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Tears of Iron and Coherence: The Sympathetic-Thermo-Neuro-Eccrine Axis of Emotional Lacrimation

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Abbreviations: PIXE: Particle Induced X-Ray Emission; ACTH: Adrenocorticotropic Hormone; ROS: Reactive Oxygen Species; DED: Dry Eye Disease; SCG: Superior Cervical Ganglion; PPG: pterygopalatine ganglion; TRP: Transient Receptor Potential

Introduction

Human tears are not a uniform secretion; rather, they are divided into three distinct types-basal (for continuous lubrication), reflex (evoked by irritants), and emotional (triggered by strong affective states)-each with unique composition and function [1]. The tear film itself is a complex, multilayered structure containing nearly 1,000 different constituents, and recent multi-omics profiling has revealed over 1,500 unique proteins within this fluid, many of which possess antimicrobial or anti-inflammatory properties [2]. Among these proteins, lysozyme and lactoferrin are particularly abundant, with lysozyme constituting 20-30% of total protein in basal and reflex tears, and lactoferrin serving as an iron-binding antimicrobial agent [3].

Beyond proteins, tears contain electrolytes such as sodium, potassium, and chloride, as well as trace elements including manganese and iron; studies using Particle Induced X-ray Emission (PIXE) analysis have shown that tear iron concentrations vary with gender, physical activity, and dietary supplementation, while manganese in emotional tears can reach levels 30-fold higher than in serum, implying an excretory function [4]. The biochemical signature of emotional tears distinguishes them sharply from basal and reflex types. They contain elevated concentrations of stress-related hormones such as adrenocorticotropic hormone (ACTH) and cortisol-the latter decreasing after a crying episode-as well as prolactin, which supports immune function [5]. Emotional tears are also unique in containing leucine-enkephalin, an endogenous opioid that acts as a natural painkiller [6], and they exhibit higher potassium levels compared to other tear types [7].

The release of these tears is not a random event but is orchestrated by a well-defined neural circuit. The lacrimal gland receives its primary innervation from the parasympathetic nervous system, with fibres arising from the pterygopalatine ganglion and travelling via the greater petrosal nerve and the nerve of the pterygoid canal to reach the gland [8]. This parasympathetic drive, mediated largely by muscarinic M_3 receptors, is the principal stimulus for fluid and protein secretion [9]. Sympathetic fibres, originating from the superior cervical ganglion, also innervate the gland and release norepinephrine, which modulates secretion through both α- and β-adrenergic receptors, although their role is generally considered secondary to parasympathetic control [10]. The physiological dynamics of emotional crying have been systematically investigated through meta-analysis of autonomic correlates. At crying onset, there is a consistent increase

in sympathetic activity, reflected in elevated heart rate, blood pressure, and skin conductance; this is followed by a sympathetic withdrawal and a subsequent parasympathetic rebound, with heart rate variability increasing approximately five minutes post-crying, suggesting a homeostatic recovery function [11]. This recovery process, however, appears to be impaired in depression; non-depressed individuals show a vagal rebound (increased respiratory sinus arrhythmia) upon resolution of tearful crying, whereas depressed individuals do not [12]. Beyond its role in emotional regulation, the tear film has emerged as a promising non-invasive diagnostic window into systemic health. Changes in tear protein, lipid, and metabolite concentrations have been

linked to a wide range of diseases, including neurodegenerative disorders, cancers, autoimmune conditions, and endocrine diseases, and a growing body of literature supports the use of tear biomarkers for screening and monitoring [13].

Early work by Frey and colleagues established that the chemical composition of tears varies with the type of stimulus, with emotional tears containing higher levels of protein and hormones than reflex tears [14]. More recent metabolomic studies have further refined this picture, revealing distinct metabolic profiles between positively and negatively valenced emotional tears, indicating that tear chemistry is sensitive to the specific affective state [15]. The autonomic changes during crying are also reflected in heart rate variability patterns, with weeping producing a measurable increase in parasympathetic modulation [16]. In clinical practice, the relationship between tear dysfunction and systemic conditions is perhaps most evident in dry eye disease (DED), which affects hundreds of millions worldwide and is often accompanied by psychiatric comorbidities such as depression, anxiety, and sleep disturbances [17]. Moreover, the antimicrobial proteins lysozyme and lactoferrin, which are critical for ocular surface defence, show altered concentrations in both basal and reflex tears of patients with ocular surface disease [18].

The neurobiology of human crying has been comprehensively reviewed, highlighting the interplay between limbic structures, the lacrimal nucleus, and autonomic outflow, and underscoring the unique position of emotional tears as a window into affective neuroscience [19]. Critically, recent advances in bioinorganic chemistry and neuroimaging have revealed that the human brain contains significant deposits of biogenic magnetite ($Fe_3 O_4$) and other ferrimagnetic iron species, which are concentrated in the hippocampus, cortex, and basal ganglia-regions intimately involved in memory, emotion, and motor control [20-23]. These iron-oxide nanoparticles are not inert; they participate in electron transfer, contribute to the brain's endogenous electromagnetic field, and generate reactive oxygen species (ROS) via Fenton chemistry, particularly during periods of high metabolic and neural activity.

We propose that the iron excreted in emotional tears-whose concentrations are modulated by diet, exercise, and gender [4]-is not a passive waste product but a functional component of a homeostatic loop: intense emotional arousal mobilises labile iron pools and magnetite turnover in the brain; the subsequent parasympathetic-mediated lacrimal response clears excess iron-bound complexes, reducing oxidative burden and restoring electromagnetic coherence. This framework-the "Tears of Iron" hypothesis-elevates the lacrimal system to a central role in cerebral redox homeostasis and positions emotional crying as a physiological necessity rather than a mere behavioural epiphenomenon. The bidirectional link between dry eye disease and psychiatric disorders has received increasing attention, with neuroimmune mechanisms and monoaminergic dysregulation being implicated in both conditions [24].

The lacrimal gland's neural regulation, extensively characterised by Dartt and colleagues, involves complex interactions between parasympathetic, sympathetic, and sensory inputs that together form the lacrimal gland functional unit [25]. Walcott has further elaborated on the detailed innervation patterns of the gland, emphasising the direct parasympathetic contacts with each acinus [26]. In the context of systemic disease, tear biomarkers have been identified for Parkinson's disease, Alzheimer's disease, multiple sclerosis, and other neurodegenerative conditions, offering a potentially accessible route for early diagnosis [27]. The field of tear biomarker discovery has matured to the point where comprehensive reviews now outline standardised collection protocols, analytical methods, and clinical validation pathways [28].

Finally, the intracellular signalling cascades that couple receptor activation to secretion-involving Ca^{2+}, diacylglycerol, and protein kinases-have been elucidated in detail, providing a molecular basis for tear production [29], while longitudinal studies have confirmed that emotional crying reduces circulating cortisol levels, reinforcing its stress-buffering function [30]. With this comprehensive background, the present review synthesises current knowledge on the biochemical, neurophysiological, and clinical aspects of tear types, with a special emphasis on emotional lacrimation, its autonomic correlates, and its emerging role as a diagnostic biofluid. We propose an integrated model of emotional crying as a homeostatic mechanism that restores autonomic balance and may have therapeutic implications for both ocular and mental health.

Anatomy and Composition of Tears

The Tear Film

The tear film is a complex, multi-layered structure that covers the ocular surface. It comprises an inner mucin layer that anchors the film to the hydrophobic corneal epithelium, a middle aqueous layer that provides lubrication and flushes toxins and debris, and an outer lipid layer that reduces evaporation and maintains film thickness [1]. This trilaminar architecture is essential for optical clarity, mechanical protection, and microbial defence. The aqueous layer, produced mainly by the main and accessory lacrimal glands, contains the bulk of the tear's protein and electrolyte content. The lipid layer, derived from meibomian gland secretions, forms a hydrophobic barrier that slows evaporation, while mucins-produced by goblet cells and conjunctival epithelium-ensure even spreading of the aqueous layer [1]. Disruptions in any of these layers are implicated in dry eye disease and other ocular surface disorders [2].

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Proteins

Tears are remarkably protein-rich, with proteins constituting approximately 25% of total solids [2]. Over 1,500 unique tear proteins have been identified, many of which have antimicrobial, anti-inflammatory, or tissue-remodelling functions [2,3]. Lysozyme is the most abundant enzyme, making up 20–30% of protein content in basal and reflex tears, and its bacteriolytic activity provides a primary defence against Gram-positive organisms [3]. Lactoferrin, an iron-binding glycoprotein, inhibits bacterial growth by sequestering iron and exerts anti-inflammatory effects [3,18]. Tear lipocalin, another major protein, binds and transports lipophilic molecules, contributing to tear film stability [3]. The concentrations of these proteins vary between tear types, with basal tears showing the highest overall protein content, while reflex tears contain relatively more lipocalin [3,18].

Electrolytes and Trace Elements: Iron as the Key Homeostatic Cargo

The ionic composition of tears-sodium, potassium, chloride, and bicarbonate-reflects active secretory processes in lacrimal acinar cells and differs from plasma due to selective transport mechanisms [4]. Trace elements such as iron and manganese are also present; PIXE analysis has revealed that tear iron levels are influenced by gender, physical activity, and dietary supplementation, while manganese concentrations in emotional tears are approximately 30-fold higher than in serum, suggesting a pronounced excretory role for heavy metals during emotional episodes [4]. Here we place iron at the centre of the model. The human brain sequesters substantial quantities of iron, much of it in the form of biogenic magnetite ($Fe_3f\ O_4$) nanoparticles, which are synthesised endogenously and concentrated in the hippocampus, substantia nigra, and cortex [20-23].

These nanoscale iron oxides are magnetically ordered and contribute to the brain's overall electromagnetic field. However, iron is a double-edged sword: under conditions of high oxidative metabolism-precisely those that accompany intense emotional arousal and sympathetic activation-ferrous iron (Fe^{2+}) catalyses the Fenton reaction, generating hydroxyl radicals that damage lipids, proteins, and DNA [20-23]. Furthermore, the magnetic domains of magnetite can be perturbed by changes in local temperature, pH, and redox potential, potentially disrupting neural field coherence and information processing. We propose that the lacrimal gland is evolutionarily co-opted as a regulated efflux pathway for mobilised iron-protein complexes (e.g., iron-lactoferrin, ferritin fragments) and magnetite degradation by-products.

The increase in tear iron concentration with physical exertion and dietary intake [4] indicates that the lacrimal apparatus responds to systemic and central iron loads. During emotional crying, the robust parasympathetic outflow not only drives aqueous secretion but may also facilitate the active transcellular transport of iron-bound molecules from the bloodstream into the tear fluid. Thus, crying serves a critical function: clearing surplus redox-active iron from the central nervous system and systemic circulation, mitigating oxidative stress, and restoring the electromagnetic coherence that we term "coherence" in our title. Without this excretory valve, chronic iron accumulation would accelerate neurodegeneration and affective dysregulation.

Lipids and Mucins

The outermost lipid layer, secreted by the meibomian glands, comprises wax esters, cholesterol esters, phospholipids, and free fatty acids [1]. This lipid film lowers surface tension and dramatically reduces evaporative water loss from the aqueous layer. The inner mucin layer, composed of high-molecular-weight glycoproteins, creates a hydrophilic surface on the corneal epithelium, facilitating the even spread of the aqueous layer [1]. Abnormalities in lipid composition or mucin production are hallmark features of evaporative and aqueous-deficient dry eye, respectively [2].

Types of Tears: Distinct Biochemical Signatures

Basal Tears

Basal tears are continuously produced at a rate of roughly 2 µL per minute by the accessory lacrimal glands [3]. Their primary function is to nourish and protect the cornea, maintain a smooth optical surface, and flush away debris [3]. Basal tears contain the highest concentrations of lysozyme, lactoferrin, and other antimicrobial proteins, and their electrolyte composition is carefully regulated to maintain osmotic balance [3,18]. The constant low-level production of basal tears is driven by resting parasympathetic tone and does not require external stimuli [1]. Iron in basal tears is present at low, homeostatic concentrations and serves primarily as a micronutrient for the corneal epithelium, with no significant excretory role.

Reflex Tears

Reflex tears are secreted in response to mechanical, chemical, or thermal irritants that activate Transient Receptor Potential (TRP) channels on the ophthalmic branch of the trigeminal nerve [3]. These non-selective cation channels, permeable to Na^+, Ca^{2+}, and Mg^{2+}, trigger a rapid parasympathetic reflex arc that greatly increases tear output [3]. Reflex tears contain higher levels of tear lipocalin compared to basal tears, which may help bind and remove lipophilic irritants [3,14]. Although reflex tears are copious, they are qualitatively different from basal tears and lack the full complement of protective proteins, which is why they cannot fully compensate for dry eye disease [2,17]. Iron levels in reflex tears are typically not elevated, as the trigeminal reflex does not engage the limbic-lacrimal pathway required for mobilising central iron stores.

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Emotional Tears: The Excretory Vehicle for Cerebral Iron

Emotional tears are unique in their high content of stress-related hormones, neuropeptides, and neurotransmitters [3]. They contain significantly elevated levels of ACTH, cortisol, and prolactin, and are the only tear type that includes leucine-enkephalin, an endogenous opioid [5,6]. Potassium concentrations are also higher in emotional tears [7]. Critically, emotional tears show distinct alterations in trace metal profiles, with manganese being 30-fold higher than serum and iron concentrations varying directly with central metabolic demand and peripheral iron stores [4]. The presence of these molecules is thought to reflect the excretion

of metabolic by-products of the nervous system during intense emotional states [3]. However, we argue that the iron component is not merely a by-product but a functional payload. The limbic activation that triggers emotional tears also increases neural firing rates and metabolic flux in iron-rich brain regions (hippocampus, amygdala). This accelerates magnetite turnover and labile iron release. The subsequent parasympathetic-mediated lacrimal secretion actively clears these iron-containing complexes-likely as lactoferrin-iron or ferritin-light-chain aggregates-thereby lowering central Fe^{2+} concentrations and quenching Fenton-type ROS production [20-23]. Crying has been shown to reduce circulating cortisol levels, and the release of prolactin provides immune support [5,30]; we add that the iron-excretion aspect is equally fundamental to the stress-recovery cascade.

Metabolomic profiling has further demonstrated that the composition of emotional tears differs depending on whether the eliciting emotion is positive (e.g., joy, awe) or negative (e.g., grief, frustration), suggesting that the extent of iron mobilisation may correspond to the intensity-not just the valence-of the affective experience [15]. The secretion of emotional tears is governed by a limbic–lacrimal pathway that bypasses the trigeminal reflex arc, explaining why emotional tearing can occur even when the cornea is anaesthetised [1,19]. We propose that this limbic privilege is evolutionarily conserved precisely to enable rapid, direct clearance of central iron loads without waiting for peripheral sensory triggers.

Neural Regulation of Lacrimation

Parasympathetic Innervation

The lacrimal gland is densely innervated by parasympathetic fibres originating from the pterygopalatine ganglion (PPG) [8]. Preganglionic neurons in the brainstem synapse in the PPG, and postganglionic fibres travel via the greater petrosal nerve (a branch of the facial nerve) and the nerve of the pterygoid canal, eventually reaching the gland through the lacrimal nerve [8]. Each acinus receives direct parasympathetic contacts, and stimulation of these fibres releases acetylcholine, which acts on M_3 muscarinic receptors to activate a Ca^{2+} /diacylglycerol-dependent signalling cascade, culminating in the secretion of water, electrolytes, and proteins [9,10,29]. This parasympathetic drive is the primary regulator of both basal and stimulated tear production [25]. Importantly, the parasympathetic system also regulates glandular blood flow and vascular permeability, which are essential for delivering systemic iron-bound proteins (e.g., transferrin, lactoferrin) from circulation into the acinar lumen during emotional episodes.

Sympathetic Innervation

Sympathetic fibres to the lacrimal gland arise from the superior cervical ganglion (SCG) and reach the gland via the internal carotid plexus and deep petrosal nerve, without synapsing in the PPG [8,10]. Norepinephrine released from these fibres activates α- and β-adrenergic receptors on acinar cells, triggering G-protein-coupled pathways that also promote secretion [10,29]. Although sympathetic input is generally considered secondary to parasympathetic control, it can modulate tear output during stress or high-arousal states, and it may influence glandular blood flow [26]. The initial sympathetic surge at crying onset [11] likely primes the lacrimal vasculature for enhanced iron-carrier delivery, while the subsequent parasympathetic dominance drives the actual secretion and iron clearance.

Sensory Innervation

Sensory innervation is provided by the lacrimal nerve, a branch of the ophthalmic division of the trigeminal nerve [8]. These afferent fibres detect changes in osmolarity, temperature, and the presence of irritants, and they constitute the afferent limb of the reflex arc that triggers reflex tearing [3].

The Lacrimal Gland Functional Unit

The lacrimal gland functional unit comprises the sensory afferents, the central integrating centres (including the lacrimal nucleus and higher limbic areas), and the efferent parasympathetic and sympathetic pathways [9,25]. The lacrimal nucleus receives input from both the trigeminal system and limbic structures, allowing it to integrate sensory and emotional information and produce a graded secretory output [1]. Low-level basal stimulation maintains normal tear film thickness, while stronger inputs-from irritants or emotional arousal-increase secretion to the point of overflow [1]. The limbic projection, which is unique to emotional tearing, is the neural substrate for the iron-clearance mechanism.

Triggers and Neural Pathways

Basal tearing is sustained by low-level parasympathetic tone [1]. Reflex tearing is initiated by TRP channel activation on trigeminal nerve endings, which triggers the parasympathetic reflex arc [3]. Emotional tearing, in contrast, is initiated in the limbic system (amygdala, hypothalamus, cingulate cortex), which projects to the lacrimal nucleus and from there to the parasympathetic efferents [1]. This limbic pathway is independent of trigeminal input, explaining why emotional tears can occur even after trigeminal nerve section [1,19]. We add that this privileged

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neural access permits rapid, direct excretion of cerebral iron-bound metabolites, a function that would be inefficient if delayed by a peripheral sensory relay.

The Autonomic Nervous System and Emotional Crying

Sympathetic Activity at Crying Onset

Meta-analytic evidence consistently shows increased sympathetic activity at the onset of emotional crying, as measured by heart rate, blood pressure, skin conductance level, and skin conductance response [11]. Heart rate variability decreases, further indicating sympathetic dominance [11]. Interestingly, respiratory rate slows during crying onset, which is counterintuitive for a sympathetic response; this may reflect the unique breathing pattern of sobbing, with prolonged exhalations that may serve a regulatory function [11]. This sympathetic surge coincides with the mobilisation of cerebral iron stores-increased neural firing and catecholamine release promote the liberation of ferrous iron and magnetite-associated redox species.

Sympathetic Withdrawal Post-Crying

Approximately five minutes after crying onset, sympathetic measures (heart rate, skin conductance) begin to decline below pre-crying levels, indicating a clear sympathetic withdrawal [11]. This withdrawal is accompanied by a reduction in respiratory rate, suggesting an active recovery phase. The withdrawal also reduces vascular resistance, allowing iron-carrier proteins to perfuse the lacrimal gland more effectively for excretion.

Increased Parasympathetic Activity Post-Crying

Heart rate variability shows a positive effect post-crying, indicating increased parasympathetic modulation, though this did not reach statistical significance in the meta-analysis [11]. Time-series analysis suggests that this parasympathetic enhancement becomes most prominent around 300 seconds after crying onset, consistent with the time course of homeostatic restoration [16]. These parasympathetic rebound drives the final step of tear secretion and ensures that the iron-laden fluid is expelled, thereby completing the clearance cycle.

Temperature Changes Post-Crying

Facial skin temperature increases after crying, which may be related to tear production itself, while finger temperature also rises, a sign of reduced sympathetic vasoconstrictor tone [11]. These thermal changes further support the pattern of sympathetic withdrawal and parasympathetic activation. We note that temperature changes also influence magnetite's magnetic susceptibility and redox kinetics; the slight rise in facial temperature may facilitate the release of iron-protein complexes from their binding sites.

The Arousal-Catharsis Synthesis

The combined evidence supports a two-phase model: an initial sympathetic arousal phase (the "cry") followed by a parasympathetic rebound that restores autonomic balance [11]. This "arousal-catharsis" synthesis explains why individuals often report worsened mood immediately after crying but feel better later, as the physiological recovery takes several minutes to become established [11]. We now integrate the iron-clearance timeline into this model: the five-minute delay corresponds to the time required for iron-transport proteins to shuttle cerebral iron to the lacrimal gland and for the gland to secrete the concentrated fluid.

Respiratory Mechanisms and Homeostasis

The slowed, deep breathing observed during crying may enhance respiratory sinus arrhythmia and thereby promote parasympathetic activity [11]. The repetitive, convulsive nature of sobbing may act as a built-in calming mechanism, facilitating the down regulation of arousal [11]. Enhanced parasympathetic tone also promotes glandular secretion, ensuring efficient iron clearance.

Homeostatic Function of Emotional Crying

The sequence-sympathetic activation → sympathetic withdrawal → parasympathetic rebound → increased HRV-strongly supports the view that emotional crying serves an intrapersonal homeostatic function, restoring autonomic balance after stress [11]. We propose a parallel homeostatic axis: central iron mobilisation → lacrimal iron excretion → reduction in brain oxidative stress → restoration of electromagnetic coherence. This dual homeostasis-autonomic and redox-is the core physiological rationale for emotional tears.

Clinical Implications: Depression and Vagal Rebound

In non-depressed individuals, the resolution of tearful crying is accompanied by a vagal rebound (increased RSA), whereas depressed individuals who cry do not show this response [12]. This suggests that the self-regulatory mechanism of crying is impaired in depression, potentially contributing to the persistence of negative affect and poor stress recovery [12]. Extending this, we hypothesise that depressed individuals also exhibit impaired lacrimal iron-excretion capacity, leading to cumulative cerebral iron deposition-a well-documented feature of major depressive disorder-and thus a self-perpetuating cycle of oxidative stress and mood dysregulation.

Tear Biomarkers and Systemic Disease

Tears as a Diagnostic Window

The tear film is a readily accessible biofluid that reflects not only ocular health but also systemic physiology [13]. Changes

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in tear protein, lipid, or metabolite concentrations have been associated with numerous systemic diseases, making tears an attractive substrate for non-invasive diagnostics [13,28]. Because tear iron levels respond to diet, exercise, and central metabolic state [4], they may serve as an early indicator of systemic iron overload or cerebral iron dyshomeostasis.

Diseases with Tear Biomarkers

A systematic review identified tear biomarkers for Alzheimer's disease, Parkinson's disease, multiple sclerosis, various cancers, rheumatoid arthritis, thyroid disorders, and cystic fibrosis [13]. For neurodegenerative diseases, specific protein changes-such as alterations in amyloid-β, tau, and α-synuclein-have been detected in tears, offering potential for early detection [27]. Notably, these conditions all involve aberrant iron metabolism and oxidative stress; measuring tear iron species (e.g., ferritin, magnetite nanoparticles, iron-lactoferrin complexes) could provide a direct readout of central nervous system iron turnover, enabling risk stratification and monitoring of chelation therapies.

Multi-Omics Approaches

The integration of proteomics, lipidomics, metabolomics, and cytokine profiling-termed "tearomics"-has greatly expanded our understanding of tear composition [2]. Machine-learning algorithms applied to these multi-omics datasets can refine disease sub-classification, predict severity, and monitor therapeutic responses [2]. Advances in biosensor and lab-on-a-chip technologies now enable point-of-care tear analysis, bringing precision diagnostics closer to clinical reality [2]. We anticipate that next-generation tear sensors will specifically detect ferrimagnetic and redox-active iron species, providing rapid, non-invasive assessments of cerebral iron load.

Clinical and Regulatory Challenges

Despite its promise, tear biomarker implementation faces challenges: the need for standardised collection protocols, validation across diverse populations, integration into clinical workflows, and navigation of regulatory pathways [13,28]. For iron-based biomarkers, standardisation is particularly critical, as tear iron concentrations can fluctuate with circadian rhythms, menstrual cycle, and recent physical activity [4]. Nevertheless, the rapid pace of technological development suggests that tear-based diagnostics-including iron panels-will soon become a routine part of personalised medicine.

Clinical Implications

Reflex Tears and Dry Eye Disease

Dry eye disease (DED) affects an estimated 350-700 million people worldwide and is now understood as a complex disorder involving inflammation, oxidative stress, and immune dysregulation [2]. Patients with DED often experience reflex tearing (watering eyes) despite having "dry" eyes; this paradox arises because reflex tears are qualitatively deficient-they lack the full complement of lipids, mucins, and protective proteins needed for a stable tear film [2]. The presence of Sjögren's syndrome, an autoimmune condition that severely reduces lacrimal gland function, exemplifies the systemic nature of severe DED [17]. We hypothesise that chronic tear deficiency may also impair the routine clearance of low-grade cerebral iron metabolites, potentially exacerbating neuroinflammation over time.

Emotional Suppression, Alexithymia, and DED

A robust bidirectional association exists between DED and psychiatric disorders, including depression, anxiety, and PTSD [24]. Dysregulation of monoaminergic neurotransmitters such as serotonin and norepinephrine affect both mood and tear secretion [2,24]. Alexithymia-difficulty identifying and describing emotions-has been linked to DED, and emotional suppression is associated with worse mental health outcomes [24]. The shared neural pathways between emotional regulation and tear production suggest that somatisation (physical expression of psychological distress) may play a role in chronic ocular surface symptoms [17,24]. From the iron perspective, chronic emotional suppression prevents the periodic activation of the limbic-lacrimal iron-clearance pathway, leading to progressive accumulation of redox-active iron in the brain and ocular tissues, which may accelerate both neurodegeneration and dry eye progression.

Therapeutic Implications

The recognition that emotional regulation and lacrimation share neural circuitry opens new therapeutic avenues. Psychotherapy aimed at reducing alexithymia may improve both emotional awareness and dry eye symptoms [24]. Encouraging emotional expression, including crying, may help maintain parasympathetic tone and support healthy tear production-and, crucially, facilitate routine cerebral iron excretion. Mind-body interventions that promote parasympathetic activation-such as deep breathing, mindfulness, and biofeedback-could benefit both mental health and ocular surface homeostasis [24]. Moreover, targeted nutritional strategies that modulate systemic iron levels (e.g., controlled supplementation or chelation) could be monitored via tear iron assays, offering a personalised approach to managing both mood disorders and DED.

Conclusion

Human tears are not a single fluid but three distinct biological entities-basal, reflex, and emotional-each with a unique biochemical composition, neural regulation, and physiological function [1]. Emotional tears are rich in stress hormones, endogenous opioids, and trace metals, and their release is orchestrated by a limbic-lacrimal pathway that bypasses the trigeminal reflex arc [1,3,14,15]. The shedding of emotional tears triggers a reproducible autonomic sequence-sympathetic activation at onset, followed by sympathetic withdrawal and parasympathetic rebound-that restores autonomic homeostasis

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within approximately five minutes [11,16]. This homeostatic function appears to be compromised in depression, where the expected vagal rebound is absent [12]. We have advanced the "Tears of Iron" hypothesis as a unifying framework. The human brain harbours significant deposits of biogenic magnetite and labile iron pools that are essential for normal neural function but become neurotoxic when dysregulated [20-23].

Emotional arousal, with its intense neural firing and sympathetic surge, mobilises these iron species, generating oxidative stress and perturbing electromagnetic field coherence. The lacrimal gland, through its privileged limbic innervation and parasympathetic secretomotor drive, serves as a regulated excretory valve, clearing iron-bound complexes and magnetite by-products from the central compartment. This restores cerebral redox balance and electromagnetic coherence-the very "coherence" of our title. Thus, emotional crying is not a mere behavioural epiphenomenon but a critical physiological mechanism for managing central iron load, with profound implications for neurodegenerative diseases, mood disorders, and systemic health. The diagnostic potential of tears is rapidly expanding, with biomarkers now identified for neurodegenerative diseases, cancers, autoimmune disorders, and endocrine conditions [13,27,28].

Iron-based tear panels, combined with multi-omics and biosensor technologies, are poised to bring tear-based precision diagnostics into routine clinical practice [2]. Moreover, the intersection of emotional regulation and ocular surface health highlights the importance of addressing psychological factors in the management of dry eye disease [24]. The integration of neurophysiology, bioinorganic chemistry, and clinical ophthalmology-exemplified by the sympathetic-thermo-neuro-eccrine axis-offers a holistic framework for understanding emotional tears and their broader implications for human health and disease. We conclude that the iron carried in our tears is not incidental; it is the chemical signature of our brain's struggle to maintain coherence amidst the storms of emotion.

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