Generalised mosaicism for *TSC2* mutation in isolated lymphangioleiomyomatosis

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To the Editor:

Lymphangioleiomyomatosis (LAM) is a rare, slowly progressive pulmonary disease causing cystic lung destruction and respiratory failure. It affects predominantly premenopausal women, and rarely men. It can occur as a sporadic condition (sporadic LAM) or in association with tuberous sclerosis complex (TSC) [1]. LAM is caused by biallelic inactivation of the tumour suppressor gene *TSC2* in LAM cells, which leads to hyperactivation of mammalian target of rapamycin complex (mTORC)1, resulting in anabolism and LAM cell proliferation [2]. Sirolimus and everolimus, mTORC1 allosteric inhibitors, have been shown to retard progression of LAM [3].