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## Biochemical markers of antioxidant and anti-inflammatory activities of *Momordica charantia* in human studies

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### Abstract

*Momordica charantia* L. (bitter gourd or karela) is widely consumed as a vegetable and therapeutic agent in Asia, Africa, and Latin America. Traditionally employed in Ayurveda, Chinese medicine, and folk healing systems for the treatment of diabetes and infections, bitter gourd has attracted growing scientific interest for its antioxidant and anti-inflammatory properties. These effects are mediated by a broad spectrum of phytochemicals including cucurbitane-type triterpenoids, phenolics, flavonoids, and peptides. In human studies, bitter gourd supplementation has been associated with modulation of biochemical markers such as superoxide dismutase (SOD), glutathione peroxidase (GPx), catalase, malondialdehyde (MDA), nitric oxide (NO), C-reactive protein (CRP), tumor necrosis factor-alpha (TNF- $\alpha$ ), and interleukins (IL-6, IL-1 $\beta$ , IL-10).

This paper reviews available clinical and interventional studies to analyze how bitter gourd influences antioxidant enzyme activity and inflammatory cytokine regulation in humans. Evidence indicates that supplementation with juice, extracts, or capsules of *M. charantia* enhances endogenous antioxidant defenses, reduces lipid peroxidation, and lowers systemic inflammation. Although findings are promising, variability in sample sizes, study duration, extract standardization, and biomarker selection limits the strength of conclusions. Future clinical trials with rigorous methodology and comprehensive biomarker panels are needed to establish bitter gourd as a scientifically validated nutraceutical for oxidative stress and inflammation-related disorders.

**Keywords:** *Momordica charantia*, antioxidant enzymes, inflammatory cytokines, human studies, biochemical markers

### Introduction

Chronic oxidative stress and low-grade inflammation are increasingly recognized as central drivers of non-communicable diseases, including diabetes mellitus, cardiovascular disorders, metabolic syndrome, and neurodegenerative conditions. The interplay between reactive oxygen species (ROS) and inflammatory cytokines creates a vicious cycle in which oxidative stress triggers inflammatory pathways and vice versa. The assessment of biochemical markers such as enzymatic antioxidants, lipid peroxidation products, and inflammatory mediators provides important insights into disease progression and therapeutic efficacy.

*Momordica charantia* L. (bitter gourd) is a tropical and subtropical plant belonging to the family Cucurbitaceae, consumed both as a food and a medicinal herb. Traditional medicine systems have long employed bitter gourd for diabetes (*Prameha* in Ayurveda), infections, digestive disorders, and skin ailments. In the modern context, phytochemical investigations have demonstrated that bitter gourd contains cucurbitane-type triterpenoids (charantin, momordicosides), flavonoids (quercetin, rutin), phenolic acids (gallic acid, chlorogenic acid), alkaloids, peptides (polypeptide-p), and saponins, all of which contribute to its bioactivity.

While antioxidant and anti-inflammatory properties of *M. charantia* are well established in animal and *in vitro* studies, evidence from human trials is of greater clinical importance. Biochemical markers measured in human studies, including superoxide dismutase (SOD), catalase, glutathione peroxidase (GPx), malondialdehyde (MDA), C-reactive protein (CRP), nitric oxide (NO), and cytokines like tumor necrosis factor-alpha (TNF- $\alpha$ ) and interleukins (IL-6, IL-10), provide objective measures of the plant's biological effects.

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This paper presents a comprehensive review of human studies investigating the influence of *M. charantia* on biochemical markers of antioxidant and anti-inflammatory activities. The objectives are to synthesize current evidence, identify gaps, and suggest directions for future research to establish bitter gourd as a validated nutraceutical in oxidative stress and inflammation management.

### Phytochemical Basis of Antioxidant and Anti-Inflammatory Activities

The antioxidant effects of bitter gourd arise largely from its polyphenolic and flavonoid content. Quercetin, catechins, rutin, and gallic acid scavenge free radicals and enhance antioxidant enzyme activity. Cucurbitane-type triterpenoids, including charantin and momordicosides, activate AMP-activated protein kinase (AMPK), which indirectly modulates oxidative stress and inflammation.

Anti-inflammatory properties are attributed to saponins, peptides, and triterpenoids that inhibit nuclear factor-kappa B (NF- $\kappa$ B) signaling, downregulate pro-inflammatory cytokines, and upregulate anti-inflammatory mediators. Lectins in bitter gourd also stimulate immune responses and regulate cytokine production.

These phytochemicals influence not only direct radical scavenging but also gene expression of antioxidant enzymes and inflammatory mediators, making biochemical markers in human studies a meaningful measure of their activity.

### Biochemical Markers in Human Studies

#### Antioxidant Markers

**Superoxide dismutase (SOD):** SOD is a key antioxidant enzyme that converts superoxide radicals into hydrogen peroxide. Several clinical trials show that bitter gourd supplementation significantly increases SOD activity in patients with type 2 diabetes, reflecting enhanced antioxidant defense.

**Catalase (CAT):** Catalase decomposes hydrogen peroxide into water and oxygen. Studies report elevated catalase activity after four to twelve weeks of bitter gourd supplementation, suggesting improved detoxification of ROS.

**Glutathione peroxidase (GPx):** GPx reduces peroxides using glutathione. Human studies indicate increases in GPx activity, correlating with reduced oxidative stress markers.

**Malondialdehyde (MDA):** MDA is a marker of lipid peroxidation. Bitter gourd supplementation has consistently reduced MDA levels in human studies, confirming its role in limiting lipid oxidative damage.

### Anti-Inflammatory Markers

**C-reactive protein (CRP):** CRP is an acute-phase protein elevated in systemic inflammation. Some trials demonstrate reductions in CRP after bitter gourd supplementation, though findings are inconsistent due to short intervention periods.

**Tumor necrosis factor-alpha (TNF- $\alpha$ ):** TNF- $\alpha$  is a pro-inflammatory cytokine linked to insulin resistance and chronic disease. Bitter gourd extracts have been shown to lower TNF- $\alpha$  levels, particularly in metabolic syndrome and diabetic patients.

**Interleukins (IL-6, IL-1 $\beta$ , IL-10):** IL-6 and IL-1 $\beta$  are pro-inflammatory cytokines, while IL-10 has anti-inflammatory roles. Studies report reductions in IL-6 and IL-1 $\beta$ , with simultaneous increases in IL-10, suggesting immunomodulatory effects.

**Nitric oxide (NO):** Bitter gourd influences NO levels, balancing vascular homeostasis and reducing inflammatory stress. Enhanced NO availability in human studies is associated with improved endothelial function.

### Evidence from Human Studies

#### Clinical Trials in Diabetic Patients

The majority of human studies focus on diabetic populations. In a randomized controlled trial involving 100 type 2 diabetic patients, supplementation with 2000 mg/day of bitter gourd extract for 12 weeks significantly increased SOD, CAT, and GPx activities while lowering MDA and TNF- $\alpha$  levels. Another trial using fresh juice (50 mL/day) for 4 weeks reported improvements in fasting blood glucose accompanied by reductions in MDA and CRP, highlighting both metabolic and biochemical effects.

#### Studies in Metabolic Syndrome

In individuals with metabolic syndrome, supplementation with standardized capsules of *M. charantia* led to reductions in oxidative stress markers, with significant decreases in MDA and CRP and increased antioxidant enzyme activity. Improvements in lipid profiles were also observed, suggesting integrated effects on oxidative stress and inflammation.

### General Population and Dietary Supplementation

Few studies have assessed bitter gourd supplementation in healthy individuals. One small study administering bitter gourd tea for six weeks found moderate increases in SOD and GPx activity, indicating that even in non-diseased populations, antioxidant defenses can be enhanced. However, changes in inflammatory markers were less pronounced.

**Table 1:** Biochemical markers of antioxidant and anti-inflammatory activities in human studies on *M. charantia*

Study Population	Intervention (Form & Duration)	Antioxidant Markers Improved	Anti-Inflammatory Markers Improved
Type 2 Diabetes	Extract 2000 mg/day, 12 weeks	↑ SOD, ↑ CAT, ↑ GPx, ↓ MDA	↓ TNF- $\alpha$ , ↓ CRP
Type 2 Diabetes	Fresh juice 50 mL/day, 4 weeks	↑ SOD, ↓ MDA	↓ CRP
Metabolic Syndrome	Capsules 1.5 g/day, 8 weeks	↑ SOD, ↑ GPx, ↓ MDA	↓ IL-6, ↑ IL-10, ↓ CRP
Healthy Adults	Bitter gourd tea, 6 weeks	↑ SOD, ↑ GPx	Minimal effect on cytokines

### Discussion

The findings from human studies collectively support the role of *Momordica charantia* as a modulator of oxidative stress and inflammation. Improvements in SOD, GPx, and

catalase activities reflect enhanced endogenous antioxidant capacity, while reductions in MDA demonstrate protection against lipid peroxidation. These changes are consistent with phytochemical evidence showing that flavonoids and

triterpenoids activate antioxidant gene expression and scavenge free radicals.

The anti-inflammatory profile, characterized by reductions in CRP, TNF- $\alpha$ , and IL-6 and increases in IL-10, aligns with known mechanisms by which bitter gourd compounds inhibit NF- $\kappa$ B signaling and modulate cytokine expression. This suggests that bitter gourd does not simply suppress inflammation but also restores immunological balance.

However, limitations persist. Sample sizes in most clinical studies remain small, often below 150 participants. Duration of intervention rarely exceeds 12 weeks, which restricts understanding of long-term effects. Standardization of extracts is inconsistent, with some studies using fresh juice and others employing capsules of varying phytochemical composition. Furthermore, not all biomarker panels are comprehensive, with many studies focusing only on one or two antioxidant enzymes or inflammatory cytokines.

Despite these challenges, the consistency of results across diverse populations strengthens the conclusion that *M. charantia* exerts measurable biochemical effects in humans. The modulation of oxidative and inflammatory markers is particularly relevant for chronic diseases where these processes underlie pathology.

### Future Directions

Future research should address several gaps. Large, multicenter randomized controlled trials with standardized extracts are required to confirm efficacy. Comprehensive biomarker panels including oxidative DNA damage markers, advanced lipid peroxidation products, and a broader cytokine profile would provide more detailed mechanistic insights. Long-term studies are needed to evaluate safety and sustained efficacy. Additionally, pharmacokinetic studies on the absorption and metabolism of key phytochemicals like charantin and momordicosides will help explain inter-individual variability in biomarker responses.

Integration of nutrigenomic approaches, analyzing gene expression changes in response to bitter gourd supplementation, could further clarify molecular mechanisms. Finally, combining bitter gourd with other antioxidant-rich foods or herbal extracts in dietary interventions may reveal synergistic effects, enhancing therapeutic outcomes.

### Conclusion

Human studies investigating the antioxidant and anti-inflammatory activities of *Momordica charantia* consistently demonstrate beneficial effects on biochemical markers. Supplementation enhances antioxidant enzyme activity (SOD, GPx, catalase), reduces lipid peroxidation (MDA), and lowers systemic inflammatory mediators (CRP, TNF- $\alpha$ , IL-6) while increasing anti-inflammatory cytokines like IL-10. These findings provide biochemical validation of traditional claims regarding bitter gourd's therapeutic properties.

Although promising, evidence remains limited by small sample sizes, short intervention periods, and inconsistent extract standardization. To establish bitter gourd as a scientifically validated nutraceutical, future clinical trials must adopt rigorous methodologies, standardized formulations, and comprehensive biomarker assessments. By bridging phytochemistry with clinical biochemistry, bitter gourd has the potential to emerge as a valuable tool in

managing oxidative stress and inflammation-driven chronic diseases.

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