and nintedanib are licensed treatments but are only available at specialised centres. We have previously shown that patients whose local hospital was a prescribing centre (PC) were more likely to be receiving AFDs than those whose local hospital was not a prescribing centre (NPC).[1]

Objectives We set out to test the hypothesis that this indicates differences in ability to travel or to seek specialist care which might be reflected in differences in indices of multiple deprivation (IMD).

Methods We obtained a full list of patients who received AFDs since 2013 and obtained their postcodes from hospital databases. We additionally obtained markers of socio-economic status based on the IMD score obtained from government websites. Data were recorded in January 2016 and July 2017 and compared with non-parametric statistics.

Results The number of patients per 1 00 000 population in each postcode area started on AFDs increased from a median (range) of 3.04 (0–15.86) in 2016 to 8.81 (1.16–33.87) in 2017 ($p=4x10^{-6}$). In both 2016 ($p=0.0119$) and 2017 ($p=0.0089$), there were more patients on AFDs per postcode area where the local hospital was a PC compared to a NPC. Looking at the distribution of IMD in 2017, there was a small difference ($p=0.057$) that did not appear fully to explain the difference in AFD prescriptions between PC and NPC.

Conclusions The NHS constitution requires equality in access to therapy regardless of where the patient lives. Although AFD prescriptions have increased significantly between 2016 and 2017, we have again demonstrated inequality of access to AFDs depending on patient location. These differences do not appear fully explained by differences in indices of multiple deprivation.

Refernce 1. Woodhead FA, Townsend S, Desai D. P171 Health inequality exists in pirfenidone prescription for idiopathic pulmonary fibrosis in the English Midlands according to patient location. *Thorax* 2016, Dec 1;71(3):A176–7.

M27 EFFECT OF PIRFENIDONE ON BREATHLESSNESS AS MEASURED BY THE UCSD-SOBQ SCORE IN PATIENTS WITH IDIOPATHIC PULMONARY FIBROSIS (IPF) WITH MODERATE LUNG FUNCTION IMPAIRMENT

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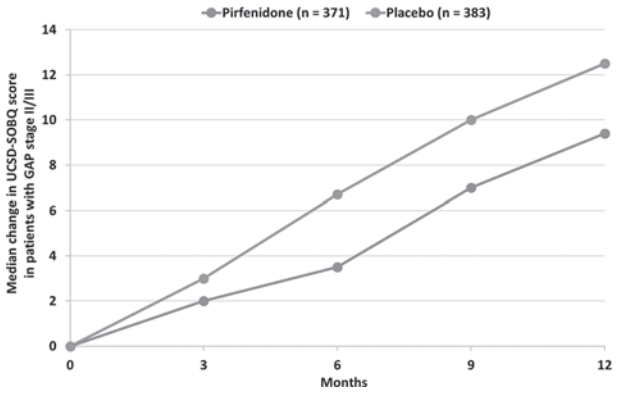
Introduction Treatment of IPF with pirfenidone slows disease progression as measured by changes in forced vital capacity (FVC), independent of baseline FVC values. In a previous analysis of patients with limited vs more advanced lung function impairment, increases in University of California, San Diego Shortness of Breath Questionnaire (UCSD-SOBQ) scores were more pronounced in patients with Gender Age Physiology index (GAP) stage II/III vs GAP stage I and in patients with baseline FVC <80% vs FVC ≥80%0·1 We examined the effect of pirfenidone on UCSD-SOBQ in these subpopulations.

Methods 1247 patients in ASCEND (NCT01366209) and CAPACITY (NCT00287716; NCT00287729) were randomised to pirfenidone 2403 mg/d or placebo. Patients were stratified by GAP stage I vs stage II/III and by baseline%–predicted FVC. The effect of pirfenidone on UCSD-SOBQ score was assessed by continuous and categorical changes from baseline over 12 months, and by multiples of the minimal clinically important difference of 5 points for UCSD-SOBQ.



Abstract M27 Figure 1 Median increase in UCSD-SOBQ scores from baseline in patients with GAP stage II/III.

Results Pirfenidone-treated patients with GAP stage II/III had higher UCSD-SOBQ scores after 12 months than those with GAP stage I (median increase from baseline: 9.4 vs 5.0); similar Results occurred with placebo (12.5 vs 4.3). GAP stage II/III patients treated with pirfenidone had less increase in median UCSD–SOBQ score at 12 months compared with those receiving placebo (9.4 vs 12.5; median difference −3.5, 95% CI −6.2,–0.5; $p=0.0161$) with the curves diverging after 3 months (figure 1). Evaluation of categorical change for patients with GAP stage II/III demonstrated that pirfenidone reduced the proportion of patients with UCSD-SOBQ score increases of ≥15 points (45.6% vs 38.4%; $p=0.0449$) and ≥20 points (37.7% vs 28.6%; $p=0.0089$) at 12 months compared with placebo; increases of ≥5 or≥10 points were similar between treatment groups. Results in patients with% FVC ≤80% were comparable to GAP stage II/III.

Conclusions In patients with IPF with moderate lung function impairment, pirfenidone reduced the progression of breathlessness compared with placebo. Patients receiving pirfenidone showed less change from baseline in UCSD-SOBQ score and a lower proportion of patients had more pronounced increases in UCSD-SOBQ scores at 12 months.

REFERENCE

1. Albera C *et al*. *Eur Respir J* 2016;48:843–851.

M28 DEFERRING TREATMENT WITH PIRFENIDONE RESULTS IN LOSS OF LUNG FUNCTION THAT IS NOT RECOVERED BY LATER TREATMENT INITIATION

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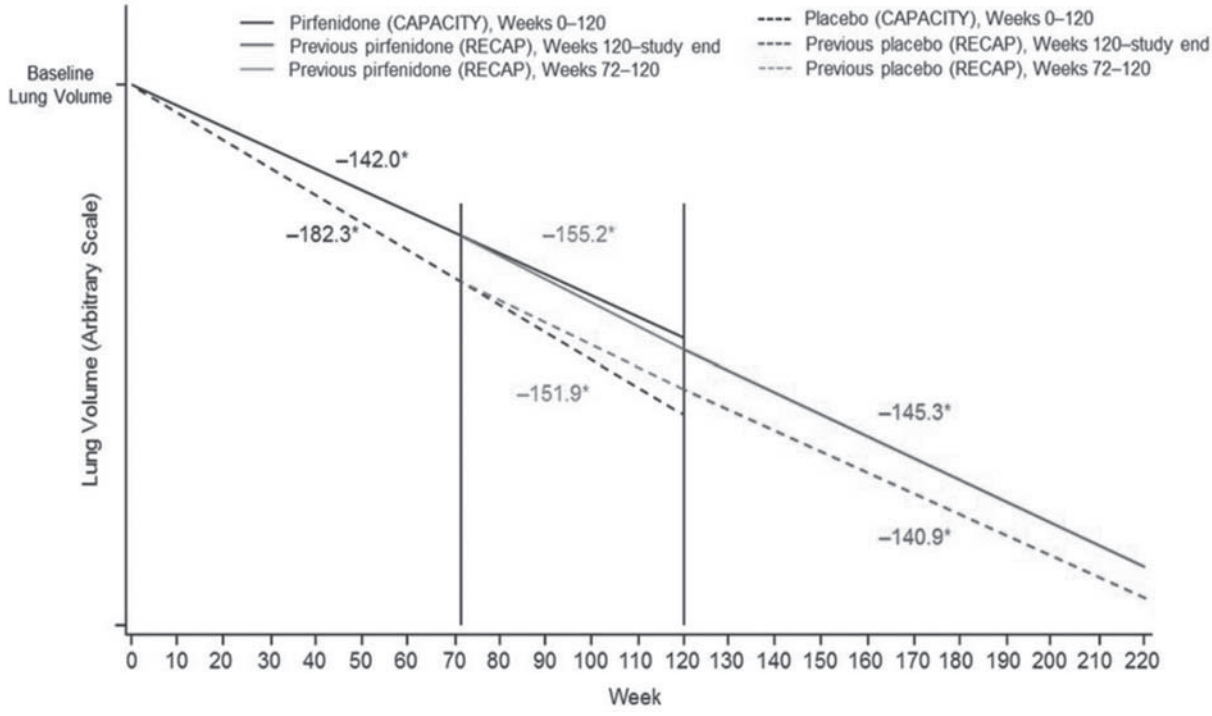
Introduction Idiopathic pulmonary fibrosis (IPF) is characterised by a progressive loss of lung function. Intervention with an anti-fibrotic, such as pirfenidone, as early as possible in the disease course may be the most appropriate strategy to preserve lung capacity.[1] In this analysis, data from the pivotal CAPACITY (004/006; NCT00287716/NCT00287729) trials and subsequent RECAP (012; NCT00662038) rollover trial were used to investigate the impact of deferring pirfenidone treatment on annual decline in lung function (forced vital capacity [FVC]) in patients with IPF.

Methods The annual rate of lung function (FVC; mL) decline was calculated for all treated patients who completed CAPACITY and RECAP. Weeks 0–120 included all patients randomised in CAPACITY to pirfenidone 2,403 mg/day or placebo. Weeks 72–120 included data for the transition period, when patients either continued pirfenidone or switched to pirfenidone in RECAP. After Week 120, only data for patients in RECAP were included. Patients randomised to pirfenidone in CAPACITY were compared with those who received placebo in CAPACITY before initiating pirfenidone in RECAP.

Results From Week 0 to 120, the annual rate of lung function decline (FVC) was −142.0 mL (n=345) in patients who received pirfenidone in CAPACITY and −182.3 mL (n=347) in those who received placebo. During the transition period (Weeks 72–120), these values were −155.2 mL (n=236) and −151.9 mL (n=249) in patients who continued and switched to pirfenidone, respectively. In RECAP (≥Week 120), the annual rate of lung function decline in patients who received pirfenidone in both trials was −145.3 mL (n=219) and −140.9 mL (n=218) in those who switched from placebo to pirfenidone.

Conclusions These data show that loss of lung function during CAPACITY was not recovered during RECAP. Therefore, failure to initiate pirfenidone treatment in IPF as early as possible may lead to an irrecoverable loss of lung volume during the period without treatment.

Funding F. Hoffmann-La Roche, Ltd./Genentech, Inc.



Abstract M28 Figure 1 Annual rate of decline in lung volume in CAPACITY and RECAP.

REFERENCE

1. Albera C *et al*. *Eur Respir J* 2016;48:843–851.

M29 FVC DECLINE OVER 1 YEAR PREDICTS MORTALITY BUT NOT SUBSEQUENT FVC DECLINE IN PATIENTS WITH IPF

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Introduction In the INPULSIS trials, nintedanib reduced disease progression by reducing FVC decline vs placebo in patients with IPF. Patients who completed an INPULSIS trial could receive open-label nintedanib in the extension trial INPULSIS-ON.

Aim To assess the impact of FVC decline in INPULSIS on FVC decline and mortality in INPULSIS-ON.

Methods Descriptive analysis of the proportions of nintedanib-treated patients who had FVC decline <10% or≥10% predicted (pred) from baseline to week 52 of INPULSIS and the proportions of patients in these groups who had FVC decline <10% pred,≥10% pred, or died in the first year of INPULSIS-ON.

Results 430 patients received nintedanib in both INPULSIS and INPULSIS-ON. Of these, 89.1% had FVC decline <10% pred from baseline to week 52 of INPULSIS. FVC decline from baseline to week 52 in patients treated with nintedanib in INPULSIS did not predict FVC decline in the first year of INPULSIS-ON. Most patients (77.2%) had FVC decline <10% pred in the first year of INPULSIS-ON. Patients who had