Receptor-mediated Decapentaplegic (Dpp) degradation is seen by biologists to play an important role in allowing for the formation of relatively stable PMad patterns of signaling Dpp gradient in (and thereby the development of) the wing imaginal disc of a Drosophila fruit fly. To the extent that receptors act as a "sink" for BMP proteins such as Dpp, localized expression of ectopic (or an abnormal amount of) the Dpp receptor Thickvein (tkv) would cause a net flux of free Dpp morphogens toward the site of receptor over-expression. One possible consequence would be a depression of Dpp signaling in adjacent areas since less Dpp morphogens are now available for binding with the same concentration of receptors at the adjacent areas. However, recent experiments on Dpp-Sog interaction designed to examine this possible effect were inconclusive. The principal goal of the present talk will be to address this problem. The talk will consist of three parts: 1) a brief introduction to the tissue patterning phase of biological development of an organism; 2) a description of the controversy at the turn of this century that attracted the speaker to work in this field and the possible resolution he and his two collaborators provided by modeling, analysis and computation, and 3) formulation of a mathematical model that accounts for the essential biological processes responsible for a possible depression of Dpp signaling outside the area of elevated tkv in a Drosophila embryo and the information extracted from this model by the method of matched asymptotic expansions about the question of reduced signaling.