Retinal neovascularisation is a major clinical complication of diabetic retinopathy that occurs late in the disease process. It constitutes the most damaging phase and results in visual loss. Neovascularisation is defined as the growth of new blood vessels which, in a disease process such as diabetic retinopathy, occurs in abnormal retinal locations. Long term consequences of retinal neovascularisation include vessel leakage, formation of epiretinal membranes and retinal detachment. However, to date there has been few reports on changes in the choroidal vasculature in diabetic retinopathy. A major problem is that changes in the choroidal vasculature cannot readily be detected using conventional ophthalmoscopic techniques; in addition, such techniques view the vasculature from its vitread (inner) aspect, precluding detection of changes in the sclerad (outer) aspect.

We recently generated several transgenic mouse lines with retinal neovascularisation using the human isoform of vascular endothelial growth factor (hVEGF$_{165}$) and the murine rhodopsin promoter$^1$. VEGF was selected since it is the most potent known stimulator of blood vessel growth and permeability. Here we characterised pathological changes in the trVEGF029 line to determine its suitability as a model for retinal neovascularisation comparing wildtype littermate controls (−/−) with transgenic heterozygotes (+/−). We used confocal microscopy of *Griffonia simplicifolia* lectin-stained wholemounts to reveal the retinal vasculature and scanning electron microscopy of Mercox corrosion casts to reveal the choroidal vasculature. In wildtype littermate controls at 4 weeks, the retinal vasculature is mature and composed of 3 regularly arranged capillary beds in the retinal ganglion cell layer/nerve fibre layer and along the inner and outer aspects of the beds inner nuclear layer. However, in transgenics, abnormal vessels resembling microaneurysms seen in clinical diabetic retinopathy extended towards the photoreceptor layer, the source of the hVEGF$_{165}$. Other changes included haemorrhage, vessel leakage and capillary dropout. Scanning electron microscopy of choroidal vascular casts revealed irregularities in vessel caliber, vessel tortuosity and leakage, dilatation of capillaries and vortex veins, narrowing of arteries and hairpin loop formation in precapillary arterioles. Abnormal vessels formation including outpouching and microaneurysms were also observed on the sclerad aspect.

Our study shows that trVEGF029 displays a number of clinical features characteristic of retinal neovascularisation found in human diabetic retinopathy. Detection of damage to the choroidal vasculature indicates that post-mortem clinical studies are necessary to determine whether similar changes are found in diabetic retinopathy and to evaluate further the relevance of the trVEGF029 model.