rate: 0.4%; failure: 99.6%).[185] During the past 15 years no new drugs have been approved for the treatment of AD and the available drugs are not cost-effective.[186] Therefore, the pharmacogenetics of AD is very limited, circumscribed to cholinesterase inhibitors and memantine (Table 5.1), remaining stuck in a primitive stage of underdevelopment due to the lack of novel therapeutic options. Although many studies on the pharmacogenetics of AD have been published since the early 2000s,[64,65] many of them are redundant and contradictory, focusing mainly on the *APOE* gene and, to a lesser extent, on some *CYP* family genes and other minor genes.[63] In this context, several considerations are pertinent regarding further steps to be followed in order to achieve a more mature profile of AD pharmacogenomics: (i) a better characterization of the roles played in drug efficacy and safety by genes involved in the pharmacogenomic network is necessary; (ii) since most genes are under the influence of the epigenetic machinery, pharmacoepigenomics is becoming an attractive field that deserves special attention; (iii) drug–drug interactions represent a problematic issue in over 80% of AD patients; (iv) since the neurodegenerative process underlying AD neuropathology starts 20–30 years before the onset of the disease, novel therapeutics should be addressed to prevent premature neuronal death; (v) specific biomarkers for AD are necessary in 3 different contexts: predictive markers before disease onset, early diagnosis in initial stages, and drug monitoring (in both preventive and/or therapeutic strategies); and (vi) physicians should be aware of the usefulness of pharmacogenomics to prescribe more accurately, avoid adverse reactions and optimize the limited therapeutic resources available for the treatment of dementia.[8,187]

During the past 10 years, over 1000 different compounds have been studied as potential candidate drugs for the treatment of AD.[9,11,18,188] About 50% of these substances are novel molecules obtained from natural sources.[9,11] The candidate compounds can be classified according to their pharmacological properties and/or the AD-related pathogenic cascade to which they are addressed to halt disease progression. In addition to the FDA-approved drugs since 1993 (tacrine, donepezil, rivastigmine, galantamine, memantine) (Table 5.1), most candidate strategies fall into 6 major categories: (i) novel cholinesterase inhibitors and neurotransmitter regulators, (ii) anti-Aβ treatments (APP regulators, Aβ breakers, active and passive immunotherapy with vaccines and antibodies, β- and γ-secretase inhibitors or modulators), (iii) anti-tau treatments, (iv) pleiotropic products (most of them of natural origin), (v) epigenetic intervention, and (vi) combination therapies.[8,9,11,18]

In more global terms, prospections of diverse natural sources (vegetal, marine, animal) have allowed the identification and characterization of novel bioproducts with potential utility in the prevention and treatment of a vast array of age-related pathological phenotypes and NDDs as well. Prototypal examples of these biotechnological products are LipoFishins and Atremorine.[23]