and poorly associated with BMI.2 Hence, if BMI is unhelpful in planning therapy, is it possible that more specific measurement of body composition would allow us to tailor treatment accordingly?

Ding et al have hypothesised in AP&T that body composition can indeed predict the outcome of anti-TNF therapy.3 There are several reasons why this may be the case. Both skeletal muscle and adipose tissue are known to release pro- and anti-inflammatory cytokines, and disease activity may be associated with malabsorption and malnutrition.4 Using CT scanning software, Ding et al assessed body composition in patients with Crohn’s disease undergoing anti-TNF therapy. Of the group studied, 24.5% had a primary nonresponse to anti-TNF and 57.7% of these were demonstrated to be myopenic (OR 4.69). There was, however, no association with body composition and secondary loss of response. Interestingly, although demonstrated in another observational study,5 obesity (BMI >30 in this study) did not predict primary or secondary loss of response and was poorly correlated with all of the composite markers of body composition.

The lack of drug and antibody levels available at the time of this observational retrospective cohort study makes it impossible to assess causality, but it suggests that myopenia in Crohn’s patients is a significant factor in predicting anti-TNF primary response.

However, we should remember that not all patients with myopenia had a primary nonresponse and that this, therefore, represents only one of the factors we need to consider. With the advent of increasingly available biomarkers, body composition may well be something we should be considering when tailoring the treatment of patients with Crohn’s disease. The authors also suggested that we should consider muscle-building exercise in Crohn’s patients to improve response to the treatment. Although not specifically looked at in this paper, holistic individualised care should be the aim of all of our therapies, and perhaps, we are one step closer to being able to choose the right biologic at the right time for the right patient.

ACKNOWLEDGEMENTS

Declaration of personal and interests: None.

REFERENCES


DOI: 10.1111/apt.14409

Editorial: anti-TNF therapy and myopenia in Crohn’s disease—another step towards personalised medicine. Authors’ reply

We thank Dr Murray for his insightful editorial on our retrospective cohort study examining body composition parameters and their associations with anti-TNF therapy outcome.1,2 As the therapeutic targets for Crohn’s disease are still limited, it is vital to unravel the many causes of primary nonresponse and secondary loss of response to current biologic therapies.

With pharmacologic and clinical studies into anti-TNF therapy demonstrating loss of response secondary to possible infliximab clearance and leakage across the gut membrane, escalated dosing has been proposed as appropriate for induction management in ulcerative colitis.3,4 Other parameters such as severity of disease are important considerations as it results in malabsorption and an increased catabolic
state. Interestingly, in an article by Dotan et al, patients with low weight (< 40 kg) were more likely to have low trough levels as infliximab clearance increased with lower body weight. It is indeed true that albumin, body weight and the presence of antibodies to infliximab are all factors which can influence primary nonresponse.

We agree with Dr Murray that due to the lack of anti-TNF levels, we are unable to assess causality. Our data also demonstrated that each quartile of myopenia resulted in an increasing rate of primary nonresponse and large variations in dosing when using lean body mass rather than weight.

The potential of muscle to act as a reservoir for infliximab following administration and its anti-inflammatory properties by releasing myokines may explain the impact of muscle mass on outcomes in Crohn’s disease. Furthermore, inadequate dosing may lead to increased immunogenicity and greater drug clearance emphasising the importance of adequate dosing of patients to prevent primary nonresponse.

The difficulties in aiming for muscle-building during a severe flare of inflammatory bowel disease are certainly appreciated. However, nutritional support in conjunction with medical therapy may also offer patients a more individualised and optimised approach to care.

Ultimately, we hope that precision medicine using all the available tools to determine the appropriate dose and correct drug will be important in achieving the goals of treatment with mucosal healing as proposed by the STRIDE guidelines. Body composition parameters may be another important variable in the data obtained in future randomized control trials to demonstrate efficacy.

ACKNOWLEDGEMENTS
The authors’ declarations of personal and financial interests are unchanged from those in the original article.

FUNDING INFORMATION
None.

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REFERENCES

DOI: 10.1111/apt.14399

Editorial: avoiding corticosteroids in the treatment of inflammatory bowel disease

Corticosteroids have remained a cornerstone of therapy for inflammatory bowel disease (IBD) for decades, but short- and long-term side effects limit their practical utility. Data from the TREAT registry indicated that prednisone use was a significant predictor of both infection and mortality in patients with Crohn’s disease. Recent guidelines from medical societies recommend against routine first-line use of corticosteroids for the management of mild-to-moderate ulcerative colitis (UC), and when prescribed for moderate-to-