

## IN BRIEF

**DEVELOPMENT****DNA methylation from oocyte to placenta**

Maternal DNA methylation by DNA methyltransferase 3A (DNMT3A) and DNMT3B has an important yet uncharacterized role in the development of trophoblast (extra-embryonic) tissues. Branco *et al.* generated maternal, oocyte-specific *Dnmt3a* and *Dnmt3b* double-knockout (mDKO) mice and found pronounced reduction in the adhesion of trophoblast giant cells in mDKO conceptuses, with direct effects on the expression of trophoblast differentiation and adhesion genes. Cultured *Dnmt*-null trophoblast stem cells (TSCs) formed less-distinct colonies with decreased surface adherence and exhibited gene expression changes similar to those in mDKO early trophoblasts. *Scml2*, encoding a subunit of Polycomb repressive complex 1, was highly upregulated in both *Dnmt*-null cell types and responsible for downregulation of a placenta morphogenesis marker. *Scml2* deletion in *Dnmt*-null TSCs restored their surface adherence. Thus, oocyte-derived DNA methylation is essential for placental development.

**ORIGINAL ARTICLE** Branco, M. R. *et al.* Maternal DNA methylation regulates early trophoblast development. *Dev. Cell* **36**, 152–163 (2016)

**PLANT CELL BIOLOGY****Root endodermis adapts to nutrient availability**

The endodermis of the root serves as a border between the outer cortex and the inner vascular system and is involved in regulating the uptake of nutrients from the soil. Endodermis can transition from an absorbing to a protective epithelium, and this is associated with the deposition of a hydrophobic polymer called suberin. Suberization has been linked to drought and salt resistance and was thought to be permanent. Barberon *et al.* show that suberization is in fact a reversible process, and that suberin is deposited or degraded in response to changing nutritional needs. They further reveal that suberization is controlled by plant hormones, with abscisic acid stimulating suberin deposition, and ethylene inhibiting it and inducing suberin degradation. This previously unanticipated plasticity is physiologically relevant and serves as an adaptive response to nutritional stress.

**ORIGINAL ARTICLE** Barberon, M. *et al.* Adaptation of root function by nutrient-induced plasticity of endodermal differentiation. *Cell* **164**, 447–459 (2016)

**GENE EXPRESSION****CRISPR–Cas screening for enhancers**

Korkmaz *et al.* describe a CRISPR–Cas9-based screening method for the functional analysis of endogenous enhancers. The method is based on bioinformatics analyses to delineate sets of potential enhancers of interest, followed by cloning of single guide RNA (sgRNA) libraries to target the enhancers by CRISPR–Cas9 and screening for enhancers that are active in specific cellular settings. Using this method, the authors discovered a new p53-bound enhancer (p53<sup>enh3507</sup>) of the p53 effector gene *CDKN1A*, which is required for p53-dependent, oncogene-induced senescence (OIS) in immortalized human cells, as well as a new *CCND1* enhancer that mediates ER $\alpha$ -dependent breast cancer cell proliferation. Additionally, the use of CRISPR–Cas9 with sgRNAs tiling the 2 kb region surrounding p53<sup>enh3507</sup> enabled pinpointing new domains that are not bound by p53 but that are nevertheless required for p53-dependent OIS.

**ORIGINAL ARTICLE** Korkmaz, G. *et al.* Functional genetic screens for enhancer elements in the human genome using CRISPR–Cas9. *Nat. Biotechnol.* **34**, 192–198 (2016)