



## Molecular Research of Chronic Lymphocytic Leukemia (CLL)

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### Message from the Guest Editor

Current thinking supports a paradigm whereby CLL cells cyclically traffic from secondary lymphoid and bone marrow environments to the blood and back again. Importantly, CLL cells in tissues receive signals for survival and proliferation. However, when they are in the blood, they are destined for death unless they can re-enter tissues in a timely way. Moreover, the longer CLL cells remain in tissues, the more likely it is that disease will be progressive and resistant to therapy.

We know that a principal regulator of CLL cell tissue residency is the expression of the integrin  $\alpha 4 \beta 1$ , moreover, B cell receptor (BCR), is important for activating  $\alpha 4 \beta 1$ -mediated adhesion to its ligands VCAM and fibronectin which has therapeutic implications. What is unclear is how CLL cells make decisions to remain, leave, or even re-enter tissues, and whether pathogenetic features such as constitutive signals, chromosomal deletions, and/or the mutation of key genes contribute to this decision making. This Special Issue invites research articles and reviews to advance our knowledge of CLL cell biology and broaden our understanding of progressive disease.





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## Message from the Editor-in-Chief

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