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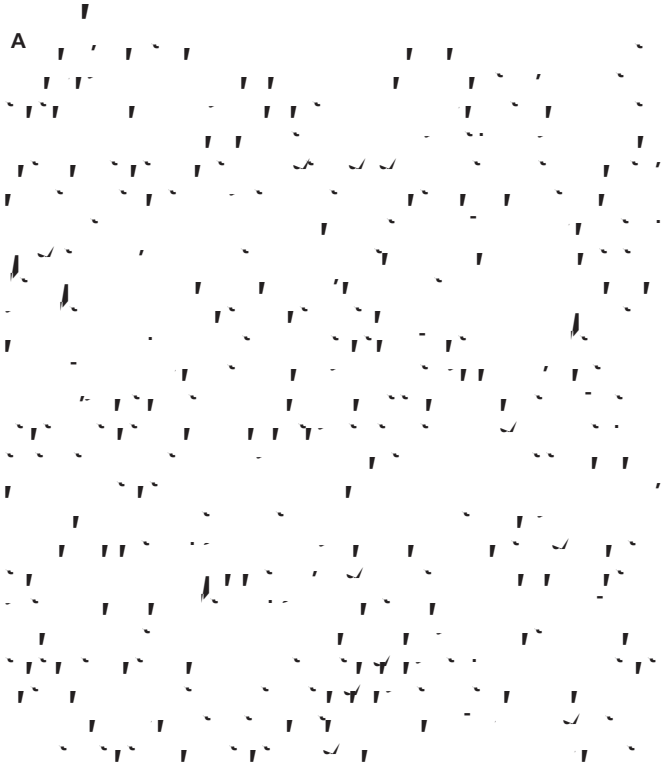

generations) and observed that aged cells form significantly larger colonies on galactose, demonstrating a significant growth advantage even at intermediate age (Figs 1F and S3, Supporting information).

Time-course and redilution experiments provided no evidence for permanent genetic adaptation to galactose (Figs 2A and S4, Supporting information); instead, aged cells have a transient advantage rapidly lost in progeny. This could be explained either by aged cells growing more rapidly than young cells on galactose or by aged cells resuming growth more rapidly after an environmental change. To distinguish these, we compared cells aged in galactose prior to outgrowth in glucose or galactose (Fig. 2B). As before, aged cells outcompeted young cells in galactose showing that an environmental change is not required. Furthermore, we did not observe an accelerated galactose response in aged cells or a dependence on nutrient storage (Fig. S5, Supporting information).

This implies that aging cells divide faster than young cells in galactose. We measured cell cycle times on glucose and galactose of wild-type (non-MEP) diploid cells aged for ~14 generations by micromanipulation (cf. 16–30 generations for competition assays), and also of the daughters of these cells (Figs 2C and S6, Supporting information). On glucose, the cell division times of mothers and daughters were similar, although division time heterogeneity increased with age as reported (Liu et al., 2015). On galactose the opposite was observed: division time decreased significantly with age while heterogeneity lessened. To independently confirm this remarkable observation, we measured average cell division time across 24-h aging by bud scar counting and again observed that aged cells divide faster than young cells on galactose (Fig. 2D).

Our experiments show that aging does not entail a simple decline in fitness for yeast. Rather, aging cells lose glucose specialization as gene expression studies have suggested (Lin et al., 2001; Lesur & Campbell, 2004), but gain fitness for other carbon sources. This is very surprising, but is consistent with conflicts between optimal life-history strategies evident from mutants with reduced fitness on glucose but improved

causal to) the aging process. Either way, it creates a selective pressure for the evolution of aging regulatory systems as aged cells would be under positive selection in non-glucose and fluctuating environments. Consequently, as nutrient responsive signalling pathways evolved in early eukaryotes, manipulation of the aging process may have provided a way to tune growth strategy for current and future nutrient availability, and indeed aging is accelerated by galactose (Liu