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"Active Charcoal+: Phytochemical Composition, Mechanistic Rationale, and Evidence Appraisal of a Five-Component Ayurvedic Formulation — A Critical Narrative Review"

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Abstract: Active Charcoal+ is formulated as a 500-mg Ayurvedic capsule containing *Andrographis paniculata* (130 mg), *Aloe barbadensis* (83.5 mg), *Allium sativum* (85.5 mg), menthol (75 mg), and Triphala Masi (75 mg), together with excipients. This critical narrative review examines how the published evidence for these individual components relates to digestive function and comfort, intestinal ecology, immune-inflammatory regulation, metabolic and hepatic physiology, antioxidant defence, and detoxification-associated processes. Triphala Masi is classified as a thermally carbonised Triphala preparation and is kept conceptually distinct from both uncarbonized Triphala and pharmaceutical activated charcoal. The human evidence varies markedly according to preparation, dose, and endpoint. Individual trials of standardised *Andrographis* products report gastrointestinal activity, but pooled findings are less consistent. Garlic has the broadest human evidence across metabolic, hepatic, and immune-inflammatory outcomes. Peppermint-oil trials offer indirect clinical context for menthol, whereas human-colon experiments support a direct spasmolytic action of menthol through reduced L-type Ca²⁺ influx and separate TRPM8-related effects on colonic motor function. Aloe findings are strongly dependent on the leaf fraction and processing method. Studies of ordinary Triphala show measurable but non-uniform changes in the microbiome and cannot be directly transferred to Triphala Masi. Evidence specific to Masi remains limited to pharmacognostic and in-vitro observations. Overall, the formulation has a biologically coherent ingredient-level rationale for gastrointestinal, inflammatory, and metabolic support, but current data do not establish finished-product efficacy or generalised systemic detoxification.

Keywords: Active Charcoal+; Triphala Masi; *Andrographis paniculata*; *Aloe barbadensis*; *Allium sativum*; menthol; digestive health; gut-liver axis

Abbreviations

ALT, alanine aminotransferase; AST, aspartate aminotransferase; BET, Brunauer-Emmett-Teller; CI, confidence interval; CYP, cytochrome P450; GI, gastrointestinal; HbA1c, glycated haemoglobin; HDL, high-density lipoprotein; HOMA-IR, homeostatic model assessment of insulin resistance; IBS, irritable bowel syndrome; IL-6, interleukin-6; LDL, low-density lipoprotein; LPS, lipopolysaccharide; MAPK, mitogen-activated protein kinase; NF-κB, nuclear factor kappa B; NK, natural killer; NNT, number needed

to treat; Nrf2, nuclear factor erythroid 2-related factor 2; RCT, randomized controlled trial; TNF- α , tumor necrosis factor-alpha; TRPM8, transient receptor potential melastatin 8.

1. Introduction

Gastrointestinal physiology extends well beyond digestion: epithelial integrity, microbial metabolism, mucosal immune activity, enteric sensory pathways, and portal delivery to the liver are closely interconnected. This integrated biology provides a more appropriate framework for evaluating digestive-wellness formulations than considering any one organ or pathway in isolation. In particular, the gut-liver axis links intestinal barrier function and microbial products with hepatic exposure through the portal circulation, while bile acids, cytokines, and microbial metabolites participate in bidirectional signalling between the two compartments^[1-5].

Active Charcoal+ brings together five pharmacologically distinct components: *A. paniculata* (Kalamegh), *A. barbadensis* (Kumari), *A. sativum* (Lasuna), menthol, and Triphala Masi. The product label specifies 75 mg of Triphala Masi (charcoal) in each capsule. That distinction matters because Masi is produced by heating botanical material until it becomes carbonised, so it cannot be treated as merely powdered Triphala in another physical form. Ayurvedic literature describes Mashi as a combustion-derived dosage form whose properties depend on the conditions of preparation^[6]. In Triphala Masi, microscopy across increasing preparation temperatures shows progressive loss of recognisable plant structure^[7]. Other work on experimentally prepared Triphala Masi/Mashi has identified residual extractable phenolics and tannins together with antimicrobial activity^[8].

The carbonised component must also be distinguished from pharmaceutical activated charcoal. Medical activated charcoal is deliberately processed to create defined adsorptive properties and has been studied clinically at gram-scale doses in toxicology^[9,10]. Simply carbonising Triphala does not establish comparable surface area, pore structure, or binding capacity. Activated-charcoal studies can therefore illustrate general adsorption principles, but they cannot be used as direct evidence that 75 mg of Triphala Masi behaves in the same way.

The remaining ingredients contribute different evidence streams. Standardised *Andrographis* preparations have been tested in randomised ulcerative colitis studies, although pooled analyses are less definitive than the individual positive trials^[11-13]. Peppermint oil has supportive evidence from major systematic and network meta-analyses, but a multi-component essential oil delivered in a specific formulation is not clinically interchangeable with isolated menthol^[14-16]. Garlic has randomised human data relevant to hepatic steatosis, insulin resistance, oxidative-stress markers, and related metabolic outcomes^[17-20]. Aloe has both gastrointestinal and metabolic human studies, but the results vary substantially with the material studied, including gel, leaf, and standardised preparations^[21-28].

Accordingly, this review appraises the biological rationale, human findings, safety, dose relevance, and formulation-level applicability of the principal Active Charcoal+ ingredients. The term detoxification is used only for defined processes such as sequestration within the gastrointestinal lumen, hepatic transformation of xenobiotics, cellular antioxidant/cytoprotective responses, and physiological elimination. It is not used as a substitute for generalised cleansing or unspecified toxin removal. Throughout the review, readers should distinguish evidence that a source ingredient is biologically active from evidence that the finished formulation itself produces the same effect.

2. Literature Search and Evidence Appraisal

This manuscript was developed as a critical narrative synthesis with emphasis on evidence quality and relevance to the labelled formulation. Priority was given to peer-reviewed primary studies, especially randomised trials, controlled human investigations, pharmacokinetic studies, and validated analytical work. PubMed/MEDLINE served as the main bibliographic verification source, and article details were checked against publisher or DOI records; Scopus and Web of Science indexing were considered when available. Systematic reviews and meta-analyses were used when they added necessary context or pooled interpretation, with preference for established clinical and pharmacology journals. Literature available through 2026 was considered. Because the evidence base for Triphala Masi itself is small, preparation-specific studies were retained even when published outside major clinical journals.

Methodological rigour and relevance to Active Charcoal+ were judged separately. For each clinically important study, the botanical source, plant part, processing or extraction method, degree of standardisation, administered dose, treatment duration, study population, and measured endpoint were compared with the labelled product. Studies of ordinary Triphala were treated only as evidence for the parent formulation, pharmaceutical activated-charcoal studies as comparator evidence, and peppermint-oil trials as indirect evidence for isolated menthol. This approach prevents strong data generated with a materially different intervention from being mistaken for finished-product efficacy. Terms such as moderate, limited, preliminary, and mechanistic only are used descriptively rather than as formal GRADE categories. Four cited sources were not indexed in PubMed/MEDLINE; one Triphala Masi paper and one older Triphala clinical report were retained because no equivalent indexed studies of the carbonised preparation were available, while two *Journal of Functional Foods* papers are indexed in Scopus and Web of Science but not MEDLINE. Their contribution is therefore treated as supporting rather than definitive evidence.

3. Active Charcoal+: Product Composition and Formulation Rationale

Table 1 presents the confirmed 500-mg capsule composition. The formulation spans several physiological levels: Triphala Masi is most relevant to luminal physicochemical hypotheses; menthol to gastrointestinal sensation and smooth-muscle behaviour; Andrographis and Aloe to mucosal or inflammatory biology; and garlic to the strongest human metabolic and hepatic evidence among the ingredients. This pattern makes mechanistic complementarity plausible, but no finished-formulation data demonstrate pharmacological synergy. Figure 1 identifies the marketed product and label, while Figure 2 summarises the ingredient-level domains considered in the review.

Table 1: Confirmed composition and principal evidence domains of Active Charcoal+.

Ingredient	Amount	Preparation	Principal evidence domain	Key limitation
<i>Andrographis paniculata</i> (Kalamegh)	130 mg	Plant material; diterpene-lactone source	GI inflammatory biology; immune modulation; hepatic/antioxidant mechanisms	Andrographolide content not specified on label
<i>Aloe barbadensis</i> (Kumari)	83.5 mg	Leaf	Bowel/mucosal physiology; metabolic evidence	Leaf fraction and anthraquinone/aloin specification not stated
<i>Allium sativum</i> (Lasuna)	85.5 mg	Bulb	Metabolic, inflammatory and hepatic evidence	Research preparations often use much higher doses or standardised products
Menthol/peppermint satva	75 mg	Monoterpene component	Sensory signalling; smooth-muscle physiology; digestive comfort	Peppermint-oil clinical evidence is indirect for isolated menthol
Triphala Masi (Triphala charcoal)	75 mg	Carbonised Triphala preparation	Luminal physicochemical rationale; limited Masi-specific evidence	Adsorption surface area and binding capacity not established for product



Fig 1: Active Charcoal+ product and manufacturer-supplied label. The label lists Kalamegh 130 mg, Kumari 83.5 mg, Lasuna 85.5 mg, menthol 75 mg, and Triphala Masi (Charcoal) 75 mg per 500-mg capsule. The image is included for formulation identification; scientific interpretation is derived from the cited academic literature.

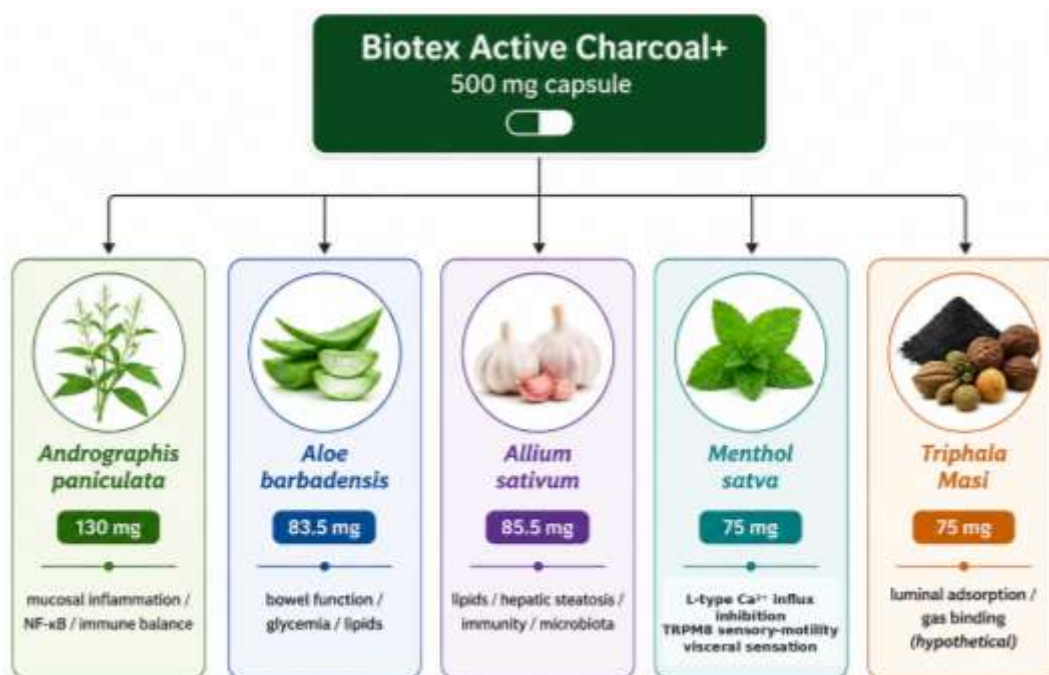


Fig 2: Ingredient-level formulation map for Active Charcoal+. The diagram displays the labelled quantities of the five principal ingredients and the scientific domains evaluated in this review. Functional phrases denote ingredient-level or mechanistic evidence and should not be interpreted as finished-product clinical efficacy. For menthol, direct human colon spasmolysis is linked to L-type Ca^{2+} influx inhibition, whereas TRPM8 is presented as a separate sensory/motility context.

4. Triphala Masi: Carbonised Botanical Component

4.1 Botanical precursor and carbonisation

Classical Triphala consists of the fruits of *Terminalia chebula*, *Terminalia bellirica*, and *Phyllanthus emblica*, the same three-fruit composition used in the human microbiome study^[29]. Triphala Masi

represents a different material because the parent mixture is exposed to heat until substantial carbonisation occurs. In a pharmacognostic comparison across preparation temperatures, recognisable plant architecture progressively diminished between 300 and 350 °C, and material heated to 400 °C was predominantly black and carbonised^[7]. Ayurvedic descriptions of Mashi similarly identify it as a heat-derived dosage form prepared under process-dependent open or closed conditions^[6]. These changes justify treating Mashi as a distinct preparation rather than as raw Triphala with a different appearance.

Thermal carbonisation may alter volatile compounds, organic phytochemicals, moisture, ash content, surface chemistry, and microstructure. The experimental Triphala Mashi examined by Biradar et al. still yielded phenolic- and tannin-containing aqueous and alcoholic extracts, and those extracts showed antimicrobial activity in vitro^[8]. Thus, carbonisation did not render that preparation chemically inert. However, these observations do not demonstrate clinical digestive benefit, microbiome improvement, toxin sequestration, or systemic detoxification. Figure 3 therefore separates what has been documented after carbonisation from the properties that remain unknown for the finished product.

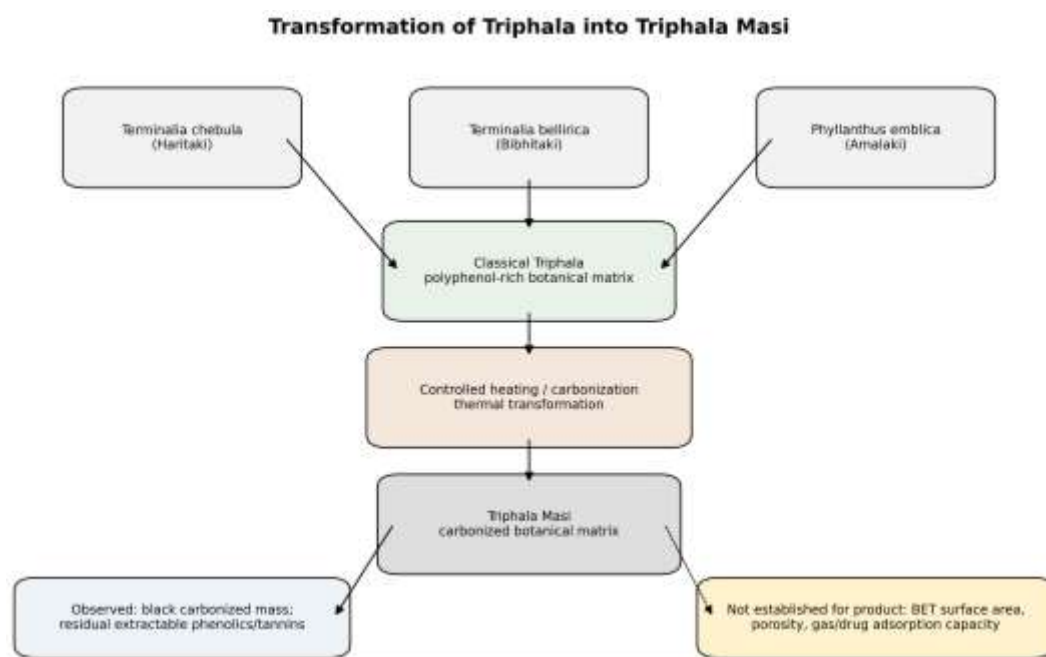


Fig 3: Transformation of ordinary Triphala into Triphala Masi and evidence boundaries. The figure separates documented carbonisation-related transformation and residual extractable constituents from properties not established for the finished product, including BET surface area, porosity, and gas- or drug-adsorption capacity.

4.2 Ordinary Triphala versus Triphala Masi

Findings obtained with ordinary Triphala should be interpreted only as evidence for the non-carbonised parent formulation. In a four-week double-blind pilot, participants receiving 2,000 mg/day of ordinary Triphala showed measurable changes in faecal microbial profiles, but the microbiome sample was small, and responses varied considerably within and between individuals^[29]. A later human-derived colon model also produced mixed taxonomic effects: *Akkermansia muciniphila* increased, whereas *Bifidobacterium* spp. decreased, alongside shifts in phenolic metabolites and fermentation products^[30]. Reviews describe ordinary Triphala as a polyphenol-rich gastrointestinal formulation^[31,32], and an older gram-dose study reported changes in bowel-related symptoms^[33]. None of these observations establishes that 75 mg of carbonised Triphala Masi produces the same exposure or microbiome response.

4.3 Triphala Masi versus activated charcoal

Pharmaceutical activated charcoal is manufactured as a high-surface-area adsorbent and is used in selected acute oral poisonings^[9,10]. Triphala Masi is prepared differently, and no product-specific measurements of BET surface area, pore volume, adsorption kinetics, or drug binding were identified for Active Charcoal+.

Human gas studies with conventional activated charcoal are also inconsistent. Some trials reported attenuation of post-meal flatus and breath hydrogen^[34], and a double-blind study conducted in United States and Indian cohorts found lower breath hydrogen together with less bloating and cramping^[35]. Two randomised primary-care trials of combination products containing activated charcoal reported improvement in dyspeptic symptoms^[36,37], but both formulations also contained simethicone, so the charcoal contribution cannot be isolated. Other trials were negative: 4 g of activated charcoal did not differ from placebo for breath hydrogen or flatus after a bean meal^[38]; repeated 0.52-g doses did not reduce sulfurous gases, total gas release, or abdominal symptoms^[39]; charcoal 400 mg twice daily did not improve breath hydrogen or symptoms in a double-dummy comparison in which rifaximin was effective^[40]; and a simethicone-charcoal product performed worse than metronidazole in a pilot study of flatus incontinence^[41]. EFSA consequently accepted a narrowly defined post-prandial flatulence claim for 1 g before and 1 g after a meal but did not substantiate a bloating claim^[42]. That reference dose is about twenty-seven times the Triphala Masi content of one Active Charcoal+ capsule. In addition, 12-25 g/day of activated charcoal did not produce a meaningful microbiome shift in a healthy-volunteer feasibility study^[43]. Meta-analytic and pharmacokinetic data nonetheless confirm that a sufficiently adsorptive charcoal preparation can modify drug exposure under defined conditions^[44,45]. These findings establish comparator principles only and do not demonstrate equivalent gas binding, adsorption, or medicine interaction for 75 mg Triphala Masi.

5. *Andrographis paniculata*

A. paniculata contains labdane diterpene lactones, with andrographolide among the best-characterised constituents^[46]. The clinically relevant gastrointestinal studies used standardised HMPL-004 rather than small amounts of unstandardised plant material. One placebo-controlled trial enrolled 224 adults with mild-to-moderately active ulcerative colitis and tested HMPL-004 at 1,200 or 1,800 mg/day for eight weeks; clinical response at week 8 was significantly greater than placebo at 1,800 mg/day (60% versus 40%; $P = 0.018$) but not at 1,200 mg/day (45% versus 40%; $P = 0.592$), and clinical remission did not differ significantly at either dose^[11]. A separate 120-patient double-blind comparison used HMPL-004 at 1,200 mg/day and sustained-release mesalazine at 4,500 mg/day; because there was no placebo arm and the trial was not designed as an equivalence study, similar group outcomes do not independently prove efficacy^[12]. More recently, a 2024 meta-analysis did not find a significant pooled advantage for *A. paniculata* in clinical response or remission, despite positive findings in some individual trials^[13]. The totality of evidence therefore supports biological activity while also emphasising the importance of dose and preparation.

Experimental studies provide a mechanistic basis for anti-inflammatory activity. Andrographolide can react chemically with the Cys62 residue in the p50 component of NF- κ B, an interaction that interferes with NF- κ B recognition of its DNA target sequence^[47]. In macrophage models, andrographolide also reduced LPS-triggered inflammatory cytokine output together with NF- κ B/MAPK pathway activation^[48]. Broader pharmacology reviews describe additional anti-inflammatory and metabolic targets for andrographolide and related diterpenes^[49-51]. These mechanisms are relevant to mucosal inflammatory biology, but they do not establish that the 130-mg plant quantity in Active Charcoal+ produces comparable systemic or mucosal exposure.

The safety literature likewise illustrates why formulation matters. A meta-analysis focused on oral *A. paniculata* identified gastrointestinal and cutaneous reactions among the more frequently reported adverse events^[52], whereas a broader review found that serious reactions were concentrated largely in injectable andrographolide-derivative products rather than conventional oral herbal preparations^[53]. Experimental data also indicate possible effects on hepatic CYP activity^[54]. Human liver-safety observations are mixed. In a cross-sectional cohort of 810 Thai gastroenterology outpatients, recent *A. paniculata* use was associated with a higher frequency of alanine-aminotransferase increase (44.5% vs 32.2%; $P = 0.018$), with the association remaining after multivariable adjustment (adjusted odds ratio 1.62; $P = 0.042$); fewer users had cirrhosis, which the investigators interpreted as likely reflecting clinical avoidance of herbal products in patients with established cirrhosis rather than a protective effect^[55]. In a separate pharmacokinetic study of 23 healthy volunteers, one week of aqueous extract delivering andrographolide-equivalent doses of 180 or 360 mg/day was followed by

ALT elevations in 30.4% and AST elevations in 26.1% of participants; these changes were judged probably treatment-related, were not clearly dose-dependent, and resolved after discontinuation^[56]. These observations reinforce the need to interpret Andrographis according to the actual preparation and exposure^[46].

6. *Aloe barbadensis*

Aloe does not represent a single standardised pharmacological entity. Inner-leaf gel contains abundant polysaccharides, including acemannan, whereas latex-containing tissues are richer in hydroxyanthracene compounds associated with stimulant-laxative activity. Whole-leaf preparations differ further according to processing and decolourisation^[28,57]. Because the Active Charcoal+ label identifies Aloe leaf without specifying the fraction, results from one Aloe material cannot be assumed to apply to another.

Gastrointestinal evidence includes a small double-blind placebo-controlled study of oral Aloe vera gel in 44 evaluable patients with mild-to-moderately active ulcerative colitis. Participants received 100 mL twice daily for four weeks. A secondary endpoint, clinical response, favoured Aloe (47% vs 14%; $P < 0.05$), whereas the prespecified remission comparison was not statistically significant (30% vs 7%; $P = 0.09$), and sigmoidoscopic scores were similar between groups^[21]. Evidence for bowel function is less straightforward. Positive constipation findings come mainly from combination products: a preparation containing celandin, Aloe, and psyllium improved stool frequency and consistency^[58], whereas a prebiotic blend composed chiefly of inulin and lactitol with only 3.9% Aloe gel did not significantly change gastrointestinal symptoms compared with placebo^[59]. When Aloe was given alone in 33 patients with refractory constipation-predominant IBS, pain and flatulence scores improved, but stool frequency, urgency, and consistency did not; the uncontrolled design also prevents separation of treatment effects from expectancy effects^[60]. Randomized IBS trials have generally failed on their primary endpoints, including studies of 58 patients^[61], 110 patients in a crossover design^[62], a 68-patient standardised-extract pilot^[63], and a larger trial in which symptom severity improved similarly with Aloe and control^[64]. A post hoc analysis suggested benefit mainly in diarrhoea-predominant IBS and for pain rather than stool outcomes^[65]. This helps explain the inconsistent pooled literature: a 2018 meta-analysis of three trials reported a modest benefit (SMD 0.41; 95% CI 0.07-0.75)^[66], whereas a 2026 synthesis of 60 studies involving 6,329 patients found no significant Aloe effect (RR 1.25; 95% CI 0.76-2.06), even though peppermint oil was effective in the same analysis^[67].

Controlled human studies have also evaluated defined Aloe preparations in prediabetes, metabolic-syndrome phenotypes, hyperlipidemic type 2 diabetes, and early untreated diabetes^[22-25]. Meta-analytic results suggest possible glycemic benefit but are heterogeneous. One analysis of five randomized trials involving 415 participants estimated reductions in fasting glucose of 30.05 mg/dL (95% CI -54.87 to -5.23; $P = 0.02$) and HbA1c of 0.41 percentage points (95% CI -0.55 to -0.27), while still characterizing the evidence as limited by the small number and quality of trials^[27]. Another meta-analysis of eight studies and 470 participants found improvement in fasting glucose mainly in prediabetes with no clear HbA1c effect there, while in type 2 diabetes the fasting-glucose effect was marginal and HbA1c improved significantly (-11 mmol/mol; 95% CI -19 to -2; $P = 0.01$)^[26]. That same type 2 diabetes estimate, equivalent to approximately -1.0%, is what places Aloe vera leaf gel among the better-performing plant interventions for HbA1c in an overview of 25 meta-analyses (pooled mean difference -0.99%; 95% CI -1.75 to -0.23), so the two figures are not independent findings^[68]. By contrast, a randomised trial of 1,000 mg/day Aloe powder for two months in 44 patients with type 2 diabetes reported no significant changes in fasting glucose, HbA1c, or lipid measures^[69]. The pattern therefore appears to depend more on preparation and exposure than on the label term Aloe alone. The older constipation study involving Aloe, celandin, and psyllium also cannot isolate an Aloe-specific contribution because the intervention was multi-ingredient^[58].

Aloe safety is equally preparation-dependent. Materials rich in anthraquinones, including latex-containing preparations, are associated with stimulant-laxative effects such as diarrhoea and abdominal cramping, and toxicology reviews repeatedly distinguish these products from inner-leaf gel and other processed forms^[28,57]. Oral Aloe has also been linked to rare cases of acute hepatitis, with improvement after withdrawal described in case reports and small series^[70-72]. These reports do not imply a uniform risk across all Aloe products; instead, they underscore the need to know the exact raw material used in a leaf-based formulation.

7. *Allium sativum*

Garlic chemistry changes substantially with processing. Tissue disruption permits alliin to be converted to allicin, while drying, ageing, heating, and extraction alter the relative abundance of allicin, ajoene, allyl sulfides, and more stable compounds such as S-allyl cysteine. Reviews of organosulfur chemistry therefore treat raw garlic, garlic powder, aged garlic extract, and standardised tablets as chemically distinct interventions rather than interchangeable forms^[73].

Randomized trials and pooled analyses provide the main human metabolic evidence for garlic. In non-alcoholic fatty liver disease, garlic powder has been associated with improvements in insulin resistance, fasting glucose, oxidative-stress measures, and aspects of body composition^[19]. In metabolic syndrome, one registered three-month trial randomised 90 participants to 1,600 mg/day garlic powder or placebo; the 2021 report from that trial described metabolic-syndrome components, insulin resistance, fatty-liver index, and appetite^[20], and a 2023 publication reported intestinal-transit and cardiometabolic outcomes from the same trial dataset^[80]. These are therefore two reports of one clinical trial rather than independent cohorts. Meta-analyses of garlic powder and other randomised garlic interventions generally show modest favourable effects on lipid and broader cardiometabolic markers^[74-76]. However, these studies used substantially larger or more standardised exposures than the 85.5 mg bulb quantity in Active Charcoal+.

Garlic also has direct human data in hepatic steatosis. One randomised trial used 800 mg/day of garlic powder for 15 weeks and found a higher proportion of steatosis improvement than with placebo, together with changes in associated metabolic measures^[17]. In another double-blind trial, 90 participants received 1,600 mg/day for 12 weeks; hepatic steatosis and several liver enzymes decreased, as did total cholesterol, triacylglycerol, and LDL cholesterol, while HDL cholesterol also declined, which is not a favourable lipid-direction signal^[18]. A second publication from the same registered cohort reported lower insulin resistance, fasting glucose, and malondialdehyde together with greater skeletal-muscle mass^[19], because both papers derive from one trial, they should not be treated as independent studies. A subsequent meta-analysis of randomised NAFLD trials supports an overall favourable signal while also reflecting the small size of the underlying evidence base^[75].

The human microbiome literature for garlic is much thinner than common prebiotic claims imply. In an exploratory component of a hypertension trial, 1.2 g/day of aged garlic extract for 12 weeks was associated with greater microbial richness and diversity^[77]. A double-blind study of 800 mg/day garlic powder for two months in women with obesity did not find significant overall microbiota changes, although trends were noted for *Akkermansia*, *Faecalibacterium*, and *Bifidobacterium*^[78]. An uncontrolled seven-person pilot of raw garlic juice likewise found little change in overall community structure despite an increase in Shannon diversity^[79]. More directly relevant to digestive function, the later report of the metabolic-syndrome trial cited above, drawn from the same 90-participant cohort and the same trial registration, evaluated intestinal transit in the 84 participants who completed the study: normal stool consistency was reported in 92.8% of the garlic group versus 64.2% of the placebo group at week 12, and in 90.4% versus 66.6% at week 6, with an overall improvement in intestinal transit time relative to control ($P = 0.001$) alongside favorable changes in lipid accumulation product and cardiometabolic indices^[80]. Transit was assessed with the Bristol Stool Form Scale, a self-reported stool-form proxy rather than an objective transit measurement, and the two reports should not be counted as independent trials. That exposure was roughly nineteen times the garlic content of a single Active Charcoal+ capsule.

Immune outcomes have also been tested in controlled human studies. A randomised placebo-controlled study in healthy adults used 2.56 g/day of aged garlic extract and detected changes in functional measures involving natural-killer and gamma-delta T cells^[81]. In 51 adults with obesity, six weeks of 3.6 g/day aged garlic extract blunted the placebo-group rise in circulating IL-6 and TNF-alpha and shifted gamma-delta T-cell and natural-killer-T-cell proportions without changing natural-killer-cell percentage^[82]. Beyond the hypertension trial already described^[77], an in vitro faecal-incubation model suggests that garlic can enhance *Bifidobacterium* in an enterotype-dependent manner without directly dosing human participants^[83]. For safety, perioperative literature raises concern about bleeding with

concentrated garlic products^[84], whereas a randomised crossover study found no impairment of platelet function after dietary-dose raw garlic in healthy volunteers^[85].

8. Menthol and Peppermint-Related Gastrointestinal Pharmacology

Menthol is a monoterpene that activates the cold-sensitive TRPM8 channel^[86], but gastrointestinal actions are not limited to TRPM8 signalling. In ex vivo circular muscle from the human distal colon, menthol suppressed both spontaneous and evoked contractions, with reduced L-type calcium entry contributing to the spasmolytic response^[87]. Separate experiments in human colonic tissue showed that activating TRPM8 itself can also decrease spontaneous contractile activity^[88].

Clinical symptom data are derived mainly from peppermint oil rather than isolated menthol. Peppermint oil contains several volatile constituents, and its effects depend partly on where and how the formulation releases them. In a randomised comparison of small-intestinal-release and ileocolonic-release peppermint oil, neither formulation achieved the prespecified primary abdominal-pain response endpoint, although some secondary measures favoured treatment^[89]. Systematic and network meta-analyses nonetheless indicate an overall short-term benefit for IBS symptoms, with heterogeneity in both efficacy and adverse events^[14-16]. Reviews of neurogastrointestinal TRP biology place TRPM8 among channels involved in gastrointestinal sensory and motor regulation^[86], and afferent experiments support a role for TRPM8 in visceral sensory processing^[90]. In healthy volunteers, peppermint oil has also been reported to prolong oro-caecal transit and reduce gallbladder emptying without materially delaying gastric emptying^[91], while another pharmacodynamic study found lower fasting intragastric pressure, reduced proximal phasic activity, and reduced appetite with little change in gastric sensitivity, tone, accommodation, or nutrient tolerance^[92]. These findings are useful mechanistic and clinical context for menthol but are not direct evidence for a 75-mg isolated-menthol dose in Active Charcoal+.

9. Integrated Effects on Digestion and Digestive Comfort

The digestive rationale is clearest when the ingredients are considered at different physiological levels. Triphala Masi is a carbonised luminal component with unverified adsorption characteristics; menthol has direct effects on gastrointestinal smooth muscle and sensory pathways; Andrographis and Aloe contribute evidence relevant to mucosal and inflammatory biology; and garlic adds metabolic and inflammatory actions together with the only directly measured digestive-function endpoint in the ingredient set. These mechanisms can be viewed as complementary, but the combination itself has not been shown to produce additive or synergistic effects.

Among the proposed domains, digestive comfort has one of the strongest mechanistic rationales, although the supporting evidence is uneven across ingredients. Menthol has direct human-colon mechanistic support^[87], whereas peppermint-oil trials provide indirect clinical symptom evidence^[14,15,89]. Andrographis and Aloe contribute human gastrointestinal findings from different disease-specific preparations^[11,12,21]. Triphala Masi, by contrast, has no validated human evidence demonstrating gas reduction, relief of bloating, or gastrointestinal absorption. The most defensible product-level interpretation is therefore biological plausibility based on constituent evidence rather than proven clinical efficacy. Conventional activated-charcoal studies also remain inconsistent for gas-related outcomes^[35,39].

10. Gut Microbiome and Intestinal Balance

Human microbiome evidence for ordinary Triphala is limited and cannot be transferred directly to the carbonised material. The randomised pilot used 2,000 mg/day of uncarbonised Triphala and included only a small microbiome sample^[29]. Because carbonisation changes the parent matrix, compounds available for microbial metabolism may be reduced or transformed. Residual phenolics in experimental Mashi^[8] do not establish prebiotic behaviour. Garlic and Aloe have additional experimental microbiome relevance, but their human data are less developed than their metabolic or gastrointestinal evidence. For Active Charcoal+, 'gut balance' is therefore best regarded as an indirect and preliminary domain. The newer colon-model study reinforces this caution because ordinary Triphala increased *Akkermansia* while decreasing *Bifidobacterium*, along with altering phenolic metabolites and fermentation products^[30]. Garlic findings are

also preparation-specific; exploratory changes seen with 1.2 g/day aged garlic extract cannot be assumed at the much smaller bulb-powder quantity in this product^[77].

11. Immune and Inflammatory Balance

The immune-inflammatory rationale rests mainly on *Andrographis* and garlic. *Andrographolide* has experimentally characterised NF- κ B/MAPK-related actions^[47-49], and standardised *Andrographis* preparations have shown activity in inflammatory gastrointestinal trials^[11,12]. Garlic contributes human immune-cell and cytokine findings from aged-extract studies^[81,82], while *Aloe* adds preparation-dependent mucosal and inflammatory context. Taken together, the evidence supports describing the formulation in terms of immune-inflammatory modulation or balance rather than claiming that it universally 'boosts immunity.'

12. Metabolic Function

Garlic carries the strongest human metabolic evidence among the ingredients. Randomized garlic-powder trials and meta-analyses report effects on insulin resistance, fasting glucose, oxidative-stress markers, lipid measures, blood pressure, and related cardiometabolic outcomes^[19,20,74-76]. *Aloe* contributes additional human data from defined gel or standardised preparations^[22-27], whereas *andrographolide* is supported mainly by preclinical metabolic research rather than directly applicable human trials^[49-51]. These findings justify an ingredient-level metabolic rationale but do not support presenting Active Charcoal+ as a treatment for diabetes, obesity, dyslipidemia, or metabolic syndrome.

13. Liver Function and the Gut-Liver Axis

The gut-liver axis provides a physiological link among epithelial integrity, microbial products, dietary metabolites, portal exposure, and hepatic metabolism^[1-5]. Within this formulation, garlic has the strongest direct human hepatic evidence, including randomised NAFLD trials and a meta-analysis^[17-19,75]. *Andrographis* contributes mainly mechanistic, pharmacokinetic, and safety information^[49-56]. *Aloe* requires a more cautious interpretation because metabolic findings coexist with rare reports of liver injury after some oral preparations^[28,70-72]. Evidence from ordinary *Triphala* cannot be assigned to *Triphala Masi* after carbonisation.

14. Detoxification: Scientific Interpretation

For scientific purposes, detoxification should be divided into discrete physiological processes rather than treated as a single undefined outcome. Binding or sequestration can occur within the gastrointestinal lumen; xenobiotics can undergo enzymatic biotransformation in the liver; antioxidant and cytoprotective systems can limit cellular injury; and excretion proceeds through gastrointestinal, biliary, renal, and other routes. These mechanisms are biologically distinct and do not justify a generalised 'cleansing' claim.

Activated charcoal provides a well-studied example of gastrointestinal sequestration. Under defined experimental conditions, randomised pharmacokinetic studies and meta-analyses show that an adequately adsorptive charcoal preparation can reduce or accelerate systemic drug exposure^[44,45]. This remains comparator evidence because the material and gram-level doses differ fundamentally from 75 mg *Triphala Masi*. A separate line of evidence concerns cellular antioxidant and cytoprotective responses, including Nrf2-regulated transcription induced by some dietary phytochemicals^[93]. Such redox effects should not be conflated with physical toxin removal. On current evidence, Active Charcoal+ can be discussed in relation to selected processes involved in gastrointestinal handling, redox defence, and metabolism, but not as a demonstrated systemic detoxification intervention.

15. Safety, Tolerability, and Herb-Drug Interaction Considerations

Safety must be interpreted ingredient by ingredient because there is no dedicated interaction study of the complete formulation. Oral *Andrographis* has been associated mainly with gastrointestinal, cutaneous, and hypersensitivity reactions^[52,53]. *Aloe* risk varies with the material used; anthraquinone-rich preparations are more strongly linked to laxative effects, and rare cases of acute hepatitis have been described after some oral *Aloe* products^[28,57,70-72]. Concentrated garlic preparations have raised perioperative bleeding concerns^[84], whereas a controlled crossover study found no platelet impairment from dietary-dose raw

garlic^[85], making relevance at the 85.5-mg bulb quantity dose- and preparation-dependent. Peppermint-oil trials report reflux, heartburn, and related gastrointestinal adverse events, which provide useful tolerability context for an oral menthol-containing formulation^[14,89].

Potential drug adsorption by Triphala Masi remains theoretical rather than demonstrated. Conventional activated-charcoal studies show that a sufficiently adsorptive carbon material can modify oral or systemic drug exposure^[9,10,44,45], but no comparable pharmacokinetic study has been performed with the 75-mg Masi component. Experimental studies of Andrographis extract and andrographolide also report CYP modulation^[54]; this supports a mechanistic interaction possibility without defining clinical magnitude for Active Charcoal+. Table 2 and Figure 4 summarise these distinctions.

Table 2: Safety and interaction considerations.

Ingredient	Main concern	Interaction context	Evidence status
Triphala Masi	Product-specific adsorption and drug-binding magnitude unknown	No direct finished-product pharmacokinetic evidence	Limitation / theoretical concern
Andrographis	GI events; rash/hypersensitivity; mild reversible transaminase elevation	Possible CYP effects vary by preparation	Systematic safety reviews and pharmacokinetic evidence ^[52-56]
Aloe	Diarrhea/cramping with anthraquinone-rich materials; rare liver injury reports	Preparation dependent	Toxicology reviews + clinical case evidence ^[28,57,70-72]
Garlic	GI intolerance; historical platelet/bleeding concern at concentrated supplemental exposures; controlled dietary-dose data are reassuring	Relevance increases with anticoagulant/antiplatelet therapy or surgery; dose and preparation matter.	Perioperative review + randomized platelet-function study ^[84,85]
Menthol	Reflux/heartburn context from peppermint preparations	Release site and formulation influence tolerability	Peppermint RCT/meta-analysis context ^[14-16,89]

Safety and Interaction Framework for Active Charcoal+

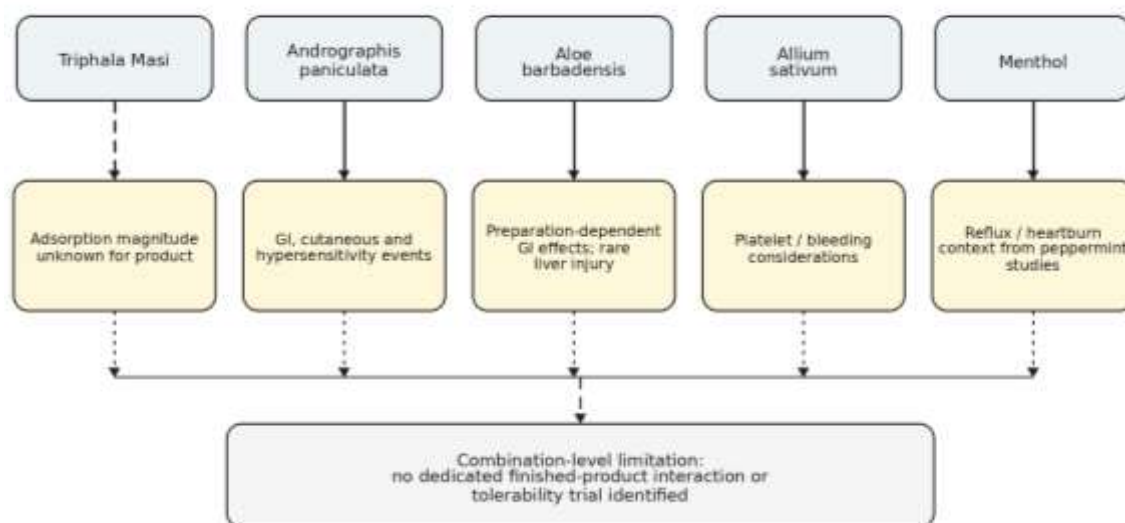


Fig 4: Safety and interaction framework for Active Charcoal+. The diagram summarises ingredient-level safety signals and distinguishes the unquantified adsorption potential of Triphala Masi from documented safety observations for the other ingredients. It does not estimate the frequency or magnitude of adverse effects for the finished formulation.

16. Human Evidence and Dose/Formulation Comparability

The main translational problem is the mismatch between the interventions studied clinically and the quantities present in Active Charcoal+. Standardised Andrographis HMPL-004 was tested at 1,200-1,800 mg/day^[11,12], compared with 130 mg of plant material in the product. Garlic liver trials generally used 800-1,600 mg/day of powder^[17-19], whereas the capsule contains 85.5 mg of bulb material. Aloe studies used defined gels or standardised preparations rather than an unspecified leaf fraction^[21-27]. Enteric peppermint-oil trials are not equivalent to isolated menthol^[14-16,89], and gram-dose ordinary Triphala studies are not equivalent to 75 mg of Triphala Masi^[29-33]. Thus, even when source studies are methodologically strong, direct applicability to the finished formulation remains limited by differences in preparation, dose, standardisation, release characteristics, and endpoints.

Table 3: Representative human evidence and applicability to the finished formulation.

Ingredient	Representative evidence	Research preparation/dose	Main observation	Product amount	Applicability
Andrographis	Sandborn et al. ^[11] ; Tang et al. ^[12] ; Iyengar et al. ^[13]	Standardized HMPL-004, 1,200-1,800 mg/day	Individual HMPL-004 trials showed clinical activity; pooled <i>A. paniculata</i> response/remission was not significant	130 mg plant	Low-direct; moderate botanical relevance
Aloe	Langmead et al. ^[21] ; Devaraj et al. ^[22] ; Huseini et al. ^[23] ; Choi et al. ^[24] ; Alinejad-Mofrad et al. ^[25]	Aloe gel / standardised Aloe preparations	GI or metabolic outcomes depended on preparation	83.5 mg leaf	Low-direct; preparation dependent
Garlic	Soleimani et al. ^[17] ; Sangouni et al. ^[18,19] (one trial, two reports) and ^[20] ; Xu et al. ^[82] ; Yu et al. ^[75]	Garlic powder 800-1,600 mg/day; aged garlic extract 3.6 g/day ^[82] ; mixed preparations in pooled analysis ^[75]	Steatosis, liver enzymes, metabolic outcomes, and immune-inflammatory biomarkers in preparation-specific trials	85.5 mg bulb	Low-direct; strong ingredient rationale
Menthol	Amato et al. ^[87,88] ; peppermint studies ^[14-16,86,89-92]	Human-colon menthol <i>ex vivo</i> ; enteric peppermint oil clinically	Human-colon spasmolytic/TRP M8 mechanisms; mixed but overall favourable peppermint-oil IBS evidence	75 mg menthol	Moderate mechanistic; indirect clinical
Triphala Masi	Peterson et al. ^[29] (human RCT); Goya-	2,000 mg/day ordinary Triphala (human); Triphala	Ordinary Triphala alters microbiota with	75 mg Masi	Indirect/preliminary

	Jorge et al. ^[30] (in vitro colon model, not a human study); Masi studies ^[7,8]	extract <i>in vitro</i> ; carbonised Masi analytical/ <i>in vitro</i>	heterogeneous taxonomic changes; no Masi human efficacy data		
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17. Evidence-Weighted Appraisal of Proposed Physiological Domains

Support is uneven across the proposed physiological domains. Digestive comfort has the clearest multi-ingredient rationale because menthol contributes direct smooth-muscle evidence, peppermint oil offers indirect clinical symptom data, and Andrographis and Aloe add gastrointestinal findings from other preparations. Metabolic and hepatic support is driven mainly by garlic. Microbiome effects remain preliminary, while detoxification is largely a mechanistic concept because adsorption by Triphala Masi has not been demonstrated.

Table 4: Qualitative evidence-weighted appraisal of proposed physiological domains.

Domain	Principal ingredients	Narrative evidence level	Product limitation	Scientific interpretation
Digestion / digestive comfort	Menthol, Andrographis, Aloe, garlic (transit); Triphala Masi rationale	Moderate ingredient-level	Direct finished-product evidence not available	Supportive/plausible
Gas/bloating	Menthol; Triphala Masi hypothesis	Limited direct; activated-charcoal comparator evidence is inconsistent	Masi adsorption not characterised; comparator gas findings include positive and negative human data ^[34,35,39]	Preliminary
Gut balance/microbiome	Ordinary Triphala context; garlic/Aloe mechanistic literature	Limited; ordinary Triphala data show measurable but non-uniform microbiome shifts	Carbonised Masi differs from parent Triphala; parent microbiome findings are heterogeneous ^[29,30]	Preliminary/indirect
Immune-inflammatory balance	Andrographis, garlic, Aloe	Moderate ingredient-level	Dose/preparation mismatch	Supportive at ingredient level
Metabolic function	Garlic, Aloe	Moderate human evidence	Research doses/preparations differ	Supportive at ingredient level
Liver / gut-liver relevance	Garlic; Andrographis context	Limited-moderate human evidence, strongest for garlic	Finished-product effect untested	Supportive rationale

Detoxification-associated physiology	Triphala Masi carbonised matrix; botanical redox mechanisms	Very limited direct	No Masi adsorption/clearance data	Mechanistic only
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18. Integrated Mechanistic Framework

The integrated framework places each ingredient at a different physiological level. Triphala Masi belongs primarily to the luminal compartment, although its adsorptive behaviour has not been quantified. Menthol has direct human colon effects on smooth muscle and separate sensory-pathway relevance. Andrographis and Aloe contribute mucosal and inflammatory evidence, while garlic provides the strongest human metabolic and hepatic signal. These pathways can converge conceptually on digestive comfort, inflammatory balance, metabolism, and gut-liver physiology, but there is no evidence that the complete combination produces additive or synergistic clinical effects. Figure 5 therefore keeps direct menthol spasmolysis separate from TRPM8-dependent sensory and motility mechanisms.

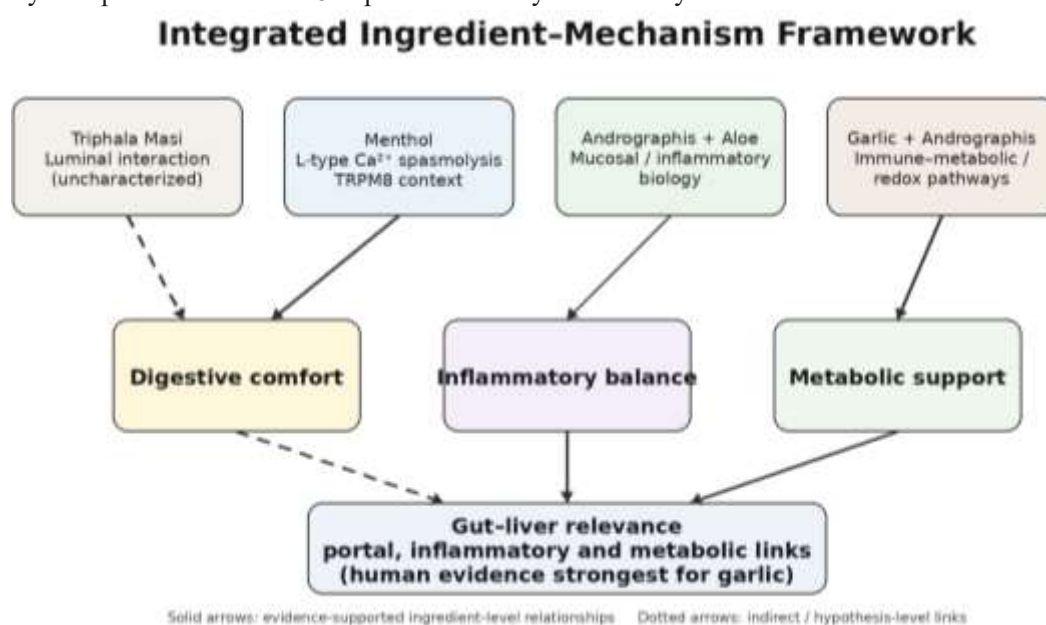


Fig 5: Integrated ingredient-mechanism framework for Active Charcoal+. Menthol is represented by two distinct evidence strands: direct human-colon spasmolysis associated with inhibition of L-type Ca^{2+} influx, and separate TRPM8-dependent sensory/motility context. Solid connections represent evidence-supported ingredient-level relationships; dotted connections represent indirect or hypothesis-level links.

19. Limitations of the Current Evidence Base

A principal consideration in interpreting this literature is that the available evidence relates to the individual ingredients, or to comparator preparations, rather than to Active Charcoal+ as a finished formulation. The label specifies the quantity of each ingredient; marker-level standardisation of andrographolide content, the Aloe leaf fraction and aloin level, the garlic sulfur-compound profile, and physicochemical characterisation of the Triphala Masi is not reported, as is common for multi-herb Ayurvedic preparations. Exposures used in the source studies therefore cannot be equated directly with intake from a single capsule.

Clinical evidence is especially sparse for Triphala Masi. Existing publications describe the effects of carbonisation on morphology, residual extractable phytochemicals, and antimicrobial activity^[6-8], but they do not establish human gastrointestinal adsorption, gas reduction, microbiome modulation, or systemic detoxification. Ordinary Triphala is chemically different after carbonisation and therefore remains only parent-formulation evidence^[29-33]. Activated-charcoal studies likewise cannot define Masi performance because they involve purpose-activated materials and much larger doses^[9,10,34,35,39,43-45]. Even

the comparator literature is internally heterogeneous, with variable microbiome responses to ordinary Triphala^[29,30] and conflicting gas findings for conventional activated charcoal^[34,35,39].

Additional limitations arise from heterogeneity of the non-carbonised ingredients. Aloe studies use materially different preparations; Andrographis, garlic, and peppermint/menthol studies often employ standardised extracts or doses substantially above those in the product. Outcomes also differ widely, ranging from ulcerative colitis activity and IBS symptoms to metabolic biomarkers, hepatic steatosis, immune-cell measurements, and pharmacokinetic endpoints. These differences restrict formulation-level inference even when the source ingredient has credible human evidence. Andrographis is a good example: individual HMPL-004 trials are favourable, whereas a recent pooled analysis did not demonstrate a significant benefit for clinical response or remission^[11-13].

20. Conclusion

Active Charcoal+ combines Triphala Masi with botanicals that have different but potentially complementary pharmacological profiles. Triphala Masi is an authentic carbonised Triphala preparation for which Mashi-specific pharmacognostic and in-vitro evidence is available^[6-8], but it should remain distinct from both pharmaceutical activated charcoal and ordinary Triphala. Menthol has direct human-tissue mechanistic support, while peppermint-oil studies provide only indirect clinical context^[14-16,86-92]. Andrographis contributes human gastrointestinal and mechanistic anti-inflammatory evidence^[11-13,46-56]; Aloe contributes preparation-dependent gastrointestinal, metabolic, and safety findings^[21-28,57,58,70-72]; and garlic has the strongest human evidence across metabolic, hepatic, and immune-inflammatory domains^[17-20,73-77,81-85].

Taken together, the most coherent ingredient-level domains are digestive comfort, inflammatory balance, and metabolic support. Hepatic relevance is supported principally by garlic, with additional mechanistic context from other ingredients. Microbiome and gas-related interpretations remain preliminary for Triphala Masi, and generalised systemic detoxification has not been demonstrated. The current literature therefore supports describing Active Charcoal+ as an evidence-informed Ayurvedic formulation with constituent-level relevance to digestive, inflammatory, metabolic, and gut-liver physiology, while clearly separating this rationale from direct clinical proof of the complete product.

Declarations

Competing interests. All three authors are employees of Biotex Life Solutions Pvt. Ltd., the developer of Active Charcoal+. This employment relationship constitutes a potential competing interest and is disclosed here in full. To mitigate it, the review appraises ingredient-level evidence separately from finished-product claims, reports null and contradictory findings alongside supportive ones, states explicitly where evidence is absent, and makes no claim of demonstrated efficacy for the finished product. The authors declare no other financial or non-financial competing interests.

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Consent for publication. Not applicable. The manuscript contains no person's data, images or case material.

Data availability. No new datasets were generated or analysed; the review is based on published literature, and all sources are listed in the References with DOI or PMID identifiers where available.

Use of artificial intelligence. Artificial-intelligence-based tools were used to assist with literature organisation, reference verification and language editing. All cited references were checked against PubMed, Crossref or publisher records, and all quantitative statements were verified against the primary sources. The authors take full responsibility for the accuracy, interpretation and integrity of the content.

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