Simplified Map of NMOSD/MOGAD

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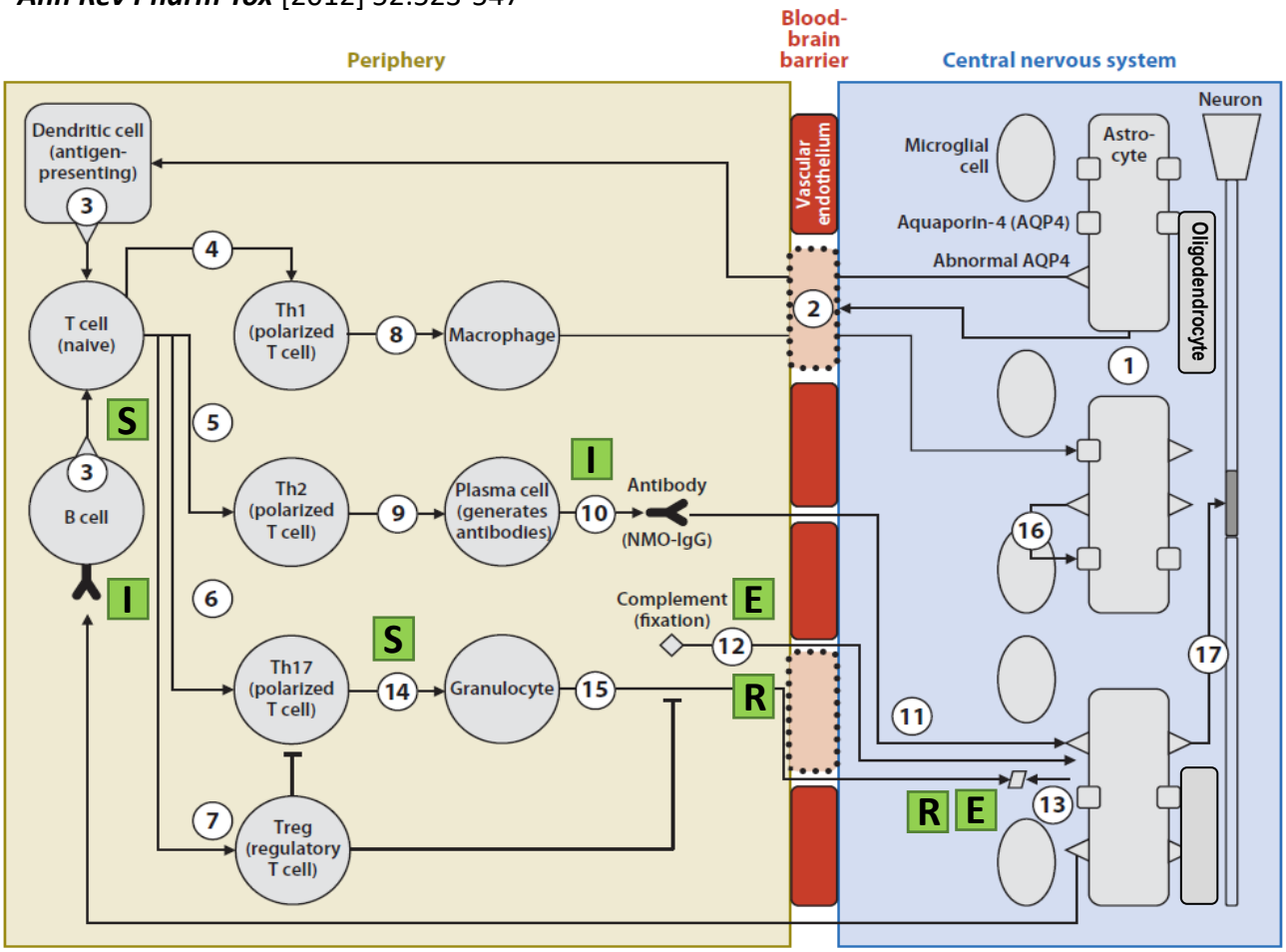

Figure 6

Hypothesized therapeutic targets and candidates in neuromyelitis optica (NMO). Each number represents a potential target for therapeutic intervention on the basis of emerging evidence: **1**, suppression of astrocyte proinflammatory signals (e.g., α-IL-6, α-TNF); **2**, restoration of the blood-brain barrier (also termed the neurovascular unit; e.g., α-VEGF-A); **3**, interference of dendritic cell, T cell, and B cell interactions and inflammatory second-signals (e.g., α-CD40/CD40L or α-CD80/CD86/CD28); **4**, interference with Th1 polarization (e.g., α-T_{bet}, α-STAT4, or α-IL-12); **5**, inhibition of Th2 or B cell activation events (e.g., α-BAFF/BLyS); **6**, inhibition of Th17 polarization (e.g., α-RORγτ or α-IL-23); **7**, activation of autoimmune-modulating systems (e.g., CTLA4 or adoptive transfer of Tregs); **8**, hypothetical blockade of Th1 cytokines (e.g., interferon-γ); **9**, inhibition of key Th2 cytokines (e.g., α-IL-4, α-IL-5, or α-IL-13); **10**, inhibition of B cell subsets such as proplasma (e.g., $CD19^+CD20^-$), naive (e.g., $CD19^+CD20^+$), and plasma (e.g., $CD19^+CD20^-CD138^+$) B cells; **11**, innovative approaches to develop protective antibody responses (e.g., beneficial monoclonal antibody or induction of α-idiotypic networks via development of tolerance); **12**, complement inhibition (e.g., α-C1q, α-C5); **13**, inhibition of specific inflammatory consequences of complement fixation (e.g., α-C5a) or related chemotactic signals (e.g., chemokines), or induction of complement-modulating mechanisms (e.g., CD59, decay-accelerating factor, or C1-INH); **14**, blockade of Th17 pathway cytokines (e.g., α-IL-17, α-IL-22, α-IL-23); **15**, inhibition of inflammatory mechanisms of granulocytes (e.g., chemotaxis, elastase); **16**, restoration of immune tolerance; **17**, repair of central nervous system injury through tissue regeneration. Abbreviations: AQP4, aquaporin-4; BAFF, B cell activating factor; BLyS, B lymphocyte stimulator; CTLA, cytotoxic T lymphocyte-associated antigen; IL, interleukin; INH, inhibitor; ROR, retinoic acid–related orphan receptor; STAT, signal transduction activator of transcription; TNF, tumor necrosis factor; Treg, regulatory T cell; VEGF, vascular endothelial growth factor. (See also the **Supplemental Visualizing NMO tool**; follow the **Supplemental Materials link** from the Annual Reviews home page at

Notes:

FDA Approved Therapies [2024]

E Eculizumab (*Soliris*)

I Inebilizumab (*Uplizna*)

S Satralizumab (*Enspryng*)

R Ravulizumab (*Ultomiris*)