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7th International Immunoglobulin Conference: Mechanisms of Action

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Summary

The mechanism of action by which therapeutic administration of intravenous immunoglobulin (IVIg) is able to provide a beneficial effect in autoimmune and inflammatory diseases is not yet fully understood, but current research is providing some answers. Signalling via receptors that interact with immunoglobulin (Ig) is crucial, and genetic polymorphisms of the Fc receptors have clear links to disease and also appear to influence the outcome of IVIg treatment. Glycosylation of the IgG, Fc- or Fab-fragments has a role in enhancing or blocking the pro- and anti-inflammatory effector functions. In addition, and independently of Fc receptors and glycosylation, Fc fragment and the constant domain of the Fab fragment contain binding sites for activated complement fragments that mediate complement-scavenging based immunomodulation. Although IgG Fc sialylation may not be critical for IVIg activity, research in some diseases suggests that it is associated with improved clinical outcomes. Therefore, further investigation of how IgG and IgA receptor expression and regulation affects the outcome of IVIg treatment may further clarify the mechanisms behind IVIg, and provide valuable guidance for future treatment paradigms.