Abstract PWE-253 Table 1 Improvement in IBDQ

<table>
<thead>
<tr>
<th>Week</th>
<th>PBO (N = 246)</th>
<th>ADA (N = 248)</th>
</tr>
</thead>
<tbody>
<tr>
<td>8</td>
<td>20 ± 36</td>
<td>29 ± 36*</td>
</tr>
<tr>
<td>32</td>
<td>20 ± 41</td>
<td>28 ± 41*</td>
</tr>
<tr>
<td>52</td>
<td>19 ± 41</td>
<td>27 ± 42*</td>
</tr>
</tbody>
</table>

*P < 0.05, p Values from ANCOVA with treatment and prior anti-TNF status as factors and baseline value as covariate.
†P < 0.05, p Values from Cochran-Mantel-Haenszel test stratified for prior anti-TNF use.

Conclusion For pts with moderate to severe UC who failed conventional therapy, ADA was more effective than PBO for inducing and maintaining improvements in HRQOL, as measured by IBDQ through 52 wks.


PWE-254 IMPACT OF INDUCTION DOZING ON MAINTENANCE OUTCOME WITH ADALUMUMAB IN CROHN’S DISEASE
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Introduction Two induction regimens of adalimumab are used in Crohn’s disease (CD): 160/80 mg or 80/40 mg at Weeks 0/2. We compared long-term efficacy for patients who received 160/80 mg vs 80/40 mg as induction therapy followed by crom maintenance therapy.

Methods Data were from two randomised, double-blinded, placebo-controlled efficacy and safety trials in moderate to severe CD. EXTEND, a 52-week study in patients with mucosal ulceration, used the 160-/80-mg induction regimen. CHARM, a 56-week study used the 160-/80-mg induction regimen. All patients who started with induction dose and were randomised to crom plus dropouts prior to Week 4 randomisation were included. Missing Crohn’s Disease Activity Index (CDAI) scores were imputed with both non-responder imputation (NRI) and last observation carried forward (LOCF). Remission (CDAI <150) and hospitalisation were compared between induction regimens. To incorporate the correlation between visits for a patient, a logistic regression with the patient-level random intercept using all the time points after Week 4 was constructed to compare likelihood of remission, controlling for baseline CDAI, fistula, prior use of an anti–tumour necrosis factor therapy, concomitant medications, CD duration, and other factors.

Results 70 patients in the 160-/80-mg group were compared with 336 patients in the 80-/40-mg group. Baseline characteristics were similar except for greater rates of rectal/anal CD in the 160-/80-mg group and greater use of concomitant steroids in the 80-/40-mg group. Compared with the 80-/40-mg group, the 160-/80-mg group had a greater percentage of time in remission from Week 0–52 (36% vs 25%; p < 0.05, NRI), significantly fewer hospitalisations per patient (0.09 vs 0.25; p < 0.05), and significantly fewer CD-related hospitalisations (p = 0.018 vs 0.18; p < 0.05). Patients in the 160-/80-mg group were significantly more likely to be in remission during Weeks 4 to 52 than were patients in the 80-/40-mg group after adjusting for baseline characteristics (adjusted OR 4.8; p = 0.001). LOCf results for remission analysis were consistently similar. The 160-/80-mg regimen did not appear to lead to a higher rate of AEs.

Conclusion The 160-/80-mg induction regimen of adalimumab was associated with a greater likelihood of remission, more time in remission, and fewer hospitalisations during crom maintenance therapy compared with the 80-/40-mg regimen.


PWE-255 5-ASA ENHANCES DUOX2 EXPRESSION IN ACTIVE ULCERATIVE COLITIS: A RISK FOR COLORECTAL CANCER?
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Introduction Colonic DUOX2 expression produces hydrogen peroxide, a reactive oxygen species (ROS), which is up-regulated in active ulcerative colitis (UC). Overproduction of hydrogen peroxide amplifies ROS-induced genetic damage and causes cellular transformation which may explain the increased colorectal cancer (CRC) risk associated with chronic UC. Mesalazine (5-ASA) has been shown to be chemo-preventative for UC associated CRC and scavenges ROS. Here, we aimed to identify and investigate the effect of 5-ASA on DUOX2 expression using human rectal cancer cell lines and mucosal tissue biopsies.

Methods Mucosal biopsies were taken from 35 patients with UC and 24 patients with normal colons for in vivo experiments, and 24 patients with UC and 14 patients with normal colons for ex vivo experiments. Total RNA was extracted and quantitative real-time PCR used to calculate expression of DUOX2. Cytometric bead array technology was used on ex vivo culture supernatants to measure cytokine profiles. In situ hybridisation for DUOX2 expression was performed on sections from eight matched pairs of non-inflamed/inflamed biopsies and five matched pairs of non-inflamed/inflamed dysplasia biopsies from UC patients. Human rectal cancer cells were
PWE-254 Impact of induction dosing on maintenance outcome with adalimumab in Crohn's disease


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