35. NEW STRATEGIES IN THE DEVELOPMENT OF ANTIPLATELET DRUGS

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Introduction. Antiplatelet medication plays an essential role in prevention and healing of thrombotic affections such as myocardial infarction or cerebrovascular accident. Despite the shown efficiency in the prevention of ischemic events, bleeding issues, thrombocytopenia, and recurrent thrombotic events serve as an impulse for the elaboration of new medicine that will remove thrombotic risks without effects on hemostasis. Platelet adhesive receptors, coagulation process phases and thrombin receptors represent the main targets in the elaboration of new antiplatelet medicine.

Aim of study. The goal is to identify the progress of new antiplatelet medicine development in scientific literature.

Methods and materials. This workpaper is realized on the analytical basis of the articles published between 2018-2022 on PubMed and Google Scholar. The most relevant articles were selected with the help of key word antiplatelet and the generic name of medicines.

Results. In current therapy are more commonly used cyclooxygenase inhibitors (acetylsalicylic acid, triflusal), P2Y12 purinergic receptors antagonists (clopidogrel, prasugrel, ticagrelor, cangrelor, regrelor, elinogrel), GPIIb/IIIa receptor antagonists (abciximab, eptifibatide, tirofiban, xemilofiban, orbofiban, sibrafiban, etc.) and PAR-1 antagonists (voraxapar, atopaxar). The new antiplatelet drugs are in preclinical and clinical trials, targeting the following platelet receptors and pathways: receptors P2Y12 (selatogrel, AZD1283, SAR21647) and P2Y1 (BMS-884775), phosphoinositide 3-kinase (AZD6482, TGX-221, idelalisib), protein disulfide-isomerase (isoquercetin, ML359), GPIIb/IIIa receptor (elarofiban, RUC-1, RUC-4), protease-activated receptor 4 (BMS-986120), GPVI (revacept, losartan, scFv9012, Troa6 și Troa10), GPIb-IX-V (caplacizumab, antifibatide), 12-lipoxygenase (ML355), P-selectin (rPSGL-Ig, PSI-697, PSI-421), CD40 (anti-CD40, Ab), prostacyclin analogs (iloprost, treprostinil, beraprost), phosphodiesterase 3 (dipiridamol, cilostazol, anagrelide, milrinone), glucagon like peptide 1 receptor (exenatide), thromboxane–prostaglandin receptor (ramatroban, ridogrel, seratrodast, terutroban), serotonin 5-HT2A receptor (APD791), prostanoid EP3 receptor, nitric oxide donors.

Conclusion. The studies on mechanism of platelet blood clots formation have developed new antiplatelet therapies that would reduce side effects and would offer new perspectives on adapting the antiplatelet therapy on patient´s pathophysiology. For the realization of this goal, new studies are needed that should evaluate the therapeutic efficiency and the ability of new medications to prevent recurrent thrombotic events.