

Abstracts

1. Effect of short-term practice of breathing exercises on autonomic functions in normal human volunteers.

[Pal GK](#)¹, [Velkumary S](#), [Madanmohan](#). [Indian J Med Res](#). 2004 Aug;120(2):115-21.

BACKGROUND & OBJECTIVES:

Practice of breathing exercises like pranayama is known to improve autonomic function by changing sympathetic or parasympathetic activity. Therefore, in the present study the effect of breathing exercises on autonomic functions was performed in young volunteers in the age group of 17-19 yr.

METHODS:

A total of 60 male undergraduate medical students were randomly divided into two groups: slow breathing group (that practiced slow breathing exercise) and the fast breathing group (that practiced fast breathing exercise). The breathing exercises were practiced for a period of three months. Autonomic function tests were performed before and after the practice of breathing exercises.

RESULTS:

The increased parasympathetic activity and decreased sympathetic activity were observed in slow breathing group, whereas no significant change in autonomic functions was observed in the fast breathing group.

INTERPRETATION & CONCLUSION:

The findings of the present study show that regular practice of slow breathing exercise for three months improves autonomic functions, while practice of fast breathing exercise for the same duration does not affect the autonomic functions.

PMID: 15347862

2. Effect of slow- and fast-breathing exercises on autonomic functions in patients with essential hypertension.

[Mourya M¹, Mahajan AS, Singh NP, Jain AK. J Altern Complement Med. 2009 Jul;15\(7\):711-7. doi: 10.1089/acm.2008.0609. PMID: 19534616 DOI: \[10.1089/acm.2008.0609\]\(#\)](#)

OBJECTIVES: Breathing exercises practiced in various forms of meditations such as yoga may influence autonomic functions. This may be the basis of therapeutic benefit to hypertensive patients.

DESIGN: The study design was a randomized, prospective, controlled clinical study using three groups.

SUBJECTS: The subjects comprised 60 male and female patients aged 20-60 years with stage 1 essential hypertension.

INTERVENTION: Patients were randomly and equally divided into the control and other two intervention groups, who were advised to do 3 months of slow-breathing and fast-breathing exercises, respectively. Baseline and postintervention recording of blood pressure (BP), autonomic function tests such as standing-to-lying ratio (S/L ratio), immediate heart rate response to standing (30:15 ratio), Valsalva ratio, heart rate variation with respiration (E/I ratio), hand-grip test, and cold pressor response were done in all subjects.

RESULTS: Slow breathing had a stronger effect than fast breathing. BP decreased longitudinally over a 3-month period with both interventions. S/L ratio, 30:15 ratio, E/I ratio, and BP response in the hand grip and cold pressor test showed significant change only in patients practicing the slow-breathing exercise.

CONCLUSIONS: Both types of breathing exercises benefit patients with hypertension. However, improvement in both the sympathetic and parasympathetic reactivity may be the mechanism that is associated in those practicing the slow-breathing exercise.

3. Heart rate variability biofeedback: how and why does it work?

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PMC4104929 PMID: [25101026](https://pubmed.ncbi.nlm.nih.gov/25101026/)

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In recent years there has been substantial support for heart rate variability biofeedback (HRVB) as a treatment for a variety of disorders and for performance enhancement ([Gevirtz, 2013](#)). Since conditions as widely varied as asthma and depression seem to respond to this form of cardiorespiratory feedback training, the issue of possible mechanisms becomes more salient. The most supported possible mechanism is the strengthening of homeostasis in the baroreceptor ([Vaschillo et al., 2002](#); [Lehrer et al., 2003](#)). Recently, the effect on the vagal afferent pathway to the frontal cortical areas has been proposed. In this article, we review these and other possible mechanisms that might explain the positive effects of HRVB.

4. M. Huston, J. Tracey. The pulse of inflammation: heart rate variability, the cholinergic anti-inflammatory pathway and implications for therapy. *Journal of Internal Medicine.* 2011; 269 (1): 45 - 53.

Biological therapeutics targeting TNF, IL-1 and IL-6 are widely used for treatment of rheumatoid arthritis, inflammatory bowel disease and a growing list of other syndromes, often with remarkable success. Now advances in neuroscience have collided with this therapeutic approach, perhaps rendering possible the development of nerve stimulators to inhibit cytokines. Action potentials transmitted in the vagus nerve culminate in the release of acetylcholine that blocks cytokine production by cells expressing acetylcholine receptors. The molecular mechanism of this cholinergic anti-inflammatory pathway is attributable to signal transduction by the nicotinic alpha 7 acetylcholine receptor subunit, a regulator of the intracellular signals that control cytokine transcription and translation. Favourable preclinical data support the possibility that nerve stimulators may be added to the future therapeutic armamentarium, possibly replacing some drugs to inhibit cytokines.

5. Libby P. Inflammatory mechanisms: the molecular basis of inflammation and disease. *Nutr Rev.* 2007 Dec;65(12 Pt 2):S140-6.

Inflammation participates importantly in host defenses against infectious agents and injury, but it also contributes to the pathophysiology of many chronic diseases. Interactions of cells in the innate immune system, adaptive immune system, and inflammatory mediators orchestrate aspects of the acute and chronic inflammation that underlie diseases of many organs. A coordinated series of common effector mechanisms of inflammation contribute to tissue injury, oxidative stress, remodeling of the extracellular matrix, angiogenesis, and fibrosis in diverse target tissues. Atherosclerosis provides an example of a chronic disease that involves inflammatory mechanisms. Recruitment of blood leukocytes characterizes the initiation of this disease. Its progression involves many inflammatory mediators, modulated by cells of both innate and adaptive immunity. The complications of established atheroma, including plaque disruption and thrombosis, also intimately involve inflammation. Mastery of the inflammatory response should aid the development of novel strategies to predict disease susceptibility, target and monitor therapies, and ultimately develop new approaches to the prevention and treatment of chronic diseases associated with aging, such as atherosclerosis.

6. Borovikova LV, Ivanova S, Nardi D et al. Role of vagus nerve signaling in CNI-1493-mediated suppression of acute inflammation. *Auton Neurosci* 2000; 85: 141–7.

CNI-1493 is a potent anti-inflammatory agent, which deactivates macrophages and inhibits the synthesis of proinflammatory mediators. The objective of the present study was to identify the role of the central nervous system (CNS) and efferent vagus nerve signaling in CNI-1493-mediated modulation of acute inflammation in the periphery. CNI-1493 was administered either intracerebroventricularly (i.c.v., 0.1-1,000 ng/kg) or intravenously (i.v., 5 mg/kg) in anesthetized rats subjected to a standard model of acute inflammation (subcutaneous (s.c.) injection of carrageenan). I.c.v. CNI-1493 significantly suppressed carrageenan-induced paw edema, even in doses at least 6-logs lower than those required for a systemic effect. Bilateral cervical vagotomy or atropine blockade (1 mg/kg/h) abrogated the anti-inflammatory effects of CNI-1493 (1 microg/kg, i.c.v. or 5 mg/kg, i.v.), indicating that the intact vagus nerve is required for CNI-1493 activity. Recording of the efferent vagus nerve activity revealed an increase in discharge rate starting at 3-4 min after CNI-1493 administration (5 mg/kg, i.v.) and lasting for 10-14 min (control activity=87±5.4 impulses/s versus CNI-1493-induced activity= 229±6.7 impulses/s). Modulation of efferent vagus

nerve activity by electrical stimulation (5 V, 2 ms, 1 Hz) of the transected peripheral vagus nerve for 20 min (10 min before carrageenan administration and 10 min after) also prevented the development of acute inflammation. Local administration of the vagus nerve neurotransmitter, acetylcholine (4 microg/kg, s.c.), or cholinergic agonists into the site of carrageenan-injection also inhibited acute inflammation. These results now identify a previously unrecognized role of efferent vagus nerve activity in mediating the central action of an anti-inflammatory agent.

7. Borovikova LV, Ivanova S, Nardi D et al. Role of vagus nerve signaling in CNI-1493-mediated suppression of acute inflammation. *Auton Neurosci* 2000; 85: 141–7.

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8. Borovikova LV, Ivanova S, Zhang M et al. Vagus nerve stimulation attenuates the systemic inflammatory response to endotoxin. *Nature* 2000; 405: 458–62

Vertebrates achieve internal homeostasis during infection or injury by balancing the activities of proinflammatory and anti-inflammatory pathways. Endotoxin (lipopolysaccharide), produced by all gram-negative bacteria, activates macrophages to release cytokines that are potentially lethal. The central nervous system regulates systemic inflammatory responses to endotoxin through humoral mechanisms. Activation of afferent vagus nerve fibres by endotoxin or cytokines stimulates

hypothalamic-pituitary-adrenal anti-inflammatory responses. However, comparatively little is known about the role of efferent vagus nerve signalling in modulating inflammation. Here, we describe a previously unrecognized, parasympathetic anti-inflammatory pathway by which the brain modulates systemic inflammatory responses to endotoxin. Acetylcholine, the principle vagal neurotransmitter, significantly attenuated the release of cytokines (tumour necrosis factor (TNF), interleukin (IL)-1beta, IL-6 and IL-18), but not the anti-inflammatory cytokine IL-10, in lipopolysaccharide-stimulated human macrophage cultures. Direct electrical stimulation of the peripheral vagus nerve in vivo during lethal endotoxaemia in rats inhibited TNF synthesis in liver, attenuated peak serum TNF amounts, and prevented the development of shock.

9. Tracey KJ. The inflammatory reflex. *Nature* 2002; 420: 853–9..

Huston JM, Ochani M, Rosas-Ballina M et al. Splenectomy inactivates the cholinergic anti-inflammatory pathway during lethal endotoxemia and polymicrobial sepsis. *J Exp Med* 2006; 203: 1623–8

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11. Tracey KJ. The inflammatory reflex. *Nature* 2002; 420: 853–9..

Inflammation is a local, protective response to microbial invasion or injury. It must be fine-tuned and regulated precisely, because deficiencies or excesses of the inflammatory response cause morbidity and shorten lifespan. The discovery that cholinergic neurons inhibit acute inflammation has qualitatively expanded our understanding of how the nervous system modulates immune responses. The nervous system reflexively regulates the inflammatory response in real time, just as it controls heart rate and other vital functions. The opportunity now exists to apply this insight to the treatment of inflammation through selective and reversible 'hard-wired' neural systems.

12. Huston JM, Ochani M, Rosas-Ballina M et al. Splenectomy inactivates the cholinergic anti-inflammatory pathway during lethal endotoxemia and polymicrobial sepsis. *J Exp Med* 2006; 203: 1623–8

The innate immune system protects against infection and tissue injury through the specialized organs of the reticuloendothelial system, including the lungs, liver, and spleen. The central nervous system regulates innate immune responses via the vagus nerve, a mechanism termed the cholinergic antiinflammatory pathway. Vagus nerve stimulation inhibits proinflammatory cytokine production by signaling through the alpha7 nicotinic acetylcholine receptor subunit. Previously, the functional relationship between the cholinergic antiinflammatory pathway and the reticuloendothelial system was unknown. Here we show that vagus nerve stimulation fails to inhibit tumor necrosis factor (TNF) production in splenectomized animals during lethal endotoxemia. Selective lesioning of the common celiac nerve abolishes TNF suppression by vagus nerve stimulation, suggesting that the cholinergic pathway is functionally hard wired to the spleen via this branch of the vagus nerve. Administration of nicotine, an alpha7 agonist that mimics vagus nerve stimulation, increases proinflammatory cytokine production and lethality from polymicrobial sepsis in splenectomized mice, indicating that the spleen is critical to the protective response of the cholinergic pathway. These results reveal a specific, physiological connection between the nervous and innate immune systems that may be exploited through either electrical vagus nerve stimulation or administration of alpha7 agonists to inhibit proinflammatory cytokine production during infection and tissue injury.

10. Oh B, Butow P, Mullan B, Clarke S, Beale P, Pavlakis N, Kothe E, Lam L, Rosenthal D. Impact of medical Qigong on quality of life, fatigue, mood and inflammation in cancer patients: a randomized controlled trial. *Ann Oncol.* 2010 Mar;21(3):608-14.

BACKGROUND:

Substantial numbers of cancer patients use complementary medicine therapies, even without a supportive evidence base. This study aimed to evaluate in a randomized controlled trial, the use of Medical Qigong (MQ) compared with usual care to improve the quality of life (QOL) of cancer patients.

PATIENTS AND METHODS:

One hundred and sixty-two patients with a range of cancers were recruited. QOL and fatigue were measured by Functional Assessment of Cancer Therapy-General and Functional Assessment of Cancer Therapy-Fatigue, respectively, and mood status by Profile of Mood State. The inflammatory marker serum C-reactive protein (CRP) was monitored serially.

RESULTS:

Regression analysis indicated that the MQ group significantly improved overall QOL ($t(144) = -5.761, P < 0.001$), fatigue ($t(153) = -5.621, P < 0.001$), mood disturbance ($t(122) = 2.346, P = 0.021$) and inflammation (CRP) ($t(99) = 2.042, P < 0.044$) compared with usual care after controlling for baseline variables.

CONCLUSIONS:

This study indicates that MQ can improve cancer patients' overall QOL and mood status and reduce specific side-effects of treatment. It may also produce physical benefits in the long term through reduced inflammation.

11. Oh B, Butow PN, Mullan BA, Clarke SJ, Beale PJ, Pavlakis N, Lee MS, Rosenthal DS, Larkey L, Vardy J. Effect of medical Qigong on cognitive function, quality of life, and a biomarker of inflammation in cancer patients: a randomized controlled trial. *Support Care Cancer*. 2012 Jun;20(6):1235-42.

PURPOSE:

Cancer patients often experience diminished cognitive function (CF) and quality of life (QOL) due to the side effects of treatment and the disease symptoms. This study evaluates the effects of medical Qigong (MQ; combination of gentle exercise and meditation) on CF, QOL, and inflammation in cancer patients.

METHODS:

Eighty-one cancer patients recruited between October 2007 and May 2008 were randomly assigned to two groups: a control group ($n = 44$) who received the usual health care and an intervention group ($n = 37$) who participated in a 10-week MQ program. Self-reported CF was measured by the European Organization for Research and Treatment of Cancer (EORTC-CF) and the Functional Assessment of Cancer Therapy-Cognitive (FACT-Cog).

The Functional Assessment of Cancer Therapy-General (FACT-G) was used to measure QOL. C-reactive protein (CRP) was assessed as a biomarker of inflammation.

RESULTS:

The MQ group self-reported significantly improved CF (mean difference (MD) = 7.78, $t(51) = -2.532$, $p = 0.014$) in the EORTC-CF and all the FACT-Cog subscales [perceived cognitive impairment (MD = 4.70, $t(43) = -2.254$, $p = 0.029$), impact of perceived cognitive impairment on QOL (MD = 1.64, $t(45) = -2.377$, $p = 0.024$), and perceived cognitive abilities (MD = 3.61, $t(45) = -2.229$, $p = 0.031$)] compared to controls. The MQ group also reported significantly improved QOL (MD = 12.66, $t(45) = -5.715$, $p < 0.001$) and had reduced CRP levels (MD = -0.72, $t(45) = 2.092$, $p = 0.042$) compared to controls.

CONCLUSIONS:

Results suggest that MQ benefits cancer patients' self-reported CF, QOL, and inflammation. A larger randomized controlled trial including an objective assessment of CF is planned.

12. Irwin MR, Olmstead R. Mitigating cellular inflammation in older adults: a randomized controlled trial of Tai Chi Chih. *Am J Geriatr Psychiatry*. 2012 Sep;20(9):764-72.

OBJECTIVES:

To evaluate the effects of a behavioral intervention, Tai Chi Chih (TCC) on circulating markers of inflammation in older adults.

DESIGN:

A prospective, randomized, controlled trial with allocation to two arms, TCC and health education (HE), 16 weeks of intervention administration, and 9 weeks follow-up.

PARTICIPANTS:

A total of 83 healthy older adults, aged 59 to 86 years.

MEASUREMENTS:

The primary endpoint was circulating levels of interleukin 6 (IL-6). Secondary outcomes were circulating levels of C-reactive protein, soluble IL-1 receptor antagonist, soluble IL-6 receptor, soluble intercellular adhesion molecule, and IL-18. Severity of depressive symptoms, sleep quality, and physical activity was also assessed over the treatment trial.

RESULTS:

Among those older adults with high levels of IL-6 at entry, a trend for a treatment group by time interaction was found ($F[1,70] = 3.48$, $p = 0.07$), in which TCC produced a drop of IL-6 levels comparable to those found in TCC and HE subgroups who had low levels of IL-6 at entry (t_{72} 's = 0.80, 1.63, p 's > 0.10), whereas IL-6 in HE remained higher than the TCC and HE subgroups with low entry IL-6 ($t_{72} = 2.47$, $p = 0.02$; $t_{72} = 1.71$, $p = 0.09$). Decreases in depressive symptoms in the two treatment groups correlated with decreases of IL-6 ($r =$

0.28, $p < 0.05$). None of the other cellular markers of inflammation changed in TCC versus HE.

CONCLUSION:

TCC can be considered a useful behavioral intervention to reduce circulating levels of IL-6 in older adults who show elevated levels of this inflammatory marker and are at risk for inflammation-related morbidity.

13. Mohan M, Saravanane C, Surange SG, Thombre DP, Chakrabarthy AS. Effect of yoga type breathing on heart rate and cardiac axis of normal subjects. *Indian J Physiol Pharmacol* 1986; 30: 334-40

Effect of inspiratory and expiratory phases of normal quiet breathing, deep breathing and savitri pranayam type breathing on heart rate and mean ventricular QRS axis was investigated in young, healthy untrained subjects. Pranayam type breathing produced significant cardioacceleration and increase in QRS axis during the inspiratory phase as compared to eupnea. On the other hand, expiratory effort during pranayam type breathing did not produce any significant change in heart rate or QRS axis. The changes in heart rate and QRS axis during the inspiratory and expiratory phases of pranayam type breathing were similar to the changes observed during the corresponding phases of deep breathing.

14. Pal G, Velkumary S, Madanmohan. Effect of short-term practice of breathing exercises on autonomic functions in normal human volunteers. *Indian J Med Res* 2004;120(2): 115–21.

BACKGROUND & OBJECTIVES: Practice of breathing exercises like pranayama is known to improve autonomic function by changing sympathetic or parasympathetic activity. Therefore, in the present study the effect of breathing exercises on autonomic functions was performed in young volunteers in the age group of 17-19 yr.

METHODS: A total of 60 male undergraduate medical students were randomly divided into two groups: slow breathing group (that practiced slow breathing exercise) and the fast breathing group (that practiced fast breathing exercise). The breathing exercises were practiced for a period of three months. Autonomic function tests were performed before and after the practice of breathing exercises.

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INTERPRETATION & CONCLUSION: The findings of the present study show that regular practice of slow breathing exercise for three months improves autonomic functions, while practice of fast breathing exercise for the same duration does not affect the autonomic functions.

PMID: 15347862

15. Lee, M. S., Huh, H. J., Kim, B. G., Ryu, H., Lee, H. S., Kim, J. M., & Chung, H. T. Effects of Qi-training on heart rate variability. *The American Journal of Chinese Medicine*. 2002; 30: 463-470.

This study investigates changes in autonomic nervous function through Qi-training. The power spectrum of heart rate variability (HRV) was examined in 20 sedentary healthy subjects and 20 Qi-trainees. It was found that Qi-training in healthy young subjects during controlled respiration increases the high frequency (HF) power and decreases the low frequency / high frequency (LF/HF) power ratio of HRV. These results support the hypothesis that Qi-training increases cardiac parasympathetic tone. In addition, Qi-trainees were found to have higher parasympathetic heart modulation compared with their age-matched, sedentary counterparts. This augmented HRV in Qi-trainees provides further support for long-term Qi-training as a possible non-pharmacological cardio-protective maneuver. In conclusion, Qi-training may stabilize the autonomic nervous system by modulating the parasympathetic nervous system.

16. Lu WA, Kuo CD. The effect of Tai Chi Chuan on the autonomic nervous modulation in older persons. *Med Sci Sports Exerc*. 2003 Dec;35(12):1972-6.

PURPOSE:

This study evaluated the effect of Tai Chi Chuan (TCC) on the autonomic nervous modulation in older persons.

METHODS:

Twenty TCC practitioners and 20 normal controls were included in this study. The stationary

state spectral heart rate variability (HRV) measures between TCC practitioners and normal controls, and the sequential changes in HRV measures after classical Yang's TCC were compared.

RESULTS:

The total power, very low-frequency power, low-frequency power, normalized low-frequency power, and low-/high-frequency power ratios in TCC practitioners were all significantly higher than those of normal controls, whereas the heart rate and systolic and diastolic blood pressures were not different between these two groups of subjects. After TCC, the normalized high-frequency power increased significantly from 22.8 +/- 14.6 normalized units (nu) before TCC to 28.2 +/- 16.1 nu 30 min after TCC and to 30.6 +/- 18.4 nu 60 min after TCC. In contrast, the low-/high-frequency power ratio decreased significantly from 2.5 +/- 2.4 before TCC to 1.8 +/- 1.4 30 min after TCC and to 2.2 +/- 2.9 60 min after TCC. The heart rate, systolic blood pressure, diastolic blood pressure, mean arterial blood pressure, and pulse pressure also decreased sequentially after TCC.

CONCLUSION:

The short-term effect of TCC was to enhance the vagal modulation and tilt the sympathovagal balance toward decreased sympathetic modulation in older persons. TCC might be good health-promoting calisthenics for older persons.