



MORPHOMETRIC TRANSFORMATIONS IN THE THYROID GLAND ASSOCIATED WITH DIGESTIVE TRACT CHEMICAL BURNS AND THEIR CORRECTION ACCORDING TO LITERATURE DATA

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Abstract: Burn injuries remain a significant global public health concern, accounting for approximately 180,000 deaths annually. The overwhelming majority of these fatalities occur in low- and middle-income countries (LMICs), with nearly two-thirds reported in the African and South-East Asian regions of the World Health Organization (WHO). Although the incidence of burn-related mortality has shown a declining trend in high-income settings, LMICs continue to exhibit disproportionately high rates, particularly among children, where mortality is estimated to be seven-fold greater than in high-income countries. Beyond fatal outcomes, non-lethal burn injuries represent a major contributor to global morbidity, resulting in prolonged hospitalization, irreversible disfigurement, and long-term disability, all of which foster social stigmatization and discrimination. In LMICs, burn injuries are recognized among the leading causes of disability-adjusted life years (DALYs) lost, underscoring their persistent clinical and socioeconomic impact.

Introduction. The burden of burn-related hospitalizations varies considerably across nations and is closely associated with the structural organization and financing of healthcare systems. In several surveyed regions, the mean duration of inpatient treatment has declined, concomitant with an increased reliance on specialized burn centers (1). Among chemical agents implicated in severe burn injuries, **acetic acid** occupies a particularly prominent role due to its ubiquitous availability, low cost, and potent corrosive properties. Toxicity may arise via multiple routes of exposure, including inhalation of vapors, dermal or mucosal contact, enteral ingestion, and parenteral administration. Of these, oral ingestion is regarded as the most catastrophic, both clinically and toxicologically, producing rapid and extensive tissue injury with systemic repercussions. The estimated lethal dose is approximately 50 mL [14].

Epidemiological data reveal alarming trends:

- Approximately 70–80% of acetic acid poisonings occur in the context of deliberate self-harm;
- Over 60% of patients present in severe or extremely severe condition upon hospital admission;
- Mortality rates range between 10–40%, establishing acetic acid among the most hazardous corrosive agents;
- Most cases involve ingestion of vinegar essence with concentrations of 70–80%;
- The majority of affected individuals are young adults between 18 and 40 years of age.

The high prevalence of such cases is directly attributable to the unrestricted commercial availability of concentrated acetic acid, its affordability, and its strong caustic potential.



Pathophysiology of Acetic Acid Poisoning

Oral ingestion of acetic acid represents the most clinically severe exposure route, associated with profound local tissue destruction and systemic toxicity. Owing to its strong corrosive action, the acid induces immediate **coagulative necrosis** of epithelial surfaces in the oral cavity, pharynx, esophagus, and gastric mucosa. The underlying pathological cascade involves several interconnected mechanisms:

- **Coagulative necrosis**, resulting from denaturation of intracellular proteins and formation of dense, non-viable tissue barriers;
- **Acute inflammatory response**, manifested as pain, edema, and microcirculatory impairment;
- **Vascular disruption**, which predisposes to hemorrhage, mucosal erosion, and possible gastrointestinal perforation;
- **Secondary infection risk**, as compromised mucosal integrity facilitates microbial translocation;
- **Hemolysis and hemoglobinuria**, secondary to the direct cytotoxic effect on erythrocytes;
- **Acute kidney injury**, attributable to hemoglobinuric nephropathy caused by tubular deposition of free hemoglobin;
- **Severe metabolic acidosis**, consequent to hydrogen ion accumulation and impaired systemic buffering capacity.

Clinical Features and Complications

The clinical spectrum of acetic acid ingestion is diverse and typically characterized by:

- Intense burning pain localized to the oral cavity, pharynx, and retrosternal region;
- Severe nausea and recurrent vomiting, frequently hematemesis;
- Hoarseness, dysphonia, and respiratory compromise secondary to laryngeal involvement;
- Dysphagia due to esophageal spasm or stricture formation;
- Gastric mucosal erosions and life-threatening gastrointestinal hemorrhage in advanced cases;
- Evidence of hemolysis, including jaundice and darkly pigmented urine;
- Renal impairment, manifesting as oliguria or complete anuria;
- Metabolic acidosis, reflected in Kussmaul-type respiration and progressive neurological deterioration.

In fulminant presentations, patients may progress to **acute toxic shock**, characterized by cardiovascular collapse, refractory metabolic acidosis, and respiratory insufficiency, necessitating immediate intensive care and advanced resuscitative interventions.

Main sections

Trends in corrosive poisonings and regulatory context

Although the overall number of poisoning cases has modestly declined in recent years, the incidence of **severe** intoxications has increased. In the European Union and North America, corrosive toxic exposures account for only about **0.4–0.5%** of all toxicological admissions, a figure largely attributable to restrictions on over-the-counter access and strict occupational safety controls [8–10]. In Uzbekistan, retail sale of acetic acid solutions with concentrations **>10%** was prohibited relatively recently, by Cabinet of Ministers Resolution No. **183/14** dated **18 November 2013**. In domestic food processing and household settings, however, **70%** acetic acid remains the most commonly encountered preparation [11,15]. The estimated lethal dose of **glacial (anhydrous) acetic acid** is **12–15 g**, corresponding to **20–40 mL** of a 90% solution, **60–80 mL** of a 70% solution, or approximately **200 g** of table vinegar at **9%** concentration.



Oxidative stress in thyroid disease

Maintenance of cellular redox homeostasis is essential for physiological function. **Reactive oxygen species (ROS)** serve as short-lived second messengers within signaling cascades [21], generated predominantly in mitochondria [22] or via enzymatic reactions involving, for example, glutathione peroxidases, superoxide dismutases, peroxiredoxins, myeloperoxidase, and catalase [21]. **Oxidative stress (OS)** is classically defined as a disequilibrium that shifts the balance toward pro-oxidants [23]. Because ROS are highly labile, their direct quantification is impractical in most biological systems [24]. Accordingly, the field relies on **damage-derived biomarkers** that index oxidative injury to proteins, lipids, and nucleic acids; frequently used markers include **8-hydroxy-2'-deoxyguanosine (8-OHdG)** [25], **malondialdehyde (MDA)** [26], and assays of **total antioxidant capacity (TAC)** [27,28].

OS participates in the pathogenesis of diverse disorders, including cancer [29], diabetes mellitus [30], cardiovascular disease [21], hepatic disease [31], Alzheimer's disease [32], neuropsychiatric conditions [33], and multiple entities affecting reproductive health [34–36]. **Hashimoto's thyroiditis (HT)** is also linked to oxidative imbalance in both adults and pediatric populations [27,37]. No single oxidative biomarker has yet achieved consensus utility as a routine clinical indicator in HT, and active work continues to identify superior candidates. Elevated **total oxidant status (TOS)** and **oxidative stress index (OSI)** have been documented in subclinical hypothyroidism and HT, suggesting that OS may contribute to progression toward overt hypothyroidism [38]. **Mitochondrial DNA copy number** and **mtDNA damage** are under investigation as additional OS-associated metrics in HT [39,40]. Small **microRNAs (miRNAs)**, which post-transcriptionally regulate gene expression, are emerging as potential biomarkers and therapeutic targets in autoimmune thyroid disease (AITD) [41]. Gene-level analyses in HT have identified OS-related transcripts with expression patterns correlated to selected miRNAs; notable associations have been observed between these signatures and immune cell subsets, including natural killer cells and B cells, underscoring immunogenetic underpinnings of disease [42]. Advanced glycation end-products (**AGEs**) have been proposed as OS-linked markers in HT, while expression of the antioxidant enzyme **paraoxonase-1 (PON-1)** appears reduced; inverse correlations between AGEs and PON-1 further emphasize pro-oxidant and inflammatory disequilibria in HT [43]. As adjunctive strategies to temper oxidative imbalance, **selenium, zinc, and vitamins D and C** are increasingly studied in thyroidal inflammation [44].

Stress, apoptosis signaling, and thyroid pathology

A substantial body of work implicates physiological and psychosocial stressors—individually and in combination—in the initiation and progression of thyroid disorders [Безрыков О.Ф., 2017; Zhang J., 2018]. Structural alterations of the thyroid under stress conditions have been linked to clinically significant outcomes, including AIT and oncogenesis. Particular attention has focused on dynamics of **Fas (FAS-R) apoptotic receptors**, whose activity contributes to the elimination of malignant thyrocytes [Cruz C., 2016; Asif F., 2018]. Multiple factors within thyroid cancer pathogenesis promote OS, thereby fostering **DNA injury, oncotransformation** of thyrocytes, and sustained **tumorigenic signaling** [Metere A., 2018]. Despite these associations, integrative, system-level syntheses quantifying the role of stress in the onset of thyroid disease remain limited, and the effects of distinct stress modalities on thyroid structure and function—with attention to sex, age, cellular population dynamics, and proliferation markers—require further clarification.

Diagnostic considerations and unmet needs in thyroid neoplasia



Mechanisms of thyroid neoplastic transformation are incompletely delineated. Latency and subsequent abrupt acceleration of previously indolent tumor clones are poorly explained. The generally favorable course of many thyroid malignancies—particularly **papillary thyroid carcinoma (PTC)** in its earliest stages—has produced enduring debate regarding the optimal **extent of surgery**. This debate has intensified as improved **fine-needle aspiration (FNA)** techniques increase referrals for operative management [Ahmed S., 2019]. Current standard evaluation integrates **ultrasonography**, **TSH/T3/T4** profiling, **calcitonin** (when indicated), and **FNA cytology** [Воробьев С.Л., 2014]. Cytologic reporting according to the **Bethesda System** stratifies risk across six categories and guides treatment planning, including operative extent [Cibas E., 2009].

Nevertheless, limitations persist. In indeterminate cytology, reliable discrimination between **benign follicular neoplasia** and **follicular carcinoma** is often not possible preoperatively due to overlapping morphologic criteria. Many patients with indeterminate results proceed to surgery, yet postoperative histology does not invariably confirm malignancy. Even in PTC, unequivocal nuclear features may not always be demonstrable in cytologic preparations [Михайлова М., 2015]. These constraints highlight the clinical importance of **biomarker discovery** and the integration of **molecular genetic investigations (MGI)** in preoperative risk assessment.

Thyroid follicular unit, aging, and functional morphology

The functional unit of the thyroid is the **follicle**, composed of follicular (thyrocyte) epithelium surrounding intraluminal **colloid**. Follicle size, shape, and epithelial cell height vary with functional state; colloid morphology also reflects synthetic activity. Aging induces morphological and functional remodeling of the gland and a progressive decline in homeostatic resilience. In male albino rats and in camels, increases in follicle size and number have been reported, whereas in humans older than **60 years** progressive **fibrosis** and **atrophy** predominate, contributing to reduced gland volume. Age-related cell loss in follicular epithelium has been described, although the extent of functional decline remains debated.

Cells with long renewal intervals accumulate genomic injury over time and are particularly susceptible to age-related dysfunction. Approximate renewal periods include neurons (~**16,425 days**), myocytes (~**5,510 days**), and hepatocytes (~**327 days**). Human thyrocytes exhibit a comparatively long renewal interval (~**3,180 days**) relative to adrenal (~**455 days**) and pancreatic (~**265 days**) cells, implying greater cumulative risk of **DNA damage** and subsequent tissue atrophy. Although several studies describe age-linked histologic shifts in the thyroid, the relationship between these changes and follicular functional activity is understudied. The present line of investigation focuses on age-dependent structural and functional remodeling within follicles and the impact of such remodeling on circulating thyroid hormone levels.

Potentilla alba extracts: antimicrobial, antiviral, antineoplastic, and thyrotropic profiles

Antimicrobial activity

Antimicrobial properties of **Potentilla alba** (white cinquefoil) rhizome extracts have been demonstrated across multiple solvent systems. In Bosnian studies, aqueous, ethanolic, acetone, and ethyl-acetate extracts were tested against **Staphylococcus aureus**, **Bacillus subtilis**, **Escherichia coli**, and **Candida albicans**. Moderate inhibition of *S. aureus* was observed with acetone (1:1) and aqueous (1:10) extracts (inhibition zones **13.4 mm** and **11.0 mm**, respectively). Acetone and ethanol preparations suppressed *E. coli* growth comparably to 2% tannic acid, with limited activity against *B. subtilis* and *C. albicans*. Subsequent work reported larger inhibition zones against *E. coli* (**21.0±1.1 mm**), *S. aureus* (**19.0±1.0 mm**), *C. albicans* (**22.0±1.1 mm**), and notable activity versus *Proteus vulgaris* (**20.0±1.0 mm**) and *Pseudomonas aeruginosa*



(23.0 ± 1.2 mm). Aqueous extract (1:10) retained efficacy against *P. aeruginosa*, *C. albicans*, and *E. coli* after two- to four-fold dilutions and, for *S. aureus* and ***Bacillus cereus***, up to **16–32-fold** dilutions, variation plausibly reflecting differences in secondary metabolite composition and strain susceptibility.

Antiviral activity

An aqueous whole-plant extract demonstrated activity against **herpes simplex virus type II** in **Vero** cell assays, whereas ethanolic extracts exhibited weaker effects.

Antineoplastic activity

Polish investigators evaluated extracts and fractions from aerial parts of *P. alba* in **HT-29 human colorectal carcinoma** and **CCD 841 CoTr** normal epithelial cells. Extracts disrupted tumor-cell membranes and reduced proliferation; fractions enriched in **caffeic-like acids** altered the cell cycle and enhanced **apoptosis**. In non-malignant cells, mitochondrial metabolism was relatively preserved, with a paradoxical increase in proliferation.

In vivo adaptogenic and anti-inflammatory effects

In mice, oral administration of aqueous rhizome extract (**36 or 72 mg/kg** for one week) prolonged forced-swim endurance in a dose-dependent manner, potentially via higher glycogen availability, and produced **anxiolytic-like** effects in light–dark box and novelty paradigms. In a mouse ear-edema model, an **acetone** rhizome extract reduced inflammation with potency comparable to **1% hydrocortisone**; the **ethanol** extract was less active.

Thyroidal relevance and endocrine physiology

The thyroid critically regulates growth, development, and metabolic homeostasis through **triiodothyronine (T3)** and **thyroxine (T4)**, under hypothalamic (**TRH**) and pituitary (**TSH**) control. Disorders span **euthyroid structural disease** (nodular disease, thyroiditis), **hyperthyroidism** (commonly Graves' disease or toxic nodular goiter), and **hypothyroidism** (iodine deficiency or hypothalamic–pituitary dysfunction). Approximately one-third of the world's population resides in regions with **iodine deficiency**; overt hypothyroidism in Europe is estimated at **0.2–5.3%**. The **EFSA** and **WHO** recommend **150 µg/day** iodine intake for adults and **200–250 µg/day** for pregnant and lactating women.

Thyrotropic activity of *Potentilla alba* extracts

Preclinical data

In animal models, a mixture of ***P. alba* root** and **Vozrozhdenie plus** balsam increased serum T3 and T4 and restored adrenergic innervation of the thyroid (Abdreshov et al., 2021). A **triterpene-rich root extract** conferred protection against **CCl₄** toxicity and **γ-irradiation**. In hypothyroid rats, extract administration elevated T3 and T4 by approximately **34%** and **30%**, respectively.

Clinical observations

Across three independent clinical studies (2012), **300 mg** rhizome extract **twice daily** reduced thyroid volume and normalized **TSH** in patients with diffuse nodular disease, hyperthyroiditis, and thyroiditis. In **77** patients with hypo- or hyperthyroidism, extract use was associated with improvement of somatic symptoms and reduction in nodule size. In another cohort of **46** patients, three months of therapy increased TSH and lowered thyroid autoantibodies. A Ukrainian multicenter series (**n=1107**) reported **≥15%** reductions in thyroid volume after six months of extract administration. Pediatric observations also noted volume reduction with hormonal normalization. In **100** patients with subclinical thyroiditis, TSH normalized and overall well-



being improved. A composite preparation (**Tireoclean**, containing *P. alba*, black chokeberry, hawthorn, and sodium selenite) improved TSH and T4 in subcompensated hypothyroidism.

Toxicological safety considerations

Despite broad medical interest, comprehensive human toxicology for *P. alba* preparations remains limited for ethical and economic reasons; rodent studies therefore provide essential proxies. **Acute oral toxicity** of aerial-part extracts in mice at **1000–4000 mg/kg** yielded an LD₅₀ of **2359.9 mg/kg body weight**; a **1/10 LD₅₀** dose (**~239 mg/kg**) given to rats for three months produced no adverse effects on survival, intake, or coat condition, supporting classification as **practically non-toxic** under **OECD** criteria [74]. Rhizome extracts showed no lethality or overt toxicity after single intraperitoneal dosing or three-month administration in mice and rats [75,76]. For ethanol–water extracts of underground parts, LD₅₀ was **6500 mg/kg** in both male and female rats [33].

Immunotoxicology studies indicated that **50 mg/kg** of dried rhizome extract did not impair humoral, cellular, or macrophage immunity in mice; **3 mg/kg** stimulated primary humoral responses in guinea pigs and did not elicit systemic or delayed-type hypersensitivity or cutaneous anaphylaxis [77]. In mice treated with the cytostatic agent **azathioprine**, co-administration of *P. alba* attenuated suppression of antibody production and cellular immune responses, suggesting **immunomodulatory** potential [78]. Caution is warranted in reproductive contexts: oral administration in pregnant rats and offspring was associated with delayed ossification of cartilaginous bone segments, and in males with reduced sperm motility, smaller Leydig-cell nuclear diameters, increased pyknotic nuclei, and a higher proportion of morphologically abnormal spermatozoa, although overall fertility with healthy females was not significantly compromised [79,80]. These findings support the need for rigorous, dose-finding clinical safety studies, especially in pregnancy and reproductive health.

Conclusion

Strengths

- *Potentilla alba* is rich in polyphenols and exhibits antioxidant, immunomodulatory, antimicrobial, antiviral, antineoplastic, and thyrotropic activities.
- Preclinical and early clinical studies suggest benefits for benign thyroid disorders, with improvements in thyroid volume, TSH/T4–T3 balance, and symptom control.

Gaps

- The main bioactive components, mechanisms of action, pharmacokinetic properties, and long-term safety remain insufficiently defined.
- Current evidence is limited by small sample sizes, heterogeneous study designs, and lack of standardized preparations.

Future Directions

- Conduct multicenter randomized clinical trials with standardized extract formulations.
- Clarify molecular pathways through integrative omics and mechanistic studies.
- Evaluate safety in vulnerable groups (pregnant women, children, long-term users).
- Develop sustainable cultivation and renewable raw-material strategies to protect natural populations.

Overall

With rigorous validation and sustainable sourcing, *P. alba* has the potential to emerge as a promising adjunct or alternative therapy in the management of thyroid diseases.



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