BIOCATALYSIS

To link zinc to adenylates

Commun. Biol. **1**, 113 (2018).

Zinc is an essential micronutrient that serves as an important enzymatic co-factor in many biochemical reactions. A deficiency in zinc can result in a variety of health issues, ranging from neurological deficits to immune disorders to poor hepatic function. Preliminary reports suggest a linkage between adenylate, along with its associated nucleotides (AMP, ADP, ATP), and zinc metabolism. In a new report in *Communications Biology*, four extra-cellular enzymes involved in hydrolysis to the ATP degradation products have been related to the biochemistry of this co-factor. By correlating metabolite levels—*in vitro* and *in vivo*—with enzyme activity levels of four extra-cellular associated enzymes, investigators found a link between zinc metabolism and levels of adenine-associated metabolites. This relationship varied by tissue, which would be consistent with expression levels of the associated enzymes. *CN*

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OSTEOPOROSIS

Boning up on turnover

Nat. Commun. **9**, 3428 (2018).

Like most physiological phenomena, bone status is a combination of creation and degradation. In older adults these processes become dysregulated and are potential targets for therapy. Amongst the proteins involved in the anabolic processes that build bone are bone morphogenetic proteins (BMPs). A new study that explored these proteins and their interaction with a ubiquitin ligase, SMURF1, found that response to interventions in mice and older adults fell into different categories, based on BMP-2 and SMURF1 response. After various experiments, including altering SMURF1 protein levels and various pharmacological interventions, there is room for clinical improvement. Specifically, decreasing levels of SMURF1 in osteoclasts—bone generating cells—holds promise as a therapeutic strategy amongst older, osteoporotic individuals. *CN*

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AGEING

Less methionine means more health

Cell Rep. **24**, 2392–2403 (2018).

Dietary modification as a method to improve lifespan and/or healthspan is an area of much interest. While there is a significant body of literature establishing caloric restriction as one method to increase longevity, much less information is available on other strategies. In *Cell Reports*, a new study describes a methionine restricted diet that extends lifespan in not only wildtype mice, but also in a mouse model of Hutchinson-Gilford Progeria Syndrome; a disease resembling premature aging. Transcriptomic analysis of hepatic tissues from both genotypes revealed significant down regulation of inflammatory pathways with methionine restriction, with changes also present in DNA repair. Lipid profiles changed as well between the control and methionine restricted diet. Moreover, addition of cholic acid to the diet improved lifespan and healthspan in the progeria mouse model. *CN*

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GENE EXPRESSION PROFILING

Worms expressing themselves

PLoS Genet. **14**, e1007559 (2018)

Caenorhabditis elegans is the organism of choice for exploring many biological phenomena due to its small size, rapid generation time, and well-described genome. Despite the multitude of studies using this worm as a model, little information was available on the transcriptome of the four tissues of adult worms—muscle, neuron, intestine, and epidermis—versus those of embryonic and larval stage animals. By busting open worms and separating tissues, investigators compared the expression profiles of the four tissues with single cell resolution. They identified mRNAs that were ubiquitously expressed across all tissues versus those that were enriched in one. In addition, they also characterized differences in alternative transcript splicing. For example, authors observed differences in ribosomal proteins and cytoskeletal proteins in the different tissues. *CN*

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