Bad mix of bacterial remnants and genetics leads to arthritis

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Here's another reason to hate leftovers. A research study appearing in the April 2009 issue of the *Journal of Leukocyte Biology* sheds light on one cause of arthritis: bacteria. In the study, scientists from the United States and The Netherlands show that a specific gene called NOD2 triggers arthritis or makes it worse when leftover remnants of bacteria cell walls, called muramyl dipeptide or MDP, are present. This discovery offers an important first step toward new treatments to prevent or lessen the symptoms of inflammatory arthritis.

"Despite recent advances in the treatment of arthritis, none target its cause," said Michael Davey, Associate Chief of Staff for Research at the Portland Oregon Veteran's Affairs Medical Center and one of the researchers involved in the study. "Our work with MDP and NOD2 is a step toward understanding the root cause of arthritis which one day may allow certain forms of arthritis to be prevented altogether."

"Now that we know that bacterial products can activate this NOD2 pathway and that this signal contributes to arthritis," said John Wherry, Ph.D., Deputy Editor of the Journal of Leukocyte Biology, "the next step is to find treatments that either rid the body of this inflammatory signal or mask it. Either way, the net effect would be the same: people would be spared from a very crippling disease. "

According to the U.S Centers for Disease Control and Prevention more that 40 million American say that they have been told by a doctor that they have arthritis or another rheumatic condition. Arthritis is the most

common cause of disability in the United States and limits activities of nearly 19 million people.

More information: H. L. Rosenzweig, M. M. Jann, T. T. Glant, T. M. Martin, S. R. Planck, W. van Eden, P. J. S. van Kooten, R. A. Flavell, K. S. Kobayashi, J. T. Rosenbaum, and M. P. Davey. Activation of nucleotide oligomerization domain 2 exacerbates a murine model of proteoglycan-induced arthritis. J Leukoc Biol 2009 85: 711. www.jleukbio.org/cgi/content/abstract/85/4/711

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