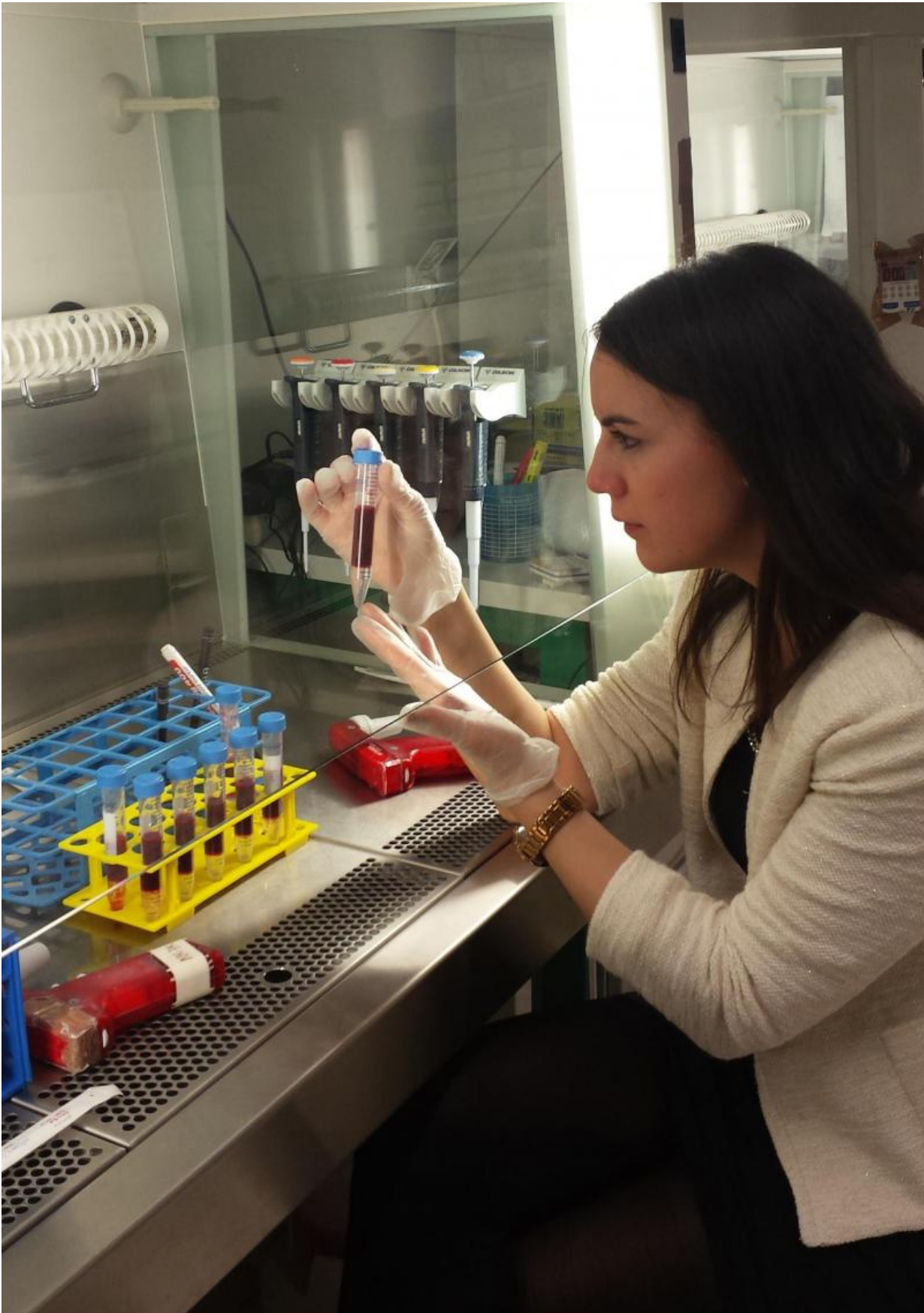


# **When the immune system promotes tumor growth**

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Dagmar Gotthardt and her colleagues have shown for the first time ever that NK cells produce a factor called VEGF-A that promotes tumor growth. Credit: Petra Kudweis

The immune system protects the body against cancer cells. The Elimination of cancer cells is an important task of NK cells. For NK cells to function properly, they require the activator STAT5.

Dagmar Gotthardt, Veronika Sexl and colleagues from the Institute for Pharmacology and Toxicology at Vetmeduni Vienna have shown that NK cells that lack STAT5 promote the growth of [cancer cells](#). "You can imagine STAT5 like an on/off switch. When STAT5 is present, it stimulates NK cells to act against cancer cells. If STAT5 is absent, NK cells do the opposite and incite the cancer cells to grow," explains Gotthardt, the first author of the study.

## **STAT5 inhibition is the treatment of choice for many types of cancer**

STAT5 is highly active in many forms of cancer such as pancreatic cancer, liver cancer and leukaemia. Drugs that inhibit STAT5 are a great hope for the treatment of many different tumor types.

Gotthardt and her colleagues now call this strategy into question. "These inhibitors not only target cancer cells but may also have a negative impact on the immune system and NK cells. They could promote the progress of the disease. That would be a dangerous cocktail for patients," Gotthardt says.

For the first time ever, the researchers have shown that NK cells produce a factor called VEGF-A (Vascular Endothelial Growth Factor A) that promotes [tumor growth](#). A number of different experiments have shown that STAT5 is required for the killing of [tumor cells](#) and normally suppresses the production of VEGF-A in NK cells. If STAT5 is inhibited, VEGF-A is produced in high amounts leading to increased [tumor progression](#). The results are of clinical relevance: One of the inhibitors that formed part of the study is already in clinical use.

**More information:** STAT5 is a key regulator in NK cells and acts as molecular switch from tumor surveillance to tumor promotion, [dx.doi.org/10.1158/2159-8290](https://doi.org/10.1158/2159-8290)

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