Pharmacological treatment of Alzheimer disease in 2028.

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Given the average duration of clinical trials (Phase I to Phase III) of 9-10 years at an average cost of 5-7 billion $ (SCOTT,2013) is not too early to consider today the possibility of a pharmacological treatment for 2028.The cause of the disease still escape us and the major investment made so far in developing anti-abeta interventions have not given clinically relevant results ,therefore, we are limited to design therapies based on results derived from molecular imaging (PET) .This approach moves from traditional neuropathological criteria into a phenotype-targeted therapy which may or may not be casually related.The multistate transition model (C.Jack et al.2016, 2017) utilizes transition rates to estimate the frequency of each state based on long- term follow up of cognitively unimpaired individuals from 50 to 90 years.The most recent one (C.Jack, 2017) combines three different imaging-based measurement in vivo: a-beta aggregation ,tauopathy and neurodegeneration ,evaluated with repeated PET tested in the same subject. .Subtyping based on these criteria makes it possible to design a differentiated therapy targeting specific pathologies in the individual patient.This approach emphasizes the critical need to continue our research in order to find the cause of the disease the best therapy within the next ten years.