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Wavelet-Based Mathematical Modelling of Regulatory T-Cell Dynamics: A Novel Framework for Understanding Immune Dysregulation in Autoimmune Disorders

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Abstract - Autoimmune disorders arise from the breakdown of immune tolerance, resulting in abnormal immune responses against the body's own tissues. Despite significant advancements in immunology and molecular biology, understanding the complex dynamics of immune regulation, particularly the role of Regulatory T (Treg) cells, remains a major challenge. Treg-mediated immune regulation involves temporal variability, spatial heterogeneity, and nonlinear feedback mechanisms that are difficult to capture using conventional mathematical approaches. This hypothesis-based research proposes a novel wavelet-based mathematical modelling framework to investigate immune dysregulation and autoimmune disease dynamics. The study integrates mathematical immunology, functional analysis, and computational wavelet techniques to examine interactions between Treg cells, effector T cells, and cytokine signalling pathways. Coupled nonlinear ordinary and partial differential equations (ODEs and PDEs) will be formulated to represent immune system interactions. Their solutions will be analysed within appropriate Hilbert spaces using orthonormal wavelet bases, enabling localized decomposition of immune responses across different temporal and spatial scales. The computational performance of the proposed framework will also be compared with conventional numerical methods to evaluate its accuracy and efficiency. This approach is hypothesized to improve the understanding of localized immune fluctuations, abrupt inflammatory transitions, and loss of immune tolerance. Ultimately, the proposed framework may contribute to improved disease monitoring, early risk stratification, and the development of personalized immunomodulatory strategies for autoimmune disorders.

Keywords: Autoimmune Disorders, Regulatory T Cells (Tregs), Wavelet-Based Mathematical Modelling, Immune Dysregulation, Hilbert Space Analysis, Nonlinear Immune Dynamics

I. INTRODUCTION

Autoimmune disorders constitute a heterogeneous group of chronic inflammatory conditions characterized by the loss of immunological self-tolerance, in which the immune system mistakenly identifies self-antigens as foreign and initiates immune responses against the body's own tissues. These conditions include rheumatoid arthritis, multiple sclerosis, type-1diabetes mellitus, systemic lupus erythematosus, and immune dysregulation, polyendocrinopathy, enteropathy, X-linked (IPEX) syndrome. Although their clinical manifestations differ, these disorders involve disturbances in immune homeostasis, abnormal immune-cell activation, and dysregulated inflammatory signalling. The development and progression of autoimmune diseases are influenced by a combination of genetic susceptibility, environmental exposures, physiological stress, lifestyle-related factors, and interactions with other pathological conditions.

The maintenance of immune homeostasis depends on a highly coordinated regulatory mechanism involving immune recognition, activation, suppression, and resolution of inflammatory responses. Regulatory T (Treg) cells play a fundamental role in this process

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by preventing excessive immune activation and maintaining tolerance towards self-antigens. The importance of Treg-mediated immune regulation was initially demonstrated by Sakaguchi et al., who identified the critical contribution of CD4+CD25+ T cells to immunological self-tolerance and the prevention of autoimmune reactions [1]. Subsequent studies established that abnormalities in this regulatory population can contribute to the development of autoimmune and inflammatory disorders [2,3].

Regulatory T cells, particularly CD4+,CD25+,FOXP3+ T cells, represent an essential component of the adaptive immune system responsible for controlling immune responses and maintaining peripheral tolerance. The transcription factor Forkhead Box P3 (FOXP3) plays a central role in Treg-cell development, differentiation, and functional stability. Genetic alterations affecting FOXP3 have been associated with IPEX syndrome, providing important evidence of the relationship between impaired regulatory T-cell function and severe autoimmune manifestations [2]. Furthermore, experimental investigations by Hori et al. and Fontenot et al. established FOXP3 as a critical regulator of Treg-cell development and suppressive activity [4,5].

Treg cells regulate immune responses through multiple mechanisms, including the secretion of immunosuppressive cytokines such as interleukin-10 (IL-10), transforming growth factor-beta (TGF-β), and interleukin-35 (IL-35), inhibition of antigen-presenting cell activity, competition for interleukin-2 (IL-2), and direct suppression of effector T-cell proliferation. These mechanisms help maintain a physiological balance between immune activation and immune suppression [6,7].

However, alterations in Treg-cell number, functional stability, differentiation, or regulatory signalling can disturb this balance and promote excessive inflammatory responses. An imbalance between Treg cells and effector T cells may therefore contribute to the breakdown of immune tolerance and the progression of autoimmune diseases. Recent studies have further highlighted the complexity of Treg-cell heterogeneity, plasticity, and functional dysfunction in autoimmune conditions [8,9,10].

Immune regulation is influenced by several interconnected genetic, environmental, and physiological factors that may alter the balance between immune activation and suppression. Genetic susceptibility can affect immune-regulatory genes and signalling pathways, whereas environmental and lifestyle-related factors, including psychological stress, sleep disturbances, radiation exposure, and other disease conditions, may influence inflammatory activity and immune-system functioning.

These factors can interact with Treg-cell differentiation, proliferation, survival, and suppressive capacity, potentially contributing to disturbances in immune homeostasis. Consequently, autoimmune disease development cannot be completely explained by the activity of a single immune-cell population or an isolated molecular pathway. Instead, it involves a complex network of interactions among Treg cells, effector T cells, cytokines, inflammatory mediators, and environmental influences.

Furthermore, immune responses exhibit considerable temporal variability and spatial heterogeneity. Inflammatory activity may fluctuate over time, while immune-cell populations and cytokine concentrations may differ across tissues and disease stages. Such characteristics indicate the importance of investigating immune regulation through dynamic mathematical approaches capable of representing nonlinear biological interactions and localized variations.

Mathematical modelling has emerged as an important quantitative approach for understanding complex biological systems, particularly when multiple interacting variables influence disease progression. Mathematical immunology provides a framework for representing immune-cell interactions, cytokine signalling, inflammatory responses, and regulatory feedback mechanisms through mathematical equations.

Several conventional mathematical techniques have been employed to investigate biological and immunological systems, including Fourier-based methods, separation of variables, finite difference methods (FDM), finite element methods (FEM), and numerical integration techniques such as Euler and Runge–Kutta methods.

Ordinary differential equations (ODEs) are useful for describing temporal changes in immune-cell populations, including Treg-cell proliferation, effector T-cell activation, and cytokine production. In contrast, partial differential equations (PDEs) can represent spatially distributed biological processes, including cytokine diffusion, inflammatory signalling, and immune-cell migration within tissues. Coupled nonlinear ODEs and PDEs can therefore provide a mathematical representation of interactions between regulatory T cells, effector T cells, and inflammatory mediators.

Previous mathematical investigations have demonstrated the potential of these approaches for understanding autoimmune disease mechanisms and predicting immune responses. A systematic review by Ugolkov et al. identified 38 mathematical models covering 13 autoimmune conditions, with nonlinear ODE-based approaches representing the majority of the reviewed models. Other models incorporated PDEs and additional computational techniques to describe different aspects of immune-system behaviour [11].

Despite their usefulness, conventional mathematical approaches may have limitations when analysing biological systems characterized by non-stationary behaviour, abrupt transitions, and localized fluctuations. In particular, global Fourier-based representations may not provide the same degree of temporal and spatial localization as wavelet-based techniques. Therefore, alternative mathematical approaches that can capture variations across multiple scales deserve further investigation.

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Wavelet Mathematics: A Novel Multiscale Approach

Wavelet analysis is a mathematical technique that enables the decomposition of complex signals and functions into localized components across different temporal, spatial, and frequency scales. Unlike conventional Fourier analysis, which represents signals through globally defined sinusoidal functions, wavelet transforms utilize localized basis functions to provide information about both the position and scale of signal variations.

The application of wavelet methods in biological and computational research has been explored for analysing complex biological sequences, molecular structures, and non-stationary biological signals. Liò demonstrated the potential of wavelet-based statistical approaches in bioinformatics and computational biology, highlighting their usefulness in analysing biological data with complex structural variations[12]. More recently, wavelet analysis has been recognized as a versatile approach for characterizing multiscale biological dynamics and transient physiological variations [13].

In the proposed research, wavelet-based mathematical representations will be incorporated within an appropriate Hilbert space framework. Immune-response variables will be represented using orthonormal wavelet bases, allowing their decomposition into localized temporal and spatial components. This approach may provide additional information about complex immune-system behaviour that could be difficult to characterize using conventional global analytical representations.

II. CONCEPTUAL INNOVATION

- **Integration of Wavelet Mathematics with Treg-Mediated Immune Regulation**

This proposed research introduces a conceptual mathematical framework that integrates wavelet-based multiscale analysis with regulatory T-cell-mediated immune regulation to investigate the complex mechanisms underlying immune dysregulation in autoimmune disorders.

Conventional mathematical models have been extensively employed to describe immune-cell interactions, cytokine signalling and inflammatory responses through ordinary and partial differential equations. However, the representation of localized fluctuations and multiscale variations in immune responses remains an important area for further methodological investigation [11].

This proposed framework will incorporate orthonormal wavelet bases and multiresolution analysis within an appropriate Hilbert space to represent the dynamic behaviour of Treg cells, effector T cells and associated cytokine interactions. This integration is intended to provide a mathematical representation of immune responses at different temporal and spatial scales.

- **Multiscale Characterization of Treg Dynamics**

A major conceptual innovation is the proposed investigation of Treg-cell behaviour across multiple biological scales, ranging from rapid cellular responses to longer-term changes in immune regulation.

Wavelet decomposition may allow different components of immune-response dynamics to be examined through their corresponding scales and coefficients. In the proposed framework:

- High-frequency components: May represent rapid changes in cellular activation and short-term immune fluctuations.
- Intermediate-frequency components: May represent variations in immune-cell interactions and cytokine-mediated signalling.
- Low-frequency components: May represent slower changes in immune regulation and longer-term population dynamics.
- Localized Identification of Immune Dysregulation

The proposed approach builds upon the localization and multiresolution properties of wavelet mathematics established by Daubechies and Mallat [14,15].

III. SCIENTIFIC RATIONALE

- **Biological Basis: Importance of Treg Cells in Maintaining Immune Homeostasis**

The immune system maintains physiological stability through a coordinated balance between immune activation and immunological suppression. Regulatory T cells, particularly CD4+,CD25+,FOXP3+ cells, are essential for maintaining peripheral immune tolerance and preventing excessive immune responses against self-antigens.

The importance of Treg cells in immune homeostasis was demonstrated by Sakaguchi et al., who established their contribution to the maintenance of immunological self-tolerance [1]. Subsequent investigations by Hori et al. and Fontenot et al. established FOXP3 as a critical transcriptional regulator of Treg-cell development and suppressive function [4,5].

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Alterations in Treg-cell differentiation, stability and suppressive activity may contribute to impaired immune regulation and autoimmune pathology. Recent research has further emphasized the complexity of Treg-cell dysfunction and its association with autoimmune diseases [8,9,10,13].

- **Mathematical Basis: Complexity of Biological Immune Interactions**

Immune-system behaviour involves multiple interconnected biological components, including regulatory T cells, effector T cells, cytokines and inflammatory mediators. Their interactions may exhibit nonlinear relationships, feedback mechanisms, temporal fluctuations and spatial heterogeneity.

Mathematical modelling provides a systematic approach to representing these interactions through quantitative relationships.

Previous mathematical investigations have employed:

- Ordinary differential equations (ODEs) to represent temporal changes in immune-cell populations.
- Partial differential equations (PDEs) to describe spatially distributed biological processes.
- Finite difference methods (FDM) and finite element methods (FEM) for numerical approximation.
- Fourier-based methods for frequency-domain representation.
- Numerical integration techniques, including Euler and Runge–Kutta methods.

A systematic review by Ugolkov et al. identified 38 mathematical models covering 13 autoimmune conditions and demonstrated the importance of mathematical modelling in investigating immune-cell interactions, cytokine signalling and disease progression [11].

- **Mathematical Basis: Relevance of Wavelet Analysis**

Wavelet analysis provides a mathematical framework for representing functions and signals at different scales while retaining information about localized variations.

Daubechies established orthonormal bases of compactly supported wavelets, providing an important mathematical foundation for wavelet decomposition [14]. Subsequently, Mallat developed the theoretical framework of multiresolution signal decomposition using orthonormal wavelet representations [15].

Liò demonstrated the potential of wavelet-based techniques in computational biology, including the analysis of biological sequences, protein structures and microarray data [12].

These developments provide a mathematical basis for exploring wavelet techniques in complex biological systems.

- **Rationale for Combining ODEs, PDEs and Wavelet Decomposition**

The proposed research will not replace conventional differential-equation-based modelling. Instead, it will investigate the integration of these approaches with wavelet decomposition.

Coupled nonlinear ODEs may describe changes in Treg-cell populations, effector T-cell activation and cytokine concentrations over time. Where spatial distribution and tissue-level interactions are considered, PDEs may be incorporated to represent spatially dependent biological processes.

The resulting mathematical system may subsequently be projected into suitable orthonormal wavelet bases within an appropriate Hilbert space.

This approach is expected to facilitate:

- Multiresolution representation of immune-response variables.
- Mathematical characterization of localized fluctuations.
- Investigation of nonlinear interactions across different scales.
- Analysis of spatial and temporal heterogeneity.
- Comparison of wavelet-based solutions with conventional numerical approaches.

IV. METHODOLOGY

Research Design

The present study proposes a theoretical and computational mathematical framework integrating nonlinear differential equations, functional analysis, Hilbert space theory and wavelet-based multiresolution techniques to investigate regulatory T-cell dynamics in autoimmune disorders. This proposed methodology will involve the mathematical representation of interactions between regulatory T cells, effector T cells and inflammatory mediators. These interactions will initially be expressed through coupled nonlinear ordinary differential equations (ODEs). Subsequently, spatial variations may be incorporated using partial differential equations (PDEs).

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The resulting mathematical system will be projected into suitable orthonormal wavelet bases within an appropriate Hilbert space to investigate temporal and spatial variations across multiple scales.

Identification of Biological Variables

Initially, three major components of immune regulation will be considered:

- Regulatory T cells (Treg): Responsible for suppressing excessive immune activation and maintaining immune tolerance.
- Effector T cells (Teff): Responsible for immune activation and inflammatory responses.
- Cytokines: Signalling molecules involved in regulating interactions between immune cells.

For mathematical modelling, the following variables are proposed:

Variable	**Biological representation**
R(t)	Regulatory T-cell population
E(t)	Effector T-cell population
P(t)	Pro-inflammatory cytokine concentration
S(t)	Immunoregulatory cytokine concentration

The cytokine variables represent simplified functional groups rather than individual cytokines. In an expanded model, specific mediators such as IL-2, IL-10 and TGF-β can be incorporated separately. This model parameters will represent cellular proliferation, natural cell death, cytokine production, cytokine degradation and suppressive interactions.

Development of the Mathematical Model

a. Equation 1: Regulatory T-cell dynamics

This equation represents Treg-cell population changes through basal recruitment or production, immunoregulatory signalling and natural cell loss.

b. Equation 2: Effector T-cell dynamics

This equation represents effector T-cell activation and proliferation in response to inflammatory signalling, natural cell loss and suppression by Treg cells.

c. Equation 3: Pro-inflammatory cytokine dynamics

This equation represents pro-inflammatory cytokine production by effector immune cells, natural cytokine degradation and regulatory suppression.

d. Equation 4: Immunoregulatory cytokine dynamics

This equation represents immunoregulatory cytokine production associated with Treg-cell activity and its subsequent degradation.

Extension to Spatially Dependent PDEs

Although ODEs can describe changes in immune-cell populations over time, they do not independently represent spatial variations within biological tissues.

Therefore, the proposed framework may be extended into coupled PDEs by incorporating spatial diffusion and migration terms.

For example:

Here:

- represent effective diffusion or spatial transport coefficients.
- represents the spatial Laplacian operator.
- represent the corresponding biological interaction terms from the proposed ODE model.

This extension will allow the investigation of spatial heterogeneity, cytokine distribution and localized immune-cell interactions.

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Selection of Orthonormal Wavelet Basis

After developing the mathematical equations, the next step will involve selecting an appropriate wavelet basis for representing the immune-response variables.

Wavelet analysis is particularly useful because it provides localized information about variations across different scales.

The proposed study will investigate suitable wavelet families, such as:

- Haar wavelets.
- Daubechies wavelets.
- Symlets.
- Coiflets.

The final selection will depend on the smoothness of the biological variables, computational efficiency, boundary conditions and ability to represent localized fluctuations.

Daubechies wavelets will be considered because of their compact support and orthonormal properties [14].

The mathematical variables will be represented through wavelet expansions:

Where:

- represents an immune-response variable.
- represents the selected wavelet basis function.
- represents the resolution or scale level.
- represents the spatial location.
- represents the corresponding wavelet coefficient.

This representation will allow the mathematical system to be analysed through localized components at different scales.

Multiresolution Analysis within Hilbert Space

The selected wavelet functions will be used to construct a suitable Hilbert space representation of the immune-response variables.

A Hilbert space provides a mathematical framework in which functions can be represented through inner products, orthogonal bases and convergent expansions.

The proposed framework will employ multiresolution analysis to decompose immune-response variables into different levels of approximation and detail.

Conceptually, this will involve:

1. Representing the biological variables as functions in an appropriate function space.
2. Selecting a suitable orthonormal wavelet basis.
3. Decomposing the functions into coarse and fine resolution components.
4. Obtaining wavelet coefficients at different scales and locations.
5. Reconstructing the mathematical representation from these coefficients.

The mathematical foundation of this approach is based on orthonormal wavelet theory and multiresolution decomposition developed by Daubechies and Mallat [14,15].

Projection of the Nonlinear Mathematical System

The coupled nonlinear differential equations developed in the previous stages will be projected into the selected wavelet basis.

For a general mathematical equation:

- The wavelet expansion will be substituted into the equation.
- The resulting expression will be projected onto each selected wavelet basis function using the corresponding inner product.

The procedure will involve:

- Projection of the time-derivative term.
- Projection of the diffusion or spatial interaction term.
- Projection of linear growth and degradation terms.
- Projection of nonlinear cellular interaction terms.
- Evaluation of interactions between different wavelet scales.

Numerical Solution and Computational Implementation

The resulting coefficient-based mathematical system will be solved using suitable numerical techniques.

The proposed computational workflow will include:

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1. Defining initial conditions for Treg cells, effector T cells and cytokines.
2. Selecting wavelet families and resolution levels.
3. Computing the required wavelet coefficients and projection matrices.
4. Constructing the reduced nonlinear coefficient equations.
5. Solving the resulting system using suitable numerical integration techniques.
6. Reconstructing the original immune-response functions.
7. Analysing the resulting temporal and spatial variations.

Numerical techniques such as Runge–Kutta methods may be used for solving the coefficient equations.
The reconstructed results will be examined to investigate changes in Treg-cell populations, effector T-cell activity and cytokine concentrations.

Comparative Evaluation with Conventional Mathematical Methods

To assess the usefulness of the proposed approach, its performance will be compared with selected conventional numerical and mathematical methods.

The comparison will include:

Mathematical method	**Purpose of comparison**
Fourier-based methods	Comparison of global frequency representation and localized signal analysis
Finite Difference Method (FDM)	Comparison of numerical approximation and spatial resolution
Finite Element Method (FEM)	Comparison of spatial representation in complex domains
Euler method	Comparison of numerical integration performance
Runge–Kutta method	Comparison of temporal numerical accuracy
Wavelet-based method	Evaluation of multiscale representation and localized variations

The following parameters will be considered:

- Numerical accuracy and approximation error.
- Computational time.
- Convergence behaviour.
- Stability of numerical solutions.
- Ability to represent localized fluctuations.
- Performance at different resolution levels.

All methods should be evaluated using comparable initial conditions, model parameters, boundary conditions and numerical tolerances wherever applicable.

Biological Interpretation and Potential Validation

The mathematical outputs will be interpreted in relation to known biological mechanisms of immune regulation.
Potential biological datasets may include:

- Flow cytometry measurements of CD4+,CD25+,FOXP3+ Treg cells and effector T-cell populations.
- Cytokine concentrations obtained through appropriate molecular assays.
- Microscopy-derived spatial distributions of immune cells.
- Longitudinal measurements from relevant autoimmune disease datasets.

Where suitable datasets are available, model parameters may be estimated through parameter-fitting techniques and the predicted mathematical behaviour may be compared with observed biological measurements.

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Wavelet-derived coefficients will be investigated as potential analytical features associated with changes in immune regulation.

MECHANISM OF WORKING OF THE PROPOSED FRAMEWORK

Biological immune interactions are first converted into mathematical equations, then transformed into wavelet representations and finally interpreted as multiscale immune-response patterns.

Proposed working mechanism

- Stage 1: Biological input

Treg cells, effector T cells and cytokine interactions

- Stage 2: Mathematical formulation

Development of coupled nonlinear ODEs and PDEs

- Stage 3: Wavelet decomposition

Selection of orthonormal wavelet bases and multiresolution expansion

- Stage 4: Wavelet projection

Projection of linear, nonlinear and spatial interaction terms into wavelet space

- Stage 5: Numerical computation

Solution of the reduced coefficient-based mathematical system

- Stage 6: Multiscale reconstruction

Reconstruction of immune-response variables across scales

- Stage 7: Biological interpretation

Investigation of immune fluctuations, regulatory imbalance and potential dysregulation patterns

The proposed methodology is expected to produce:

Component	**Expected output**
Biological modelling	Mathematical representation of Treg–Teff interactions
ODE/PDE development	Coupled nonlinear immune-regulation equations
Wavelet decomposition	Scale-dependent mathematical coefficients
Hilbert space representation	Orthonormal mathematical representation
Nonlinear projection	Reduced coefficient-based equations
Numerical computation	Simulated immune-response dynamics
Comparative analysis	Evaluation against conventional methods
Biological interpretation	Potential mathematical indicators of immune dysregulation

Software and Computational Requirements

Software/tool	**Proposed application**
MATLAB	Mathematical modelling and numerical simulations
Python	Wavelet analysis and computational implementation
PyWavelets	Wavelet decomposition and reconstruction
NumPy/SciPy	Numerical calculations and differential equations
Matplotlib	Graphical representation of mathematical outputs

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SPSS (optional)	Statistical analysis if biological datasets are obtained

V. FEASIBILITY

- Established Mathematical Techniques: Wavelet analysis, Hilbert space theory, nonlinear differential equations and numerical modelling are well-established mathematical approaches that can be integrated into the proposed framework.
- Computational Feasibility: The proposed model can initially be developed and simulated using MATLAB or Python, without requiring expensive laboratory infrastructure.
- Availability of Scientific Literature: Existing research on Treg-cell biology, autoimmune disorders and mathematical immunology provides a sufficient theoretical foundation for developing the proposed model.
- Availability of Biological Parameters: Initial model parameters can be obtained from published experimental studies and relevant biological datasets, subject to appropriate validation.
- Modular Model Development: The research can be conducted progressively, beginning with coupled ODEs and subsequently extending to spatial PDEs and wavelet-based multiresolution analysis.
- Established Wavelet Frameworks: Orthonormal wavelet families, including Haar and Daubechies wavelets, provide suitable mathematical foundations for the proposed computational methodology.
- Comparative Validation: The proposed framework can be evaluated against conventional methods such as Fourier analysis, FDM, FEM and numerical integration techniques using measurable computational parameters.
- Interdisciplinary Collaboration: Collaboration between mathematics, computational science and medical researchers can facilitate mathematical development and biological interpretation.
- Scope for Future Experimental Validation: The theoretical findings can subsequently be evaluated using flow cytometry, cytokine measurements and microscopy-derived datasets to investigate their biological relevance.

VI. CONCLUSION AND HEALTHCARE IMPACT

The proposed research presents a novel theoretical and computational framework for understanding regulatory T-cell (Treg) dynamics in autoimmune disorders through wavelet-based mathematical modelling. Treg cells play a crucial role in maintaining immune tolerance, and their interaction with effector T cells and inflammatory mediators involves complex, nonlinear and multiscale processes. Conventional mathematical approaches may not always adequately represent localized and time-dependent variations in these interactions. By integrating wavelet analysis with mathematical models based on ordinary and partial differential equations, this framework aims to examine immune-regulatory changes across different spatial and temporal scales. It may help identify patterns associated with disturbances in immune homeostasis, including fluctuations in Treg activity and inflammatory responses. The proposed approach also offers scope for integrating biological parameters obtained from flow cytometry, cytokine assays and other experimental methods in future studies. Comparative computational assessment against conventional techniques will be essential to establish its accuracy, efficiency and practical value. Overall, this hypothesis-based research seeks to establish a foundation for interdisciplinary investigation at the intersection of immunology, mathematics and computational biology. Its proposed applications remain theoretical and require computational testing, experimental validation and clinical evaluation before any diagnostic or therapeutic use can be considered.

- Improved understanding of immune dysregulation: May provide a mathematical approach to studying disturbances in Treg-mediated immune regulation in autoimmune disorders.
- Multiscale analysis of immune responses: Could help characterize immune-cell fluctuations and inflammatory changes occurring over different spatial and temporal scales.
- Identification of potential early warning patterns: May help explore mathematical patterns associated with changes in immune homeostasis and disease activity.
- Support for autoimmune disease research: Could provide a computational framework for investigating conditions such as systemic lupus erythematosus, rheumatoid arthritis and multiple sclerosis.
- Integration of biological and computational data: May facilitate the combined analysis of flow-cytometry findings, cytokine measurements and longitudinal immune-cell data.
- Potential contribution to disease monitoring: Wavelet-derived features could be investigated as candidate analytical indicators of disease activity, subject to independent validation.

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- Foundation for personalized research approaches: May support future investigations into individual variations in immune-regulatory dynamics and disease progression.
- Interdisciplinary research advancement: Could encourage collaboration between medical science, immunology, mathematics and computational biology to develop improved approaches for studying autoimmune diseases.

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