Nature Reviews Clinical Oncology **10**, 182 (2013); published online 19 February 2013;
doi:10.1038/nrclinonc.2013.23;
doi:10.1038/nrclinonc.2013.24;
doi:10.1038/nrclinonc.2013.25

IN BRIEF

HAEMATOLOGICAL MALIGNANCIES

Risk groups defined for chronic myelomonocytic leukaemia

Patients with chronic myelomonocytic leukaemia (CMML) have a very variable prognosis and, until now, there has been no widely accepted scoring system that could be used to determine the prognosis of these patients. A study has now used data from a cohort of 558 patients with CMML to develop a CMML-specific prognostic scoring system. This system was then validated in an independent cohort of 274 patients. The study showed that the most relevant variables were the CMML subtypes as outlined by the WHO and the French–American–British classification, as well as CMML-specific cytogenetic risk classification and the level of a patient's dependency on red blood cell transfusions. The prognostic score developed by the team identified four separate risk groups according to overall survival and risk of evolution into acute myeloid leukaemia. It remains to be seen how this prognostic score would affect patient treatment.

Original article Such, E. *et al.* Development and validation of a prognostic scoring system for patients with chronic myelomonocytic leukemia. *Blood* doi:10.1182/blood-2012-08-452938

BASIC RESEARCH

General mechanism for halting cancer progression found?

Treatment with immunotherapy frequently does not cause apoptosis of cancer cells, but rather the halting of growth, or slow tumour regression. This growth arrest and regression has been associated with tumour-specific IFN- γ -producing CD4⁺ T-helper-1 cells. Furthermore, patient profiling also identified a critical role for TNF in cancer control. Now, studies in cell culture and genetically modified mouse models have shown that a combination of IFN- γ and TNF can drive cancer cells into a senescent state. The induction of senescence is dependent on STAT1 and TNFR1 signalling. The researchers showed that this mechanism of senescence induction functioned in a number of mouse and human cancer cell lines, and they claim that the mechanism they have identified explains clinical responses to immunotherapy in cervical cancer and disseminated melanoma. This finding might be of broad relevance for cancer control.

Original article Braumüller, H. *et al.* T-helper-1-cell cytokines drive cancer into senescence. *Nature* doi:10.1038/nature11824

GENETICS

New disease entity identified for meningioma

Although one-third of primary tumours that occur in the central nervous system of adults are meningiomas, it is only in a small number of patients that these tumours occur at multiple sites. Most patients with these multiple-site meningiomas also have type 2 neurofibromatosis (associated with mutations in *NF2*). To determine if there were other mechanisms underlying this manifestation of the disease, the exomes of three unrelated individuals with familial multiple-site meningiomas were sequenced. Heterozygous loss-of-function mutations were found in the *SMARCE1* gene in two of the individuals, and similar mutations were found in six further individuals. Tumours in these patients were a clear-cell histological subtype, and the authors suggest that this represents a new disease manifestation.

Original article Smith, M. J. *et al.* Loss-of-function mutations in *SMARCE1* cause an inherited disorder of multiple spinal meningiomas. *Nat. Genet.* doi:10.1038/ng.2552