

A new approach to osteoporosis treatment Disabling a bone formation inhibiting protein molecule

How can the human body be stimulated to build more bone, thus helping those affected by osteoporosis? Researchers in Ulm have investigated the role played by the protein molecule Axl in this process. Their efforts were successful: in a study, they demonstrated that Axl inhibits bone formation – and that cells produce significantly more bone tissue when the molecule is blocked using medication. There are no discernible harmful side effects. The findings have been published in the highly prestigious journal Bone Research.

Osteoporosis is one of the most common age-related conditions, and it is set to become even more prevalent in an increasingly ageing population. It particularly affects post-menopausal women. When bones lose density and stability, they break more easily – often even under minimal strain or following minor falls. Medication that helps combat bone loss often leads to bones becoming too rigid, or is expensive, cumbersome to use, and can often only be initiated for a limited period or has side effects. Researchers in Ulm are therefore searching for new active substances that stimulate the body to rebuild more bone tissue. The scientists have now discovered a promising research avenue: the protein molecule Axl exerts an influence on how well bone-forming cells develop.

The Ulm study focuses on a group of protein molecules on the surface of cells, known as TAM receptors, which can influence bone formation – including Axl. The researchers wanted to find out what effect Axl has, and whether bone formation can be specifically promoted by switching it off using genetic engineering or a special active substance. To this end, they used bone cells in the laboratory as well as a mouse model. “Our findings show for the first time that the Axl protein molecule inhibits bone formation and that this effect can be specifically counteracted with medication,” says Professor Jan Tuckermann, Head of the Institute of Molecular Endocrinology and Physiology. “This opens up a completely new avenue for the development of drugs to treat osteoporosis.” Tuckermann led the study together with Professor Anita Ignatius, Director of the Institute of Orthopaedic Research and Biomechanics, and Dr Mubashir Ahmad, a postdoctoral researcher at the same institute.

Significantly increased bone mass

The less Axl present in the bone cells, the better they can grow. When the protein molecule was blocked, cells formed significantly more bone tissue in the laboratory. This is because a gene becomes more active, which, via a signalling pathway, ensures that bone cells develop more effectively. This effect was also observed in mice: after just two to four weeks of treatment with the active substance BGB324, the animals had significantly more bone mass in their femurs and vertebrae. “It was particularly important for us to demonstrate that blocking Axl also works in a living organism. The significant increase in bone mass gives us confidence that this approach could also be relevant for human patients,” said Professor Anita Ignatius.

Apart from a slight, harmless reduction in white blood cells, no discernible harmful side effects were observed during the treatment of the mice. Lead author Dr Mubashir Ahmad emphasised: “A major benefit of our discovery is that the active substance we used, BGB324, is already being tested in clinical trials for cancer. As data on safety and tolerability are already available, this could pave the way for faster application in osteoporosis treatment.”

In addition to Ignatius and Tuckermann’s institutes, several other departments at Ulm University Medicine were involved in the study: the Institute of Immunology, the Clinic for Neurology, the German Centre for Neurodegenerative Diseases and the German Centre for Child and Adolescent Health, all based in Ulm.

The work was funded, amongst others, by the German Research Foundation (DFG) and the Federal Ministry of Research, Technology and Space. The active substance BGB324 used in the study was provided free of charge by the Norwegian company BerGenBio.

Publication details:

Ahmad, M., Kölbl, C., Jan, I. et al. *Inhibition of AXL receptor tyrosine kinase increases osteoblast function and bone mass*. Bone Res 14, 71 (2026). DOI: 10.1038/s41413-026-00554-0

Press release

16-Oct-2026

Source: Ulm University

Further information

Prof. Dr Jan Tuckermann,

Head of the Institute of Molecular Endocrinology and Physiology

Email: jan.tuckermann@uni-ulm.de

► [Ulm University](#)