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 (ΔS6 mice). While the ΔS6 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 (ΔS6:c-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 ΔS6 mouse.

Image credit: R.E. Hammer and S.A. Comerford