Vitronectin (Vn) plays an important role in a number of physiological and pathophysiological processes. It promotes the adhesion and spreading of a wide variety of cell types and is a subcomponent of the soluble SC5b-9 complex of complement where it protects bystander cells from cytolysis. Vn also plays an important role in fibrinolysis by stabilizing PAI-1 in its active conformation which otherwise rapidly converts to a latent form.

Vn was previously known as serum-spreading factor or S-protein, and is a plasma and serum glycoprotein with a normal concentration ranging from 200 – 400 ug/ml. It exists in both a 75 kDa single-chain form and a 65 + 10 kDa two-chain form. Vn can exist in at least two different conformational forms. The majority of Vn found in the circulation is present in the native (“closed”) form. In this form, most of the binding sites for other ligands are cryptic. The second form of Vn, the denatured (“open”, multimeric) form, is a result of a conformational change in the native protein induced by denaturants such as urea, adsorption onto surfaces, low pH or reduction and alkylation. This conformational change leads to exposure of the heparin binding site, formation of disulfide-bonded multimers and rupture of the disulfide bond that links the 10 kDa light chain to the 65 kDa heavy chain of the two chain form. The liver is the primary site of vitronectin synthesis, however, Vn is also found in platelets, megakaryocytes, monocytes and macrophages.