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Qualitative Content Analysis of Antioxidant-Centred Mechanisms Reported for Trigonella foenum-graecum: Linking Phytochemical Classes to Disease-Relevant Pathways

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ABSTRACT

Background. Fenugreek (*Trigonella foenum-graecum* L.) seed extracts and their isolated constituents are credited with a very wide range of health-related effects, but the supporting studies are scattered across models, extract types and endpoints, and it is unclear how antioxidant activity relates to the other effects attributed to the plant.Objective. To examine, by qualitative content analysis, how experimental and clinical studies explain the pharmacological behaviour of fenugreek and its main constituents, with particular attention to antioxidant mechanisms. Methods. A codebook of phytochemical classes and outcome domains was built deductively and extended inductively while reading a corpus of in vitro, animal and human studies. Coded segments were condensed into categories and then themes. Trustworthiness was supported by an audit trail, repeated re-reading of the source texts and a negative-case check.Results. Six themes emerged: (1) redox buffering as the most common upstream explanation; (2) compound-specific signalling; (3) convergence of oxidative, inflammatory and metabolic pathways; (4) dependence of effect on extract type and plant part; (5) a favourable but incompletely characterised safety profile; and (6) translational gaps, above all the scarcity of pharmacokinetic data. Mechanistic explanations rested mainly on two compounds, 4-hydroxyisoleucine and diosgenin, whereas trigonelline, flavonoid C-glycosides, sotolone and galactomannan were comparatively under-examined. Antioxidant claims were usually inferred from enzyme and lipid-peroxidation markers rather than shown at compound level.Conclusions. Antioxidant activity functions in the literature as a connecting explanation rather than a demonstrated cause. Future work should characterise extracts chemically, test single compounds and combinations against defined redox and signalling endpoints, and add pharmacokinetic measurements before clinical claims are extended.

Introduction

Plants protect themselves against pests and climatic stress by synthesising secondary metabolites, and many of these compounds have been adopted as medicines. Traditional plant-derived medicines remain the primary source of health care for a large share of the world's population. Fenugreek (*Trigonella foenum-graecum* L., family Fabaceae) is a short-lived annual that is grown in Asia, Africa and Europe and used as a food, spice and herbal remedy, including in Ayurvedic and traditional Chinese medicine (Nagulapalli Venkata et al., 2017). Its seeds are the best-studied part: they are ground into a condiment, and the endosperm yields a galactomannan gum that is used as a soluble fibre and as a stabiliser in baked products.

The pharmacological literature on fenugreek is large. Reported activities include antioxidant, anti-inflammatory, glucose-lowering, lipid-lowering, anti-obesity, hepatoprotective, hormonal and anti-proliferative effects (Khorshidian et al., 2016; Nagulapalli Venkata et al., 2017). These effects are attributed to a chemically diverse set of constituents: steroidal saponins and their sapogenins, the non-proteinogenic amino acid 4-hydroxyisoleucine (HIL), the pyridine alkaloid trigonelline, flavonoid and phenolic glycosides, and the galactomannan fibre of the endosperm (Fuller & Stephens, 2015).

What is less clear is how these findings fit together. Oxidative stress is widely invoked as a shared mechanism, because reactive oxygen species damage lipids, proteins and DNA and are implicated in inflammation, diabetes and cancer. Yet authors working with different models and different extracts rarely state whether antioxidant behaviour is the cause of the observed benefit, a parallel property, or merely a convenient explanation. A close reading of how the studies themselves reason about mechanism is therefore needed, in addition to a count of what they found.

This study takes a qualitative approach to that problem. Rather than pooling effect sizes, it treats published experimental and clinical reports as texts and asks how they construct the link between a phytochemical, a cellular mechanism and a health outcome. Three research questions guided the analysis:

- **RQ1.** Which phytochemical classes are most consistently connected to antioxidant outcomes, and on what kind of evidence?
- **RQ2.** How do the studies explain the connection between redox balance and metabolic, inflammatory, hormonal and anti-proliferative outcomes?
- **RQ3.** What features of design, reporting and safety data limit the translation of these findings?

Materials and Methods

Study design and reporting

A qualitative content analysis was carried out following the conventional and directed approaches described by Hsieh and Shannon (2005) and the stepwise process of Elo and Kyngäs (2008). Categories were first derived from existing knowledge of fenugreek phytochemistry (directed, deductive coding) and then extended whenever segments did not fit (conventional, inductive coding). Higher-order themes were developed in the manner of thematic analysis (Braun & Clarke, 2006). The report follows the Standards for Reporting Qualitative Research (O'Brien et al., 2014) as far as they apply to a document-based design. No human participants were recruited and no animals were used; all material is secondary and published.

Corpus

The analytic corpus consisted of primary studies and meta-analyses on fenugreek and its constituents that had been located through Google Scholar and PubMed and compiled in the authors' working literature database. A report was retained when it described an antioxidant, anti-inflammatory, metabolic, hormonal, antimicrobial, anti-proliferative or safety outcome for a fenugreek preparation or a named constituent. Studies on other species, on livestock nutrition without a fenugreek intervention, and general methodological papers were excluded from coding. Reviews were read for context but not coded as evidence. The unit of analysis was a meaning unit: a sentence or short passage in which the authors of a study state a finding, a proposed mechanism or a limitation.

Coding procedure

Coding proceeded in five steps. (i) All texts were read through for familiarisation. (ii) A starting codebook was drawn up with two axes: phytochemical class (Table 1) and outcome domain (glycaemic, lipid and weight, tissue protection, hormonal, anti-proliferative, antimicrobial, safety). (iii) Meaning units were coded to one or more codes, and a new code was created whenever a unit expressed a mechanism or limitation that the codebook could not hold, for example "combination with a second agent strengthens the effect". (iv) Codes were condensed into categories, and categories were compared across study types (in vitro, animal, human) to build themes. (v) Each draft theme was tested against the source texts by searching for negative cases, meaning studies that contradicted it or reported a null result.

Credibility, dependability, confirmability and transferability were addressed following Lincoln and Guba (1985) and Graneheim and Lundman (2004). Credibility was supported by returning to the original passages whenever a category was revised. Dependability was supported by an audit trail that recorded every merge, split and rename of a code. Confirmability was supported by keeping the wording of each coded passage next to the interpretive label. Transferability is discussed in Section 5. Because a single analytic

team handled the corpus, interpretive bias cannot be excluded, and the themes should be read as a structured reading of the literature rather than as a measurement.

Results

The phytochemical framework

Six phytochemical classes organised the deductive codebook (Table 1). Steroidal saponins are the leading class of seed constituents; most of the saponins isolated from fenugreek are of the furostanol type, and the sapogenins diosgenin and yamogenin arise from them by enzymatic conversion and acid hydrolysis (Pang et al., 2015; Challinor & De Voss, 2013). HIL is the dominant free amino acid in dry seed (Aljuhaimi et al., 2018), and the galactomannan of the endosperm has a mannose-to-galactose ratio close to 1:1, which makes it more soluble than the galactomannans of guar or locust bean (Nagulapalli Venkata et al., 2017). Flavonoids and phenolic acids occur largely as glycosides, with apigenin C-glycosides such as vitexin and isovitexin repeatedly reported (Benayad et al., 2014; Baba et al., 2018).

Table 1. Deductive codebook: phytochemical classes and the bioactivity codes attached to them in the corpus

Phytochemical class	Representative compounds	Bioactivity codes assigned
Steroidal saponins and sapogenins	Protodioscin, diosgenin, yamogenin	Anti-proliferative; anti-inflammatory; lipid-lowering; androgenic and anabolic; adipogenesis
Free amino acids	4-Hydroxyisoleucine (HIL), canavanine	Insulinotropic; insulin-sensitising; anti-inflammatory
Alkaloids	Trigonelline	Glycaemic; neuroprotective; adipocyte differentiation
Flavonoids and phenolics	Vitexin, isovitexin, apigenin C-glycosides, quercetin, kaempferol, coumarins	Radical scavenging; COX inhibition; antimicrobial
Dietary fibre	Galactomannan (soluble), insoluble fraction	Delayed carbohydrate absorption; bile-acid excretion; satiety
Volatile lactones and oils	Sotolone, N-linoleoyl-2-amino-butyro lactone	β-cell protection; GLP-1 signalling

Source: coded from the corpus; class membership follows Fuller and Stephens (2015) and Nagulapalli Venkata et al. (2017).

Overview of the coded evidence

Table 2 lists the six themes with their constituent categories and a qualitative judgement of the evidence behind each. Table 3 and Table 4 show representative experimental and human records that were coded under them. The tabulated records are illustrative rather than exhaustive; the themes were built from the full corpus. Figure 1 summarises the structure of the explanations that the studies offer.

Table 2. Themes, categories and analytic judgement of evidence

Theme	Categories	Evidence base	Analytic judgement
T1. Redox buffering as upstream explanation	Lower lipid peroxidation; restored SOD, CAT, GPx; protection against toxicants; polyphenol–activity correlation	Rodent and in vitro; no direct radical measurement	Consistent in direction, weak on causal attribution
T2. Compound-specific signalling	HIL and insulin signalling; diosgenin and cell-cycle control; trigonelline	In vitro and rodent; some human	Strongest for HIL and diosgenin; thin for the rest

	and lactones with GLP-1; fibre and absorption		
T3. Convergence of redox, inflammation and metabolism	ROS–NF-κB/MAPK–IRS-1 axis; COX inhibition by apigenin C-glycosides; TNF-mediated insulin resistance	In vitro and rodent	Coherent narrative; largely inferred
T4. Extract and matrix dependence	Solvent effects; plant part; germination; standardised proprietary extracts	Cross-study comparison	Limits comparability between studies
T5. Safety signals	Negative genotoxicity assays; no overt organ toxicity; minor gastrointestinal events	Rodent toxicology; short human studies	Reassuring but short-term
T6. Translational gaps	Little pharmacokinetic data; few compounds studied; synergy untested; quality control	Authors' own stated limitations	Main barrier to clinical claims

Evidence base and analytic judgement are the authors' interpretive ratings, not statistical measures.

Theme 1: redox buffering as the upstream explanation

The most frequent explanatory move in the corpus was to attribute benefit to antioxidant action. Typical evidence was a fall in lipid-peroxidation markers (thiobarbituric acid reactive substances or malondialdehyde) together with recovery of superoxide dismutase (SOD), catalase (CAT) and glutathione peroxidase (GPx) activity. In diabetic rats, fenugreek restored these enzymes, alone or combined with vanadate (Kumar et al., 2015), reduced peroxidation in a way that resembled glibenclamide (Middha et al., 2011), and raised cardiac SOD, CAT and GPx when paired with swimming training (Arshadi et al., 2015). Protection was also reported against toxicant injury, for example deltamethrin in rats, where haematological and biochemical indices were preserved and peroxidation fell dose-dependently (Abdel-Daim et al., 2014), and carbendazim-induced testicular damage, where SOD and CAT improved and malondialdehyde fell (Lamfon, 2012).

Two features of this theme deserve emphasis. First, extract type mattered: aqueous extracts showed stronger antioxidant activity than ethanolic ones in the study of Chaturvedi et al. (2013), which the authors linked to water-soluble polyphenols and flavonoids, and polyphenol content followed the order husk, whole seed and endosperm in line with radical-scavenging strength (Naidu et al., 2011). Second, the claims were almost always indirect. Studies inferred antioxidant mechanisms from downstream enzyme and lipid markers and seldom measured radical species or tested a single compound against the same endpoint. Chemical and cell-based antioxidant assays measure different things (López-Alarcón & Denicola, 2013), so a reduction in a single marker is a limited basis for naming a mechanism.

Theme 2: compound-specific signalling

Mechanistic detail was concentrated on two molecules. For HIL, studies reported activation of AMP-activated protein kinase, PI3K/Akt-dependent glucose uptake, suppression of inflammation-driven serine phosphorylation of IRS-1 and reversal of TNF-induced insulin resistance in muscle and liver cells (Maurya et al., 2014; Gautam et al., 2016; Gao et al., 2015). Diosgenin was linked to G1 cell-cycle arrest, inhibition of Akt and its downstream targets, suppression of STAT3 signalling and apoptosis in breast, colon and hepatocellular carcinoma cells, as well as attenuation of Wnt signalling in breast cancer stem-like cells (Bhuvanalakshmi et al., 2017; Fuller & Stephens, 2015). Crude seed extracts also reduced cancer cell viability, in HepG2 cells through p53, Bax, PCNA and caspase-3 (Khalil et al., 2015) and in other lines without a defined mechanism (Alsemari et al., 2014; Al-Oqail et al., 2014).

Other constituents were comparatively under-examined. Trigonelline was shown to restrain adipocyte differentiation and lipid accumulation in 3T3-L1 cells (Ilavenil et al., 2014). A lactone isolated from seed acted as a positive modulator of GLP-1 signalling (King et al., 2015). The soluble fibre fraction improved glucose handling in diabetic rats by delaying carbohydrate digestion and absorption (Hannan et al., 2007). Flavonoid C-glycosides appeared mainly as COX inhibitors and radical scavengers (Liu et al., 2012). In human studies, furostanol-enriched extracts were associated with higher testosterone and better sperm profile (Bagchi et al., 2017; Maheshwari et al., 2017), and fenugreek powder with higher haemoglobin in women of reproductive age (Doshi et al., 2012). Antimicrobial results were reported at concentrations far above those used in metabolic studies, for example with ethanolic seed extract against Gram-positive and Gram-negative bacteria (Fezea et al., 2015).

Table 3. Representative in vitro and animal records coded in the analysis

Reference	Model	Preparation, dose, duration	Coded outcome	Theme
Hamza et al. (2012)	High-fat-fed C57BL/6J mice	Aqueous seed extract, 2 g/kg/day, 20 weeks	Lower plasma glucose and insulin; reduced insulin resistance	T2, T4
Hannan et al. (2007)	STZ-diabetic rats	Soluble fibre, 0.5 g/kg in diet, 28 days	Lower fructosamine; reduced platelet aggregation	T2
Kumar et al. (2015)	Alloxan-diabetic rats	Seed powder, 5% of diet, 21 days, ± vanadate	Lower glucose, higher insulin; stronger in combination	T1, T4
Jin et al. (2014)	STZ-diabetic rats	Seed powder, 9 g/kg in diet, 12 weeks	Lower glucose, albuminuria, HbA1c, BUN and creatinine	T1
Chaturvedi et al. (2013)	Hyperlipidaemic rats	Alcoholic seed extract, 200 mg/kg	Cholesterol down 26.2%; triglycerides down 36.6%	T2
Ikeuchi et al. (2006)	Mice	Seed extract, 300 mg/kg, 4 weeks	Longer swimming time to exhaustion	T2
Arshadi et al. (2015)	STZ-diabetic rats	Seed extract + swimming training	Higher cardiac SOD, CAT, GPx	T1
Abdel-Daim et al. (2014)	Deltamethrin-treated rats	Seed extract	Preserved haematology and biochemistry; less peroxidation	T1
Lamfon (2012)	Carbendazim-treated rats	Seed extract	Higher SOD and CAT; lower malondialdehyde	T1
Naicker et al. (2016)	HepG2 cells	Seed extract and HIL	Proximal insulin signalling, glycogenic enzymes and GLUT2 increased	T2
Gao et al. (2015)	HepG2 cells	HIL	Lower TNF-α; altered insulin signalling proteins	T3
King et al. (2015)	Cell-based assays	Seed-derived lactone	Positive modulation of GLP-1 signalling	T2

STZ, streptozotocin; HbA1c, glycated haemoglobin; BUN, blood urea nitrogen; SOD, superoxide dismutase; CAT, catalase; GPx, glutathione peroxidase.

Theme 3: convergence of oxidative, inflammatory and metabolic pathways

Many studies placed redox balance, inflammation and insulin action on one chain of reasoning. In the account given by Maurya et al. (2014), HIL lowered ROS generation and the activation of NF-κB, JNK, ERK and p38 MAPK in muscle cells exposed to palmitate, which in turn reduced inhibitory serine phosphorylation of IRS-1 and restored insulin sensitivity. Gautam et al. (2016) described a related route through AMPK activation and suppression of SOCS3 in TNF-treated cells. Liu et al. (2012) separated seed fractions by solvent and showed that hexane and aqueous fractions inhibited COX-2 whereas the ethyl acetate fraction inhibited COX-1; apigenin 8-C-glucopyranoside was the strongest COX-2 inhibitor among three C-glycosides tested.

This chain is persuasive, but most of its links come from different studies using different cell types and different compounds. No study in the corpus followed a single preparation through all the steps from oxidative marker to inflammatory signal to insulin response. The convergence is therefore best described as a hypothesis assembled across the literature.

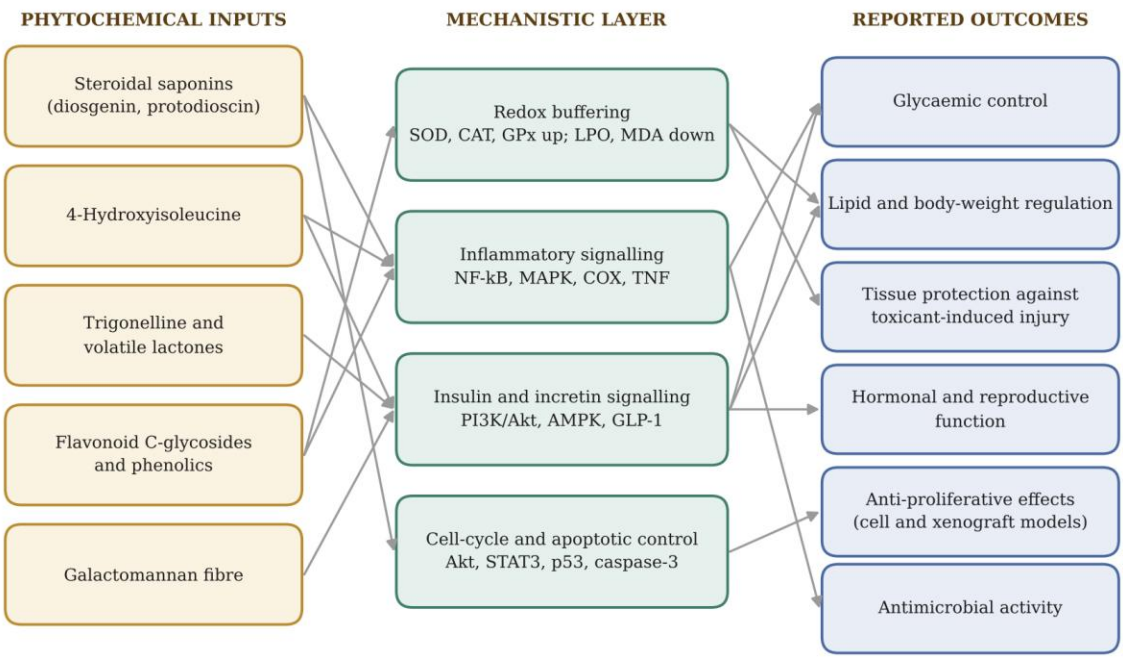


Figure 1. Thematic map of how the coded studies connect phytochemical inputs, mechanisms and outcomes for fenugreek. Links reflect explanations proposed in the source studies.

Theme 4: dependence on extract type and plant part

Effects depended heavily on how the material was prepared. Aqueous and ethanolic extracts differed in antioxidant and antimicrobial activity (Fezea et al., 2015; Chaturvedi et al., 2013), hexane, aqueous and ethyl acetate fractions differed in COX selectivity (Liu et al., 2012), and seed composition varied with growing location (Aljuhaimi et al., 2018; Baba et al., 2018). Studies used seed, leaf, sprout, germinated seed, crude powder, soluble fibre and branded standardised extracts such as a furostanolic saponin preparation (Hua et al., 2015). Doses were reported as grams per kilogram of diet in some studies and milligrams per kilogram by gavage in others. Together these differences make direct comparison of potency across studies unreliable.

Theme 5: safety signals

Safety evidence was generally reassuring. Essential oils, oleoresins and natural extracts of fenugreek are listed as generally recognised as safe under United States federal regulations, and mutagenicity and micronucleus assays of extracts were reported as negative. A furostanolic saponin extract caused no toxicity at 5 g/kg in female rats (Hua et al., 2015), and a 28-day oral study of a standardised glycoside extract in mice found no adverse effects (Kandhare et al., 2015). A human-equivalent oral dose range of 21.16 to 48.64 g for a 60 kg adult has been calculated (Ouzir et al., 2016). In people with diabetes, seed powder at 25 g/day for 24 weeks was reported without hepatic, renal or haematological abnormalities, and mild events such as migraine and indigestion occurred in four of 80 women in one set of trials. The qualification is that nearly all of this evidence comes from rodents or from trials lasting weeks to months.

Table 4. Representative human studies and syntheses coded in the analysis

Reference	Population and design	Intervention	Coded outcome	Theme
Gaddam et al. (2015)	People with prediabetes; 3-year randomised trial	Fenugreek	Lower cumulative diabetes incidence; lower fasting and postprandial glucose	T2

Kaur et al. (2016)	Type 2 diabetes	Seed powder as add-on to metformin	Efficacy and tolerability evaluated	T2, T5
Neelakantan et al. (2014)	Meta-analysis of clinical trials	Fenugreek intake	Glycaemic improvement; effect noted at higher seed-powder doses	T2, T4
Gong et al. (2016)	Meta-analysis, diabetes and prediabetes	Fenugreek	Improved fasting blood glucose	T2
Bagchi et al. (2017)	Healthy volunteers	Protodioscin-enriched extract	Higher free testosterone; improved sperm profile	T2, T4
Maheshwari et al. (2017)	50 male volunteers; 12 weeks	Furosap™ extract	Higher testosterone; improved sperm profile	T2, T4
Park et al. (2018)	Testosterone deficiency syndrome; randomised, double-blind	Fenugreek and *Lespedeza cuneata* mixed extract	Efficacy and safety assessed	T2, T5
Doshi et al. (2012)	Women of child-bearing age	Fenugreek powder	Higher haemoglobin	T2
Bae et al. (2015)	Overweight women	Fennel and fenugreek tea	Reduced short-term subjective appetite	T2
Pant et al. (2018)	Human volunteers	Germinated seeds	Lower total and LDL cholesterol; HDL and triglycerides unchanged	T2
Kiss et al. (2018)	Healthy volunteers	Fenugreek seeds	Insulin-sensitising effect with changes in plasma MCH	T2

LDL, low-density lipoprotein; HDL, high-density lipoprotein; MCH, melanin-concentrating hormone.

Theme 6: translational gaps

The corpus itself pointed to the limits of the evidence. Pharmacokinetic information on fenugreek products was described as scarce, so absorption, distribution and the active forms reaching tissue remain largely unknown. Only a few compounds, chiefly HIL and diosgenin, had been studied in depth, while other constituents await investigation. Possible synergy between constituents was proposed but not tested, and the supply of purified compounds for biological work is constrained because some can be obtained only in small amounts. Raw-material variation makes quality control and standardisation important, and delivery technologies such as nano-formulations and liposomes were suggested as a way to improve efficacy. Although branded extracts are sold as nutraceuticals, and meta-analyses report improvements in fasting glucose (Gong et al., 2016; Neelakantan et al., 2014), the authors of the original studies themselves called for further work before fenugreek is positioned as a medical food or drug.

Discussion

The central finding of this analysis is that antioxidant activity works in the fenugreek literature as a unifying narrative more than as a tested cause. Studies in very different areas, from diabetic nephropathy to toxicant protection, converge on the same markers and the same explanation. That convergence is informative, because it shows a reproducible pattern: fenugreek preparations repeatedly shift peroxidation and antioxidant-enzyme markers in the protective direction. It also exposes the weakness of the evidence, since the same markers are affected by almost any change in metabolic state, and a reduction in glucose or lipid load can itself relieve oxidative stress. Without experiments that separate redox effects from metabolic effects, for example by giving a pure antioxidant control or blocking a redox pathway, the direction of causation remains open.

The second finding concerns uneven knowledge. Mechanistic reasoning leans on HIL and diosgenin, and this matches the emphasis of earlier syntheses (Fuller & Stephens, 2015). By contrast, the flavonoid C-glycosides that are most likely to account for in vitro

radical scavenging and COX inhibition, along with trigonelline, sotolone and the galactomannan fibre, have been tested in far fewer systems. A reader might therefore overestimate how much of fenugreek's activity is understood. The thematic map in Figure 1 should be read with this in mind: some arrows rest on experiments with the pure compound, and others on proposals.

The third finding is that extract identity is itself a mechanistic variable. Because aqueous, ethanolic and hexane preparations contain different compound mixtures, and because branded extracts are enriched in particular saponins, results cannot be transferred freely between them. Studies that report only a plant part and a solvent, without marker-compound content, leave the reader unable to say what dose of which constituent produced the effect. This limitation interacts with the absence of pharmacokinetic data: without knowing which forms reach tissue, in vitro concentrations cannot be compared with what a human consuming 5 to 25 g of seed powder is likely to experience.

Several practical recommendations follow from the themes. Experimental reports should state the chemical profile of the extract, including marker compounds such as HIL, diosgenin or trigonelline. Studies should pair crude extract with isolated compounds and with combinations so that synergy can be tested. Redox claims should be backed by more than one assay family and, where possible, by a direct measure of reactive species. Human trials of standardised extracts should include oxidative and inflammatory biomarkers alongside glucose and lipid endpoints, and should be accompanied by pharmacokinetic sampling. Finally, long-term safety follow-up is needed to extend the reassuring short-term findings.

Limitations

This was a document-based qualitative study, and several limits apply. The corpus came from the authors' own compiled literature and was not built through a registered, systematic search, so some relevant studies may be missing and the balance of themes may reflect what was collected. Coding was done by a single analytic team, which leaves room for interpretive bias even with an audit trail. The records were heterogeneous in model, dose and endpoint, so no effect sizes were pooled and the evidence ratings in Table 2 are interpretive. Much of the material is preclinical. Finally, because the analysis relied on how authors described their own findings, errors or over-statements in the primary reports would have carried through unchecked.

Conclusions

A qualitative reading of the fenugreek literature shows a consistent but largely inferential link between antioxidant activity and the plant's metabolic, inflammatory, hormonal and anti-proliferative effects. The most secure mechanistic knowledge concerns 4-hydroxyisoleucine and diosgenin, whereas the contribution of other constituents, of whole-extract synergy and of pharmacokinetics is still poorly understood. Safety data are encouraging but short-term. The next step is not more descriptive studies of crude extracts, but chemically characterised preparations, compound-level and combination experiments, and human trials that measure redox and signalling endpoints together with exposure. Such designs would turn the present narrative of antioxidant benefit into a testable account.

Declarations

Ethical approval. Not applicable. The study analysed published literature and involved no human participants or animals.

Data availability. The codebook and the coded passages supporting the themes are available from the corresponding author on reasonable request.

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