UCLouvain

PRESS RELEASE

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Research

# UCLouvain team discovers link between sugar metabolism and Parkinson's disease

**IN BRIEF:**

- **Parkinson's disease** is the second most common neurodegenerative disease, but **we still do not understand why this disease occurs.**
- Scientists from the **UCLouvain de Duve Institute** have discovered a **new type of molecular damage** that is **linked to the metabolism of sugar** (glycolysis).
- They also discovered a **mechanism that allows cells to prevent this type of damage**. A deficiency in this mechanism leads to the accumulation of damage and Parkinson's disease.
- Understanding the molecular origin of Parkinson's disease **might help develop therapies that treat not only the symptoms but also the cause** of the disease.

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A **UCLouvain de Duve Institute research team**, led by **Prof. Guido Bommer**, has just discovered the **role of an enzyme – Park7 – which is inactive in some patients with Parkinson's disease**: this enzyme prevents cellular damage caused by the metabolism of sugars in glycolysis, the main pathway of this metabolism. They suspect that **this type of damage represents one of the first steps in the development of the disease**. The study is published in the **journal *PNAS*** (*Proceedings of the National Academy of Sciences*, USA) and was carried out with the financial support of WELBIO, the European Research Council (ERC) and the Fondation Roi Baudouin.

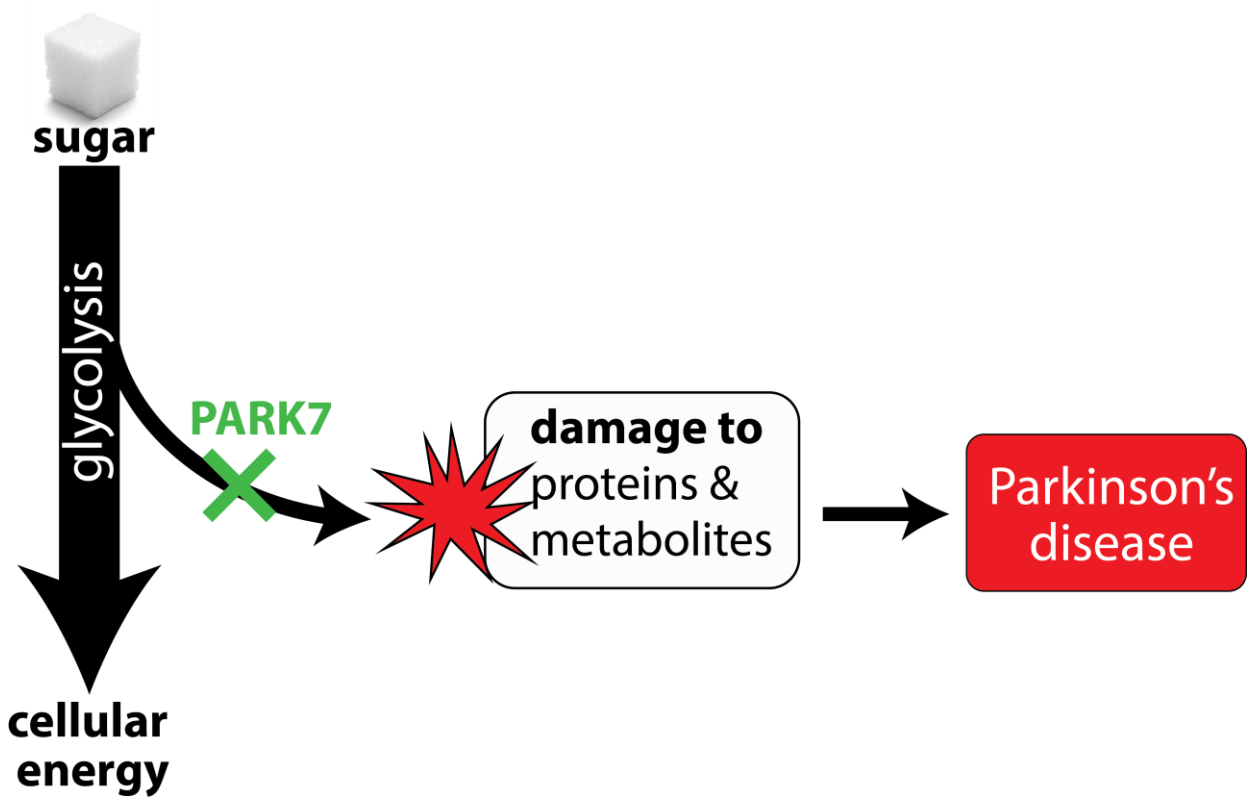
Like Alzheimer's disease, **Parkinson's disease** is a common and very debilitating neurodegenerative disease. It is characterised by the accumulation of insoluble proteins and the death of certain brain cells, but it is still not known why this occurs. It was long suspected that the accumulation of molecular damage over a lifetime plays a role, but **the precise nature of this damage was not known until now.**

Prof. Bommer's team has just d**iscovered a close link between the metabolism of sugars and a new type of cellular damage**, which **seems to play a role in certain cases of Parkinson's disease**. This damage **concerns both proteins** (the workhorses of our cells) **and many metabolites** (the small molecules that serve as building blocks for proteins, among others). In our cells, sugars are consumed via glycolysis – a series of chemical reactions that break down glucose by converting it successively into a series of metabolites. One of these metabolites is **spontaneously converted into a highly reactive compound**, cyclic 1,3-phosphoglycerate, **which has never been described before** and damages proteins and metabolites.

Prof. Guido Bommer's group **also found that all our cells and almost all living cells have an enzyme – called PARK7 – that can destroy this reactive compound**, thereby preventing the damage from occurring. They observed that **inactivation of PARK7 causes damage to accumulate in systems as diverse as human cells, mice and even flies**. **Some cases of Parkinson's disease are due to genetic inactivation of the PARK7 enzyme**. However, despite thousands of scientific papers published on the subject, the function of PARK7 remained unknown. The new findings offer a mechanistic explanation of how PARK7 deficiency causes Parkinson's disease. Since PARK7 is

easily inactivated by oxidative stress that can be triggered for multiple reasons, **the same damage could also play a role in patients with an intact PARK7 gene.**

In keeping with the de Duve Institute's motto of "Better understanding for better healing", the researchers **hope that a better understanding of the mechanisms behind Parkinson's disease could lead to new treatments that target the origin of the disease** rather than just its symptoms. This is a source of great satisfaction for Prof. Bommer, who also emphasises, "It's **exceptional to make such a fundamental discovery on a subject that has been studied so much for so long**."





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