# Article information:

Characterization of the adipocyte cellular lineage in vivo | Nature Cell Biology
<https://www.nature.com/articles/ncb2696>

# Article summary:

1. The cellular lineage of adipocytes in white adipose tissue (WAT) is tightly regulated and requires a mechanistic understanding for homeostasis and growth.

2. Various methods have been used to study adipocyte precursors, including culture of the stromal-vascular fraction (SVF), fluorescence-activated cell sorting (FACS), and genetic approaches using Cre recombinase.

3. PdgfRα labels all mature adipocytes in WAT depots, and both the CD24+ and CD24− populations are part of the in vivo adipocyte cellular lineage. Adipocytes are not derived from endothelial or hematopoietic lineages under normal conditions, but some adipocytes may be derived from circulating cells of hematopoietic origin in transplant and injury models.

# Article rating:

May be slightly imbalanced: The article presents the information in a generally reliable way, but there are minor points of consideration that could be explored further or claims that are not fully backed by appropriate evidence. Some perspectives may also be omitted, and you are encouraged to use the research topics section to explore the topic further.

# Article analysis:

The article "Characterization of the adipocyte cellular lineage in vivo" published in Nature Cell Biology discusses the various methods used to study adipocyte precursors and their relationship to adipocyte lineage cells in vivo. The article provides insights into the mechanisms of white adipose tissue (WAT) homeostasis and growth, which is essential for understanding obesity and related diseases.

The article highlights that mature adipocytes are post-mitotic, and a change in adipocyte number occurs through disruption of the balance between rates of adipogenesis and adipocyte death. Therefore, characterization of the adipocyte cellular lineage is required for a mechanistic understanding of WAT homeostasis and growth. The article discusses various methods used to study adipocyte precursors ex vivo and in vivo, including culture of the whole stromal-vascular fraction (SVF) from adipose tissues, fluorescence-activated cell sorting (FACS), and genetic approaches.

The article reports that two cell populations derived from WAT, defined by the marker profiles Lin−:CD34+:CD29+:Sca-1+:CD24+ (CD24+) and Lin−:CD34+:CD29+:Sca-1+: CD24− (CD24−), are adipogenic in vitro but only the CD24+ population is capable of generating a functional WAT depot following transplantation into a residual WAT depot of lipodystrophic mice. This indicates that the CD24+ population contains adipocyte progenitors.

The article also discusses genetic approaches used to investigate the adipocyte cellular lineage. A previous study showed that Cdh5–Cre labels mature adipocytes, suggesting an endothelial origin for white adipocytes as Cdh5 labels endothelial lineages. However, for studies of WAT, the cellular specificity of reporters that stain the cytoplasm is difficult to delineate given the paucity of cytoplasm in mature adipocytes and the high vascularity of WAT. To overcome this limitation, the article employed a mouse strain harbouring a fluorescent-membrane dTomato/membrane eGFP (mT/mG) Cre reporter construct that marks Cre excision by a heritable switch from membrane-targeted tdTomato expression to membrane-targeted eGFP expression.

The article reports that PdgfRα labels all mature adipocytes in all major WAT depots and is expressed in WAT-resident cells that produce brown-like adipocytes in response to β-adrenergic stimulation and white adipocytes on HFD feeding. The article also highlights that the SVF of PdgfR α–Cre:mT/mG mice contains a low percentage of GFP+ cells in CD31+ and CD45+ populations. In contrast, both the CD24+ and CD24− cells are nearly completely traced by PdgfR α–Cre, indicating that these cell populations are potential adipocyte precursors.

Overall, the article provides valuable insights into the mechanisms of WAT homeostasis and growth. However, it is important to note that the study has some limitations. For example, the study only focuses on one potential adipocyte precursor population (CD24+) derived from WAT, which may not represent all adipocyte precursors. Additionally, while the study provides evidence for PdgfRα as a marker for mature adipocytes and potential adipocyte precursors, further studies are needed to confirm these findings.

In conclusion, while the article provides valuable insights into the characterization of the adipocyte cellular lineage in vivo, it is important to consider its limitations and potential biases when interpreting its findings.

# Topics for further research:

* Adipocyte precursors other than CD24+ population
* Mechanisms of adipocyte death in white adipose tissue
* Role of PdgfRα in adipocyte differentiation and homeostasis
* Comparison of ex vivo and in vivo methods for studying adipocyte lineage
* Genetic approaches for investigating adipocyte cellular lineage
* Relationship between adipocyte lineage and obesity-related diseases

# Report location:

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