

FIGURE 28.3 (A) Concentration-dependent instantaneous acceleration of A β (1–40) misfolding in the presence of atheronal-B (3). In the absence of Ath-B, no detectable aggregation of A β is observed within the time shown. (B) Fibrils formed from A β with atheronal-B upon agitation adopt a classic amyloidlike network of fibrils as observed by AFM. (From [], with permission. Copyright © Wiley-VCH Verlag GmbH & Co. KGaA.)

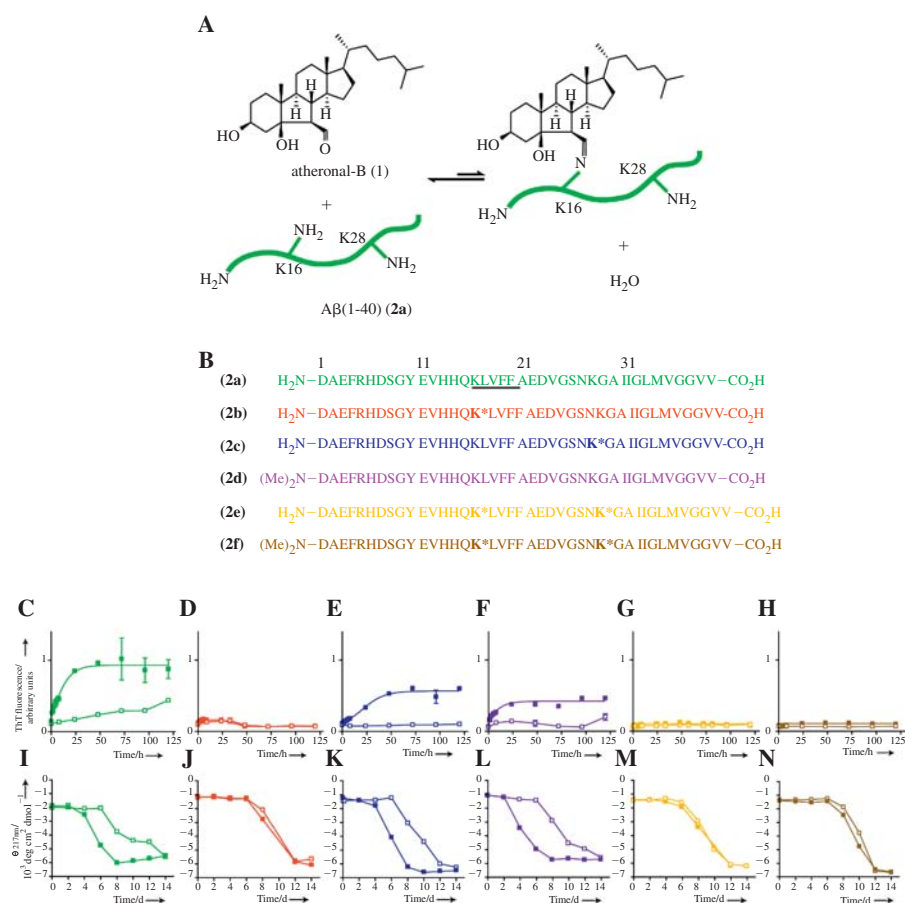


FIGURE 28.4 (A) Schiff base formation between atheronal-B and primary amines on Aβ; (B) sequences of mono-, bis-, and tris-dimethylated peptide analogs (2b to f) to native Aβ(1-40) (2a); (C-N) kinetics of atheronal-B-induced aggregation of amyloid-β peptides 2a to f. (C-H) ThT analyses, ex: 440 nm and em: 485 nm reported as mean ± SD; (I-N) far-UV CD analyses (reported as an average of three scans) of mean residue ellipticity (θ) at 217 nm, of peptides 2a (—, wild-type); of peptides 2a (—, wild-type); 2b (—, K*16); 2c (—, K*28); 2d (—, Me₂N-D1); 2e (—, K*16, K*28); 2f (—, Me₂N-D1, K*16, K*28). In each case, the peptide (100 μM) in phosphate-buffered saline, pH 7.4, is incubated quiescently in the presence (filled squares) or absence (open squares) of aldehyde 1 (100 μM) at 37°C. (From [], with permission. Copyright © Wiley-VCH Verlag GmbH & Co. KGaA.)