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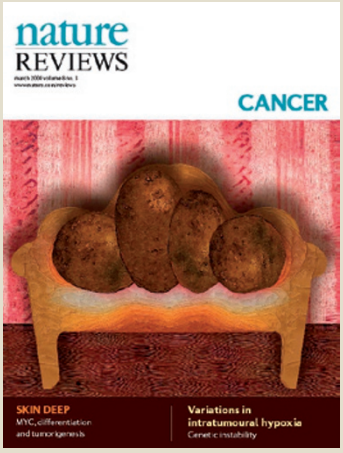

▸ **COVER:** 'Couch potatoes' by Manuel Enriquez Ramirez, inspired by the Review on p205.


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Since the isolation of the MYC oncogene in the early 1980s, numerous concepts that try to explain how this transcription factor contributes to tumorigenesis in so many different types of cancer have been postulated. However, new ideas about MYC-dependent responses and how these might be involved in tumorigenesis are still emerging. For example, the focus of MYC research has primarily been its role as a transactivator of genes associated with cell proliferation, whereas recent data point toward activities of MYC that are independent of this role.

This current excitement surrounding MYC in the cancer research field has prompted us to produce a series of specially commissioned articles that discuss some of the key issues and new insights surrounding MYC and cancer — from strategies that target MYC-mediated tumorigenesis for therapeutic intervention to an expansion of the repertoire of signalling pathways that are induced downstream of MYC activation, such as DNA damage response signalling.

Key concepts implicit in the consideration of how MYC might function as an oncogene are also likely to be relevant to other oncogenes and transcription factors — for example, MYC target genes do not appear to change according to cell type, although the biological outcome does. Also, the strength and duration of MYC activation, which can be regulated in part by MYC cofactors, can also determine the biological outcome of MYC activation. Such concepts are exemplified in the first article of the MYC series: a Perspective by Fiona Watt and colleagues on page 234, which discusses how MYC induces differentiation in epidermal stem cells, a paradox to the traditional view of MYC as a promotor of cell proliferation and tumorigenesis.