created to examine adolescence in detail (0–5, 6–12, 13–15, 16–19, 20–23, 24–29 and ≥ 30 years). A longitudinal analysis (2008–2013) of weight change investigated potential explanatory variables between the sexes, such as diabetic status (CFRD) and FS.

Results Cross-sectional Boys 13–15 years had lower mean BMIP than girls (41.2 vs 50.5 $p < 0.001$). Boys had a lower Z-score than girls for age 13–23 years. Mean BMI was significantly lower in females (21.9 vs 22.5 $p < 0.001$). More females were underweight (BMI < 19) than males (18.9% vs 14.0% $p < 0.001$), this was specifically observed in ages ≥ 24 years. Underweight individuals died younger than individuals with BMI ≥ 19 (29.3 years vs 36.0 years $p = 0.007$). Lower weight was associated with more IVABx days. Males had higher rates of FS (34.7% vs 28.9% in females $p < 0.001$) specifically when ≥ 16 years. These sex differences were not explained by differences in ethnicity, genotype or socio-economic status.

Longitudinal Boys had a greater fall in BMIP (8.3 vs 4.1 in females $p = 0.002$). In adulthood, females had significantly less increase in BMI (0.20 vs 0.57 in males $p < 0.001$). FEV₁ decline was greater in females (7.4% vs 5.9% in males $p = 0.0016$) not receiving FS, with no difference in change in FEV₁ between the sexes in those receiving FS.

Conclusion From ≥ 16 years boys changed from having lower BMIP to higher mean BMI and lower rates of underweight status in adulthood. Higher rates of FS in adolescent boys might explain this. Lower weight is associated with earlier death and increased IVABx use. In individuals without FS females have a greater decline in FEV₁ than males, this is not seen in individuals on FS. This has not been previously shown and warrants further analysis.

Managing pleural disease: from intervention to conservation

S128 EXPLORING THE BEHAVIOUR OF MESOTHELIOMA IN A POST HOC ANALYSIS FROM THE TIME 1 TRIAL

¹RM Mercer, ²J Macready, ²H Jeffries, ³N Speck, ⁴NI Kanellakis, ⁵N Maskell, ⁶J Pepperell, ⁷T Saba, ⁸A West, ⁹N Ali, ¹⁰RF Miller, ¹JC Corcoran, ¹RJ Hallifax, ¹R Asciak, ¹M Hassan, ¹I Psallidas, ¹NM Rahman. ¹Oxford Centre for Respiratory Medicine and Oxford Respiratory Trials Unit, Oxford, UK; ²University of Oxford, Oxford, UK; ³University of Zurich, Zurich, Switzerland; ⁴Laboratory of Pleural Translational Research, Nuffield Department of Medicine, University of Oxford, Oxford, UK; ⁵Academic Respiratory Unit, Department of Clinical Sciences, Southmead Hospital, University of Bristol, Bristol, UK; ⁶Somerset Lung Centre, Musgrove Park Hospital, Taunton, UK; ⁷Blackpool Teaching Hospitals NHS Foundation Trust, Blackpool, UK; ⁸Medway Maritime Hospital, Gillingham, UK; ⁹King's Mill Hospital, Mansfield, UK; ¹⁰Research Department of Infection and Population Health, Institute of Epidemiology and Healthcare, University College London, London, UK

10.1136/thoraxjnl-2017-210983.134

Introduction Malignant Pleural Mesothelioma (MPM) is the only malignancy which develops primarily in the pleural space and is a common cause of a malignant pleural effusion (MPE). MPE associated with MPM is managed similarly to any other malignancy, but it is unclear if the underlying mechanisms of fluid accumulation are the same and whether different treatment strategies should therefore be employed. The TIME 1 trial enrolled patients with MPE who underwent pleurodesis. This post hoc analysis compares the outcomes of patients with MPM to the rest of the trial population.

Abstract S128 Table 1

	Mesothelioma	Other	Significance
Pleurodesis Success	77/107 (72.0%)	142/173 (82.1%)	$\chi^2 = 3.97$, 1 df, $p = 0.046$
Trapped lung	19/91 (20.9%)	26/177 (14.7%)	$\chi^2 = 1.65$, 1 df, $p = 0.199$
Mean total fluid drained (95% CI)	2208 (1917–2498)	2345 (2119–2570)	$p = 0.621$
Mean change in CRP (Day 0–1)	58 (SD 43)	32 (SD 50)	$p < 0.001$
Mean change in WCC (Day 0–1)	2.53 (SD 2.83)	1.89 (SD 3.31)	$p = 0.13$
Median enrolment pain VAS	4 (IQR 6)	5 (IQR 15)	$p = 0.016$

SD=Standard Deviation, df=degrees of freedom, CI=confidence interval, and IQR=Interquartile Range

Methods 298 patients had available data on their final diagnosis. A number of different variables were compared, including pleurodesis success, systemic inflammation, the prevalence of trapped lung, total fluid volume drained and baseline pain Visual Analogue Score (VAS).

Results Of the 298 patients included in the analysis 110 patients had mesothelioma (36.9%). Post pleurodesis, MPM patients had a significantly greater rise in CRP than those with other underlying pathologies but had a significantly lower rate of successful pleurodesis. Patients with MPM had a lower pain VAS score on enrolment. There was no significant difference in the rates of trapped lung, the total volume of pleural fluid drained or the change in White Cell Count (WCC) between the groups.

Conclusion There are significant differences in the outcomes of patients with MPM and those with other MPE. Patients with MPM had a lower pleurodesis rate but a significantly greater change in C-reactive protein levels post pleurodesis, signifying a higher inflammatory response to pleurodesis, which has been assumed to associate with pleurodesis success. The mechanisms causing the increased inflammatory response in MPM are unclear. The basis for the lower rates of pleurodesis is unexplained, especially as there was no significant difference in the rates of trapped lung. Patients with MPM had a lower level of pain VAS scores on enrolment but further analyses are needed to determine whether this is clinically relevant and reproducible. These data indicate that MPM behaves differently to other forms of MPE and treatment strategies should be tailored towards MPM as a separate entity.

S129 DOES INFLAMMATION PREDICT SUCCESSFUL PLEURODESIS? A POST HOC ANALYSIS FROM THE TIME 1 TRIAL

¹RM Mercer, ²J Macready, ²H Jeffries, ³N Speck, ⁴NI Kanellakis, ⁵N Maskell, ⁶J Pepperell, ⁷T Saba, ⁸N Ali, ⁹A West, ¹⁰RF Miller, ¹R Asciak, ¹R Hallifax, ¹JC Corcoran, ¹M Hassan, ¹I Psallidas, ¹NM Rahman. ¹Oxford Centre for Respiratory Medicine and Oxford Respiratory Trials Unit, Oxford, UK; ²University of Oxford, Oxford, UK; ³University of Zurich, Zurich, Switzerland; ⁴Laboratory of Pleural Translational Research, Nuffield Department of Medicine, University of Oxford, Oxford, UK; ⁵Academic Respiratory Unit, Department of Clinical Sciences, Southmead Hospital, University of Bristol, Bristol, UK; ⁶Somerset Lung Centre, Musgrove Park Hospital, Taunton, UK; ⁷Blackpool Teaching Hospitals NHS Foundation Trust, Blackpool, UK; ⁸King's Mill Hospital, Mansfield, UK; ⁹Medway Maritime Hospital, Gillingham, UK; ¹⁰Research Department of Infection and Population Health, Institute of Epidemiology and Healthcare, University College London, London, UK

10.1136/thoraxjnl-2017-210983.135