

The safety and efficacy of retreatment with IFX after a short “drug holiday” was recently demonstrated in the STORI trial. However, data regarding re-treatment after a long drug holiday (>1 year) are few. With increased use of biologics, the number of patients who have lost response to both biologics is increasing. These patients have limited therapeutic options but retreatment with IFX has been proposed.

**Methods** We performed a retrospective review of patients with Crohn’s disease who had been re-treated with IFX after a period off treatment of at least 1 year. Patients were identified from our biologics database and their records were reviewed. Patient details and clinical outcome measures were extracted into a standardised form.

**Results** 24 patients (14 male) were studied with a median age of 38 years (range: 21–61 years). 15 patients had responded to their first course of treatment; IFX was stopped due to episodic treatment being the norm at that time (n=9), patient choice (n=3), failure to re-attend for planned treatment (n=1) and development of strictures requiring surgery (n=2). The median time between stopping IFX and retreatment was 35 months (range 14–102). In this cohort, 80% responded to retreatment (12/15), 2 developed infusion reactions and 1 developed secondary loss of response. Median follow-up among continued responders was 20 months (range 2–43). Nine patients stopped their first course of IFX for either primary non-response (n=5), secondary loss of response (n=2) or infusion reaction (n=2). The median time between treatments was 32 months (range 18–42). In this cohort, 78% of patients responded to retreatment (7/9); 2 had infusion reactions. Follow-up among ongoing responders was for a median of 11 months (range: 2–84 months). All infusion reactions occurred on the second retreatment dose despite premedication with hydrocortisone (200 mg iv).

**Conclusion** Re-treatment with IFX after a drug holiday of at least 1 year was frequently successful, whether the patients had initially responded to IFX or not. The main limiting factor was the development of infusion reactions. We conclude that retreatment with IFX is a viable option in people with limited therapeutic options even if they failed to respond to their first course of treatment or have previously lost response.

Abstract PTU-102 Table 1

Disease duration (y) (median (range))	36 (21–59)
Age at diagnosis	22 (11–50)
% Ileal disease	4% (1/24)
% Ileocolonic	25% (6/24)
% Colonic disease	71% (17/24)
% B1 (non-stricturing/penetrating)	21% (5/24)
% B2 (stricturing)	38% (9/24)
% B3 (penetrating)	42% (10/24)
% p (perianal)	38% (9/24)
Concurrent immunosuppressants	63% (15/24)

**Competing interests** None declared.

#### PTU-103 ANTI-INFLIXIMAB ANTIBODIES: PREVALENCE, INFUSION REACTIONS, IMMUNOSUPPRESSION AND RESPONSE, A META-ANALYSIS

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**Introduction** Infliximab is a chimeric monoclonal antibody directed against tumour necrosis factor  $\alpha$ . When used in inflammatory bowel

disease (IBD), primary non-response is seen in at least 10% of patients with secondary loss of response occurring in a further 10%–15% per year. It has been suggested that this may in part be a result of the development of anti-infliximab antibodies (ATIs). The prevalence of ATIs varies according to the frequency of administration of infliximab and the use of immunosuppressants. ATIs have also been linked with infusion and hypersensitivity reactions. We aimed to perform a meta-analysis of the prevalence of ATIs in people with IBD receiving infliximab, the effect of immunosuppressive drugs on prevalence of ATIs and the effects of ATIs on infusion reactions and remission rates.

**Methods** MEDLINE and EMBASE databases were systematically searched from 1948 and 1980 respectively to October 2011. Inclusion criteria included randomised controlled trials, cohort studies or case series reporting on anti-infliximab antibodies in adult or juvenile IBD. Data from eligible studies were extracted into a standardised form and a meta-analysis performed.

**Results** 18 studies involving 3326 patients were included. The prevalence of ATIs was 45.8% when episodic infusions of infliximab were given and 12.4% when maintenance infliximab was given. Rates of infusion reactions were significantly higher in patients with ATIs (RR: 2.07, 95% CI 1.61 to 2.67). Immunosuppressants resulted in a 50% risk reduction in prevalence of ATIs. The prevalence of ATIs was 17.5% in patients using immunosuppressants and 37.7% in those who were not (RR: 0.50, 95% CI 0.42 to 0.59). However, the presence or absence of ATIs did not significantly affect rates of clinical remission, which was 46.5% in patients with ATIs and 60.2% in those without (p=0.10).

**Conclusion** The prevalence of ATIs depends on the regimen of infliximab administration and the use of immunosuppressants. Patients who develop ATIs are at increased risk of infusion reactions, but the presence of ATIs does not, by itself, have an effect on rates of clinical remission.

Abstract PTU-103 Table 1

	Number of ATIs	95% CI	RR
Episodic Infliximab	95/128 (74.2%)	0.66 to 0.81	
Episodic Infliximab + immunosuppression	77/171 (45.0%)	0.38 to 0.53	0.60
Maintenance Infliximab	148/423 (35.0%)	0.31 to 0.40	
Maintenance Infliximab + immunosuppression	32/315 (10.2%)	0.07 to 0.14	0.31

**Competing interests** None declared.

#### PTU-104 THIUGUANINE NUCLEOTIDES: OPTIMISING THIOPURINE THERAPY IN INFLAMMATORY BOWEL DISEASE (A DGH PERSPECTIVE)

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**Introduction** Azathioprine (AZA) and 6-mercaptopurine (6MP) have been used in treatment of inflammatory bowel disease since introduction in the 1960s.<sup>1</sup> 6-thioguanine nucleotides (TGN) levels are related to therapeutic response with likely minimum threshold value of 235–250 pmol/8×10<sup>8</sup> RBC.<sup>2–5</sup> Thiopurine metabolism is affected by thiopurine methyltransferase (TPMT) and its activity is therefore important. Methylated derivatives like 6 methyl mercaptopurine<sup>6</sup> (MMP) are possibly partially responsible for hepatotoxic effects and maximum threshold value has been established at 5700 pmol/8×10<sup>8</sup> RBC in a paediatric IBD population.<sup>2</sup>