1. The Game of Memory

Imagine that inside every brain there lies an enormous library — millions of shelves, billions of books, and pathways connecting every room. Now imagine a peculiar rule: each time you revisit a book, its pages brighten; each time you ignore it, the ink fades. One day, you start noticing that some rooms are dimmer than others, and some books vanish entirely. This "game" — watching memories weaken, fragment, or disappear — is not just a biological phenomenon. It is a mathematical one. To explore dementia from a mathematical perspective, let us pose the following question:

If a memory is a mathematical quantity, what rules determine how fast it fades? And what happens to the network of memories as more pieces are lost?

At first glance, it might appear impossible to express something as human as forgetting in equations. Yet, as we will soon see, memory decay follows patterns found in exponential functions, probability theory, graph connectivity, and even stochastic processes. Each of these tools allows us to reframe dementia not merely as the deterioration of cells, but as the collapse of a mathematical structure.

Consider a simplified model of human memory in which each memory may be represented by a mathematical object:

$$M = (c, w, r, t)$$

where

- c denotes the informational content,
- $w \in \mathbb{R}^+$ is the memory's current strength or weight,
- $r \in [0,1]$ is its instantaneous recall probability, and
- t is the time elapsed since encoding.

This object evolves over time according to deterministic and stochastic rules that govern how biological memory degrades. The central question motivating this essay is the following:

Given a memory represented by $M(t)$, what mathematical laws determine its decay, recall probability, and eventual disappearance under dementia?

To build intuition, imagine an enormous internal library containing millions of interconnected "books." Each time a book is retrieved, its weight w increases; each time it is forgotten, w decays. As t increases, some books dim, others vanish, and connections between shelves begin to break. This qualitative metaphor corresponds precisely to quantitative decay laws.

In this essay, we will step through these mathematical ideas — from algebraic decay to network theory — and ultimately see how they informed the design of a real-world assistive technology: AlzCare, a smart pill system built to reduce medication errors for people living with dementia.

1. Exponential Forgetting: Memory as a Decaying Function

To build on the conceptual model introduced in the previous section, consider a single memory — for instance, the name of a relative or the location of a familiar object. We can formalize its strength at time $t$as a real-valued function:

$$M(t),$$

where $M(t) \in [0,1]$represents the accessibility or intensity of the memory at time t.

A simple first-order approximation assumes that memory fades continuously over time, with a rate proportional to its current strength. This leads naturally to the **exponential decay differential equation**:

$$\frac{dM}{dt} = -\lambda M,$$

where:

- $M$is the memory strength,
- $\lambda > 0$is the decay constant, determining how quickly the memory fades.

Solving this ordinary differential equation yields the classical exponential forgetting curve:

$$M(t) = M(0)e^{-\lambda t},$$

where $M(0)$represents the initial memory strength.

In healthy individuals, $\lambda$is small, reflecting gradual forgetting. Memories diminish slowly over time, and repetition or reinforcement can temporarily restore strength. In dementia, however, the effective decay rate increases significantly; λmay rise such that memories that previously persisted for months or years now decay within days or even hours.

This exponential formulation captures a crucial insight: **forgetting is not random** — it follows a predictable mathematical law. The decay curve provides a quantitative measure of memory reliability over time and establishes a foundation for more advanced probabilistic modeling.

Yet, real-world cognitive decline is more complex than this simple model. Not all memories decay at the same rate; some are remarkably resilient while others vanish abruptly. Furthermore, memory retrieval is probabilistic — even a memory that exists may fail to be recalled reliably. To capture these nuances, we must incorporate probability theory into our analysis, defining the **probability of recall** as a function of memory strength:

$$P_r(t) = 1 - \exp\left(-k\, M(t)\right),$$

where $k$is a sensitivity constant reflecting how accessible the memory is.

This formulation allows us to model both the deterministic decay of memory and the stochastic nature of recall. It lays the groundwork for understanding critical thresholds of memory strength — the points at which memories become effectively inaccessible — and ultimately connects to clinical observations in dementia.

2. Probability of Recall

Memory recall is inherently probabilistic, meaning that even if a memory exists in the brain, the likelihood of retrieving it at a given time is not guaranteed. This can be formally expressed as:

$$Pr(t) = 1 - \exp\left(-kM(t)\right),$$

where $M(t)$represents the strength of the memory at time $t$and $k > 0$is a parameter that quantifies retrieval sensitivity. The function shows that as memory strength $M(t)$decreases, the probability of successfully recalling the memory also decreases. There is a critical memory threshold, M_c, below which recall becomes effectively negligible:

$$M(t) \leq M_c \Rightarrow Pr(t) \approx 0.$$

This threshold reflects the point at which a memory is effectively lost for practical purposes, even if traces of it still exist in the brain. To illustrate, consider a numerical example where the initial memory strength is $M(0) = 1$, the decay rate is $\lambda = 0.05$, and the retrieval sensitivity is $k = 3$. The half-recall time, T_c, is the time at which the probability of recall drops to 50%, defined by:

$$1 - e^{-3e^{-0.05T_c}} = 0.5.$$

Solving this equation gives $T_c \approx 13.86$. This means that after approximately 13.86 time units, the memory has decayed enough that there is only a 50% chance of recalling it. In this way, the model formalizes the concept of memory fading and provides a quantitative framework for understanding how recall probability declines over time, capturing the interplay between memory strength, decay, and retrieval sensitivity.

3. The Brain as a Network

The brain can be understood as a complex network, with memories encoded within a dynamic web of neurons and their connections. Formally, this neural network can be represented as:

$$G(t) = (V(t), E(t), w(t)),$$

where $V(t)$ is the set of neurons, $E(t)$ represents the synaptic connections between them, and $w(t)$ denotes the strengths or weights of these connections. Memories are stored as patterns of activation across this network, and the integrity of the network is essential for proper cognitive function.

In conditions such as dementia, this network gradually deteriorates. Neurons are lost at a rate α, and synapses at a rate β, leading to a shrinking network over time:

$$|V(t)| = |V(0)| - \alpha t, |E(t)| = |E(0)| - \beta t.$$

As neurons and synapses disappear, the network becomes fragmented. One important concept in network theory is the giant component, which is the largest connected subgraph of the network. In a healthy brain, the giant component spans most of the network, allowing efficient communication between neurons. However, as nodes and edges are progressively lost due to neurodegeneration, the giant component collapses.

The breakdown of this connected structure mirrors clinical cognitive decline, as the ability of the brain to integrate information diminishes. Functions that rely on widespread neural coordination, such as memory, attention, and reasoning, are disproportionately affected.

By modeling the brain as a network, researchers can quantify the relationship between structural loss and cognitive impairment. This framework provides both a theoretical understanding of neurodegenerative processes and a potential guide for interventions aimed at preserving network connectivity to slow the progression of dementia.

4. Stochastic Progression via Markov Chains

Cognitive decline in neurodegenerative diseases is highly variable, both across individuals and over time. One way to model this variability is through a Markov chain, where the disease is represented as a sequence of discrete cognitive states. In a simple model, we can define three states: S_0 for mild impairment, S_1 for moderate dementia, and S_2 for severe dementia. The progression between these states is probabilistic and captured by a transition matrix:

$$P = \begin{pmatrix} 1-p_0 & p_0 & 0 \\ 0 & 1-p_1 & p_1 \\ 0 & 0 & 1 \end{pmatrix}.$$

Here, p_0 is the probability of progressing from mild to moderate impairment, and p_1 the probability of moving from moderate to severe dementia. The final state, S_2, is absorbing, meaning that once reached, further progression is irreversible. To account for patient variability, the transition probabilities can be modeled as stochastic variables:

$$p_i = \mu_i + \sigma_i Z, Z \sim N(0,1)$$

where μ_i is the average progression rate, σ_i captures variability, and Z is a standard normal random variable. This formulation reflects that some patients decline rapidly, while others progress more slowly.

Over repeated iterations, the Markov chain predicts that all patients will eventually reach the absorbing state S_2, consistent with the irreversible nature of severe dementia. This framework allows researchers and clinicians to simulate disease trajectories, estimate expected time spent in each cognitive state, and assess interventions aimed at slowing progression. Markov modeling thus provides a rigorous mathematical approach to capturing the stochastic and patient-specific nature of cognitive decline.

5. Medication Risk and AlzCare

Memory decline significantly affects a patient's ability to adhere to medication regimens, which can have serious clinical consequences. To formalize this, we define the risk of mismanagement as:

$$R(t) = P_m(t) \cdot C, P_m(t) = 1 - P_r(t),$$

where $P_m(t)$ is the probability of forgetting a dose, $P_r(t)$ is the probability of recalling the medication at time t, and C represents the clinical consequence associated with missed or incorrect dosing. Incorporating memory decay over time, we can express recall probabilistically as $P_r(t) = 1 - \exp(-kM(t))$, where $M(t)$ denotes memory strength and k is a retrieval sensitivity parameter. Substituting this into the risk equation gives:

$$R(t) = e^{-kM(0)e^{-\lambda t}} C,$$

highlighting that as memory weakens, the risk of mismanagement grows exponentially. This emphasizes the need for timely interventions to prevent adverse outcomes.

AlzCare is an AI-enabled pill dispenser designed to mitigate these risks by integrating memory-aware predictive modeling with automated adherence support. The system is structured as a discrete-event system, where each action—dispensing a dose, retrieving it, or issuing an alert—is modeled as a state transition:

$$S_{t+1} = f(S_t, E_t),$$

with S_t representing the current state and E_t external events, such as sensor inputs or patient interactions. Dosing schedules are maintained through modular arithmetic, ensuring doses occur at regular intervals:

$$t \equiv 0 (\mathrm{mod} h),$$

where h is the prescribed dosing interval. Threshold sensors confirm whether the patient has retrieved the medication:

$$\text{Retrieve}(t) = \begin{cases} 1 & \text{if sensor reading} \geq \theta \\ 0 & \text{otherwise} \end{cases},$$

where θ is a predefined retrieval threshold.

Beyond immediate monitoring, AlzCare employs predictive AI to estimate future mismanagement probabilities:

$$P_{\text{future}} = g(M(t), R(t), \text{sensor history}),$$

leveraging real-time memory estimates, current risk levels, and historical adherence data. This allows proactive interventions, such as reminders, caregiver alerts, or adjusted dosing schedules, tailored to the patient's cognitive state.

By integrating memory decay models with stochastic risk assessment and AI-driven automation, AlzCare provides a comprehensive, patient-centered approach to medication management. This system not only reduces the probability of missed doses but also enables clinicians to quantify and respond to adherence risk dynamically, ensuring better outcomes for individuals with cognitive decline.

6. **Conclusion**

Dementia can be understood as a complex interaction between multiple mathematically describable processes. First, the exponential decay of memory strength models how individual memories weaken over time, capturing the predictable yet accelerating loss associated with neurodegeneration. Second, probabilistic recall formalizes the inherent uncertainty in retrieving memories, illustrating that even partially retained information may fail to be accessed reliably. Third, network degradation represents the progressive loss of neurons and synaptic connections within the brain's neural graph, highlighting how fragmentation of the giant component corresponds to global cognitive decline. Finally, stochastic state transitions, as captured through Markov chains, model the variability of disease progression across individuals, accounting for the probabilistic and irreversible nature of dementia's trajectory.

By integrating these frameworks, it becomes possible to quantify risk, anticipate cognitive decline, and simulate patient-specific trajectories, providing actionable insight for interventions. This mathematical understanding directly informs technologies such as *AlzCare*, which applies decay models, probabilistic thresholds, and network considerations to optimize medication adherence and reduce clinical risk. In this sense, mathematics transcends theoretical description, offering practical tools to anticipate, mitigate, and manage the consequences of forgetting, bridging abstract models with real-world patient care and enabling evidence-driven solutions for those affected by dementia.