days (for further breakdown see Table1). The remaining (64%) were benign till to date.

Conclusion The data demonstrates that a dedicated pleural service has the ability to rapidly review and diagnose patients with suspected MPE (especially the cytology positive). There is a need for improvement in patient's timeline for those referred for VATS. Perhaps a dedicated pleural multidisciplinary meeting may help to reduce the delay and improve patient care.

Abstract P241 Table 1

Mean time from referral to Pleural clinic	5 days
Mean time from referral to pleural clinic to diagnosis (pleural fluid cytology positive)	17 days
Mean time from diagnosis (pleural fluid cytology positive) to treatment	26 days
Mean time from pleural clinic to VATS referral	10.2 days
Mean time from VATS referral to VATS procedure	22.6 days
Mean time from VATS to diagnosis	11.6 days

P242 THE PROGNOSIS OF PATIENTS DIAGNOSED WITH PULMONARY ADENOCARCINOMA AT LOCAL ANAESTHETIC THORACOSCOPY (LAT): THE ROLE OF PRIMARY T STAGE

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Background Adenocarcinoma is the commonest type of lung cancer and may present with metastatic malignant pleural effusion (MPE)[1] We observed that some patients with pulmonary adenocarcinoma diagnosed at LAT did not have radiological evidence of primary lung parenchymal lesion. We hypothesised that these patients may have a better prognosis than those with lung nodules or masses due to reduced tumour burden.

Methods We retrospectively reviewed all patients who underwent LAT from 2006–2016 and screened those diagnosed with pulmonary adenocarcinoma. We reviewed these patients' radiology, age, gender, TNM staging and prognosis.

Results 491 patients underwent LAT from 2006–2016. 69/491 (14.05%) were diagnosed with pulmonary adenocarcinoma on histology of parietal pleura. 8 patients out of 69 (3 females, 5 males; mean age 68.25 years) did not have any radiologically detectable lung parenchymal lesion. The TNM staging (7th edition) of these eight patients without lung parenchymal lesion was T0N0M1a, T0N2M1a, T0N2M1a, T0N3M1a, T0N3M1a, T0N0M1b, T0N1M1b, T0N2M1b. Overall prognosis of MPE with lung parenchymal lesion was 331.7+/-63.14 days and without lung parenchymal lesion was 143.5 +/– 32.8 days, p=0.31 (figure 1)

Conclusion We have demonstrated no significant difference in the prognosis of patients with MPE secondary to pulmonary adenocarcinoma in the absence or presence of a radiologically determined primary lung parenchymal lesion. Although not statistically different (p=0.31) those patients without a primary lung parenchymal lesion may have a worse prognosis and this requires further investigation in larger cohorts as this may prove to be an important prognostic factor for MPE.

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P243 SURVIVAL PREDICTION IN MALIGNANT PLEURAL MESOTHELIOMA: FUNDAMENTAL LIMITATIONS OF ROUTINELY AVAILABLE CLINIC PREDICTORS

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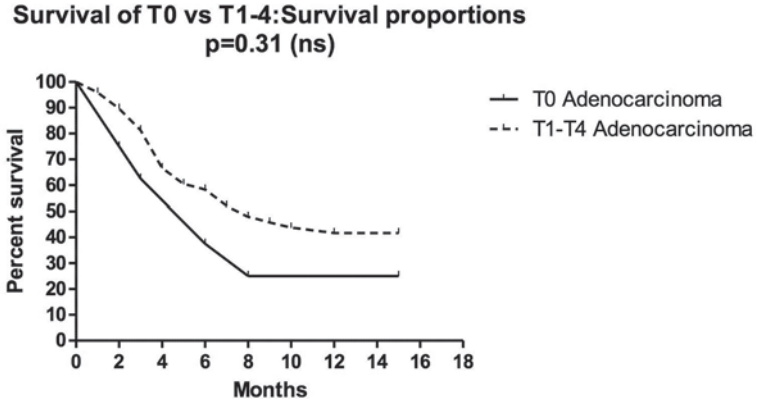
Introduction Accurate prognostication is difficult in Malignant Pleural Mesothelioma (MPM). Published prognostic models antedate optimal staging, a range of emerging predictors and use methods that cannot be up-scaled to incorporate these. Most existing models allocate patients to risk groups rather than precisely predicting survival. We developed robust computational models that can be up-scaled and provide quantitative statistics regarding the predictions offered. Here we report their performance using routinely available clinical data, on which previous models are based.

Materials and Methods Baseline information regarding 20 candidate predictors was collected for 269 MPM patients diagnosed in the West of Scotland (January 2008 – April 2014). Patients were allocated to balanced training (n=169) and validation sets (n=100). Prognostic signatures (minimal length best-performing multivariate trained models) were generated

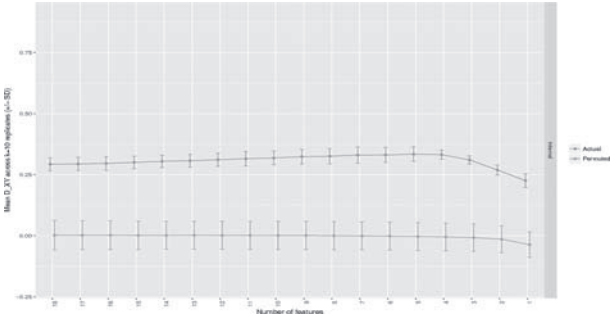


Abstract P242 Figure 1

by Least Absolute Shrinkage and Selection Operator (Lasso) regression for Overall Survival (OS), OS<6 months and OS<12 months. OS prediction was quantified using Somers D_{XY} statistic, which varies from 0 to 1, with increasing concordance between observed and predicted outcomes. 6- and 12 month survival were described by area under the curve (AUC) scores.

Results Median OS was 270 (IQR 140–450) days. The primary OS model assigned high weights to 4 predictors: age, performance status, white cell count and serum albumin, and after cross-validation performed significantly better than would be expected by chance (mean D_{XY} 0.332 (+/-0.019) figure 1). However, validation set D_{XY} was only 0.221 (0.0935–0.346), equating to a 22% improvement in survival prediction than would be expected by chance. 6- and 12 month OS signatures included the same 4 predictors, in addition to epithelioid histology plus platelets and epithelioid histology plus C-reactive protein (mean AUC 0.758 (+/-0.022) and 0.737 (+/-0.012), respectively). The <6 month OS model demonstrated 74% sensitivity and 68% specificity. The <12 month OS model demonstrated 63% sensitivity and 79% specificity. Model content and performance were generally comparable with previous studies.

Discussion The prognostic value of the basic clinical information contained in these, and previously published models, is fundamentally of limited value in accurately predicting MPM prognosis. The methods described are suitable for expansion using emerging predictors, including tumour genomics and volumetric staging.



Abstract P243 Figure 1 Model performance as a function of the number of features for actual (red line) and permuted (blue line) data. Performance for Overall Survival (OS) is measured by D_{XY} for OS. Standard deviations from replicates of cross-validation are shown for each point as bars.

Clinical implications of cystic fibrosis

P244 EXTENDED CFTR SCREENING FOR PATIENTS WITH A CLINICAL DIAGNOSIS OF CF BUT ONLY ONE GENE ON INITIAL SCREENING

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Introduction Standard CF genotyping only identifies 94% of CF genes, resulting in the emergence of a "cystic fibrosis screen positive, inconclusive diagnosis" (CFSPID) designation. This, and the advent of genotype-specific CFTR directed therapies has highlighted the need for more comprehensive genotyping, particularly to identify rarer genes when only a single gene is found on initial screening. However, extended CFTR screening is an expensive investigation and we wished to assess its use and yield.

Methods Between 2014 and 2016 we identified 40 people with CF attending our large regional adult unit without two known pathogenic CFTR genes and offered them extended CFTR screening. We looked at the yield in terms of additional genes identified and their clinical significance in 37 of these (3 refused/did not attend).

Results A new molecular diagnosis (i.e., two pathogenic genes) was made in 18 (48.5%) people with CF. Genes associated with CFTR related disorders were found in a further 2 (5.5%), genes of uncertain pathogenicity were found in 3 (8.1%), and one or no genes were found in 14 (37.8%). Of the 18 people with CF with additional identified genes, 8 had those associated with responsiveness to the CFTR potentiator ivacaftor (4 × 3272–26 A≥G, 1 × 711+3 A≥G, 1 × R347H, 1 × 2789+5G≥A, 1 × S945L) and 2 of these (R347H and S945L) have recently been approved for ivacaftor use by the U.S. Food and Drug Administration.

Conclusions In people with a clinical diagnosis of CF but only one pathogenic gene on initial screen, extended CFTR screening identified a second gene in nearly half of cases. Furthermore, a significant proportion of the identified genes have been reported to respond to ivacaftor. It is therefore important that all people with CF without two known mutations undergo extended mutation screening in order to establish who may benefit should the current license for ivacaftor be expanded in the UK.

P245 INFLUENZA B OUTBREAK AT A LARGE ADULT CF CENTRE: CLINICAL CONSEQUENCES AND POTENTIAL CONTRIBUTING FACTORS

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Introduction In May 2016 an outbreak of influenza was declared on the adult cystic fibrosis (CF) ward at the Manchester Adult Cystic Fibrosis Centre (MACFC), where all patients are treated in individual rooms and strongly encouraged not to mix. The aim of this study was to investigate the outbreak, its clinical consequences, and potential contributing factors.

Method Notes of all patients admitted to the CF ward in May 2016 were retrospectively reviewed and data recorded included: patient location, respiratory viral PCR results, spirometry data (baseline measurements in the 6 month prior to onset, at onset of influenza, and at 3 months post infection), influenza vaccination status (all staff and patients), and measurements of ventilation in patient rooms.

Results Ten patients tested positive for influenza B; all were shown to have been infected with the same strain of the virus: B/Brisbane/60/2008. An outbreak timeline identified the likely index case (only patient admitted within the incubation period of influenza, first to develop symptoms and test positive for influenza B). Subsequently, 8 patients whose rooms