Unnatural fusion of API2 and MALT1 proteins leads to deregulated transcription in MALT lymphoma.

Normal, healthy cells use specific proteins called transcription factors to regulate the activit of their genes. Importantly this is done in a tightly controlled manner. When the transcription factor NF-κB is functioning normally, it is capable of being turned off and on, and thus cellular function is retained. In many instances of cancer, however, NF-κB has lost its "off switch," which leads to unregulated gene activity (transcription).

Scientists at the University of Michigan have discovered a key factor leading to NF-κB deregulation in mucosa-associated lymphoid tissue (MALT) lymphoma. The proteins API2 and MALT1 are fused together to form an unnatural new protein. This fusion oncoprotein binds to NF-κB-inducing kinase (NIK) and cleaves it, thus removing the regulatory region of the kinase and leading to constant activity of the NF-κB pathway. Neither API2 nor MALT1 appear capable of cleaving NIK on their own. It is only the fusion protein that has this activity.

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