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Patients receiving immunosuppression to prevent organ transplant rejection are at a greatly increased risk of developing nonmelanoma skin cancer. In recent years a correlation has been identified between the class of immunosuppressant that these patients receive and their subsequent cancer risk; in particular, patients switched from calcineurin inhibitors to mammalian target of rapamycin (mTOR) inhibitors not only displayed a dramatic reduction in new tumor formation but also in some cases a regression of their existing lesions. Studies of cancer models in mice and cell lines in the laboratory have attributed these discrepancies in cancer risk to the ability of immunosuppressants such as mTOR inhibitors to elicit direct anticancer effects, including suppressing angiogenesis and increasing autophagy-mediated DNA repair. Recent evidence from the immunological literature however, suggests a significant alternative contribution of mTOR inhibitors; namely the promotion of memory T-cell function. Recent advances in understanding memory T-cell establishment and the demonstration of their critical role in long-term immunity make it timely to review the available evidence as to whether the improved nonmelanoma skin cancer outcome shown by patients switched to mTOR inhibitor treatment regimens may be associated with the retention of memory T-cell function.