Table 1 OC-113 Data represents basal time-points pre-iron load

	Healthy Controls (n=15)	C282Y/C282Y (n=9)	p
Mean age, ($\pm$ SD)	28.4 ($\pm$ 4.1)	53.8 ($\pm$ 13.4)	
Hepcidin (ng/ml)	32.9 (24.8–43.7)	13.1 (7.2–23.9)	<0.0001*
Iron (μ mol/l)	14.6 (12.7–16.8)	27.9 (20.2–38.6)	0.0002*
Ferritin (μ g/l)	51.2 (34.7–75.5)	50.9 (39–66.6)	0.53
%TSAT	25.3 (21.5–29.8)	52.3 (37.1–73.8)	0.0001*
Hepcidin: ferritin ratio	1.1 (0.8–1.5)	0.3 (0.1–0.5)	0.0044*

Data are presented as GM (95%CI) by unpaired t test (* p <0.05 considered significant)

Figure 1 OC-113

under the curve (AUC) was calculated and compared between the two groups.

Results The basal serum hepcidin levels in patients with *HFE*-H were significantly low as compared to HC ($p=0.0002$) (table 1). Incremental serum hepcidin response seen in HC reached significance at 4 h post iron challenge ($p=0.0085$). There was no significant hepcidin response in *HFE*-H at 4 h ($p=0.294$) (figure 1). The overall hepcidin response was significantly lower in *HFE*-H as compared to HC (AUC: $p=0.0127$).

Conclusion Failure to mount a rapid hepcidin response to an oral iron challenge is one of the key mechanisms of iron accumulation in spite of prevailing excess body iron in *HFE*-H patients with C282Y/C282Y mutation.

Competing interests None.

Keywords hereditary haemochromatosis, iron.

OC-113

BLUNTED INCREASE IN SERUM HEPCIDIN AS RESPONSE TO ORAL IRON IN *HFE* – HEMOCHROMATOSIS

doi:10.1136/gut.2011.239301.113

A Sangwaiya,¹* V Manglam,¹ M Busbridge,² M Thursz,³ J Arnold¹ ¹Department of Gastroenterology, Ealing Hospital NHS Trust, Southall, UK; ²Department of Clinical Chemistry, Charing Cross Hospital, London, UK; ³Department of Hepatology, St Marys Hospital, London, UK

Introduction Majority of patients with *HFE* hemochromatosis (*HFE*-H) are homozygote for C282Y mutation. Studies suggest iron accumulation in most types of hemochromatosis is due to hepcidin deficiency. The precise link between hepcidin levels and iron absorption in *HFE*-H patients has been poorly understood. We measured hepcidin response to oral iron challenge (200 mg ferrous sulphate), in *HFE*-H (C282Y/C282Y) patients and compare to healthy controls (HC).

Methods Nine patients with C282Y/C282Y *HFE*-H along with 15 HC were recruited for the study. All *HFE*-H were iron depleted and studied at a time distant to phlebotomy. Hepcidin was measured using a published immunoassay method. Serum iron, ferritin and transferrin saturation (%TSAT) were measured using standard methods. The area