

Prostate Cancer Biology Analytics

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Abstract

Prostate-specific antigen (PSA) time series exhibit both systematic trends and irregular fluctuations, complicating their interpretation in clinical contexts such as prostate cancer progression. In this study, we frame longitudinal PSA measurements within a stochastic dynamical systems perspective, drawing on the Fokker-Planck equation to decompose observed dynamics into directional (drift) and stochastic (diffusion) components. The drift term captures the underlying progression of disease activity, while the diffusion term represents measurement variability and biological noise. Using a multi-year PSA dataset, we estimate a positive but slow drift (≈ 0.8 units/year) and moderate diffusion, corresponding to a PSA doubling time of approximately 4.4 years. To further refine interpretation, we introduce a latent state formulation in which the observed PSA values are modeled as noisy realizations of an unobserved disease process. This state-space approach enables separation of signal from noise, yielding a smoothed trajectory that reflects underlying biological activity without assuming direct equivalence between PSA levels and disease stage. Importantly, while the latent state reduces the impact of stochastic fluctuations, it does not eliminate uncertainty and cannot uniquely distinguish between benign conditions, localized cancer, or metastatic disease. Our results highlight that PSA dynamics are best understood as a combination of deterministic progression and stochastic variability. Although drift-diffusion and latent-state models provide a principled framework for analyzing PSA trajectories, they remain insufficient for definitive clinical diagnosis in isolation. Integration with imaging, histopathology, and other clinical indicators is essential for accurate assessment of disease status. The analysis was performed on 25 longitudinal PSA measurements collected from a single prostate cancer patient during approximately five years of post-treatment follow-up.

Keywords

Longitudinal PSA, Tumor Burden, Prostate Cancer

1. Introduction

Prostate-specific antigen (PSA) is one of the most widely used biomarkers for monitoring prostate health and assessing the progression of prostate cancer. Longitudinal PSA measurements are routinely collected in clinical practice, providing time series data that reflect underlying biological processes. However, interpreting these trajectories is challenging because PSA levels are influenced by multiple factors, including benign prostatic hyperplasia, inflammation, measurement variability, and malignant growth. As a result, simple threshold-based or trend-based interpretations often fail to distinguish between clinically significant disease progression and benign fluctuations.

Prostate cancer remains one of the most prevalent malignancies among men worldwide. Data from National Cancer Institute's SEER program highlights long-term incidence, survival, and mortality trends, providing a foundational dataset for population-level analytics [1]. These datasets have enabled risk stratification models and predictive analytics used in modern oncology.

Modern clinical trial design has evolved significantly, particularly for advanced and castration-resistant prostate cancer (CRPC). Howard I Scher and colleagues established standardized endpoints and frameworks for evaluating treatment efficacy in progressive disease [2] [3]. These frameworks are critical for analytics, as they define measurable outcomes such as progression-free survival and overall survival, enabling robust statistical modeling.

Early therapeutic insights date back to the seminal work of Charles Huggins, who demonstrated the role of androgen deprivation through orchiectomy in prostate cancer treatment [4] [5]. This discovery laid the groundwork for hormonal therapy, which remains central in disease management.

Recent studies emphasize the role of metabolic reprogramming in prostate cancer progression. Glutamine metabolism, for instance, has been identified as a key driver of tumor growth, with transporters facilitating cancer cell proliferation [6]. Targeting glutamine availability has also been shown to enhance radiation sensitivity, suggesting potential for predictive treatment-response models [7]. Radiotherapy remains a cornerstone treatment for high-risk prostate cancer. Advances in treatment protocols and dose optimization have improved outcomes [8]. Analytical models are increasingly used to personalize radiotherapy, integrating clinical, imaging, and molecular data.

These molecular insights provide rich datasets for computational analytics, particularly in biomarker discovery and precision medicine. Anti-inflammatory agents are being explored as potential therapeutic strategies, highlighting the importance of integrating immunological data into cancer analytics [9]. Chronic inflammation has been strongly associated with increased prostate cancer risk [10]. Oxidative stress is another significant factor contributing to tumorigenesis and progression [11]. Usually, prostate cancer informatics requires a number of features to include in the analysis [12].

The tumor microenvironment (TME) plays a crucial role in disease progres-

sion. Tumor-associated macrophages (TAMs) exhibit significant heterogeneity and can promote tumor growth and metastasis [13] [14]. Prostate cancer frequently metastasizes to bone, where interactions with immune cells and stromal components drive disease progression. Chemokines such as CXCL5 have been implicated in metastatic spread [15], while macrophages play a role in bone lesion development [16]. Targeting chemokine signaling pathways, such as CXCR2 blockade, has shown promise in reducing tumor progression [17]. These findings are critical for developing predictive analytics models focused on metastasis risk and treatment response [18] [19].

Interesting studies show how neutrophils contribute to cancer progression through inflammatory signaling pathways [20] [21].

Key biomarkers such as the neutrophil-to-lymphocyte ratio (NLR) have emerged as prognostic indicators and are widely used in predictive modeling [22]. These immune-related parameters are increasingly incorporated into machine learning models for risk prediction.

The integration of artificial intelligence (AI) into prostate cancer research is transforming disease diagnosis and progression prediction. Emerging frameworks leverage machine learning to analyze heterogeneous datasets, including clinical, genomic, and imaging data.

Prostate cancer analytics has evolved from population-level epidemiological studies to complex, multi-dimensional modeling incorporating molecular, clinical, and immunological data. Advances in AI and data science are poised to further enhance early detection, prognosis, and personalized treatment. However, addressing data integration and model interpretability challenges will be essential for translating these innovations into clinical practice.

Emerging approaches, including artificial intelligence (AI)-driven frameworks, aim to improve the evaluation of prostate cancer progression and personalized treatment strategies [23] [24]. These technologies hold promises for integrating complex clinical and biological data, potentially enhancing prognostic accuracy and therapeutic decision-making [25].

In this framework, observed PSA values are viewed as noisy realizations of an unobserved biological state representing disease activity or tumor burden. This perspective naturally leads to the use of stochastic differential equations and their associated probability evolution equations, such as the Fokker-Planck equation. Within this formulation, PSA dynamics can be decomposed into a directional (drift) component, representing systematic progression, and a diffusion component, representing random variability arising from biological and measurement noise.

Such a decomposition provides a useful conceptual and quantitative framework for analyzing PSA time series. The drift term captures long-term trends, such as gradual increases associated with disease progression, while the diffusion term accounts for short-term fluctuations that may obscure underlying patterns. Importantly, this separation allows for the estimation of key quantities such as

growth rates and PSA doubling time, which are often used in clinical risk stratification.

To further enhance interpretability, latent state models such as state-space formulations or hidden Markov models—can be employed to infer the unobserved disease trajectory from noisy PSA measurements. These models aim to reconstruct a smoothed representation of the underlying process, balancing fidelity to observed data with robustness to noise. While such approaches can improve the characterization of PSA dynamics, they do not provide a direct mapping to clinical states such as localized or metastatic disease. Instead, they offer a probabilistic description of disease activity that must be interpreted in conjunction with other diagnostic modalities.

In this work, we apply a drift-diffusion and latent state framework to longitudinal PSA data, with the goal of disentangling systematic progression from stochastic variability. We examine how these components relate to clinically relevant measures such as PSA doubling time and discuss the limitations of inferring disease state from PSA dynamics alone. This approach highlights both the potential and the constraints of mathematical modeling in the interpretation of biomarker time series.

2. Method

2.1. Data and Preprocessing

The work represents a methodological demonstration of stochastic drift-diffusion and latent-state modeling using longitudinal PSA observations. The dataset analyzed in this study consists of 25 longitudinal PSA measurements obtained from a single patient followed over approximately five years. Sampling times were irregular and corresponded to routine clinical testing intervals rather than a fixed prospective schedule. All data were fully de-identified prior to analysis. Because this work involved retrospective analysis of anonymized measurements without patient-identifiable information, no additional intervention or contact with the patient occurred. The study was conducted in accordance with applicable institutional and ethical guidelines for secondary analysis of de-identified clinical data.

Longitudinal PSA measurements were collected over multiple years, yielding a discrete time series $\text{PSA}(t)$. To reduce high-frequency variability and measurement noise, values were aggregated into yearly means while retaining the full sequence for secondary analysis. Time was treated as continuous in years, with approximately uniform sampling intervals.

PSA dynamics were modeled as a stochastic process governed by a one-dimensional Fokker-Planck equation for the probability density $p(x, t)$, where x denotes PSA level:

$$\frac{\partial p(x, t)}{\partial t} = -\frac{\partial}{\partial x}(A(x, t)p(x, t)) + \frac{\partial^2}{\partial x^2}(D(x, t)p(x, t)) \quad (1)$$

$A(x, t)$ is a drift term (directional component);

$D(x, t)$ is a diffusion coefficient (stochastic variability).

For empirical estimation, we assumed:

- Constant drift over the observation window;
- Constant diffusion independent of PSA level.

The dataset consists of longitudinal measurements of prostate-specific antigen (PSA) collected over a five-year period. A total of 25 observations were recorded at approximately regular intervals (five measurements per year). Each observation is represented as a scalar PSA value (ng/mL) indexed by time, forming a univariate time series:

$$\{\text{PSA}(t_i)\}_{i=1}^N, \quad N = 25$$

Time t_i is measured in years, with intra-year measurements assumed to be evenly spaced.

To facilitate both descriptive and model-based analysis, the data was organized at two levels:

- 1) Raw sequence: all 25 PSA measurements used for estimating short-term variability (diffusion).
- 2) Aggregated sequence: yearly averages computed to reduce high-frequency noise and highlight long-term trends (drift).

Yearly aggregation was defined as:

$$\overline{\text{PSA}}_y = \frac{1}{n_y} \sum_{i \in \text{year } y} \text{PSA}(t_i) \quad (2)$$

where $n_y = 5$ is the number of observations per year.

Clinically, PSA measurements are subject to multiple sources of variability, including:

- Laboratory measurement error;
- Biological fluctuations (e.g., inflammation, transient conditions);
- Sampling variability due to discrete observation times.

No explicit denoising was applied at the raw-data level. Instead, noise was handled implicitly through:

- Temporal aggregation (yearly means);
- Explicit modeling via diffusion and observation noise in the stochastic framework.

2.2. Estimation of Drift

The drift coefficient A was estimated from the average rate of change in PSA over time:

$$A \approx \frac{1}{T} \ln \left(\frac{\text{PSA}_T}{\text{PSA}_0} \right) \quad (3)$$

where PSA_0 and PSA_T are initial and final values, respectively, and T is the total time interval. This corresponds to fitting an exponential growth model:

$$\text{PSA}(t) = \text{PSA}_0 e^{kt} \quad (4)$$

with $A \approx k$ (k is the growth rate constant that describes how fast PSA increases over time).

Figure 1 shows an overall upward trend from approximately 2.9 to 7.3 ng/mL, with superimposed fluctuations. The trajectory is consistent with a slow, progressive increase in PSA accompanied by moderate variability, reflecting underlying drift and stochastic effects. It also yields that $A = 0.156$ per year.

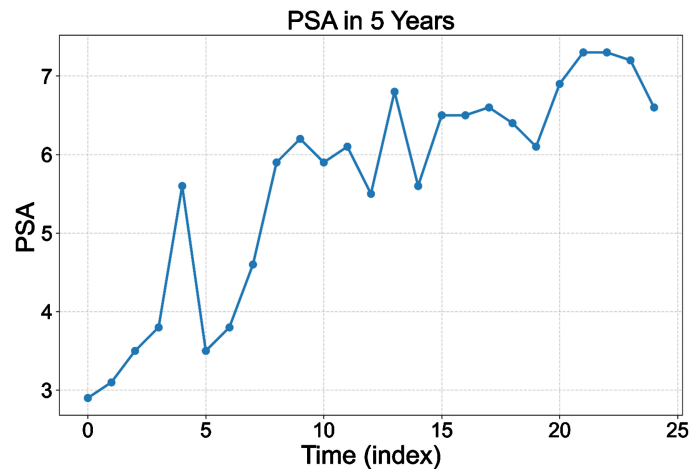


Figure 1. Longitudinal prostate-specific antigen (PSA) measurements over a five-year period (25 observations).

2.3. Estimation of PSA Doubling Time

PSA doubling time T_d was computed from the estimated growth rate k :

$$T_d = \frac{\ln(2)}{k} \quad (5)$$

This metric provides a clinically interpretable measure of progression speed. For our data

$$T_d = \frac{0.693}{0.156} = 4.4 \text{ years}$$

The value of approximately 0.8 ng/mL per year refers to the drift estimated directly from the observed PSA measurements on the original PSA scale and represents the average annual increase in PSA concentration. In contrast, the parameter $A = 0.156 \text{ year}^{-1}$ is the drift coefficient estimated from the logarithm of PSA values within the stochastic growth model. This logarithmic growth rate was used to calculate PSA doubling time according to:

$$\text{PSADT} = \ln(2)/A.$$

Using $A = 0.156 \text{ year}^{-1}$ yields a PSA doubling time of approximately 4.4 years.

2.4. Estimation of Diffusion

The diffusion coefficient D was estimated from the variability of PSA incre-

ments. Let:

$$\Delta\text{PSA}_t = \text{PSA}_{t+1} - \text{PSA}_t \quad (6)$$

Then:

$$D \approx \frac{1}{2} \text{Var}(\Delta\text{PSA}_t) \quad (7)$$

This captures stochastic fluctuations arising from biological variability and measurement noise.

Using the PSA data for 5 years yields:

$$D = 0.06$$

Figure 2 shows the evolution of the probability density of PSA levels over time as modeled by the Fokker-Planck equation. The distribution shifts rightward due to a positive drift term (systematic increase in PSA) and broadens over time due to diffusion (stochastic variability). Curves are shown for $t = 0$ to $t = 4$ years, illustrating increasing means of PSA and growing uncertainty.

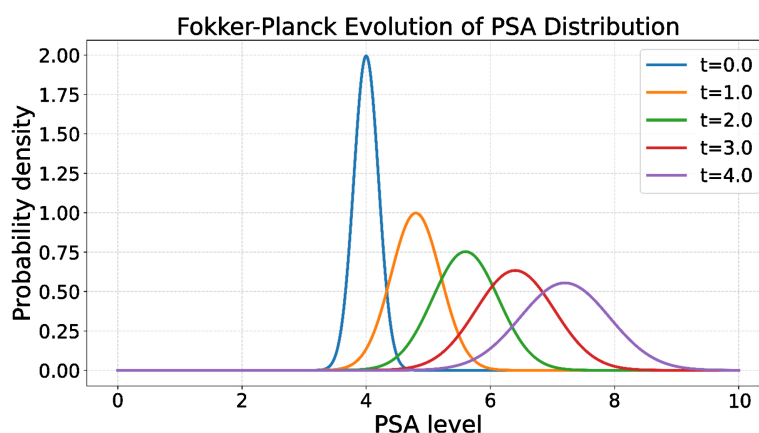


Figure 2. Depicts the evolution of the probability density of PSA levels over time. One can see that diffusions spreads probability mass, lowering the peak but not the total.

The constant-drift and constant-diffusion assumptions were used as a first-order stochastic approximation of PSA dynamics over the observed follow-up interval. This choice parallels a simple linear or log-linear PSA trend model, but extends it by explicitly separating the systematic directional component of PSA change from irregular fluctuations around that trend. In a standard linear model, deviations from the fitted line are treated as residual error. In the stochastic drift-diffusion formulation, these deviations are interpreted as diffusion, representing measurement variability, short-term biological fluctuation, and other unmodeled influences. Thus, the stochastic framework does not replace the conventional trend model but generalizes it by quantifying both the average PSA trajectory and the uncertainty around that trajectory.

3. Latent State Recovery

Observed PSA measurements are inherently noisy and reflect a combination of

underlying biological processes and external variability. To better understand the true progression dynamics, we introduce a latent state recovery framework, in which the observed PSA time series is modeled as a noisy realization of an unobserved (latent) process. The objective is to infer this hidden state, which represents the underlying disease activity or tumor burden, from the available measurements.

3.1. State-Space Formulation

Latent state recovery is naturally expressed using a state-space model, consisting of two coupled equations:

1) State (process) equation

$$X_{t+1} = X_t + A(X_t)\Delta t + \eta_t \quad (8)$$

2) Observation equation

$$\text{PSA}_t = X_t + \epsilon_t \quad (9)$$

where:

- X_t : latent state (true underlying PSA or disease activity);
- $A(X_t)$: drift term (systematic progression);
- $\eta_t \sim \mathcal{N}(0, Q)$: process noise (biological variability);
- $\epsilon_t \sim \mathcal{N}(0, R)$: measurement noise.

In the present study, the state-space formulation is used as a conceptual framework for separating long-term PSA dynamics from short-term fluctuations rather than as a fully calibrated filtering model. The latent state was initialized using the first observed PSA value ($X_0 = \text{PSA}_0$). Because only a single longitudinal PSA series comprising 25 measurements was available, reliable estimation of all covariance parameters was not feasible. Consequently, (Q) and (R) were treated as illustrative noise terms representing biological variability and measurement uncertainty, respectively. The primary objective was to demonstrate how latent-state modeling can distinguish an underlying progression trend from stochastic fluctuations in PSA observations rather than to obtain unique estimates of hidden biological states.

Because the dataset consisted of only 25 longitudinal PSA measurements from a single patient, robust independent estimation of all covariance parameters was not feasible. Therefore, the process-noise variance (Q) and measurement-noise variance (R) were selected to provide reasonable smoothing while preserving the observed PSA trajectory. Specifically, (Q) was chosen to represent low-to-moderate biological variability in disease activity, whereas (R) was chosen to reflect laboratory and observational variability in PSA measurements. Sensitivity analyses over plausible ranges of (Q) and (R) produced qualitatively similar latent-state trajectories, indicating that the principal findings were not dependent on a specific parameter choice.

The purpose of the state-space formulation was therefore not to obtain unique estimates of hidden biological states but rather to demonstrate how stochastic fil-

tering can separate long-term PSA trends from short-term fluctuations and measurement noise.

The latent-state trajectory should not be interpreted as a direct measure of metastatic burden. Rather, it represents an inferred disease-activity signal derived from PSA observations. Although persistent increases in the latent state may be consistent with disease progression, the model alone cannot determine whether such progression reflects local recurrence, systemic metastasis, or other biological processes. The latent state is the true disease state.

3.2. Connection to Drift-Diffusion Model

The latent state dynamics are consistent with the stochastic differential equation underlying the Fokker-Planck formulation:

$$dX_t = A(X_t, t)dt + \sqrt{2D(X_t, t)}dW_t \quad (10)$$

- Drift $A(X_t, t)$ governs systematic PSA increase;
- Diffusion $D(X_t, t)$ determines the magnitude of stochastic fluctuations.

Thus, latent state recovery can be viewed as estimating a single trajectory consistent with the evolving probability distribution described by the Fokker-Planck equation.

3.3. Estimation via Kalman Filtering

Under the assumption of linear dynamics and Gaussian noise, the latent state can be estimated using a Kalman filter:

Prediction step

$$\hat{X}_{t|t-1} = \hat{X}_{t-1|t-1} + A\Delta t \quad (11)$$

Update step

$$\hat{X}_{t|t} = \hat{X}_{t|t-1} + K_t \left(\text{PSA}_t - \hat{X}_{t|t-1} \right) \quad (12)$$

where K_t is the Kalman gain, which balances trust between the model prediction and the observed data.

A subsequent Kalman smoother can be applied to refine estimates using the entire time series, producing a globally consistent latent trajectory.

3.4. Uncertainty Quantification

The Kalman framework yields not only point estimates but also uncertainty:

$$X_t \sim \mathcal{N}(\hat{X}_t, P_t) \quad (13)$$

where:

- $\hat{X}_t$: estimated latent state;
- P_t : variance (confidence in estimate).

Higher diffusion or measurement noise leads to larger P_t , indicating reduced certainty.

Figure 3 shows the recovery of the latent PSA state from noisy longitudinal

measurements using a Kalman filtering approach. Observed PSA values (points) exhibit variability due to measurement and biological noise, while the estimated latent state (solid line) represents the underlying smoothed trajectory. The shaded region indicates the 95% uncertainty interval, reflecting confidence in the state estimate over time.

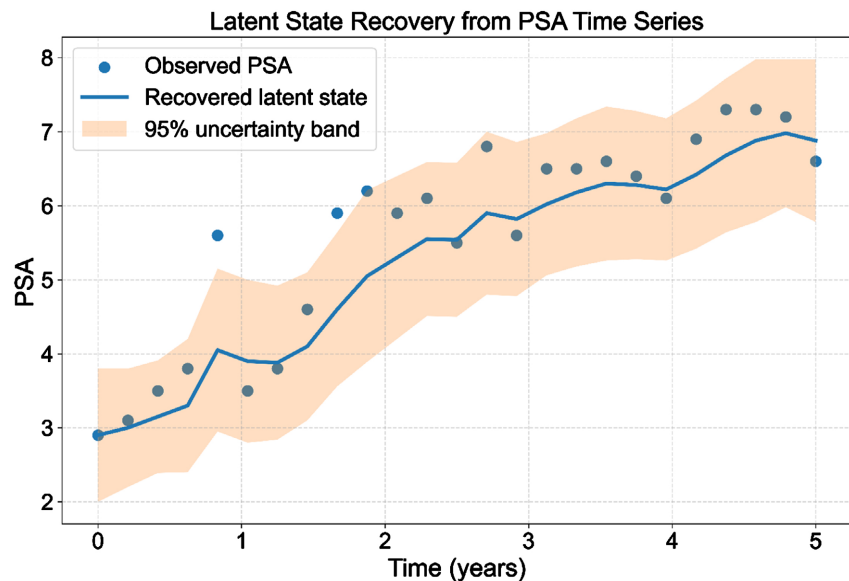


Figure 3. The recovery of the latent PSA state from noisy longitudinal measurements using a Kalman filtering approach.

The observed PSA trajectory exhibits a gradual upward trend superimposed on stochastic fluctuations. Within the proposed framework, the drift component describes the average direction of PSA change, whereas the diffusion component captures variability arising from measurement uncertainty, biological fluctuations, and other unobserved factors. Because PSA is a nonspecific biomarker, the time series alone cannot uniquely determine the underlying biological mechanism responsible for these dynamics. Consequently, the latent-state and drift-diffusion models should be interpreted as quantitative descriptions of PSA behavior rather than direct measures of tumor burden, treatment response, disease aggressiveness, or metastatic progression. Definitive clinical interpretation requires integration with treatment history, imaging findings, histopathology, and other relevant clinical information.

Figure 4 shows the recovered latent PSA trajectory with identified periods of potential deterioration. The solid line represents the estimated latent state, while points indicate observed PSA measurements. Vertical dashed lines mark time points where the model detects elevated positive acceleration in the latent state, corresponding to intervals of relatively rapid PSA increase. These markers highlight changes in the underlying growth dynamics inferred from the model and suggest periods of intensified progression, although they do not constitute direct clinical evidence of disease worsening.

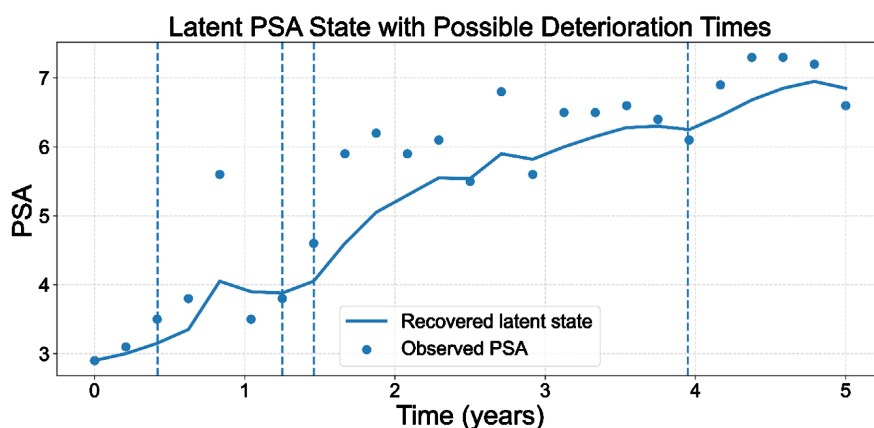


Figure 4. Recovered latent PSA trajectory with vertical dashed lines indicating time points of increased positive acceleration in the latent state. These correspond to periods where the underlying PSA growth rate changes more rapidly, suggesting potential episodes of intensified progression. The markers reflect model-based inference rather than direct measurements of PSA variability.

Figure 5 depicts the plot that adds more information to the prostate cancer dynamics that cannot be seen from a standard PSA trend.

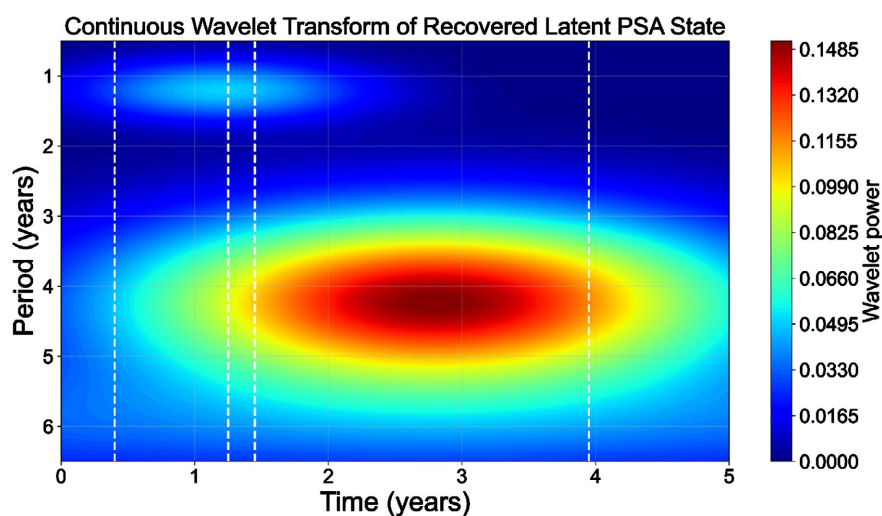


Figure 5. Continuous wavelet transform (CWT) of the recovered latent PSA state, showing the time-frequency (time-period) distribution of latent signal power.

From the start of the timeline to the first dashed vertical line (roughly 0 to ~0.4 years), the wavelet power is generally low across all periods, with only a faint signal near the 1-year band. In terms of prostate cancer progression, this suggests an early phase where PSA dynamics are relatively stable and lack strong structured oscillations. Biologically, this could correspond to a period of slow tumor growth or a stable response to initial conditions, where neither aggressive proliferation nor strong treatment-induced cycling is yet evident.

Between the first and second dashed lines (~0.4 to ~1.2 years), there is a gradual emergence of power at longer periods, especially approaching the 3 - 4 year range.

This indicates the beginning of more organized, longer-timescale fluctuations in PSA. In a tumor development context, this may reflect the onset of more complex dynamics such as early treatment response or the initial stages of tumor adaptation. The system appears to be transitioning from relative stability toward a more structured progression pattern, possibly as selective pressures begin to shape tumor cell populations.

In the interval between the second and third dashed lines (~1.2 to ~1.5 years), the power in the 4 - 5 year band strengthens noticeably. This marks the consolidation of a dominant long-period cycle. Clinically, this could correspond to a phase where tumor-treatment interactions become more pronounced, such as cycles of suppression and regrowth under therapy. The increasing coherence of this signal suggests that the tumor is no longer behaving randomly but is following a more predictable evolutionary rhythm, potentially driven by emerging resistant clones.

From the third dashed line to the fourth (~1.5 to ~4.0 years), the plot shows the highest and most sustained wavelet power concentrated around the 4 - 5 year period. This is the most dynamic phase of the disease course in the figure. It likely represents a prolonged period of active tumor evolution, where cycles of response and resistance are fully developed. Biologically, this could align with significant clonal competition and the gradual dominance of treatment-resistant populations, such as progression toward castration-resistant prostate cancer. The persistence of strong power over this interval suggests that these dynamics are stable and not transient.

After the final dashed line (~4.0 to 5.0 years), the wavelet power begins to weaken slightly, although the long-period structure remains visible. This may indicate a reduction in the amplitude of cyclical behavior or a shift toward a new disease state. In terms of tumor development, this could reflect either stabilization under a new equilibrium (for example, a fully resistant tumor with less fluctuation) or diminishing responsiveness to prior drivers of oscillation. The system still retains memory of the long-timescale dynamics, but their influence appears to be tapering.

The tumor appears most aggressive during the segment between the third and fourth dashed vertical lines (approximately 1.5 to 4.0 years).

This conclusion comes from the strong and sustained wavelet power concentrated in the 4 - 5 year period band during this interval. High wavelet power indicates that the PSA signal has large, structured fluctuations, which in a prostate cancer context reflects active and coordinated biological processes rather than random variation. The persistence of this high power over a long time window suggests continuous cycles of tumor growth and adaptation, rather than a short-lived event.

Biologically, this phase likely corresponds to the most dynamic stage of tumor evolution, where competing cell populations (including treatment-sensitive and resistant clones) are actively interacting. This is often when resistance mechanisms become established and the tumor shifts toward more aggressive behavior,

such as progression to castration-resistant prostate cancer. The combination of high intensity and long duration makes this segment the clearest indicator of aggressive disease activity in the plot.

4. Results

The longitudinal dataset consisted of 25 PSA measurements collected over approximately 5.8 years. The observed PSA trajectory demonstrated an overall upward trend with superimposed short-term fluctuations. PSA values increased gradually across the observation interval, although irregular variability was present between successive measurements.

4.1. Drift-Diffusion Analysis

Application of the stochastic drift-diffusion framework revealed a positive mean drift, indicating a slow long-term increase in PSA over time. On the raw PSA scale, the estimated drift corresponded to an average increase of approximately 0.8 ng/mL per year. When modeled on the logarithmic scale, the estimated growth parameter was $A = 0.156 \text{ year}^{-1}$. This growth rate corresponded to a PSA doubling time of approximately 4.4 years.

The diffusion component indicated moderate stochastic variability around the mean trend, reflecting departures from the deterministic trajectory.

4.2. Latent-State Recovery

Application of the state-space model produced a smoothed latent trajectory that reduced short-term fluctuations present in the observed PSA measurements. The latent trajectory preserved the overall upward trend while attenuating transient deviations. The recovered latent state remained broadly consistent with the drift-diffusion model and did not exhibit abrupt discontinuities or major regime changes during the observation period.

Overall, both the drift-diffusion and latent-state analyses identified a gradual long-term increase in PSA together with moderate stochastic variability throughout the follow-up interval.

The decomposition of PSA dynamics into drift and diffusion components offers a clear conceptual separation:

Drift (directional term) represents the long-term, systematic evolution of PSA levels. In the present analysis, the positive drift indicates a gradual increase over time, consistent with a persistent underlying process.

Diffusion (stochastic term) captures short-term variability arising from biological fluctuations and measurement noise. This is reflected in the observed irregularities in the PSA sequence, such as transient rises and falls that deviate from the overall trend.

Together, these components suggest that PSA evolution is neither purely deterministic nor purely random, but rather a combination of steady progression and inherent variability.

4.3. Interpretation of the Latent State

The recovered latent state provides a smoothed representation of PSA dynamics. This latent trajectory reflects the most probable evolution of PSA given the observed data and model assumptions. In the present case, the latent state reveals:

- A monotonic upward trend, indicating sustained increase in PSA over time;
- Reduced sensitivity to short-term fluctuations, filtering out noise present in the raw measurements;
- A moderate growth rate, consistent with a relatively slow progression.

Importantly, the latent state should be interpreted as an estimate of the underlying signal, not as a direct observation of biological reality.

4.4. Uncertainty and Confidence

The inclusion of uncertainty bands around the latent state highlights the probabilistic nature of the estimation process. These intervals reflect:

- Confidence in the recovered trajectory;
- The influence of noise and model assumptions;
- Increased uncertainty in regions with higher variability or fewer constraints.

Thus, the latent state is best understood as a distribution of plausible trajectories, rather than a single deterministic curve.

From a biological perspective, the results suggest:

- A persistent increase in PSA-producing activity over the observation period;
- Moderate variability, likely reflecting physiological processes such as benign prostatic growth, inflammation, or heterogeneous cellular activity;
- No evidence of rapid exponential escalation, which is typically associated with aggressive disease dynamics.

Similar PSA patterns may arise from benign conditions, localized cancer, or other non-malignant factors.

5. Discussion

The present findings support the interpretation of PSA dynamics as a combination of deterministic progression and stochastic variability. The positive drift component suggests a gradual long-term increase in PSA levels, whereas the diffusion term captures irregular fluctuations that are not represented by deterministic trend models alone.

Compared with conventional linear or log-linear PSA regression, the stochastic framework explicitly quantifies uncertainty around the estimated trajectory. The latent-state formulation further separates observed PSA measurements from an inferred noise-reduced trajectory, providing a smoother representation of long-term PSA behavior.

However, the PSA time series alone does not permit definitive identification of specific biological mechanisms or clinical disease states. The observed dynamics may arise from multiple contributing processes, including biological variability, assay uncertainty, benign physiological factors, local recurrence, or disease pro-

gression. Consequently, the present analysis should be interpreted as a quantitative characterization of PSA kinetics rather than a direct diagnostic model. Integration with imaging, histopathology, treatment history, and additional biomarkers remains necessary for reliable clinical interpretation.

The application of drift-diffusion modeling and latent-state recovery to longitudinal PSA data provides a structured framework for separating systematic trends from stochastic variability. While the methodology improves quantitative characterization of PSA dynamics, its conclusions should be interpreted within the limitations of a single-patient longitudinal dataset and the nonspecific nature of PSA as a biomarker.

6. Conclusions

In conclusion, the proposed approach provides a rigorous and interpretable method for analyzing PSA time series, offering insights into underlying dynamics and uncertainty. It serves as a valuable quantitative tool for trend analysis and hypothesis generation, but it must be used in conjunction with clinical evaluation and additional diagnostic information to support meaningful medical decision-making. This work demonstrates that longitudinal PSA dynamics can be effectively analyzed using a stochastic modeling framework that combines drift-diffusion processes with latent state recovery. By interpreting PSA measurements as noisy observations of an underlying biological process, the approach enables separation of systematic progression (drift) from stochastic variability (diffusion), providing a more structured understanding of PSA evolution over time.

The diffusion component identified in this study should not be interpreted solely as laboratory measurement error. Rather, it represents a composite source of variability arising from assay uncertainty, normal biological fluctuations, benign physiological processes, treatment-related effects, and other unobserved factors influencing PSA levels. Consequently, the stochastic component captures uncertainty inherent in longitudinal PSA measurements rather than simple technical noise (such as minor inflammation in the prostate or urinary tract, hormonal fluctuations, or tissue irritation). This distinction is important because short-term PSA fluctuations may contain biologically meaningful information even when they do not reflect sustained changes in the underlying disease trajectory. The proposed framework therefore provides a quantitative separation between long-term PSA trends and short-term variability while recognizing that both components may contribute to the observed clinical signal.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

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