

The Movement of Red Blood Cells

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INTRODUCTION

We still use terms like "mean red blood cell volume" MCV, "mean corpuscular hemoglobin concentration" MCHC, and other "mean" RBC indices and properties including deformability, density, and longevity in clinical settings. We also compare one "stable state" to the new one that all cells mysteriously acquire at the same time in our test tubes when we work with bulk responses of RBCs to stimuli. Is it always reasonable? Based on what we've learned about RBCs over the last few decades, there are many fractions of RBCs in our blood, each with its own set of traits and sensitivity to stressors. The quantity of RBCs that make up each of these fractions differs across healthy people and varies dramatically in patients with various illnesses. We don't know much about the variables that control this variability, except that it can't be explained only by RBC age. This information gap necessitates greater research and the use of both traditional (such as density centrifugation or fluorescence based cell sorting) and new (such as single cell high throughput technologies) technical ways to gain readouts of subpopulations or even individual cells. Information on a few thousand RBCs will provide an overview of the cell populations. Another key issue is defining a "steady state condition," as we commonly refer to it, and determining if it exists at all. RBCs are dynamic entities that choose one of several preferred states and can be found in any of them at any point in time when in flow, according to advances in live cell imaging. They move, their Ca^{2+} levels fluctuate, as do their ion content and pre-membrane ATP levels. The dynamics of these processes

are poorly understood. We've begun to say goodbye to snapshot-style techniques, but there are still obstacles to overcome. It's still unknown how RBC heterogeneity and its dynamic adaptive character affect blood rheology and gas exchange. Without the development of new integrated multidisciplinary experimental and theoretical modelling methodologies, it will be impossible to answer these concerns. Any stress circumstances, including acute or chronic stimulation of de novo RBC synthesis, are thought to impact signaling pathways that control the proliferation and differentiation of erythroid precursor cells, according to a new theory. As a result, the characteristics of newly generated RBCs may differ from those found in species with unstressed erythropoiesis. What are the mechanisms behind stress erythropoiesis, and what are the features of RBCs produced in response to stress, as well as the function these cells play in environmental challenge adaptation, are all questions that need to be answered thoroughly. Stress erythropoiesis is recognized for patients with chronic haemolytic state, hence research in this field will have a huge impact at the translational level. Despite decades of research, we still don't have a good grasp of how RBCs maintain their form and function over the 100–120 days they spend in circulation. Protein turnover in nucleated cells normally takes hours to days, a far shorter time period than the months that the same proteins stay functional in RBCs. This remarkable cell lifespan was not thoroughly investigated. The maintenance of RBC function under stressful conditions in the circulation is outstanding and not fully understood due to a lack of translation.

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