Preface

Weight loss – in many cases, as little as 4 kg – with its pleiotropic benefits and optimal safety profile appears as the most effective means of managing type 2 diabetes (T2DM) (Harris 1991; Pories et al. 1995; Sjostrom et al. 2004; Pontiroli et al. 2005; Ratner et al. 2005; Pi-Sunyer et al. 2007; Dixon et al. 2008). In addition, the pharmacotherapeutic armamentarium seems well equipped with ten different classes of antidiabetic drugs; providing potent tools to achieve predefined HbA1c goals (i.e. insulin, sulphonylureas, metformin, thiazolidinediones, alpha-glucosidase inhibitors, glinides, GLP1-analogues, DPP-4 inhibitors, pramlintide, and colesevelam) (Rodbard et al. 2009).

What then are we struggling for? First, in most of the T2DM patients, present clinical praxis fails to attain sustained weight loss and glycemic control (Nathan et al. 2009). Second, even if optimal management of HbA1c, lipid profile and blood pressure could hypothetically be supplied, increased morbidity and mortality rates would still leave much room for improvement (Mourad and Le Jeune 2008). Third, unravelling the molecular pathophysiology of nutrient excess should allow to target the thrifty genotype roots of obesity and T2DM directly and should thus facilitate the development of highly efficient novel therapies (Neel 1999). Respectively, distinct encouragement evolves from potential mechanisms underlying treatments through metformin and bariatric surgery (Cummings et al. 2004; Foretz et al. 2010).

The chapters of this book report cutting-edge research on molecular events in adiposity and T2DM, thus opening the way for innovative drug-based therapeutic strategies. Beyond that, profound insights and exciting ideas are unveiled. Please, go ahead and explore!

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References


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