

Hepatic ribosomal protein S6 (Rps6) insufficiency results in failed bile duct development and loss of hepatocyte viability; a ribosomopathy-like phenotype that is partially p53-dependent

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c-Myc ameliorates the hepatobiliary disease and growth defect caused by hepatic Rps6 deficiency.

A modest increase in the level of hepatic c-Myc provided by an Albumin-c-Myc transgene rescues the neonatal growth defect and significantly improves hepatobiliary disease in mice with hepatic ribosomal protein S6 (Rps6) insufficiency ($\Delta S6$ mice). While the $\Delta S6$ mouse (second from left) is runted and has a yellow-tinged coat due to a failure of bile duct development and cholestatic-induced hepatocyte death, the mouse expressing a higher level of c-Myc ($\Delta S6:c\text{-Myc}$ mouse) (far right) is indistinguishable from the wild-type (WT) mouse (far-left) or its Albumin-c-Myc littermate (third from left) in terms of size and lacks the jaundiced (yellowed) coat of the $\Delta S6$ mouse. Image credit: R.E. Hammer and S.A. Comerford