Troubleshooters that block cancer

The research was carried out on breast cancer cells. Scientists have shown how a family of "limpet-like" proteins play a crucial role in repairing the DNA damage which can lead to cancer.

They hope the finding could pave the way for a new type of drug which could help kill cancer cells, and promote production of healthy replacements.

The proteins seem to have a remarkable ability to zero in on damaged areas.

The breakthrough, uncovered independently by two teams, appears in the journal Nature.

The family of Small Ubiquitin-like Modifier (SUMO) proteins track down sites in the body where DNA damage has occurred.

"This is the first step towards developing drugs which may protect normal cells from the side effects of chemotherapy, or improve the effectiveness of current breast cancer treatments."

Dr Jo Morris
King's College London

They attach themselves to normal proteins, and guide them in to fix the genetic faults.

Using this method, the proteins are even able to repair double strand DNA breaks - the most severe type of DNA damage.

When their work is done, the proteins detach themselves and move on.

Breast cancer gene

One of the study teams was able to follow this process of repair taking place on the BRCA1 gene, which, if damaged, is associated with a very high risk of breast cancer.

SUMO was shown to attach to the damaged gene, and switch it back on - helping prevent
The research was carried out on breast cancer cells. Scientists have shown how a family of “limpet-like” proteins play a crucial role in repairing the DNA damage which can lead to cancer. They hope the finding could pave the way for a new type of drug which could help kill cancer cells, and promote production of healthy replacements.

The proteins seem to have a remarkable ability to zero in on damaged areas. The breakthrough, uncovered independently by two teams, appears in the journal Nature. The family of Small Ubiquitin-like Modifier (SUMO) proteins track down sites in the body where DNA damage has occurred. This is the first step towards developing drugs which may protect normal cells from the side effects of chemotherapy, or improve the effectiveness of current breast cancer treatments.

Dr Jo Morris, from King's College London, said: "This new insight is the first step towards developing drugs which may protect normal cells from the side effects of chemotherapy, or improve the effectiveness of current breast cancer treatments."

Dr Lesley Walker, of Cancer Research UK, which part-funded the study, said: "DNA damage, particularly double strand DNA breaks, are a fundamental cause of cancer and we know that people who have mutations in the BRCA1 gene have a higher risk of developing some kinds of cancer."

"Discovering that these limpet-like proteins play such an important role in repair may provide new opportunities to stop cancer from growing."

But she added: "This is an extremely complex and intricate biological process so it may be many years before we can use this knowledge to safely intervene and help treat cancer patients."