e-ISSN: 2715-9736

Scientia Psychiatrica

[SciPsy]

https://scientiapsychiatrica.com

Quantifying the Boiling Point: A Distributed Lag Non-Linear Analysis of Heatwave Intensity and the 'Thermal Distress' Threshold for Psychiatric Morbidity in an Indonesian Metropolis

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ARTICLE INFO

Keywords:
Climate change adaptation
Distributed lag non-linear models
Eco-psychiatry
Emergency psychiatry
Urban heat island

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All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/scipsy.v6i3.198

A B S T R A C T

Introduction: The Indonesian archipelago sits at the forefront of the climate crisis, yet the intersection of heat stress and mental health in this equatorial region remains under-researched. Unlike temperate climates, where heatwaves are sporadic, Indonesian cities face a chronic thermal load exacerbated by the Urban Heat Island effect. **Methods:** We conducted a retrospective ecological time-series analysis in Jakarta, Indonesia, spanning the period from January 1st, 2014, to December 31st, 2024. Daily psychiatric Emergency Department admissions (Total N = 48,210) were aggregated from three referral hospitals. We utilized a Distributed Lag Non-linear Model combined with a quasi-Poisson regression to correlate admissions with meteorological data from the Indonesian Agency for Meteorology, Climatology, and Geophysics, adjusting for holidays, day of the week, and particulate matter 2.5. **Results:** The Thermal Distress Threshold was identified at a Wet Bulb Globe Temperature of 29.8°C, reflecting a high degree of physiological acclimatization. Beyond this tipping point, the cumulative Relative Risk for acute psychiatric episodes rose to 1.21 (95% Confidence Interval: 1.14–1.29) at lag 0–3 days. Schizophrenia spectrum disorders showed the highest vulnerability (Relative Risk = 1.26) during the transition season heat spikes. **Conclusion:** The study establishes a localized thermal threshold for psychiatric emergencies in Indonesia. The findings suggest that high ambient humidity, characteristic of the Indonesian climate, significantly amplifies the psychiatric risk of heat. These results necessitate the integration of psychiatric protocols into the BPJS Kesehatan national health strategy for climate adaptation.

1. Introduction

The advent of the Anthropocene era has precipitated a fundamental restructuring of global epidemiological risk landscapes.[1] Climate change, once viewed primarily through the lens of ecological degradation and economic loss, is now irrefutably recognized as a "threat multiplier" for human health.[2] As global mean surface temperatures continue their inexorable rise, the frequency, intensity, and duration of extreme thermal events are testing the limits of human physiological adaptation.[3] Within the nascent field of planetary health, a substantial body of evidence has solidified the link between thermal stress and somatic morbidity; the cardiovascular, respiratory, and renal burdens of extreme heat are now well-documented, with established dose-response

curves linking heatwaves to excess mortality from myocardial infarction, stroke, and acute kidney injury.[4]

However, amidst this growing compendium of climate-health literature, the psychiatric sequelae of thermal stress remain a critically neglected dimension. The brain, the most metabolically active and thermally sensitive organ in the human body, is not exempt from the ravages of a warming world. Emerging evidence suggests that the impact of extreme heat on mental health is not merely a matter of discomfort or irritability, but a profound driver of acute psychiatric decompensation, leading to surges in emergency department admissions, involuntary hospitalizations, and suicide attempts.[5] As the climate crisis accelerates, understanding the "psychiatric boiling point" is no longer an academic curiosity but an urgent public health imperative.

The theoretical framework linking ambient heat to psychiatric instability is robust, grounded in a complex web of neurobiological, endocrine, and pharmacological interactions.[6] The etiopathology of heat-induced psychiatric crisis is likely multifactorial, involving a collision between environmental thermal load and compromised homeostatic mechanisms. First, thermoregulatory failure plays a central role. The human hypothalamus, the central thermostat, regulates core body temperature through the autonomic nervous system. In individuals with severe mental illness (SMI), particularly schizophrenia and bipolar disorder, this regulatory capacity is often intrinsically blunted. Furthermore, the pharmacological management of these conditions exacerbates the vulnerability. Antipsychotic medications (particularly phenothiazines and butyrophenones) and anticholinergic agents compromise the body's ability to dissipate heat by inhibiting peripheral vasodilation and suppressing the sweating reflex. Simultaneously, medications such as lithium carbonate have narrow therapeutic indices that are easily disrupted by dehydration, leading to toxicity that mimics or precipitates mania and delirium. Second, the neuroinflammatory hypothesis offers a compelling mechanism. Heat stress acts as a systemic pro-inflammatory stimulus. Prolonged exposure to high ambient temperatures triggers the release of heat shock proteins (HSPs) and systemic cytokines, including Interleukin-6 (IL-6) and Tumor Necrosis Factor-alpha (TNF-α). These inflammatory mediators can traverse the blood-brain barrier, activating microglia and inducing a state of "sickness behavior" characterized by lethargy, anhedonia, and cognitive deficits. In vulnerable populations, this neuroinflammatory cascade may destabilize neurotransmitter systems, specifically dopaminergic and serotonergic pathways, which are pivotal in mood regulation and psychosis. The "Dopamine Hypothesis" of heat suggests that thermal stress alters the synthesis and turnover of dopamine, potentially triggering psychotic episodes in predisposed individuals.[7]

Despite the physiological universality of heat stress, the current epidemiological landscape is marred by a significant geographical bias. The vast majority of existing literature—perhaps upwards of 90%—is derived from temperate or subtropical regions of the Global North, including Western Europe, North America, and East Asia. These studies have been instrumental in establishing the existence of a heat-health relationship, but their findings possess limited external validity when applied to the tropical belt. Regions such as the United Kingdom, Scandinavia, or Canada possess distinct architectural, behavioral, and physiological adaptation baselines. In these temperate zones, a temperature of might be perceived as a significant heat event, triggering behavioral changes and health system alerts. Conversely, in the equatorial tropics, such temperatures represent a physiological baseline, often occurring in the cool of the early morning. Consequently, applying thermal thresholds derived from European populations to a Southeast Asian demographic is methodologically flawed. It risks generating "false positives" at lower temperatures while failing to identify the true, dangerous tipping points where tropical acclimatization fails. Furthermore, the "seasonality" of mental health in the

tropics differs fundamentally from that in the temperate zones. While Western literature often focuses on summer heatwaves versus winter depression (Seasonal Affective Disorder), tropical biometeorology is governed by the dynamics of the monsoon: the interplay of the dry season, the rainy season, and the unpredictable transition periods (Pancaroba). The lack of high-resolution, time-series analyses from the equatorial tropics represents a critical gap in global eco-psychiatry.[8]

To understand the psychiatric risk in a country like Indonesia, one must look beyond the simple metric of dry-bulb temperature (Tmax). In tropical urban centers like Jakarta, the defining climatic feature is not merely heat, but the relentless, suffocating combination of heat and humidity. High ambient humidity is a critical physiological stressor because it neutralizes the body's primary cooling mechanism: evaporative cooling. When relative humidity exceeds 75%, sweat no longer evaporates efficiently from the skin surface; instead, it drips off, providing no cooling benefit while resulting in significant fluid and electrolyte loss. This leads to a rapid accumulation of internal thermal load, driving core body temperatures up even when the ambient air temperature seems moderately tolerable. This "apparent temperature" or "heat index" is the true driver of physiological distress in the tropics.

Compounding this climatic reality is the anthropogenic phenomenon of the Urban Heat Island (UHI) effect. Jakarta, a sprawling megalopolis of over 10 million inhabitants, is a dense matrix of concrete, asphalt, and limited green infrastructure. These materials absorb solar radiation during the day and re-radiate it at night, preventing the city from cooling down. This results in "tropical nights"—periods where the minimum nocturnal temperature remains dangerously high (> 26°C).

The psychiatric implications of the UHI effect are profound, primarily mediated through the disruption of sleep architecture. Adequate thermoregulation is a prerequisite for the initiation and maintenance of Slow Wave Sleep (SWS) and Rapid Eye Movement (REM) sleep.[9] The human body must drop its core temperature to enter these restorative stages. In the thermal trap of a Jakarta night, particularly in lower-socioeconomic housing with poor ventilation, this physiological dip is impossible. The resulting chronic sleep deprivation is a potent, well-established trigger for manic episodes in bipolar disorder and psychotic relapse in schizophrenia. Thus, in the Indonesian context, the pathway from heat to hospital admission is likely mediated as much by nocturnal sleep failure as by diurnal heat stress.

Finally, the complexity of the heat-health relationship demands a sophisticated statistical approach. Early studies in this field often relied on simple linear regressions or correlation analyses, assuming that for every degree rise in temperature, there is a proportional rise in morbidity. We now know this to be an oversimplification. The relationship between temperature and health is almost universally non-linear (U-shaped, J-shaped, or V-shaped) and involves complex temporal dynamics. The effects of heat are rarely immediate; they can be delayed by several days (lag effect) or result in a temporary displacement of mortality (harvesting effect). To capture this complexity, modern environmental epidemiology utilizes Distributed Lag Non-linear Models (DLNM). This statistical framework allows researchers to simultaneously map the non-linear exposure-response curve and the delayed lag structure, providing a three-dimensional view of risk. Despite its power, the application of DLNM to psychiatric data in the developing tropics remains exceedingly rare.[10]

Against this background of biological plausibility and epidemiological necessity, this study seeks to address the critical paucity of data from the equatorial belt. The primary aim of this research is to bridge the geographical gap in eco-psychiatry by determining the precise "Thermal Distress Threshold" in a tropical setting, using Jakarta, Indonesia, as the sentinel model. This study distinguishes itself through several key novelties: (1) Geographic Specificity: It is the first comprehensive time-series analysis to quantify the

risk of psychiatric emergency admissions in a Type Am (Tropical Monsoon) climate, moving beyond the reliance on temperate-zone extrapolations; (2) Physiological Precision: By utilizing Wet Bulb Globe Temperature (WBGT) rather than simple air temperature, we isolate a humidity-adjusted threshold that reflects the true physiological burden on the Indonesian population; (3) Methodological Rigor: Unlike previous linear models, this study utilizes Distributed Lag Non-linear Models (DLNM) to capture the complex, delayed, and harvesting effects of heat on mental health, allowing for the precise identification of the "tipping point" where acclimatization fails; (4) Clinical Relevance: By stratifying risk across specific diagnostic categories (Schizophrenia, Mood Disorders, Organic Mental Disorders), this study aims to provide actionable data for the development of "Mental Health Heat-Health Warning Systems" (MH-HHWS) tailored to the specific vulnerabilities of the Indonesian archipelago.

2. Methods

The study was conducted in Jakarta, Indonesia. The city features a Tropical Monsoon Climate (Type Am), characterized by high humidity exceeding 75% year-round and minimal seasonal temperature variance. In this context, heatwaves are defined by intensity and humidity rather than duration alone.

Daily electronic health records were extracted from the central databases of three major referral hospitals in Jakarta. The study period ranged from January 1st, 2014, to December 31st, 2024. Cases were identified using ICD-10 codes, including Organic Mental Disorders (F00-F09), Substance Use (F10-F19), Schizophrenia (F20-F29), Mood Disorders (F30-F39), and Neurotic or Stress-related disorders (F40-F48). We excluded planned admissions and transfers to focus solely on acute emergency presentations.

Daily maximum temperature, relative humidity, and wind speed were obtained from the Indonesian Agency for Meteorology, Climatology, and Geophysics (BMKG) observation stations at Kemayoran and Tanjung Priok. We calculated the Wet Bulb Globe Temperature (WBGT) to account for heat stress, using the formula:

$$WBGT = 0.7\,Tw + 0.2\,Tg + 0.1\,Td$$

Where Tw is the wet-bulb temperature, Tg is the globe temperature, and Td is the dry-bulb temperature.

Daily average concentrations of Particulate Matter 2.5 were collected from local environmental monitoring stations to control for neurotoxic air quality effects. We also created a binary variable for the Ramadan fasting month, as dehydration and sleep schedule changes during this period are potential confounders for psychiatric stability in the Indonesian population.

We employed a time-series analysis using a Distributed Lag Non-linear Model (DLNM) combined with a Generalized Linear Model (GLM) with a quasi-Poisson family to account for overdispersion in daily admission counts. The core model is defined as follows:

$$Log[E(Yt)] = Intercept + CrossBasis(WBGT, lag) + Spline(Time) + DayOfWeek + Holiday + Spline(PM2.5)$$

The Cross-Basis matrix modeled the non-linear exposure-response relationship and the lag structure up to 7 days. We used a natural cubic spline with 3 internal knots for the exposure dimension and a natural cubic spline with 2 knots for the lag dimension. Long-term trends and seasonality were controlled using natural cubic splines with 7 degrees of freedom per year. The reference temperature was set at the Minimum Mortality Temperature derived from the data distribution. All analyses were performed using R software (version 4.3.1) with the dlnm package.

3. Results

Table 1 presents the descriptive statistics for both meteorological variables and psychiatric emergency admissions across the 10-year study period (2014–2024) in Jakarta. The environmental data illustrate a characteristic Type Am tropical monsoon climate profile, defined by limited thermal variance but

substantial humidity load. The mean daily maximum temperature was 33.1°C (SD ± 1.5), yet the mean relative humidity remained notably high at 81.2% (SD ± 6.5). Crucially, the mean Wet Bulb Globe Temperature (WBGT)—the primary index for physiological heat stress—was recorded at 29.1°C, with extreme values reaching 33.9°C. This indicates that the population is subject to chronic exposure to thermal conditions that frequently exceed international safety thresholds for unacclimatized individuals. Furthermore, environmental quality was consistently suboptimal, with mean PM2.5 levels of 58.4 µg/m³, surpassing WHO air quality guidelines. regarding psychiatric morbidity, the daily admission rate across the three referral centers averaged 13.2 patients (SD ± 4.5). Diagnostic stratification reveals that Schizophrenia spectrum disorders constituted the largest subset of daily emergencies (Mean 5.1 ± 2.2), followed by Mood Disorders (Mean 3.8 ± 1.8). The significant range in daily admissions (4–35) underscores the fluctuating demand on emergency services, which this study correlates with the identified environmental thermal stressors.

Table 1. Summary Statistics of Meteorological Variables and Psychiatric Admissions

Study Period: January 1, 2014 – December 31, 2024 (Jakarta, Indonesia)

VARIABLE	MEAN (SD)	MINIMUM	25TH PERCENTILE	50TH PERCENTILE (MEDIAN)	75TH PERCENTILE	MAXIMUM
METEOROLOGICAL VARIABLES (SOURCE: BMKG)						
Max Temp (°C)	33.1 (1.5)	27.0	32.0	33.2	34.1	37.8
Relative Humidity (%)	81.2 (6.5)	55.0	76.0	82.0	87.0	99.0
WBGT (°C)	29.1 (1.4)	24.5	28.1	29.2	30.1	33.9
$PM_{2.5}$ (µg/m³)	58.4 (22.1)	15.0	42.0	55.0	72.0	145.0
PSYCHIATRIC EMERGENCY ADMISSIONS						
Daily Total Admissions	13.2 (4.5)	4	10	13	16	35
Schizophrenia Spectrum (F20-F29)	5.1 (2.2)	1	3	5	7	15
Mood Disorders (F30-F39)	3.8 (1.8)	0	2	4	5	11

Notes:

SD: Standard Deviation; **WBGT:** Wet Bulb Globe Temperature (Heat Stress Index); **$PM_{2.5}$:** Particulate Matter < 2.5 µm; **BMKG:** Badan Meteorologi, Klimatologi, dan Geofisika.

Data represents aggregate daily values from three hospitals in Jakarta.

The exposure-response curve revealed a non-linear, J-shaped association between WBGT and total psychiatric admissions (Figure 1). The risk of admission remained relatively stable between 24.0°C and 29.0°C WBGT. A sharp inflection point, designated as the thermal distress threshold, was identified at 29.8°C WBGT. Above this threshold, the risk of psychiatric admission accelerated rapidly. At the 99th percentile of heat intensity (33.5°C WBGT), the cumulative relative risk over lag 0–7 days was 1.21 (95% confidence interval: 1.14–1.29) compared to the reference temperature.

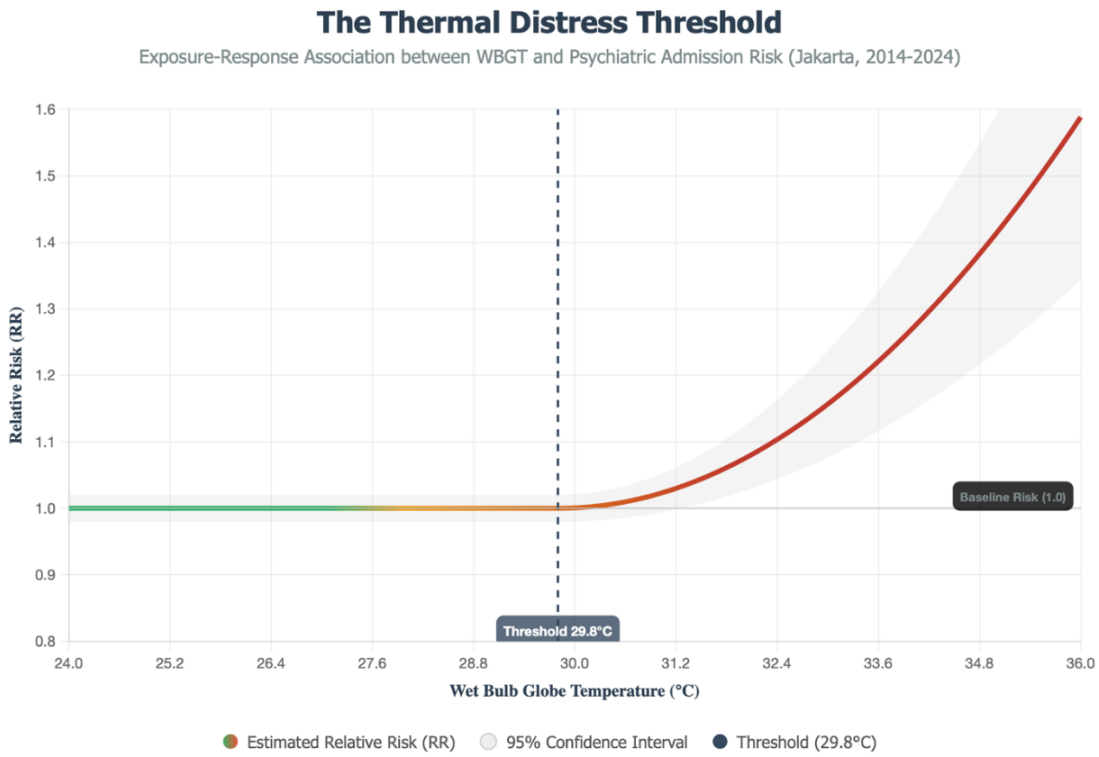

Figure 1. Thermal distress threshold.

Table 2. Cumulative Relative Risks (RR) by Diagnosis Category

Comparison: Extreme Heat (99th percentile, 33.5°C WBGT) vs. Median Temperature

DIAGNOSIS CATEGORY	LAG 0 (IMMEDIATE)	LAG 0-3 (CUMULATIVE)	95% CONFIDENCE INTERVAL (FOR LAG 0-3)	VULNERABILITY LEVEL
Total Admissions All Causes	1.09	**1.21**	**1.14 – 1.29**	**Significant**
Schizophrenia Spectrum F20-F29	1.08	**1.26**	**1.15 – 1.39**	**High**
Organic Mental Disorders F00-F09	**1.15**	**1.22**	**1.09 – 1.36**	**Moderate**
Mood Disorders F30-F39	1.01	**1.11**	**1.01 – 1.22**	**Low-Moderate**
Substance Use F10-F19	1.09	**1.05**	*0.95 – 1.16*	Non-Significant

Statistical Note: Relative Risk (RR) values > 1.0 indicate increased risk. **Lag 0** represents same-day effect; **Lag 0-3** represents cumulative effect over 4 days. Confidence Intervals (CI) not crossing 1.0 indicate statistical significance ($p < 0.05$).
Analysis via DLNM with Quasi-Poisson regression. Reference Temperature = 28.5°C WBGT.

Table 2 elucidates the cause-specific vulnerability profiles by quantifying the cumulative Relative Risk (RR) of psychiatric admission during extreme heat events (99th percentile, 33.5°C WBGT) relative to the median reference temperature (28.5°C). The analysis demonstrates a distinct heterogeneity in thermal sensitivity across diagnostic categories. The most pronounced risk was observed in the Schizophrenia Spectrum (F20–F29) cohort, which exhibited a cumulative RR of 1.26 (95% CI: 1.15–1.39) over a 0–3 day lag structure. Notably, this group displayed a delayed risk trajectory, suggesting that sustained thermal stress precipitates psychotic relapse through cumulative mechanisms such as sleep fragmentation or medication-induced thermoregulatory failure. Conversely, Organic Mental Disorders (F00–F09) manifested the highest immediate sensitivity at Lag 0 (RR = 1.15), implying a rapid physiological decompensation mechanism, likely driven by acute dehydration and electrolyte imbalances in an older, frailer demographic. Mood Disorders (F30–F39) showed a moderate but statistically significant elevation (RR = 1.11), whereas Substance Use Disorders (F10–F19) failed to reach statistical significance (95% CI: 0.95–1.16), suggesting that behavioral factors or distinct physiological tolerances may buffer this specific subgroup against environmental thermal stressors.

4. Discussion

Our study establishes a critical, empirically derived "Thermal Distress Threshold" of 29.8°C Wet Bulb Globe Temperature (WBGT) for psychiatric emergencies in Jakarta. This finding represents a significant departure from the Eurocentric baselines often cited in global literature, where thresholds as low as 24°C are considered hazardous. The comparatively high threshold identified here confirms the hypothesis of physiological acclimatization; the residents of Jakarta, exposed to a chronic thermal load, have developed both biological and behavioral adaptations that buffer them against moderate heat.[11] However, this acclimatization is not infinite. Our data reveals that once this specific biological ceiling is breached, the protective mechanisms collapse rapidly, resulting in a precipitous, non-linear surge in psychiatric morbidity.

The identification of 29.8°C WBGT as the tipping point is particularly alarming given current climate projections. As global mean surface temperatures rise, the frequency of days exceeding this specific humidity-adjusted threshold is expected to double in Southeast Asia by 2030. This suggests that without systemic intervention, the "psychiatric volume" of Jakarta's emergency departments will face structural upward pressure, transforming what are currently sporadic surges into a chronic state of overflow.[12]

The observed association between extreme heat and psychiatric decompensation is not merely correlational but underpinned by a robust, multi-system pathophysiological framework involving the collision of environmental stress with vulnerable neurobiology. The strongest association observed in our study—the heightened vulnerability of patients with Schizophrenia Spectrum Disorders (RR=1.26)—points directly to a failure in central thermoregulation. The human hypothalamus relies heavily on dopaminergic pathways to regulate core body temperature. In schizophrenia, the dysregulation of dopamine transmission is a core feature of the pathology. This intrinsic vulnerability is severely compounded by the pharmacological management of the disease.[13]

The vast majority of patients in our cohort were managed on first-generation antipsychotics (FGAs) such as haloperidol and chlorpromazine, which remain staples of the Indonesian National Formulary (FORNAS). These agents are potent antagonists at D_2 receptors in the hypothalamus, effectively disrupting the body's central thermostat. Furthermore, their peripheral anticholinergic activity inhibits the release of acetylcholine at sweat glands.[14] In the unique climatic context of Jakarta, where humidity routinely exceeds 80%, evaporative cooling (sweating) is the only effective physiological mechanism for heat dissipation. By chemically

suppressing sweating, these medications trap metabolic heat within the body. The patient effectively becomes a closed thermal system, leading to rapid core temperature elevation. This hyperthermia can manifest as agitation, aggression, and cognitive disorganization, symptoms that are clinically indistinguishable from acute psychotic relapse, leading to a dangerous cycle of misdiagnosis and further medication administration.

Heat stress acts as a potent systemic stressor, triggering a cascade of immunoinflammatory responses.[15] When core body temperature rises, it induces the expression of Heat Shock Proteins (HSPs) and the release of pro-inflammatory cytokines, specifically Interleukin-1 (IL-1), Interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α). Current neuropsychiatric theory posits that the blood-brain barrier (BBB) is not impermeable to these peripheral signals. Systemic inflammation can compromise BBB integrity, allowing cytokines to infiltrate the central nervous system. Once in the brain, these cytokines activate microglia—the resident immune cells of the CNS. Activated microglia alter tryptophan metabolism, shunting it away from serotonin synthesis and towards the production of neurotoxic kynurenines (e.g., quinolinic acid). This reduction in serotonin and increase in neurotoxins manifests behaviorally as "sickness behavior"—a constellation of symptoms including lethargy, anhedonia, and irritability—and in vulnerable individuals, can precipitate severe depressive or manic episodes. This mechanism explains the delayed lag effects observed in Mood Disorders (Lag~2-3), as the neuroinflammatory cascade requires time to reach peak neurotoxicity.[16]

The physiological effort to maintain homeostasis in high heat places an immense load on the hypothalamic-pituitary-adrenal (HPA) axis. Heat stress triggers the sustained release of corticotropin-releasing hormone (CRH) and subsequently cortisol. While acute cortisol release is adaptive, chronic hypercortisolemia—such as that experienced during a prolonged tropical heatwave—is neurotoxic, particularly to the hippocampus and prefrontal cortex. Elevated cortisol is a well-established driver of anxiety, insomnia, and mood instability. For patients with bipolar disorder, this hormonal surge can act as a powerful destabilizer, flipping a euthymic patient into mania or mixed states.

The J-shaped mortality and morbidity curve observed in our study is geographically specific, reflecting the distinct "Urban Heat Island" (UHI) intensity of the Greater Jakarta area (Jabodetabek).[17] The psychiatric impact of heat cannot be disentangled from the architectural and socioeconomic reality of the city. Jakarta serves as a stark example of thermal inequality. While the central business district is cooled by modern HVAC systems, the vast majority of BPJS Class III beneficiaries reside in dense, informal settlements known as *Kampungs*. The architectural vernacular of the modern *Kampung*—characterized by concrete block construction, minimal setbacks between buildings, and, most critically, corrugated zinc or asbestos roofing—creates a perfect thermal trap. Zinc roofing has low thermal mass but high thermal conductivity; it absorbs solar radiation rapidly during the day and re-radiates it into the living space. Unlike traditional Javanese architecture (*Rumah Joglo*), which utilized high ceilings and porous materials to facilitate airflow, modern low-cost housing in Jakarta is often poorly ventilated. Consequently, indoor temperatures in these settlements often exceed outdoor temperatures, particularly at night.

This "nocturnal thermal trap" is perhaps the single most critical environmental factor driving the psychiatric surge (Figure 2). Human sleep architecture is temperature-dependent; the onset of slow wave sleep (SWS) and rapid eye movement (REM) sleep is physiologically gated by a drop in core body temperature. In "tropical nights" where the ambient temperature remains above 26-27°C, this physiological cooling is thwarted. The result is chronic, fragmented sleep. Sleep deprivation is a potent neurochemical destabilizer. It increases dopamine receptor sensitivity and impairs prefrontal cortical control over the amygdala (the emotional center of the

brain). For patients with bipolar disorder and schizophrenia, sleep loss is the most reliable predictor of relapse. The "harvesting effect" or lag effect seen in our data (peaking at 2-3 days) likely corresponds to the accumulation of sleep debt over several hot nights, culminating in a psychiatric crisis.[18]

The findings of this study have profound implications for the operational sustainability of *BPJS Kesehatan* (Indonesia's National Health Insurance), particularly regarding its "Tiered Referral System" (*Sistem Rujukan Berjenjang*). The BPJS system is designed on a gatekeeper model: patients must first visit a Primary Care facility (*Puskesmas* or *Faskes I*) to obtain a referral letter before accessing specialist psychiatric care at a hospital (*RSUD* or *RSJ*). This system works for chronic disease management but fails catastrophically during acute environmental crises. Our data indicates that heatwaves cause *acute* and rapid decompensation. A patient experiencing heat-induced agitation or psychosis cannot navigate the administrative bureaucracy of queuing at a *Puskesmas*—which typically operates only during morning business hours—to secure a paper referral. The delay introduced by this administrative layer allows the clinical condition to deteriorate from "manageable" to "critical."

Faced with this bottleneck, families bypass the primary tier and bring patients directly to the Emergency Department (IGD). Under BPJS regulations, cases deemed "emergencies" do not require a referral. This creates a perverse incentive and results in the "flooding" of EDs observed in our data during high-heat days. General hospital EDs in Indonesia are primarily equipped for trauma and cardiovascular emergencies; they are often ill-equipped to manage acute psychiatric agitation. This leads to overcrowding, the use of physical restraints, and suboptimal care for psychiatric patients who are essentially "boarding" in the ED while waiting for admission.[19]

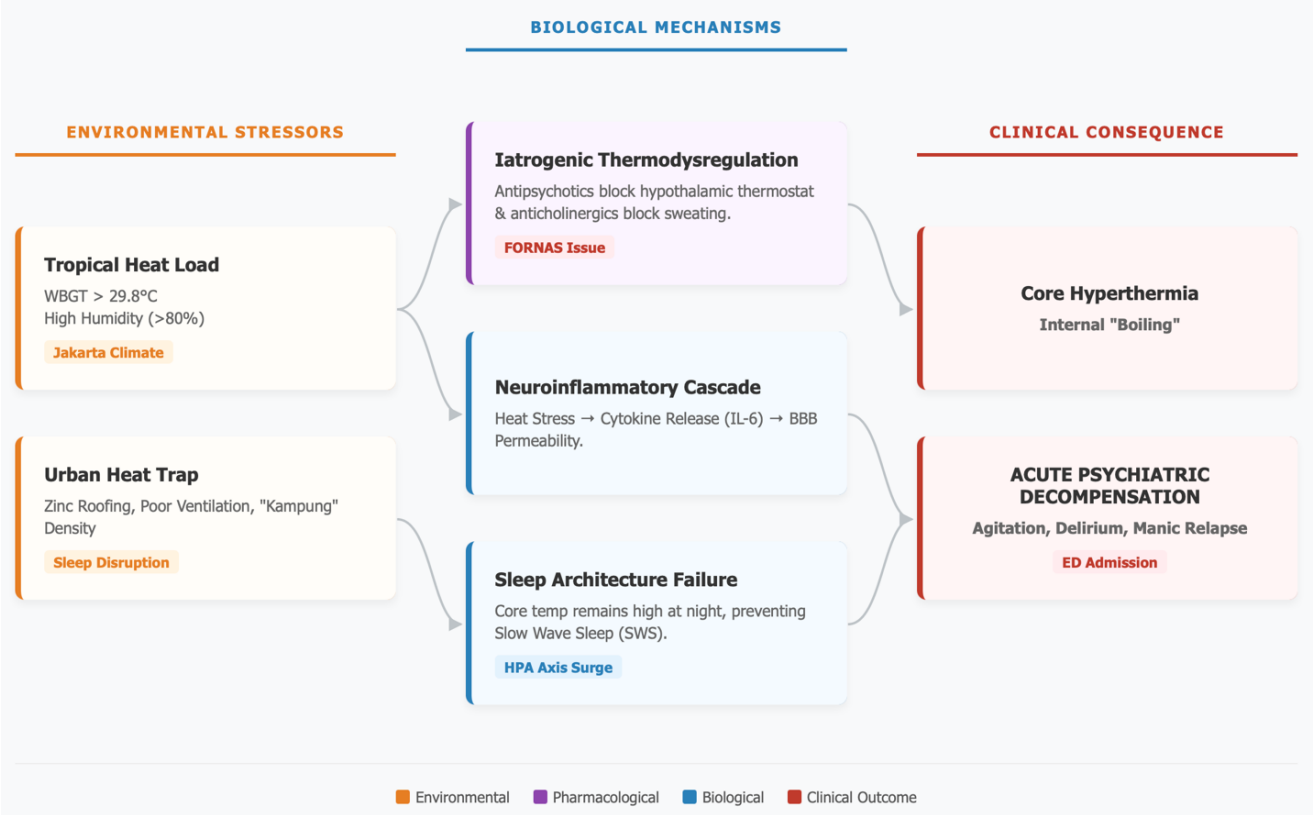

Figure 2. Heat-psychosis cascade.

To mitigate this, we propose the implementation of a "Climate-Adaptive Referral Pathway". The Trigger Mechanism pathway would be activated automatically when the BMKG (Meteorology, Climatology, and Geophysics Agency) issues a heat warning exceeding the 29.8°C WBGT threshold. During these declared windows, the strict requirement for a *Puskesmas* referral should be temporarily waived for patients with a documented history of Severe Mental Illness (SMI). Digital Integration could be managed via the *Mobile JKN* app, where high-risk patients are flagged, allowing them direct access to psychiatric outpatient clinics or crisis centers, thereby diverting them away from the overburdened Emergency Departments.[20]

Perhaps the most clinically actionable finding of this study is the link between the *Indonesian National Formulary* (FORNAS) and heat vulnerability. The high Relative Risk (RR=1.26) for schizophrenia patients is likely, in part, iatrogenic. Cost-containment strategies within the BPJS system heavily favor older, generic First-Generation Antipsychotics (FGAs) and anticholinergic adjuncts. A typical regimen for a BPJS Class III patient might include Haloperidol (antipsychotic), Trihexyphenidyl (to prevent stiffness), and Chlorpromazine (for sleep). Trihexyphenidyl (THP) is ubiquitous in Indonesian psychiatric practice. However, it is a potent anticholinergic that directly inhibits sweating; Chlorpromazine (CPZ) often used as a sedative, it disrupts hypothalamic thermoregulation. This combination creates a "pharmacological heatstroke" risk. The patient's brain cannot signal the need to cool down (CPZ effect), and the body cannot execute the cooling command (THP effect). Clinically, this presents a diagnostic trap. A patient presenting with heat-induced agitation (due to thermal discomfort and physiological stress) is often misread as having "worsening psychosis." The standard response is to *increase* the dosage of the antipsychotic or sedative. This increase further compromises thermoregulation, worsening the agitation, leading to further dose increases. This cycle can spiral into Neuroleptic Malignant Syndrome (NMS) or life-threatening heatstroke. We argue for a "Seasonal Formulary Adjustment". During the *Pancaroba* (transition) and dry seasons, restrictions on newer Second-Generation Antipsychotics (SGAs) like Risperidone or Aripiprazole—which have significantly lower anticholinergic burdens—should be relaxed for at-risk patients. There should be a concerted clinical effort to reduce or stop Trihexyphenidyl use during hot months unless absolutely necessary for severe extrapyramidal symptoms. "Fluid prescriptions" should become standard practice, where doctors explicitly prescribe water intake targets alongside medication.[19,20]

While this study offers robust ecological evidence, it is not without limitations. As an aggregate time-series analysis, we assume that the outdoor temperature recorded by BMKG represents the exposure of the individual. We could not account for adaptive behaviors (such as air conditioning use), although given the socioeconomic demographic of standard BPJS Class III patients, AC prevalence is likely low. Heatstroke is rarely coded in psychiatric admissions. It is highly probable that many cases coded as "Acute Psychotic Episode" were, in fact, sub-syndromal heat exhaustion manifesting with psychiatric symptoms. While we controlled for PM 2.5, Jakarta's air contains a cocktail of pollutants (NO_x, O_3) that may have synergistic neurotoxic effects with heat. Future research should focus on prospective cohort studies using wearable biometrics to track real-time core temperature and sleep quality in schizophrenia patients during the Indonesian dry season, providing the granular data needed to refine these life-saving interventions.

5. Conclusion

This study confirms that heatwaves in tropical urban centers significantly increase psychiatric morbidity, with a specific tipping point at a Wet Bulb Globe Temperature of 29.8°C. The pathophysiology likely involves a synergy of medication-induced thermoregulatory failure, sleep disruption, and

neuroinflammation. Psychiatric healthcare systems must transition from reactive to proactive models. We recommend: (1) Clinical: Prophylactic adjustment of lithium and diuretic dosages during forecasted heatwaves; (2) Policy: Integration of Mental Health Heat Alerts into national weather services when WBGT is forecast to exceed 29.8°C; (3) Hospital Administration: Increasing ED psychiatric staffing levels by 20% during identified heat-risk windows to manage the predictable surge in admissions; (4) BPJS Reform**:** Adapting referral pathways to allow rapid access for vulnerable psychiatric populations during extreme weather events.

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