to deficiency of Yin-blood or deficiency Yin and vigorous fire induced by it. The old saying was thus confirmed by the investigation. It has been pointed out by the author that the theory cannot serve as a determinant in making diagnosis of deficiency of Yin or Yang, but only as a reference since it is relative. Furthermore, the relationship between bodily form and constitution has been found to vary slightly with different districts and seasons. So locality and time should be taken into consideration while this theory is applied to clinical practice. In this connection Zhu Dangxi's (朱丹溪) theory on geographical background was expounded.

(Original article on page 674)

## **The Effect of Sodium Ferulate on Lipid Peroxidation of Erythrocyte Membrane**

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Sodium ferulate (SF) is an active ingredient from a Chinese herb, *Angelica sinensis*, used for inhibition of platelet aggregation. It was attempted to see if SF could inhibit lipid peroxidation of red blood cells and prevent hemolysis of paroxysmal nocturnal hemoglobinuria (PNH) erythrocytes. Normal and PNH erythrocytes were incubated with melonyl dialdehyde (MDA) and SF, percentage of hemolysis was measured, SF could remarkably reduced hemolysis induced by MDA. Sick-cell anemia erythrocytes was incubated with zymosan activated neutrophils and SF. The concentration of peroxidation product MDA was measured. It showed that the higher the concentration of SF, the lower the MDA product. SF was incubated with MDA and  $\text{H}_2\text{O}_2$  separately and concentrations of MDA and  $\text{H}_2\text{O}_2$  were measured. MDA concentration did not change after SF treatment, where  $\text{H}_2\text{O}_2$  concentration was significantly reduced. SF could combine with phosphorylethanoamine, which may be the main cause of protection of lipid peroxidation.

(Original article on page 678)