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Promising New Targeted Therapy for Acceleration of Bone Fracture Repair
Preliminary Results Indicate Increased Bone Density, Lower Toxicity, and Reduced Healing Time

WASHINGTON, Nov. 7, 2018 — There are over [six million fractures](#) per year in the U.S. with direct [costs](#) in the billions, not to mention [lost productivity](#). The only drug currently available to accelerate the healing process must be applied directly onto the fracture surface during surgery, but not all breaks require such intervention. New research, *Bone Fracture-Targeted Dasatinib Conjugate Potently Enhances Fracture Repair In Vivo*, presented today at the 2018 [American Association of Pharmaceutical Scientists \(AAPS\) PharmSci 360 Meeting](#) highlights a novel bone anabolic agent that, when injected, intravenously reduces femur fracture healing time by 60 percent without impacting the surrounding healthy tissue.

Researchers from [Purdue University](#) designed a new chemical entity (a dasatinib-aspartate₁₀ conjugate, DAC) that employs a targeted peptide attached to dasatinib, an anti-cancer drug that has been shown to promote the growth of new bones. Following systemic injection, DAC is observed to concentrate on the fracture surface, resulting in accelerated repair and increased bone density. The data shows that the healing process that typically takes eight weeks for full recovery of mechanical strength is reduced to three to four weeks when treated with the targeted drug.

“We foresee a significant need for this type of therapy,” said presenting author, Mingding Wang, Purdue University. “Even though many broken bones don’t need surgery, most require a prolonged healing process that can lead to morbidity, loss of work productivity, and in some cases even death. By developing a therapy that can accelerate bone fracture repair without damaging healthy bones or tissues, we can hopefully address these critical issues.”

The study results indicate that treatment with DAC every other day for three weeks was equally effective as daily injections of DAC, yielding a 114 percent increase in bone density, and was found to be the best treatment interval. Reducing this dosing interval to every four days, however, resulted in a measurable decline in potency. Since the blood supply to the fracture area is often disrupted immediately after a fracture, waiting a week or two for blood vessels to stabilize prior to administering DAC did not negatively impact its effectiveness or the healing speed.

While administration of nontargeted dasatinib provided some improvement in healing rate, DAC was dramatically better, doubling the bone density. In addition, since the nontargeted form of dasatinib is administered chronically to cancer patients without significant toxicity, the fracture-targeted form is expected to be even safer. That is, when dasatinib is selectivity targeted to the bone fracture surface, its presence in all other tissues should be greatly reduced.

Philip Low, principal investigator and Presidential Scholar for Drug Discovery, Purdue Institute for Drug Discovery noted, “While the use of casts, rods, or pins may still be required in some cases, the ability of this therapy to accelerate the return of a fracture patient to normal function and lifestyle could have widespread benefits to the entire orthopedic community.”

The next stage of the research is proof of efficacy of DAC in additional fractures, including long bone fractures, hip fractures, nonunion fractures, spinal fusions, and craniofacial fractures.

Editor’s Note:

All media must provide press credentials to attend this meeting and register on-site at Walter E. Washington Convention Center, main registration. To schedule an interview with the researchers, or for any other press inquiries, please contact Stacey May at 703-459-7677 or mays@aaps.org or Hillarie Turner at hillarie@vaneperen.com. For the most up-to-date program information, please click [here](#).

Bone Fracture-Targeted Dasatinib Conjugate Potently Enhances Fracture Repair In Vivo will be presented Wednesday, Nov. 7, 10:30 a.m. – 11:30 a.m. (EST) Poster Forum 2 the Walter E. Washington Convention Center.

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