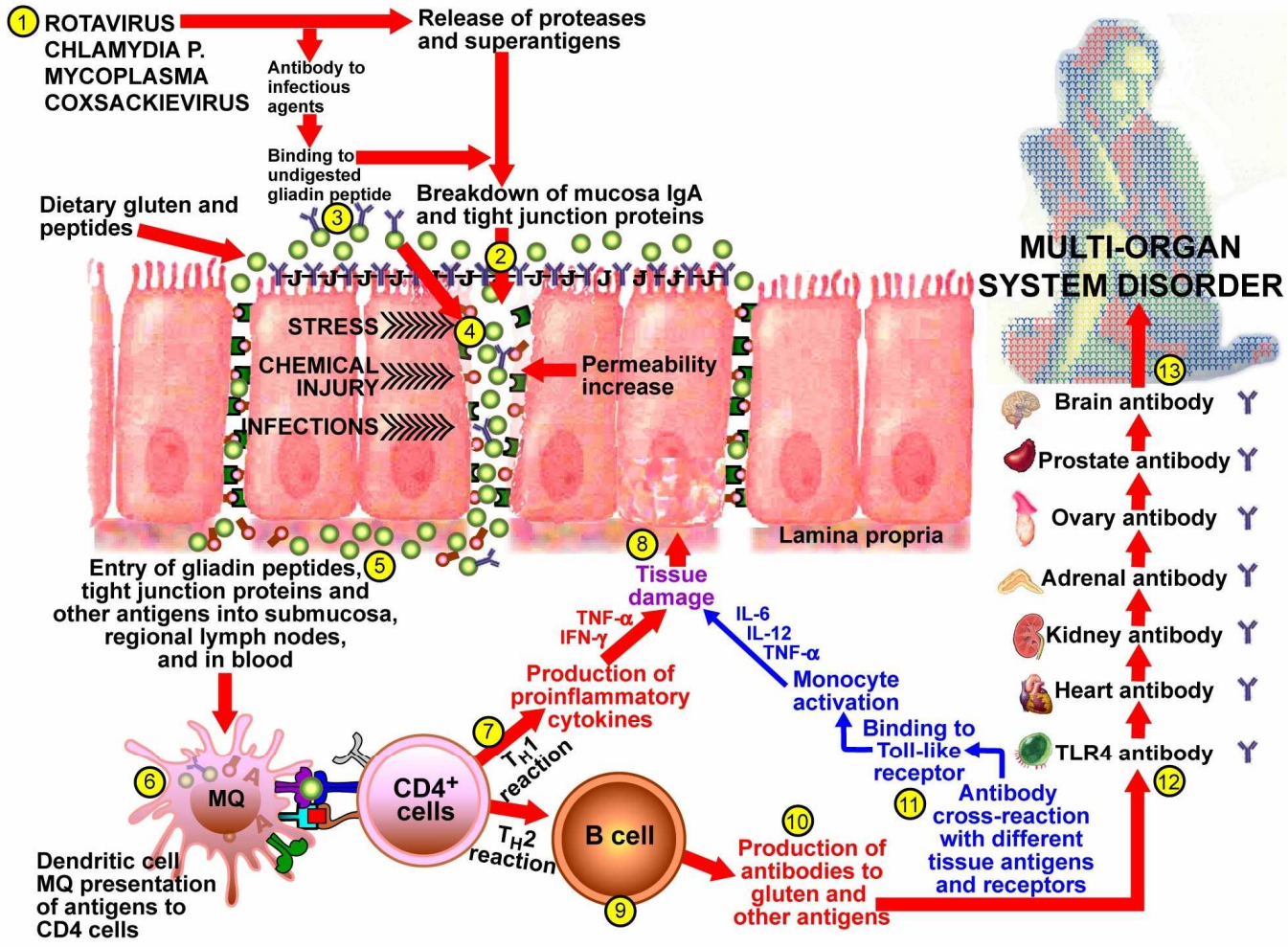

Depiction of immunological mechanisms underlying gluten intolerance and its immunopathological consequences.

Precipitation of gluten intolerance appears to be preceded by acute gastroenteritis symptoms induced by infections such as rotavirus and others(1).

Rotavirus and its super-antigens can break down mucosal IgA directly (2) or indirectly by the local production of anti-rotavirus antibody. Due to partial linear homology or cross-reactivity between rotavirus protein and a-gliadin, the anti-rotavirus antibody binds to gliadin and forms complexes with it (3).

The combination of infection antibody cross-reactivity with gliadin and additional stressors can severely impair mucosal integrity (4) and the entry of gliadin peptides, tight junction proteins and other antigens into the submucosa, regional lymph nodes, and the blood (5).

Gliadin peptides, rotavirus antigens A, rotavirus antibody bound to gliadin, and tight junction proteins are presented by dendritic cells with or without HLA-DQ2/DQ8 to $CD4^+$ cells (6).

This antigenic presentation results in driving the cell $CD4^+$ response either towards TH_1 reaction (7), production of proinflammatory cytokines, mucosal cell destruction and autoimmunity (8); or towards TH_2 response B-cell activation (9) and antibody production against gluten, rotavirus, and tight junction proteins (10).

Cross-reaction of these antibodies with cell receptors such as toll-like receptor (11) and tissue antigens such as heart kidney, adrenal gland, ovary, prostate, brain and others (12) results in further tissue damage and multi-organ system disorders (13).

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