Inflammatory bowel diseases (IBD) are chronic progressive diseases. Current therapeutic strategies using a classical step-up approach have not significantly altered the natural history of IBD. Persistent inflammation, which may still occur in the absence of clinical symptoms, is believed to lead to progressive bowel damage over time, which manifests with the development of strictures, fistulae and abscesses in Crohn’s disease (CD). Whether or not this concept applies to ulcerative colitis is debated. On the wake of other chronic diseases, such as rheumatoid arthritis, the goal of therapy has now shifted from mere control of symptoms to altering natural history to prevent bowel damage and disability. A new instrument, the Lemann score, is being developed to measure the cumulative structural bowel damage caused by CD over time and to assess the impact of new treatment strategies on long-term outcomes. In order to prevent accumulation of damage, a treat to target approach using endoscopic healing as a first definition of the target is now proposed together with tight control of inflammation based on monitoring of symptoms and biomarkers. Still, several questions remain regarding the definition of mucosal healing and the long-term benefit of treating patients beyond clinical symptoms. Assessment of objective measures of inflammation is an increasingly important part of the management of IBD. During follow-up, clinical decision-making is increasingly being driven by the findings of continued monitoring (for objective evidence of inflammation), with the aim of optimizing treatment for tight disease control. Here again, there are several unanswered questions around implementing this model in practice: Which monitoring tools should be used? When should they be used? How should the monitoring strategy differ in different patient scenarios? Complementary to the ‘treat to target’ concept is early intervention: losing time in high-risk patients will lead to less chance to reach the target and increased risk of further progression and bowel damage. However, early introduction of disease-modifying anti-IBD drugs cannot be recommended in all patients since disease progression is highly variable from patient to patient.
In order to reach the target, there are several unmet needs that need to be addressed in IBD. Optimization of current therapies using better understanding of pharmacokinetics is needed. Development of predictors of disease progression (clinical, serologic, genetic…) is mandatory to avoid under- and overtreating. Advances in the understanding of the roles of the adaptive and innate immune systems as well as the intestinal epithelium and endothelium have resulted in the development of multiple new biologics. However, there is a huge potential for variation in inflammatory response from patient to patient and within a single patient over time and then in response to different treatments. This uncertainty emphasizes the need for a personalized patient-based approach based on an integrated ‘omics’ approach incorporating genetic, microbiota with clinical and environmental data. Finally, in this era of increasing complexity of care, education of patients to involve them in a well-informed decision-making process should be encouraged.

To conclude, it is important to recognize that most of the new concepts in the treatment of IBD have not yet been validated, and prospective studies to evaluate their long-term impact on new end points such as bowel damage are still ongoing.