Ribosome Role in Regulating Concurrency of mRNA Translation

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Abstract. *mRNA translation involves complex concurrent processes in which many mRNA strands might be processed simultaneously, leading to parallel consumption of potentially scarce resources. Previous studies have shown that excess mRNA can harm protein synthesis efficiency. This work investigates the role of ribosomes as concurrency controllers in regulating translation dynamics. Using Petri net modeling, we demonstrate that ribosomes act as natural regulators that limit the detrimental effects of mRNA abundance by controlling the number of concurrent translation events. Our experimental results show ribosome availability is a protective mechanism against greedy consumption patterns that lead to incomplete protein synthesis. This finding provides insights into the regulatory mechanisms maintaining translation efficiency in cellular systems.*

1. Introduction

This study is the latest contribution in a series of efforts [Haeusler et al. 2023, Neto et al. 2024b, Neto et al. 2024a] aimed at using Petri Net formalisms to model processes in molecular biology. The line of research originated with foundational work that proposed an intentional semantics to molecular biology using Object Petri Nets (OPN) [Haeusler et al. 2023], providing a robust framework for modeling codon-level interactions and translation dynamics, by aligning formal concurrency theory with biomolecular processes.

Following that theoretical foundation, subsequent studies have examined the expressive adequacy of Petri Net formalisms to model complex behaviors such as race for resources that may arise during the elongation step, leading to partially translated polypeptides. The present article builds upon this framework by adding the ribosome to the model. In particular, we investigate the regulatory role of ribosomes in controlling the concurrency of translation events, demonstrating that ribosome availability is a natural safeguard against inefficiencies caused by excess mRNA explored in previous works [Neto et al. 2024b].

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Context and Motivation

mRNA translation represents one of the most fundamental processes in molecular biology, where genetic information encoded in mRNA molecules is translated into functional proteins. This process involves complex concurrent interactions between multiple molecular components, including ribosomes, mRNAs, tRNAs, and amino acids. The concurrent nature of biological processes, where various processes co-occur within the cell, introduces the possibility of race conditions that may lead to unexpected results.

Previous computational studies have revealed that excess availability of mRNA can have detrimental effects on protein synthesis efficiency [Neto et al. 2024b]. These effects manifest as non-linear protein production concerning mRNA strand availability, where increasing the available mRNA leaves room for amino acids to be consumed excessively in early translation steps, leaving insufficient resources for completing protein synthesis. This phenomenon underscores the need for regulatory mechanisms to balance translation efficiency and resource availability.

Ribosomes, as a relevant molecular translation machine, play a crucial role in controlling the concurrency of mRNA translation processes. Their availability and distribution across mRNA molecules serve as natural regulators that can mitigate the detrimental effects of mRNA abundance. Understanding this regulatory role is essential for understanding how cells maintain translation efficiency under varying conditions.

1.1. Objectives

This work aims to:

1. Quantify the protective effects of ribosome availability against detrimental mRNA abundance
2. Establish the mechanistic understanding of ribosome-mediated concurrency control
3. Demonstrate how ribosome distribution regulates translation efficiency
4. Provide experimental evidence of the ribosome's role as a natural concurrency controller

2. Theoretical Foundation

2.1. Petri Nets for Concurrency Modeling

Petri Nets provide a natural framework for modeling concurrent processes in general, with a vast range of applications, including chemical reaction networks and biological systems. The basic structure of the PNet consists of places (representing resources or states), transitions (representing events or reactions), and tokens (representing quantities or molecules). The firing of transitions consumes tokens from input places and produces tokens in output places. This simple model was proposed in [Petri 1962] (Carl Petri's seminal paper), and a vast literature has followed, encompassing applications, extensions, and analytical results, among other developments. We refer the reader to [Valk 2004, Irina and Vera 2016, Murata 1989, Dworzanski and Lomazova 2016, Venero and da Silva 2014] as a few examples.

For translation modeling, places can represent mRNA molecules, ribosomes, amino acids, and protein products, while transitions represent the various steps of the translation process. The concurrent firing of multiple transitions captures the simultaneous translation of numerous mRNA molecules by various ribosomes.

2.2. Role of Ribosomes in translation

The ribosome is a macromolecular complex responsible for protein synthesis in all living cells. It performs translation, the final step of the central dogma of molecular biology, where messenger RNA (mRNA) is decoded to produce a polypeptide chain (protein). They are composed of two subunits: the large subunit containing peptidyl transferase activity, and the small subunit responsible for decoding the mRNA. Each subunit consists of ribosomal RNA (rRNA) and ribosomal proteins.

The key steps of ribosomal activity comprise: (i) initiation, in which the small ribosomal subunit binds to the 5' untranslated region (UTR) of the mRNA, the initiator tRNA (charged with methionine or N-formylmethionine) binds to the start codon (AUG), and the large subunit joins, forming a active ribosome; (ii) elongation, where the aminoacyl-tRNAs are delivered to the A (aminoacyl) site of the ribosome by elongation factors, the ribosome catalyzes peptide bond formation between the amino acid in the A site and the growing peptide in the P (peptidyl) site using peptidyl transferase activity (a ribozyme function of the rRNA), and translocates along the mRNA (with the help of elongation factors), moving the tRNAs from A $\rightarrow$ P $\rightarrow$ E (exit site), exposing the next codon for decoding; (iii) termination, when a stop codon (UAA, UAG, or UGA) enters the A site, no corresponding tRNA binds, release factors (RFs) bind to the A site promoting hydrolysis of the ester bond linking the polypeptide to the tRNA in the P site, the ribosome disassembles and the newly synthesized polypeptide is released [Noller 2024, Jia et al. 2024].

2.3. Ribosomes as Concurrency Controllers

The key insight from previous work is that the step-by-step nature of mRNA translation raises race conditions if there is too much concurrency. In this sense, given the role of the ribosome in translation as described in section 2.2, ribosomes then act as natural concurrency controllers in translation processes. The idea being that, given the necessity of the presence of the ribosome throughout the entirety of the process, if we have, say, 10 ribosomes in a given system, the maximum amount of mRNA strands that can be processed simultaneously will be 10, regardless of any abundance of strands or amino acids.

3. Translation Modeling with Ribosomes

We kept the codon-level modeling of [Neto et al. 2024b] with the only addition being the ribosome. The ribosome attaches to the mRNA strand during the initiation phase of translation. During the elongation phase, amino acids are consumed. The ribosome-strand compound moves from the state of reading each codon i until it reaches the final codon in the the n^{th} position[1]. In the termination phase, the polypeptide is released, and the ribosome detaches from the strand, as detailed in section 2.2. The ribosome stays in the cell medium and can be reused for other translations[2].

Further analysis of the network in fig. 1 shows that we can simplify it a bit without materially affecting the dynamics of the model by fusing the $attachment$ and $t1$ transitions, leading to the network in fig. 2.

[1]In here we are simplifying the figure to make it easier to read, but the actual model needs a place for each amino acid and that amino acid will be consumed only at the appropriate transitions.

[2]This is not the most realistic assumption, as there are several steps for reusing the ribosome, but what matters for us here is that such a process for reuse exists.

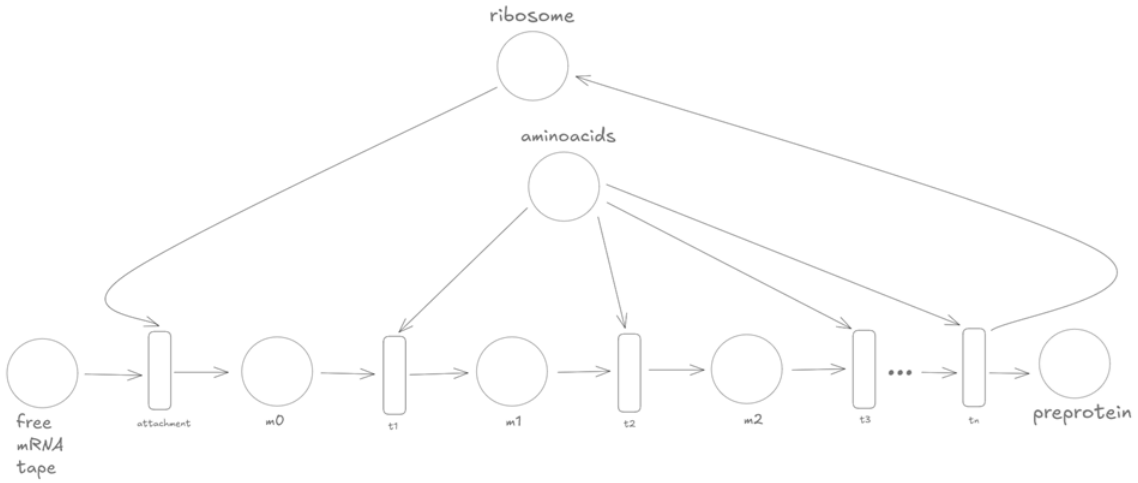

Figure 1. Petri net model for mRNA translation including ribosome dynamics. Places represent molecular components: m_0 (free mRNA strands), m_i (mRNA-ribosome complexes currently at codon i), ribosomes, amino acids, and proteins. Transitions t_i model the sequential codon-by-codon translation process.

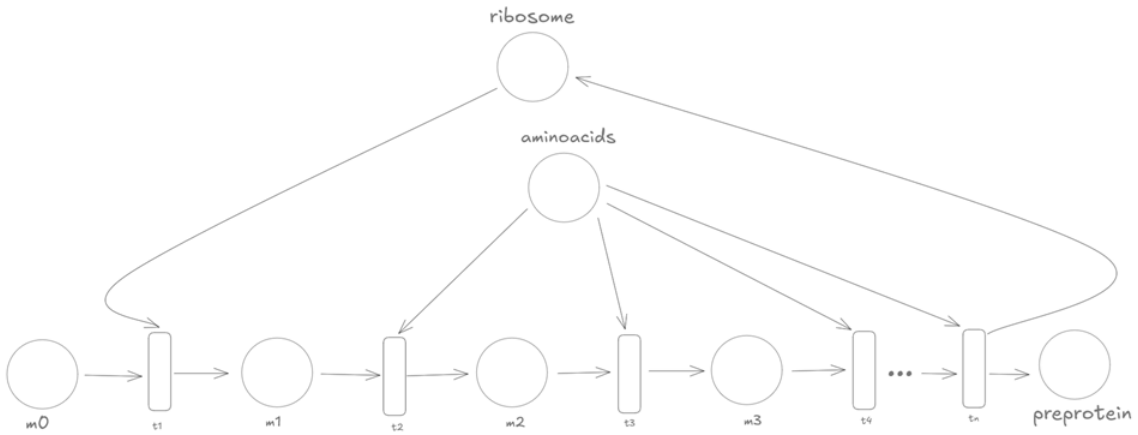

Figure 2. Simplified Petri net model used for simulation experiments. This model merges the attachment and first codon transitions (t_1) for simplicity while preserving the essential dynamics.

For clarity, we have detailed the interpretation of each place here. We will consider tokens in the ribosome, amino acids, and protein places to mean precisely that there are that many molecular compounds in the system. For the mRNA places, we will consider x tokens in place m_0 to mean that there are x "free mRNA tapes" in the system. But for the m_i places, we will consider the y tokens there to mean that the system has y mRNA-ribosome compounds currently reading the i^{th} codon of that particular mRNA tape in the system.

To illustrate, consider a ribosome binding to the free mRNA token in place m_0. Upon firing transition t_1, the system consumes one ribosome and one mRNA token and deposits a new compound token in m_1, representing the ribosome reading the first codon.

As the ribosome progresses along the mRNA, each subsequent transition t_i consumes one specific amino acid (corresponding to codon i) and moves the token from m_i to m_{i+1}, representing elongation through codon-by-codon decoding. This process continues until the final codon is reached at m_n, where the transition labeled t_n consumes the last amino acid, releases a protein token into the product place, and returns the ribosome to the pool of available units by placing a token back in the ribosome place. This cyclic flow ensures that a limited number of ribosomes constrain the total number of concurrent translation processes, enforcing an upper bound on system parallelism regardless of mRNA abundance. By tracing the movement of tokens through these places and transitions, the Petri net captures both the sequential nature of codon processing and the global concurrency limitations imposed by ribosome availability.

3.1. Model limitations

While the current model assumes ribosomes are immediately available for reuse after termination, a delay may exist in biological systems due to ribosome recycling and re-initiation. Such temporal delays in future versions could provide finer control over concurrency effects and better align with known kinetics.

We also acknowledge that the current abstraction simplifies various aspects of translation, such as codon bias, secondary mRNA structures, and tRNA availability. These factors could introduce additional concurrency bottlenecks and consist of possible future work on subsequent model refinements.

4. Methodology

We focused on simulation experiments using a Petri net implementation in Python [Neto 2025]. To run the simulation, we assume a "neutral" dynamic, in which at each epoch, every fireable transition is equally probable to happen[3].

In addition, we used the mRNA sequence for human insulin[4]

We have made the simulation data and the charts generated by plotly in https://github.com/lhcnetop/mrna_ribosome_pnet_simulation_data.

4.1. Experimental Design

We used the code in [Neto 2025] to generate a Petri net JSON specification for the insulin sequence. We fixed the number of amino acids and varied the initial tokens for the "free tape" and ribosome places for comparability. For each run, the amount of each amino acid available was precisely needed to produce 100 insulin proteins. The library used produces a parquet file with the entire history of the simulation. In general, we wanted to capture the behavior around some critical points in the parameter space:

[3]A more realistic dynamic might be to use "mass-action" in which transitions that have more tokens in the preceding places are more likely to happen, but we leave this for future works.

[4]For reference, the sequence is MALWMRLLPL LALLALWGPD PAAAFVNQHL CGSHLVEALY LVCGERGFFY TPKTRREAED LQVGQVELGG GPGAGSLQPL ALEGSLQKRG IVEQCCTSIC SLYQLENYCN [Consortium 2024], accessed in https://www.uniprot.org/uniprotkb/P01308/entry# sequences on 20 July 2025. We chose it due to its smaller size (which helps to speed up the simulation) and its extensive literature. It is worth mentioning that in [Neto et al. 2024b] we concluded that the detrimental effect of mRNA abundance increases with sequence size; therefore, our conclusions should remain even for larger sequences, albeit with slightly different values, but the overall "shape" of the data should remain the same.

- **Control experiments**: For very high (> 1000) ribosome counts, we should get a similar scenario to the experiment in [Neto 2025]
- **High mRNA scenarios**: Excess mRNA (> 100 initial "free tapes") with varying ribosome availability
- **Low ribosome scenarios**: Limited ribosome availability (< 10) with excess mRNA
- **Optimal ratio experiments**: Identification of regions of maximal protein output

Given the random nature of the simulation, each parameter combination was tested with at least 10 independent simulation runs. We calculated the mean protein output across these replicates for each data point.

4.2. Simulation Framework

We ran the Petri net for each parameter for enough steps to complete the process (as the amino acids are fixed and consumed by each transition, we can run for a maximum of *sequence length* $\times$ 100). Once the system has no fireable transitions or it reaches the maximum number of steps, it stops. For each simulation, the entire history of the network state is saved to a Parquet file. We can extract the necessary data for the analysis from these simulation runs.

4.3. Analysis Metrics

Given the various initial parameters, the primary metric we use to analyze the system is the amount of protein tokens at the last step of the simulation (or, equivalently, the maximum amount of tokens for that place in the simulation history). For scenarios where we have more amino acids than mRNA strands, we can normalize the metric to look at the percentage of the theoretical maximum (as in these scenarios, the mRNA strand is the limiting factor).

Below, we find two views of the surface plot generated. The "Excess Factor" axis refers to the ratio of initial "free tape" tokens to amino acids available (fixed at 100 by the experiment design), and the "Avg Max Protein Production" refers to the mean protein output across 10 simulation replicates for each parameter combination, representing the maximum number of tokens in the protein place at simulation completion.

We can see from figs. 3 to 6 that:

- Below the excess factor value of 1, the "free tapes" are the bottleneck for the process, and the protein output is maximal and precisely the amount of mRNA tapes available
- Beyond an excess factor of 1 and with ribosome count below the threshold of the amino acid availability of 100, the detrimental effect of the abundance of mRNA is limited by the restricted concurrency[5], and the system achieves maximum or close to maximal output.
- Until ribosome availability reaches levels close to the expected protein output of 100, the detrimental effect of mRNA abundance is relatively limited, but once ribosome availability crosses that threshold, the effects become more and more pronounced.

[5]We see from the images that the detrimental effect for ribosome availability $\leqslant 100$ remains below 5%. The HTML of the plots can be accessed in https://github.com/lhcnetop/mrna_ribosome_pnet_simulation_data

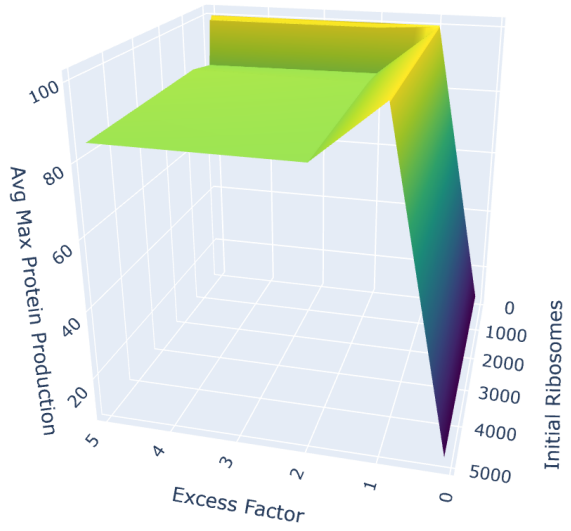

Figure 3. Three-dimensional surface plot of protein production across parameter combinations (view A). X-axis: initial ribosome count; y-axis: mRNA excess factor (ratio of mRNA strands to target protein count of 100); z-axis: average maximum protein production.

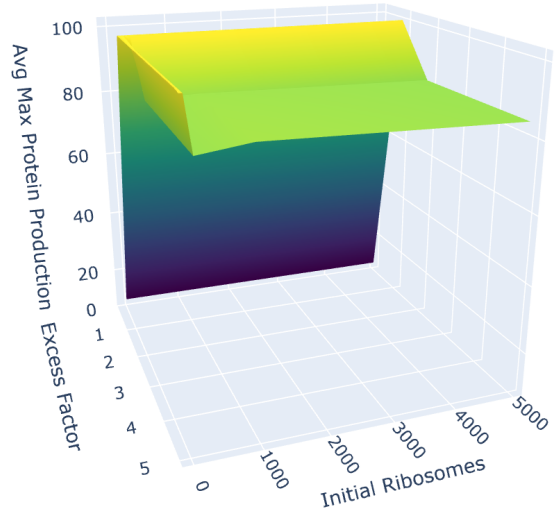

Figure 4. Three-dimensional surface plot from an alternative viewing angle (view B). Same plot as Figure 3 showing the relationship between ribosome count, mRNA excess factor, and protein production.

- For very high ribosome counts, there is no concurrency limitation and the system behaves as detailed in [Neto et al. 2024b].

These can also be seen in fig. 6, where we see that, beyond the excess factor of 1, the system with low ribosome availability and thus limited concurrency remains fairly efficient and nearly optimal. However, the mRNA abundance effect starts to show as the ribosome count increases. However, we also clearly see a dramatic difference in efficiency drop if ribosome availability remains below the 100 threshold. In contrast, the drop becomes much more pronounced when it crosses the threshold.

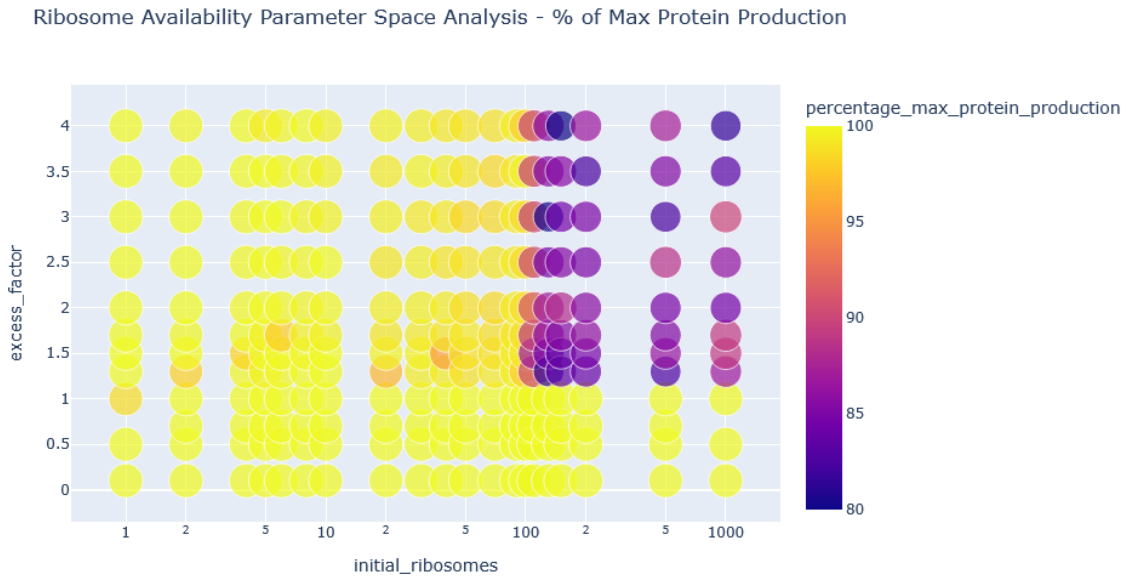

Figure 5. Scatter plot of experimental results. X-axis: initial ribosome count; y-axis: mRNA excess factor; color intensity: protein output (maximum 100). The color scale indicates the level of protein production.

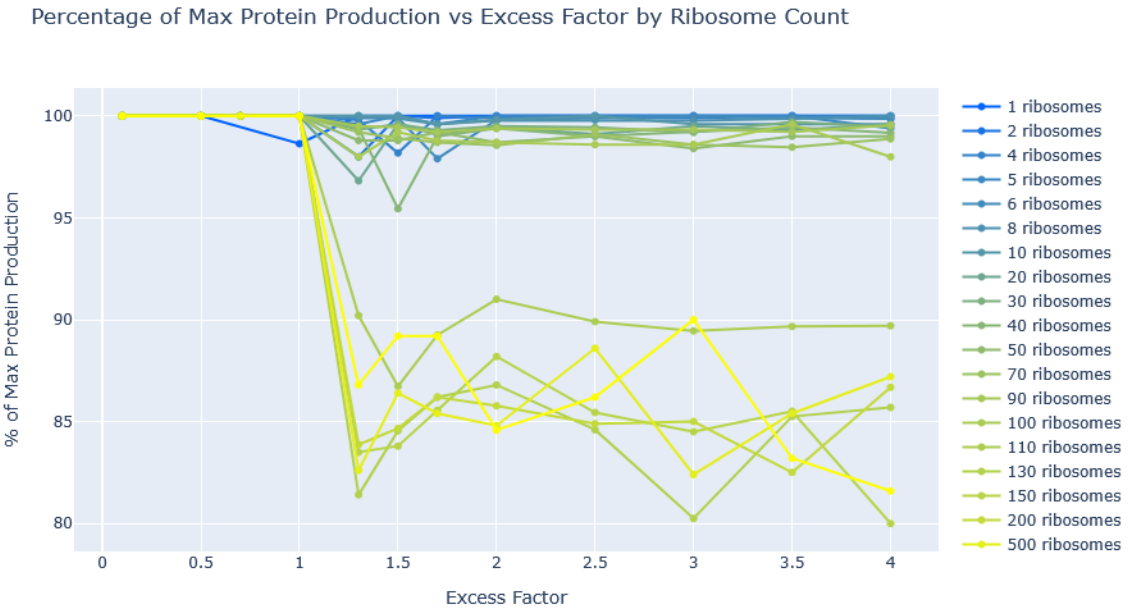

Figure 6. Line plot of protein production versus mRNA excess factor. Each line represents a different initial ribosome count. X-axis: mRNA excess factor; y-axis: protein output.

5. Results and Discussion

The simulation experiments yielded clear evidence of the regulatory role played by ribosomes in controlling the concurrency of mRNA translation and mitigating the detrimental effects of mRNA abundance. The results from the surface plots and line graphs

provide multiple insights into how ribosome availability affects overall protein synthesis efficiency across different regimes of mRNA abundance.

5.1. Ribosome Availability as a Concurrency Constraint

One of the clearest findings is that ribosome availability naturally limits the number of simultaneous translation events, acting as an upper bound on concurrency. In scenarios where the ribosome count is low (e.g., fewer than 10 ribosomes), the number of concurrent translation processes is restricted, effectively preventing the system from greedily consuming its available amino acid resources. This regulatory effect becomes significant when the mRNA excess factor exceeds 1, a situation where previous work has shown that high concurrency can lead to early depletion of amino acids and ultimately incomplete protein synthesis.

The data show that ribosome scarcity serves as a buffer in such conditions. Despite the abundance of mRNA, the bottleneck imposed by limited ribosomes results in more complete and efficient protein production. This result confirms the hypothesis that ribosomes act as natural concurrency controllers, enforcing order on an otherwise greedy resource consumption pattern.

5.2. Efficiency Plateau and Drop-Off Behavior

The protein production curve as a function of mRNA excess factor and ribosome count revealed two important behaviors:

- For excess factor $\leqslant 1$, protein output scaled linearly with mRNA count, as expected. In this scenario, ribosomes did not affect the overall system efficiency, and the bottleneck was the number of available mRNA strands.
- For excess factor > 1, particularly when ribosome availability crossed the threshold of 100 (matching the theoretical protein output maximum), the system began to exhibit a sharp decline in efficiency. This drop-off occurred due to increased concurrency, allowing too many translation processes to initiate simultaneously, quickly exhausting shared amino acid pools before polypeptides could be fully synthesized.

This non-linear behavior demonstrates a critical point in system dynamics, beyond which ribosome availability loses its regulating role and leads to significantly degraded system performance. This observation closely aligns with the concurrency theory in computer systems, where unregulated parallel execution can lead to resource contention and inefficiency.

5.3. Identification of Optimal Operational Regions

We identified optimal regions in the parameter space that maximize protein output by analyzing the heatmap and line plots. These regions are characterized by:

- Moderate mRNA excess (excess factor $\leqslant 1$)
- Sub-maximal ribosome counts (typically $\leqslant 100$ for this experiment)

5.4. Comparison with Previous Work

Compared to the results in [Neto et al. 2024b], where ribosome constraints were not explicitly modeled, the inclusion of ribosomes leads to a significant change in the system's behavior under high mRNA scenarios. In earlier models, the absence of ribosome constraints resulted in inefficient translation due to rampant resource depletion. In contrast, the updated model with ribosomes enforces concurrency limits that preserve amino acid availability and support complete protein synthesis.

This supports our central thesis: ribosomes serve not just as execution units, but as critical regulators of translation concurrency.

6. Implications and Future Work

6.1. Biological Implications

The observation that ribosomes act as concurrency controllers suggests that cellular systems may rely on the availability of ribosomes as a form of resource-based regulation for translation efficiency. This has several biological implications.

- **Intrinsic Regulation Without Signaling Overhead**: Rather than relying solely on complex feedback loops or regulatory proteins to control translation initiation, cells can leverage ribosome scarcity to throttle excessive translation automatically, ensuring amino acid pools are not prematurely exhausted.
- **Robustness in Stress Conditions**: Under nutrient starvation conditions with limited amino acid availability, maintaining a low ribosome count could protect the cell from initiating too many incomplete translations. This could represent an evolutionarily conserved strategy to improve translation robustness during fluctuating environments.
- **Implications for Ribosomopathies**: Disorders characterized by ribosome dysfunction can affect not only the rate of protein synthesis but also its completeness and fidelity. Understanding ribosome-mediated concurrency control could offer new insight into these diseases and their molecular pathology.

6.2. Computational Modeling Considerations

From a modeling perspective, it is noteworthy that breaking the system down into smaller and incomplete steps can lead to new insight and a better understanding of behavior. If we had incorporated ribosomes from the start, the effect of mRNA abundance could have been significantly less pronounced and gone unnoticed.

With this in mind, we would like to highlight that this new model, which considers the role of the ribosome, is closer to biological reality than our previous works.

Another interesting consideration is the role of modeling in allowing us to reason about the unintuitive behavior of complex systems. As we saw with the mRNA abundance work, having more of a necessary resource in a concurrent system may not improve output production but decrease it, which is unintuitive. This showcases the necessity for good modeling and simulation tools that allow us to capture such behavior and study it.

6.3. Future Research Directions

Several promising avenues stem from this work:

- **Model Refinement and Granularity**: The current model treats ribosomes as atomic entities. A logical next step is to model ribosomes in terms of their structural subunits (large and small), and potentially incorporate other components that assist in translation. Object Petri Nets (OPNs) provide a natural foundation for this decomposition, allowing for finer-grained simulation of subunit assembly, disassembly, and function.
- **Incorporation of Other Regulatory Layers**: Future models can incorporate additional biological features such as: ribosome recycling factors and energy consumption dynamics (e.g., GTP/ATP usage).
- **Complete modeling of biological information flow**: by creating a model that can, from a set of DNA sequences, build the necessary RNA and complete the translation process, we could have the model making its own mRNAs, ribosomes, tRNAs and other essential compounds, making the model more complete and allowing the possibility to study the systems that originate from these sets of sequences.

7. Conclusion

This work demonstrates that ribosomes play a critical regulatory role in translation by acting as natural concurrency controllers. By extending previous Petri net models to include ribosome dynamics explicitly, we showed that ribosome availability constrains the number of concurrent translation events, thereby mitigating the detrimental effects of mRNA abundance on protein synthesis efficiency.

Through a series of simulation experiments grounded in codon-level modeling, we quantified how ribosome scarcity prevents greedy consumption patterns that would otherwise lead to premature amino acid depletion and incomplete protein products. Our results indicate that the efficiency of protein synthesis does not always scale with the availability of translation substrates such as mRNA or amino acids, an unintuitive and interesting result. Instead, it depends crucially on the balance between the necessary substrates rather than monotonically increasing the output given increased inputs.

Beyond validating this regulatory mechanism, our findings underscore the value of formal modeling in uncovering emergent behaviors in complex biological systems. With its explicit handling of concurrency and resource constraints, the Petri net framework proves to be a powerful tool for investigating molecular processes such as translation.

Looking ahead, this work lays the foundation for richer models that decompose ribosomes into functional subunits and incorporate other regulatory actors such as helicases and energy factors. Such extensions will allow for even more realistic simulations of molecular machinery and open possibilities for synthetic biology applications.

By highlighting ribosomes' protective and regulatory roles, this study contributes to our understanding of how cells maintain efficiency and robustness in fluctuating resource availability. It also illustrates how computational tools can be used to mirror biological processes and generate new biological insights.

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