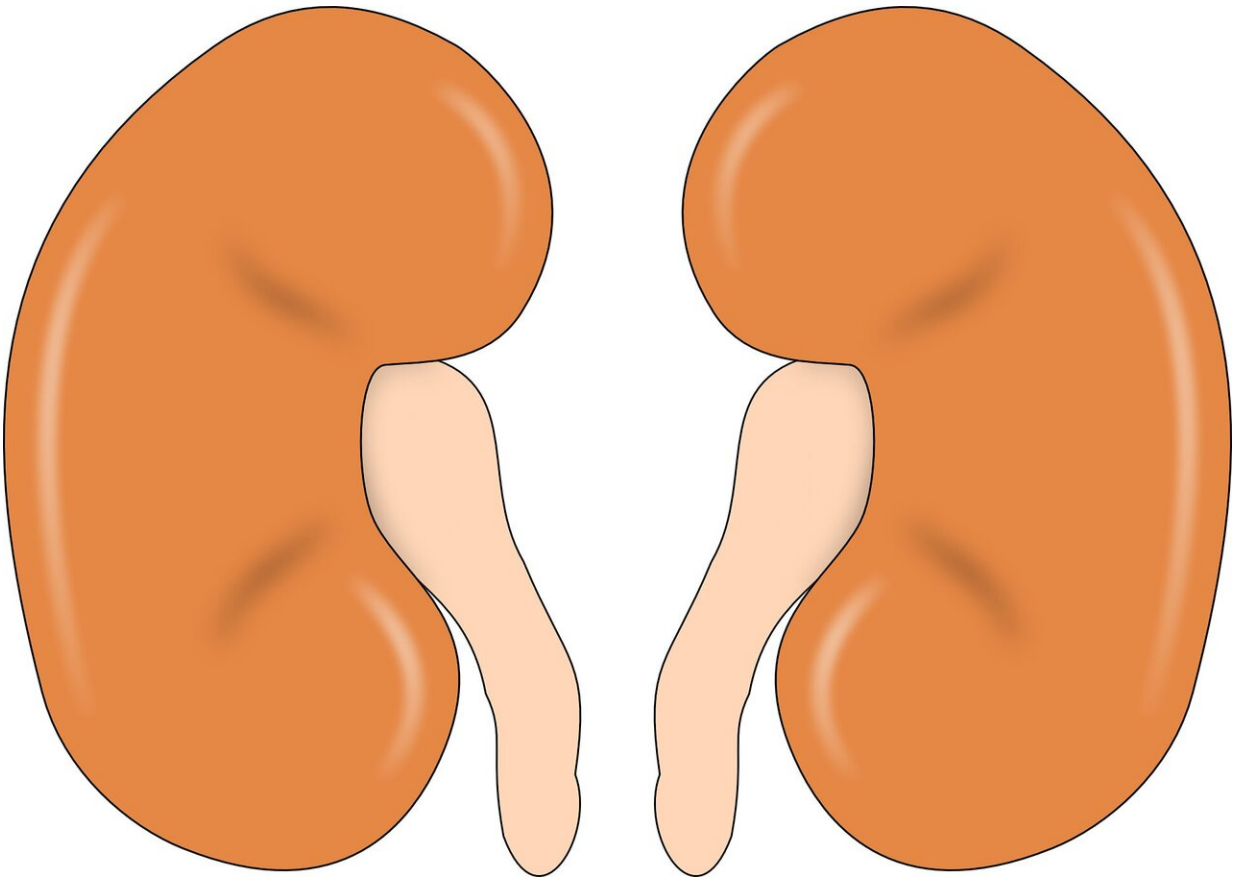
Researchers discover that a molecule of blood is effective against autoimmune kidney disease

February 3 2020



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Lupus is a chronic autoimmune disease that can damage any part of the

body. When it causes inflammation in the kidneys (called lupus nephritis), they cannot properly remove waste from the blood or control body fluids. Without treatment, nephritis can lead to scarring and permanent damage of the kidneys, and possibly final renal failure. In this case, patients need to undergo dialysis and possibly a kidney transplant. Currently, lupus nephritis patients are treated with nonsteroidal anti-inflammatory drugs and immunosuppressors, which are unsatisfactory and have side effects. Therefore, new therapeutic agents with higher potency, selectivity and safety are needed.

The research group, on immuno-inflammatory processes and gene therapy of the Bellvitge Biomedical Research Institute (IDIBELL), has published a study in the prestigious journal *Kidney International* that demonstrates the immunomodulatory therapeutic capacity of a molecule present in our blood, called C4BP (β-). These results, validated in two animal models of lupus nephritis, represent a very promising advance for patients. Also, the applicability of this discovery is being evaluated in other autoimmune disorders, such as colitis and rheumatoid arthritis.

Autoimmune diseases are caused by a malfunction of the immune system, which attacks its own cells and tissues, becoming an aggressor instead of a protector. The complement pathway is one of the components of the innate system. This, among other functions, protects the body from pathogenic organisms. The IDIBELL team, led by Dr. Josep M. Aran, has demonstrated a new activity for one of the forms of complement inhibitor protein C4BP (β-). Specifically, it has been shown that the protein is able to reprogram myeloid cells (a type of white blood cell), transforming its proinflammatory and immunogenic activity to anti-inflammatory and tolerogenic activity.

The C4BP protein (β-) can be isolated from human serum or obtained artificially (recombinantly) by culturing eukaryotic cells that express it. In mice that reproduce human lupus nephritis, it has been shown that the

administration of C4BP (β-) greatly improves the symptomatology of the disease, especially when is compared to the therapeutic action of classical immunosuppressants. In addition, it has no side effects and is also effective against dermatitis, another condition derived from lupus.

More information: Ana Luque et al, Noncanonical immunomodulatory activity of complement regulator C4BP(β-) limits the development of lupus nephritis, *Kidney International* (2019). DOI: 10.1016/j.kint.2019.10.016

Provided by IDIBELL-Bellvitge Biomedical Research Institute