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Synthesis and hypolipidemic properties of novel N-(4-benzoylphenyl) pyrrole-2-carboxamide derivatives

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Hyperlipidemia is involved in development of atherosclerosis and coronary heart disease. We synthesized two novel pyrrole carboxamide derivatives N-(4-benzoylphenyl)-4-bromo-2,5-dihydro-1H-pyrrole-2 carboxamide (1) and 4- Amino-N-(4-benzoylphenyl)-1-methyl-1H- pyrrole-2carboxamide (2) and tested them as anti-hyperlipidemic agents. The synthesized compounds were characterized using IR and NMR. Biological evaluation of compound 1 and 2 showed that compound 1 significantly decreased TG, LDL-C and TC, and mild increase in HDL-C in plasma. Contrarily, compound 2 appeared to be less potent when compared to 1; it moderately decreased TG, LDL-C and TC with mild increase of HDL-C. The NH pyrrole mediates H-bond interaction of 1 with the backbone of the target(s) protein(s) and this corresponds to the high potency of 1. The lower activity of 2 confirms that the presence of H-bond is essential to induce high activity. The finding of this work suggests that this scaffold might be promising as anti-hyperlipidemic agents for future work.

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Metabolic engineering toward enhanced LC-PUFA biosynthesis in *Nannochloropsis oceanica:* Overexpression of endogenous Δ12 desaturase driven by stress-inducible promoter leads to enhanced deposition of polyunsaturated fatty acids in TAG

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Nannochloropsis oceanica is an important source for omega-3 long-chain polyunsaturated fatty acids (LC-PUFA) such as eicosapentaenoic acid (EPA, 20:5 n-3), and a potent candidate for biofuel production, due to its outstanding capability for rapid induction of triacylglycerol (TAG) overproduction. In contrast to membrane lipids, TAG of *N. oceanica* is poor in the valuable LC-PUFA, EPA. We identified, cloned and characterized a *N. oceanica* microsomal-like Δ12-desaturase (*NoD12*) mediating the committing step of LC-PUFA biosynthesis by converting oleic acid (18:1 n-9) to linoleic acid (LA, 18:2 n-6). We generated strains of *N. oceanica* over expressing *NoD12* under the control of the stress-inducible endogenous lipid droplet surface protein (LDSP) promoter, resulting in robust expression under nitrogen starvation conditions. The overexpression of *NoD12* significantly altered fatty acid composition of total lipids and of individual lipid classes, such as a drastic increase in 18:2 proportion in phosphatidylcholine and in TAG was observed under nitrogen starvation. Some LA was converted further toward LC-PUFA resulting in a substantial increase in arachidonic acid (20:4 n-6) in TAG. Our data demonstrate the feasibility of metabolic engineering to increase LC-PUFA content in the biotechnologically important microalgae using native genes and promoters, and provide novel insights into the regulation of LC-PUFA flux to TAG under nitrogen starvation.

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