# Article information:

Bullous Pemphigoid IgG Induces Cell Dysfunction and Enhances the Motility of Epidermal Keratinocytes via Rac1/Proteasome Activation - PubMed
<https://pubmed.ncbi.nlm.nih.gov/30809225/>

# Article summary:

1. Bullous pemphigoid (BP) IgG induces internalization of type XVII collagen (ColXVII) from the plasma membrane of keratinocytes through macropinocytosis, leading to morphological and functional changes in BP IgG-stimulated cells.

2. These changes include alterations in cell membrane structure, dysfunctional mitochondria, increased production of reactive oxygen species, increased motility, and detachment.

3. Rac1 and proteasome activation are involved in the effects of BP IgG on cultured keratinocytes, and pharmacological inhibitors of these pathways can reverse the cellular alterations induced by BP IgG.

# Article rating:

May be slightly imbalanced: The article presents the information in a generally reliable way, but there are minor points of consideration that could be explored further or claims that are not fully backed by appropriate evidence. Some perspectives may also be omitted, and you are encouraged to use the research topics section to explore the topic further.

# Article analysis:

The article titled "Bullous Pemphigoid IgG Induces Cell Dysfunction and Enhances the Motility of Epidermal Keratinocytes via Rac1/Proteasome Activation" published in Frontiers in Immunology provides insights into the cellular dynamics following ColXVII internalization induced by BP IgG. The study highlights the role of keratinocyte kinetics in the direct functions of IgG in patients with BP.

The article presents a detailed analysis of the effects of BP IgG on cultured keratinocytes, including morphological and functional changes that lead to subepidermal blistering associated with BP pathogenesis. The authors report that BP IgG triggers a cascade leading to metabolic impairments and stimulates cell migration in treated keratinocytes. These cellular alterations are reversed by pharmacological inhibitors of Rac1 or the proteasome pathway, suggesting that Rac1 and proteasome activation are involved in the effects of BP IgG on cultured keratinocytes.

While the study provides valuable insights into the mechanisms underlying BP pathogenesis, there are some potential biases and limitations to consider. Firstly, the study only focuses on one aspect of BP pathogenesis, namely its effect on keratinocyte kinetics. Other factors such as genetic predisposition, environmental triggers, and immune system dysfunction may also play a role in disease development.

Secondly, while the study reports that Rac1 and proteasome activation are involved in the effects of BP IgG on cultured keratinocytes, it does not explore other potential mechanisms or counterarguments. Further research is needed to confirm these findings and explore other potential pathways involved in disease development.

Thirdly, while the article notes that BP is an autoimmune disease characterized by autoantibodies targeting type XVII collagen expressed in basal keratinocytes, it does not provide a comprehensive overview of current understanding regarding this condition. This may limit its usefulness for readers who are unfamiliar with this topic.

Overall, while this study provides valuable insights into the cellular dynamics underlying BP pathogenesis, further research is needed to confirm these findings and explore other potential mechanisms involved in disease development. Additionally, a more comprehensive overview of current understanding regarding this condition would be beneficial for readers who are unfamiliar with this topic.

# Topics for further research:

* Bullous pemphigoid genetic predisposition
* Environmental triggers of bullous pemphigoid
* Immune system dysfunction in bullous pemphigoid
* Type XVII collagen and bullous pemphigoid
* Alternative mechanisms of bullous pemphigoid pathogenesis
* Overview of current understanding of bullous pemphigoid

# Report location:

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