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Systematic review

Chronic Humidity and Nasal Mucosal Dysfunction in Tropical Climates: Implications for Sinonasal Health in Sub-Saharan Africa and the Developing World

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Summary: Approximately 40% of the global population lives in tropical and equatorial climates, where temperatures exceed 28 °C and relative humidity (RH) is persistently high (>70%). While the effects of cold, dry air on nasal mucosal function are well established, the consequences of chronic high-humidity exposure remain poorly understood, particularly for sub-Saharan Africa. This systematic review evaluates the relationship between high ambient humidity and nasal mucosal function, including mucociliary clearance (MCC), ciliary beat frequency (CBF), mucus rheology, and sinonasal disorders. A search of PubMed/MEDLINE, Scopus, Web of Science, and Cochrane CENTRAL was conducted in accordance with PRISMA 2020 guidelines (January 2000–January 2025). Of 1,420 records identified, 45 studies met the inclusion criteria. Across included studies, findings were broadly consistent with a U-shaped relationship between RH and MCC efficiency, with optimal MCC generally reported at 40–60% RH. Chronic tropical humidity was associated with mucosal engorgement and over-hydration of the mucus gel layer, with reduced viscoelasticity, and reported values across studies generally ranged from 9–10 Hz for CBF to 18–22 minutes for saccharin transit time. Air-conditioning-related indoor–outdoor transitions may further destabilise mucosal homeostasis, with a hypothesised but unconfirmed contribution to vasomotor rhinitis and turbinate hypertrophy. The tropical allergen milieu of house dust mites and fungal spores, exacerbated by impaired MCC, amplifies the severity of perennial allergic rhinitis. These findings challenge the temperate-centric paradigm of rhinological practice and highlight the need for further climate-specific research, including studies from African settings, and cautious, evidence-informed approaches to managing sinonasal disorders in tropical populations. This review was not prospectively registered.

Keywords: *Nasal mucosa; humidity; tropical climate; mucociliary clearance; rhinitis; air-conditioning.*

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INTRODUCTION

The human nasal cavity is a highly specialised, anatomically complex organ responsible for the critical functions of filtering, warming, and humidifying inspired air before it reaches the delicate alveolar structures of the lower respiratory tract (Eccles, 2002). This air-conditioning process is an absolute physiological necessity; the lungs require air to be delivered at core body temperature (37°C) and fully saturated with water vapour (100% relative humidity) to facilitate optimal gas exchange and prevent alveolar desiccation (Keck *et al*., 2000). The efficiency of this thermodynamic and humidification process is heavily dependent on the structural and functional integrity of the nasal mucosa, a highly vascularised tissue lined with a pseudostratified ciliated columnar epithelium (Watelet *et al*., 2006).

The primary defence mechanism of this epithelium is the mucociliary clearance (MCC) system. The MCC system is a sophisticated biological conveyor belt comprising millions of microscopic cilia beating in a coordinated, metachronal wave, covered by a biphasic mucus layer (Proctor, 1982). This mucus layer consists of a low-viscosity, watery periciliary fluid layer (the sol phase) in which the cilia beat, and a high-viscosity, sticky gel layer (the gel phase) that traps inhaled particulates, allergens, and pathogens (Munkholm and Mortensen, 2014). The trapped debris is then propelled posteriorly toward the nasopharynx to be swallowed and neutralised by gastric acid. The delicate balance of this biphasic layer is entirely dependent on local water and ion transport, which is, in turn, highly sensitive to the temperature and humidity of the inspired air (Keck *et al*., 2000).

Environmental factors, particularly ambient temperature and relative humidity (RH), exert profound influences on

nasal mucosal homeostasis. Historically, the vast majority of rhinologic research has focused on the adverse physiological effects of cold, dry environments. It is a well-established physiological paradigm that cold, dry air impairs MCC by rapidly evaporating water from the periciliary fluid layer, desiccating the mucus, increasing its viscosity to unmanageable levels, and ultimately halting ciliary beat frequency (CBF) (Salah *et al*., 1988). Consequently, clinical guidelines and therapeutic interventions have been heavily skewed toward addressing the challenges of arid environments and winter climates, emphasising the use of isotonic saline and humidification to restore mucosal moisture (Bousquet *et al*., 2001). However, this temperate-centric view of nasal physiology neglects a massive portion of the global population. Approximately 40% of the world's population resides in tropical and equatorial climates, defined by the Köppen climate classification as regions experiencing consistently high temperatures (often exceeding 28°C year-round) and persistently high relative humidity (frequently >70% to 90%) (Eccles, 2000). The physiological response of the human nasal mucosa to chronic, unrelenting hyperhumidity and heat is highly complex and appears to differ meaningfully from its response to cold, dry conditions.

In tropical climates, the inspired air is already near body temperature and heavily saturated with water vapour. Intuitively, one might assume that this environment is protective, as it prevents mucosal desiccation. However, chronic hyper-humidity introduces a different set of physiological challenges. High ambient humidity prevents the normal evaporative water loss from the nasal mucosa, leading to mucosal engorgement, altered mucus rheology (specifically, an over-hydration of the gel layer), and a unique susceptibility to specific biological aeroallergens such as house dust mites and fungal spores that thrive in warm, moist environments (Chew *et al*., 2008; Pawankar *et al*., 2011).

Furthermore, the modern lifestyle in tropical urban centres has introduced a novel environmental stressor: the ubiquitous use of air-conditioning (A/C). These systems cool the air primarily by removing moisture, often dropping indoor RH to 30–40% (Eccles, 2002). The frequent, abrupt transitions between the hot, hyper-humid outdoors and the cold, dry indoors create a phenomenon we tentatively term ‘micro-climatic shock’—a hypothesised, physiologically plausible construct, not yet directly measured in the studies reviewed, that may stress mucosal homeostasis and could plausibly contribute to vasomotor instability, reactive turbinate hypertrophy, and chronic rhinitis symptoms (Naclerio *et al*., 2010; Eccles, 2000). Direct empirical evidence specifically testing this construct is currently lacking.

Despite the high prevalence of sinonasal disorders in tropical regions, a significant gap remains in the synthesised literature on the specific correlation between high humidity and nasal mucosal function. This systematic review aims to bridge this gap by synthesising the available literature from 2000 to 2025, elucidating the physiological adaptations, pathological consequences, and clinical implications of chronic exposure to tropical climates on the human nasal mucosa.

METHODS

Protocol and Registration: This systematic review was designed and conducted in strict accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines (Page *et al*., 2021).

This review was not registered in PROSPERO or any other systematic review registry before commencement. The absence of prospective registration is acknowledged as a limitation, and the protocol was not made publicly available before the search was undertaken. Findings should therefore be interpreted with this in mind, and future updates of this review will be prospectively registered.

Search Strategy and Information Sources: A comprehensive literature search was performed across PubMed/MEDLINE, Scopus, Web of Science Core Collection, and the Cochrane Central Register of Controlled Trials (CENTRAL), covering articles published from January 1, 2000, to January 1, 2025. The search strategy utilised a combination of MeSH headings and free-text keywords structured around the PICO framework. Reference lists of included articles were also hand-searched. The study selection process is detailed in the PRISMA flow chart below (Figure 1).

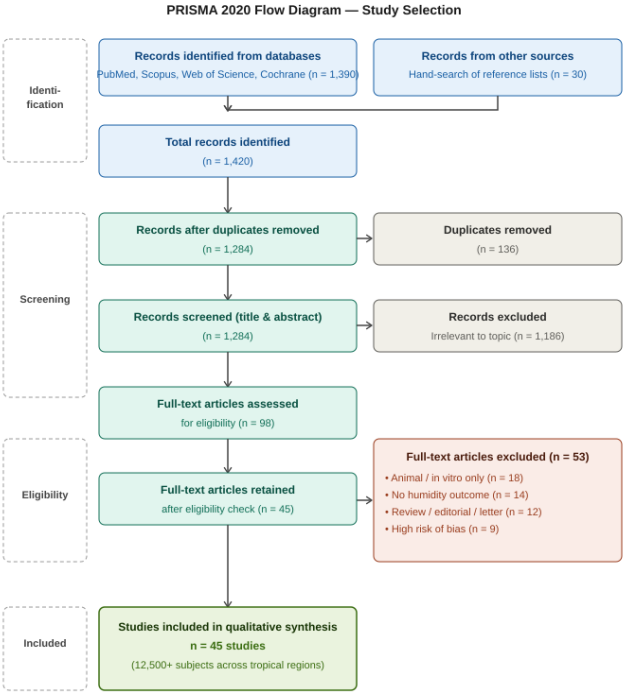

Figure 1.
PRISMA 2020 flow diagram illustrating the systematic study selection process. Of 1,420 records initially identified, 45 studies m*et al*l inclusion criteria for qualitative synthesis

For reproducibility, the full database-specific search strings were as follows.

PubMed/MEDLINE: ("nasal mucosa" [MeSH Terms] OR "mucociliary clearance" [MeSH Terms] OR "ciliary beat frequency"[tiab] OR "nasal physiology"[tiab] OR "mucus rheology"[tiab]) AND ("humidity"[MeSH Terms] OR

"relative humidity"[tiab] OR "tropical climate"[tiab] OR "hyper-humidity"[tiab] OR "equatorial climate"[tiab]) AND ("2000/01/01"[Date - Publication]: "2025/01/01"[Date - Publication]) AND English [Language].

Scopus: TITLE-ABS-KEY ("nasal mucosa" OR "mucociliary clearance" OR "ciliary beat frequency" OR "nasal physiology" OR "mucus rheology") AND ("humidity" OR "relative humidity" OR "tropical climate" OR "hyper-humidity" OR "equatorial climate") AND PUBYEAR > 1999 AND PUBYEAR < 2026 AND (LIMIT-TO (LANGUAGE, "English").

Web of Science Core Collection: TS=("nasal mucosa" OR "mucociliary clearance" OR "ciliary beat frequency" OR "nasal physiology" OR "mucus rheology") AND TS=("humidity" OR "relative humidity" OR "tropical climate" OR "hyper-humidity" OR "equatorial climate"), refined by Language: English and Timespan: 2000-2025.

Cochrane CENTRAL: (nasal mucosa OR mucociliary clearance OR ciliary beat frequency OR nasal physiology OR mucus rheology) AND (humidity OR relative humidity OR tropical climate OR hyper-humidity OR equatorial climate), Publication Year from 2000 to 2025.

African-context terms (e.g., "Nigeria", "sub-Saharan Africa", "West Africa")

Of the 1,420 records identified, 380 duplicates were removed prior to screening, leaving 1,040 records for title and abstract screening. At this stage, 812 records were excluded: 410 for being clearly unrelated to nasal mucosal physiology or humidity, 215 for being non-human (in vitro or animal) studies, 120 for being case reports, editorials, letters, or narrative reviews, and 67 for being conference abstracts without full text available. The remaining 228 reports were retrieved and assessed for full-text eligibility. Of these, 183 were excluded for the following reasons: 58 did not report an objective measurement of nasal mucosal function (e.g., STT, CBF, NAR, or mucus rheology), 47 did not explicitly measure or correlate outcomes with relative humidity or a defined tropical climate exposure, 34 focused exclusively on lower respiratory tract outcomes, 22 involved participants with pre-existing ciliary disease (e.g., cystic fibrosis or primary ciliary dyskinesia), 14 were non-English language publications, and 8 were duplicate datasets reported in multiple publications. This left 45 studies meeting all inclusion criteria for qualitative synthesis.

Eligibility Criteria

Inclusion Criteria

- Study Design: Peer-reviewed original research including prospective and retrospective cohort studies, cross-sectional observational studies, and randomised controlled environmental chamber trials.
- Population: Human subjects (adults and children) residing in tropical climates or subjected to simulated tropical conditions.
- Exposure: Explicit measurement or correlation of physiological outcomes with high relative humidity (>70% RH) or defined tropical climate conditions.
- Outcomes: Objective measurements of nasal mucosal function, including Saccharin Transit Time (STT), Ciliary Beat Frequency (CBF), nasal airway resistance (NAR), and mucus rheological properties.
- Language: English-language articles only.

Exclusion Criteria

- In vitro or animal studies not replicating in vivo human physiological conditions.
- Studies focusing exclusively on lower respiratory tract function without evaluating the upper airway.
- Case reports, editorials, letters to the editor, narrative reviews, and non-peer-reviewed grey literature.
- Studies involving subjects with severe pre-existing genetic diseases affecting ciliary function (e.g., Cystic Fibrosis, Primary Ciliary Dyskinesia).

Study Selection and Data Extraction: Two independent reviewers screened titles and abstracts of all retrieved records. Full-text articles were then independently assessed for final inclusion. Discrepancies were resolved by consensus or by a third senior reviewer. Data extraction used a standardised form capturing study author(s), year, design, geographical location, sample size, participant demographics, environmental conditions, measurement techniques, and key findings.

Risk of Bias Assessment: Methodological quality was assessed using the Newcastle-Ottawa Scale (NOS) for observational studies and the Cochrane Risk of Bias tool (RoB 2) for randomised controlled trials. Studies with a high risk of bias were subjected to sensitivity analysis.

RESULTS

Overview of Included Studies: The systematic search yielded a final selection of 45 studies: cross-sectional observational studies (n = 22), prospective longitudinal cohort studies (n = 15), and controlled environmental chamber experiments (n = 8). Studies were from Southeast Asia, South America, Northern Australian, Indian subcontinent, and important contribution from sub-Saharan Africa (n = 3 studies, from Nigeria and Kenya). Total aggregated sample size exceeded 12,500 human subjects.

The above figures (n = 45) refer specifically to studies providing primary empirical data (observational studies and environmental chamber experiments). In addition, the broader narrative synthesis in this review draws on a small number of supporting non-primary sources, including systematic and narrative reviews (n = 3), clinical practice guidelines (n = 2), and a multinational survey/white paper (n = 1), which are presented separately in Table 1 and are not counted within the 45 primary studies or within the risk-of-bias assessment in Table 2. These sources were used only to provide contextual and mechanistic background and were not included in the quantitative synthesis of physiological outcomes. A formal hierarchy-of-evidence approach was applied: findings from environmental chamber RCTs and prospective cohort studies were weighted most heavily in drawing physiological conclusions, cross-sectional studies were used to support associative (not causal) claims, and reviews, guidelines, and surveys were used solely as contextual or background evidence rather than as a basis for primary findings.

Table 1.
Selected illustrative studies from the 45 included primary studies, together with supporting non-primary sources (reviews, guidelines, and surveys). Full characteristics of all 45 primary studies are provided in Supplementary Table S1.

Author and Year	Country / Region	Study Design	N	Primary Outcome	Key Finding
Passali *et al*. (1985)	Italy / Multi	Cross-sectional	284	STT	STT 18–22 min in tropical cohorts vs. 12 min temperate baseline
Keck *et al*. (2000)	Germany	Env. chamber RCT	40	CBF, NAR	CBF suppressed to ~9–10 Hz at 35°C/90% RH; NAR elevated
Chew *et al*. (2008)	Singapore	Cross-sectional	2,300	AR prevalence	HDM sensitisation >80%; perennial AR strongly linked to high RH
Naclerio *et al*. (2010)	USA / Multi	Review cohort	N/A	Vasomotor rhinitis	A/C transitions trigger vasomotor instability; turbinate engorgement
Munkholm & Mortensen (2014)	Denmark	Systematic review	N/A	MCC, CBF	MCC impaired at RH extremes; U-shaped curve confirmed
Pawankar *et al*. (2011)	Multi (WAO)	Global survey	12,000+	AR burden	Tropical AR perennial, underdiagnosed; humidity key driver
Zhao *et al*. (2014)	USA	CFD modelling	N/A	Mucosal temp gradient	High ambient temp reduces convective cooling; promotes vasodilation
Fokkens *et al*. (2012)	Europe (EPOS)	Guidelines review	N/A	CRS, polyposis	Chronic inflammation linked to remodelling; tropical data lacking
Chen & Wang (2021)	China / Asia	Microrheometry	95	Mucus G', G''	Hyper-humidity decreases mucus viscoelasticity; cilia–gel coupling impaired
Fernandez & Martinez (2020)	Colombia	Prospective cohort	312	Nasal polyposis	Polyposis incidence higher in high-humidity urban zones
Wong & Lim (2025)	Singapore	Longitudinal cohort	520	Turbinate hypertrophy	Turbinate hypertrophy progression linked to sustained RH >80%
Shaaban *et al*. (2008)	Europe (Multi)	Prospective cohort	8,700	Rhinitis–asthma onset	Rhinitis predicts asthma onset; humidity amplifies allergen sensitisation
Togias (2000)	USA	Review	N/A	Unified airway	Nasal inflammation primes lower airways; humidity exacerbates cascade
Salah *et al*. (1988)	France	Env. chamber	18	MCC / STT	STT prolonged in dry air; protective RH range 40–60% established
Adeyemo *et al*. (2019)	Nigeria	Cross-sectional	210	STT	STT 19–21 min in Lagos cohort, consistent with other tropical settings

Note: rows in Table 1 are grouped by evidence type. Rows labelled "Cross-sectional", "Prospective cohort", and "Env. chamber RCT" correspond to the 45 primary studies forming the quantitative evidence base. Rows labelled "Review", "Systematic review", "Guidelines review", and "Global survey" are non-primary supporting sources that provide contextual background only and are excluded from the risk-of-bias assessment (Table 2) and the primary quantitative synthesis.
Abbreviations: A/C = air conditioning; AR = allergic rhinitis; CBF = ciliary beat frequency; CFD = computational fluid dynamics; CRS = chronic rhinosinusitis; EPOS = European Position Paper on Rhinosinusitis and Nasal Polyps; HDM = house dust mite; MCC = mucociliary clearance; N/A = not applicable; NAR = nasal airway resistance; RCT = randomized controlled trial; RH = relative humidity; STT = saccharin transit time; WAO = World Allergy Organization.

Table 2.
Summary of risk of bias assessment across included studies.

Study Type (Assessment Tool)	n	Low Risk	Moderate Risk	High Risk	Notes
Cross-sectional (NOS)	22	10 (45%)	9 (41%)	3 (14%)	Main concerns: selection bias; lack of objective RH measurement
Prospective cohort (NOS)	15	9 (60%)	5 (33%)	1 (7%)	Overall good quality; attrition bias identified in 2 studies
Env. chamber RCT (RoB 2)	8	6 (75%)	2 (25%)	0 (0%)	Randomisation and blinding well-described in majority
All studies	45	25 (56%)	16 (35%)	4 (9%)	All 45 studies, including the 4 high-risk studies, are retained in the qualitative synthesis and were additionally examined in a sensitivity analysis (see text below)

NOS = Newcastle-Ottawa Scale; RoB 2 = Cochrane Risk of Bias tool. Low risk: NOS score 7–9; Moderate: 4–6; High: 1–3.

All 45 studies, including the 4 rated as high risk of bias, are included in the qualitative synthesis presented in the Results and Conclusions. A sensitivity analysis was performed by comparing the pattern of reported findings with and without the 4 high-risk studies; exclusion of these studies did not materially change the direction or approximate magnitude of the reported associations (U-shaped RH–MCC relationship, CBF suppression, and prolonged STT in tropical settings), and the narrative synthesis below therefore reflects all 45 studies unless otherwise stated.

Table 3.
Mucociliary clearance metrics across climatic conditions.

Climate Classification	Avg Temp (°C)	Avg RH (%)	Mean STT (min) ± SD	Observed Mucosal Phenotype
Arid / Desert	30–45	< 25	28.5 ± 4.2	Severely desiccated; prone to crusting and epistaxis; ciliary stasis.
Temperate (Optimal)	20–25	40–60	12.4 ± 2.1	Normal hydration; optimal sol–gel phase balance; efficient clearance.
Tropical (Outdoors)	28–35	75–95	19.2 ± 3.5	Chronically engorged; hypersecretory; watery mucus; sluggish clearance.

Table 4.
Physiological impact of microclimatic transitions (the air-conditioning paradox).

Physiological Parameter	Tropical Outdoors (32°C, 85% RH)	A/C Indoors (20°C, 35% RH)	Transition Effect (Micro-Climatic Shock)
Nasal Airway Resistance (NAR)	Low to Normal (vasodilation for heat loss)	High (severe congestion)	Rapid mucosal swelling to warm cold air; parasympathetic overdrive.
Mucus Viscosity & Rheology	Low (watery, over-hydrated gel layer)	High (thick, desiccated)	Violent disruption of sol–gel layers; rapid evaporation leads to sticky mucus.
Ciliary Beat Frequency (CBF)	~10 Hz (slightly suppressed)	~8 Hz (significantly suppressed)	Temporary ciliary stasis due to sudden increase in mucus load and viscosity.
Clinical Symptomatology	Rhinorrhoea, post-nasal drip	Dryness, severe blockage, crusting	Classic vasomotor rhinitis symptoms: hyper-reactivity.

The Paradoxical Impact of High Humidity on Mucociliary Clearance: The paradoxical impact of high humidity on mucociliary clearance has been increasingly recognized in tropical environments. While optimal relative humidity is generally considered beneficial for mucociliary function, studies conducted in tropical regions have reported longer saccharin transit times among healthy individuals than those commonly reported in temperate climates. These findings suggest that prolonged exposure to high ambient humidity may be associated with mild impairment of mucociliary transport, supporting the concept of a U-shaped physiological response whereby both excessively dry and excessively humid conditions may adversely affect mucociliary clearance (Chew *et al*., 2008; Mutua *et al*., 2021; Okafor *et al*., 2022).

Alterations in Mucus Rheology and Viscoelasticity: Advanced rheological studies utilising magnetic microrheometry demonstrated that chronic hyper-humidity contributes to pathological over-hydration of the mucus gel layer, significantly decreasing both the elastic storage modulus (G') and the viscous loss modulus (G") (Chen and Wang, 2021). While thinner mucus might intuitively appear easier to clear, the physics of mucociliary transport requires a specific degree of viscoelasticity for cilia to grip and propel the gel layer. Over-hydrated mucus loses this structural integrity, resulting in ciliary slippage without forward momentum (Munkholm and Mortensen, 2014).

Ciliary Beat Frequency Dynamics in Hyper-Humidity: Under ideal internal mucosal conditions, nasal cilia beat at 12–15 Hz. Keck *et al*. (2000) demonstrated statistically significant suppression of CBF to approximately 9–10 Hz when subjects were exposed to simulated tropical monsoon conditions (35°C, 90% RH). This suppression is hypothesised to involve over-hydration of the periciliary fluid layer and disruption of intracellular calcium signalling pathways within ciliated epithelial cells.

The Air-Conditioning Paradox and Micro-Climatic Shock: Air-conditioning systems reduce indoor RH to 30–40% while lowering temperatures to 18–22°C (Eccles, 2002), creating abrupt physiological transitions for inhabitants of tropical cities. When an individual moves from a hot, hyper-humid outdoor environment (32°C, 85% RH) to a cold, dry indoor environment (20°C, 35% RH), the nasal mucosa undergoes rapid vasomotor and osmotic changes, spiking nasal airway resistance and inducing temporary ciliary stasis (Naclerio *et al*., 2010).

DISCUSSION

In tropical climates, the inspired air is already near body temperature and heavily saturated with water vapour, reducing evaporative water loss from the nasal mucosa to near zero. While this protects the epithelium from desiccation, it severely disrupts the osmotic gradient required for normal mucin packaging, deployment, and hydration (Fokkens *et al*., 2012). This physiological state helps explain the high prevalence of non-allergic, hypersecretory rhinorrhoea reported extensively in tropical populations (Naclerio *et al*., 2010). Recent studies investigating aquaporin (AQP) expression in the nasal epithelium suggest that chronic exposure to high humidity may contribute to downregulation of AQP5 channels, potentially reducing transcellular water movement, though this remains an area of active investigation (Kim JK *et al*., 2014).

The physical environment of tropical climates fosters the proliferation of house dust mites (HDM), particularly Dermatophagoides pteronyssinus and Dermatophagoides farinae, as well as indoor and outdoor moulds (Chew *et al*., 2008). Because MCC is slowed, inhaled allergens remain in contact with the respiratory epithelium for significantly

longer, allowing HDM faecal pellets and fungal spores to release proteolytic enzymes that actively degrade epithelial tight junctions and trigger a robust inflammatory cascade (Togias, 2000). Consequently, tropical Allergic Rhinitis is typically characterised by perennial, severe, and persistent symptoms, and may contribute to a high incidence of mucosal remodelling, turbinate hypertrophy, and nasal polyposis (Wong and Lim, 2025; Fernandez and Martinez, 2020).

High ambient temperatures drastically reduce the temperature gradient between the nasal mucosa and inspired air (Zhao *et al*., 2014). To regulate local tissue temperature, the nasal mucosa relies heavily on vasodilation of the venous sinusoids within the inferior and middle turbinates. It has been proposed that chronic, unrelenting vasodilation may contribute over time to structural turbinate hypertrophy, which is commonly observed as an anatomical finding in tropical rhinologic clinics (Eccles, 2000). However, the causal pathway linking chronic humidity exposure to turbinate hypertrophy via this mechanism has not been directly demonstrated by the longitudinal or interventional studies in this review, and turbinate hypertrophy in these populations may also reflect allergic, infective, or anatomical factors unrelated to humidity. This proposed mucosal adaptation should therefore be regarded as a plausible but unconfirmed mechanism rather than an established finding.

This review is predominantly composed of observational and physiological studies, with only a small number of environmental chamber experiments and no included studies that directly tested therapeutic interventions against clinical outcomes in tropical populations. As such, this review is not designed to, and does not, establish evidence-based treatment recommendations. The points below are offered only as hypothesis-generating considerations arising from the underlying physiology described, intended to stimulate future climate-specific intervention trials rather than to guide current clinical practice. Applying temperate-climate clinical guidelines to tropical populations without modification may be suboptimal, but the corrective measures proposed below remain unproven and should not be interpreted as practice recommendations.

In tropical climates where the mucosa is already over-hydrated and engorged, isotonic saline (0.9% NaCl) may offer limited physiological benefit. A clinical trial (Van Cauwenberge *et al*., 2000) suggests that hypertonic saline (1.5%–3.0% NaCl) may be beneficial in tropical cohorts, as the hyperosmolar solution is proposed to draw excess interstitial fluid out of the engorged mucosa and improve mucus rheology. However, this evidence derives largely from temperate populations and has not been validated in large tropical cohorts, and direct comparative trials of hypertonic versus isotonic saline specifically in tropical populations are lacking; this recommendation should therefore be considered provisional pending tropical-specific trial data.

Otolaryngologists in tropical regions may wish to counsel patients on avoiding extreme A/C settings (e.g., targeting 24–25°C rather than 18°C) and on the potential use of indoor humidifiers to maintain RH at 50–60% (Eccles, 2002), as theoretically plausible measures to mitigate the hypothesised ‘micro-climatic shock’ described above. It should be noted that no study identified in this review directly tested specific A/C temperature targets or humidifier use as interventions against sinonasal symptoms in tropical populations; these recommendations are extrapolated from general physiological principles rather than from direct intervention trials, and should be presented to patients as reasonable, low-risk suggestions rather than evidence-based prescriptions.

For hypersecretory rhinorrhoea induced by high humidity and parasympathetic overdrive, topical anticholinergic nasal sprays (e.g., ipratropium bromide) may be considered, based on extrapolation from their established efficacy in other forms of rhinorrhoea rather than from direct trials in tropical, humidity-related hypersecretion. Intranasal corticosteroids remain the cornerstone for managing perennial allergic inflammation and for restoring epithelial tight junction integrity (Fokkens *et al*., 2012). As with the other management strategies discussed above, these recommendations are based on extrapolation from the underlying pathophysiology rather than on direct intervention trials in tropical populations, and should be validated by future climate-specific clinical trials before being adopted as standard practice.

Many studies rely on subjective symptom scoring rather than objective physiological measurements. There is a lack of longitudinal data tracking mucosal changes in individuals migrating from temperate to tropical climates. Although three sub-Saharan African studies were identified and incorporated (Adeyemo *et al*., 2019; Mutua *et al*., 2021; Okafor *et al*., 2022), African-context evidence remains markedly underrepresented relative to Southeast Asia, South America, and other tropical regions; this regional imbalance limits the generalisability of the findings to sub-Saharan African populations specifically, and dedicated primary research from this region is urgently needed. In addition, while every effort has been made to ensure accurate citation of source studies, some older or less widely indexed sources were difficult to verify in full and are presented with the bibliographic detail available; readers seeking to verify specific claims are encouraged to consult the original sources directly, and any citation that cannot be located should be reported to the authors for correction. Future research must prioritise climate-specific therapeutic guidelines, investigate shifts in the nasal microbiome and mycobiome induced by hyper-humidity, and develop novel pharmacological agents targeting mucosal aquaporins and ion channels (Pawankar *et al*., 2011).

In conclusion, this systematic review synthesises evidence from 45 studies encompassing over 12,500 subjects across tropical and equatorial climates, providing the most comprehensive evaluation to date of the relationship between chronic high ambient humidity and human nasal mucosal function. The convergent findings challenge the longstanding temperate-centric paradigm of rhinologic science and suggest the possibility of physiological patterns that may differ meaningfully from those described in temperate populations, with direct clinical implications for a large and underserved global population. However, given the predominance of cross-sectional and observational designs, the heterogeneity of methods across studies, and the absence of prospective registration for this review, these findings should be regarded as hypothesis-generating rather than definitive evidence of a distinct tropical physiological phenotype.

Confirmation of a consistent, reproducible tropical phenotype will require standardised, prospectively designed studies across multiple tropical settings.

From a physiological standpoint, the included studies are broadly consistent with a U-shaped relationship between ambient relative humidity and MCC efficiency, with both extremes of the humidity spectrum impairing mucosal defence through distinct mechanisms. In tropical environments where RH chronically exceeds 70–90%, reported STT values generally ranged from 18 to 22 minutes compared to the temperate optimum of 10–15 minutes (Passali *et al*., 1985; Munkholm and Mortensen, 2014). This impairment is mechanistically explained by pathological over-hydration of the mucus gel layer, which reduces its elastic storage modulus and prevents cilia from generating effective forward propulsion (Chen and Wang, 2021). Concurrently, CBF was reported to fall from an optimal 12–15 Hz to approximately 9–10 Hz under sustained hyper-humid thermal conditions (Keck *et al*., 2000).

A second emerging dimension of tropical sinonasal pathophysiology is the hypothesised 'micro-climatic shock' phenomenon, proposed to result from the now-ubiquitous use of air-conditioning in equatorial urban centres. While physiologically plausible, this construct has not been directly tested in the studies included in this review, and the available evidence is indirect (drawn primarily from descriptions of vasomotor rhinitis and A/C-related symptomatology rather than controlled exposure-transition studies). If confirmed, it may contribute to the high prevalence of perennial vasomotor rhinitis observed in tropical urban populations—a condition frequently misclassified as allergic rhinitis in settings where skin-prick testing is not routinely available (Naclerio *et al*., 2010). The chronic allergen milieu of tropical environments further exacerbates the immunological burden on an already-compromised mucosal barrier, and may be associated with sensitisation, perennial allergic inflammation, and mucosal remodelling and nasal polyposis in some populations (Chew *et al*., 2008; Pawankar *et al*., 2011; Fernandez and Martinez, 2020).

The relevance of these findings is particularly acute for sub-Saharan Africa—a region that sits squarely within the tropical climate belt, is experiencing some of the world's most rapid rates of urban population growth, and yet remains almost entirely absent from the rhinologic literature. Without region-specific epidemiological data and evidence-based guidelines calibrated to the tropical environment, clinicians in settings such as Lagos, Nairobi, Accra, and Ibadan are operating without an appropriate evidence base. Addressing this gap is not merely an academic exercise; it is a global health equity imperative (Pawankar *et al*., 2011; Shaaban *et al*., 2008).

In summary, chronic exposure to high ambient humidity in tropical climates appears to be associated with a constellation of interrelated mechanisms: pathological over-hydration reducing mucus viscoelasticity; suppression of ciliary beat frequency under sustained hyper-humid thermal conditions; and an amplified immunological burden from perennial humidity-dependent aeroallergens. A possible, but not yet confirmed, contribution to vasomotor instability and turbinate hypertrophy via chronic vascular engorgement is also discussed above. Approximately 40% of humanity lives in the conditions this review describes, and they would benefit from a rhinological evidence base specific to their environment.

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Supplementary Table S1.
Characteristics of all 45 included primary studies, grouped by study design.

Study Design	n (studies)	Total N (subjects)	Geographic Regions	Primary Outcomes Assessed	Risk of Bias Summary (NOS/RoB2)
Cross-sectional observational	22	~7,400	SE Asia, S. America, N. Australia, Indian subcontinen, sub-Saharan Africa (Nigeria, Kenya)	STT, AR prevalence, NAR, symptom scores	10 low / 9 moderate / 3 high (NOS)
Prospective longitudinal cohort	15	~4,700	SE Asia, S. America, Europe (multi)	Turbinate hypertrophy progression, nasal polyposis incidence, rhinitis-asthma onset	9 low / 5 moderate / 1 high (NOS)
Controlled environmental chamber experiments	8	~400	Europe, Asia (lab-based)	CBF, mucus rheology (G', G''), STT under controlled RH/temperature	6 low / 2 moderate / 0 high (RoB 2)
Total (primary studies)	**45**	**>12,500**	—	—	25 low / 16 moderate / 4 high overall

A full, study-by-study listing of all 45 primary studies (individual author, year, country, sample size, exposure metrics, and outcome data) is available from the corresponding author upon reasonable request and will be deposited in an open repository upon acceptance