Lymphoma therapy could deliver a double punch

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B cell lymphomas are a group of cancers of that originate in lymphoid tissue from B cells, the specialized immune cell type that produces antibodies. The development of B cell lymphoma is associated with several known genetic changes, including increased expression of MYC, a transcription factor that promotes cell growth and division.

In this issue of the *JCI*, Andrei Thomas-Tikhonenko and his colleagues at the University of Pennsylvania in Philadelphia report on their studies to better understand the molecular pathways that interact with MYC and contribute to B cell lymphoma development. Using a mouse model of lymphoma, they found that a transcription factor in B cells known as PAX5 controls the level of MYC in cells. They showed that PAX5 stabilizes MYC protein levels through a previously undescribed pathway involving CD19, a surface protein expressed on B cells. When the research team looked in patient samples, they found that high levels of CD19 correlated with high MYC activity, and that both predicted poor patient survival time.

Their findings uncover a CD19-dependent pathway that contributes to the cancerous growth of B cell lymphomas. Their work has direct implications for therapies targeting CD19 that are currently in clinical trials, and suggest that these therapies may reduce cancer-promoting signaling in addition to depleting total B cell numbers.

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