investigation. The remaining 64 (75%) were subsequently admitted to 29 different locations (8 medical specialities, surgical, vascular and orthopaedic wards). Of these, 55 (86%) were seen by the lung CNS within one working day and an appropriate management plan initiated. This was aided by our live CT database, where 50 suspicious scans were coded the same day enabling early review by the lung clinician and CNS, often before formal referral from the responsible clinical team was made.

Overall 70 patients (82%) who presented as emergencies subsequently were diagnosed with a malignancy, and of these 34 (49%) had histological confirmation.

Conclusions Our Results show that, by coordinating care between the emergency and radiology departments and the lung cancer team, patients presenting unwell can be managed rapidly even if they remain in hospital. In addition, by actively seeking them out we can not only provide them with timely and appropriate investigations but also early CNS intervention, facilitating symptom management and psychological support.

P267 OPTIMISING TISSUE SAMPLING FOR THE MOLECULAR DIAGNOSIS OF LUNG ADENOCARCINOMA

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Background The development of drugs that target lung adenocarcinoma caused by epidermal growth factor tyrosine kinase (EGFR-TK) and anaplastic lymphoma kinase (ALK) mutations has focused the need to obtain sufficient tissue at biopsy to allow the detection of such molecular markers and so improve treatment options for selected patients. To investigate this further, we looked at the diagnostic yield from various biopsy techniques in our large lung cancer unit (400 cases per year, overall histological yield 77.5%).

Methods We collected data from all patients with an ultimate histological diagnosis of adenocarcinoma for the years 2014 to 2016, looking at the diagnostic method, whether tissue was analysed for molecular mutations, and whether repeat procedures were necessary for EGFR-TK and ALK testing.

Results 224 patients were identified: 42 by EBUS-TBNA, 66 by CT-guided biopsy, 44 at bronchoscopy, 66 at surgical resection, 6 from pleural fluid, and 1 by lymph node FNA. For molecular testing see Table. In addition to those patients where sampling was insufficient to make the diagnosis, a further 30 had inadequate cell numbers for mutation analysis and the reporting pathologist recommended repeat procedures. Of the 10 patients who underwent this, only 2 were retested for molecular markers, and the Results were unchanged.

Conclusion This study shows that all our pre-resection positive diagnostic samples for lung cancer do not always provide sufficient tissue for molecular analysis. Although insufficiency rates were similar between CT, EBUS and bronchoscopy, one third of CT-guided specimens had few cells for the definite exclusion of mutations. With the advent of new therapies for lung cancer, we need to optimise our diagnostic sampling techniques when testing for molecular mutations.

Abstract P267 Table 1 Rate of insufficient tissue or inadequate cell number for molecular mutation detection per sampling technique

Biopsy Technique	Insufficient tissue for EGFR testing (% of biopsy samples per sampling technique)	Insufficient tissue for ALK testing (% of biopsy samples per sampling technique)	Inadequate cell number for definitive molecular mutation detection (% of samples per sampling technique)
EBUS-TBNA	7.70%	12.80%	20%
CT-Guided Biopsy	9.50%	20%	35%
Bronchoscopy and biopsy	11%	11%	6.25%
Surgical resection	0%	0%	0%
Pleural fluid analysis	20%	20%	20%
Lymph node FNA	0%	0%	0%

Pharmacotherapies for COPD

P268 RELATIONSHIP OF INHALER ADHERENCE BEHAVIOUR TO CLINICAL OUTCOMES IN COPD: AN OBSERVATIONAL STUDY

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COPD remains a leading cause of healthcare use despite the availability of effective inhaled therapies. We examined adherence to maintenance therapy by assessing the key components of good inhaler use: habit of use and inhaler technique. The relationship between adherence patterns, specific patient characteristics and clinical outcomes at one year was examined.

We recruited 226 hospitalised patients with a diagnosis of COPD to this prospective observational study. Inhaler adherence was remotely monitored for 90 days after hospital discharge using an INCA™ audio recording device. Cluster analysis grouped patients by their adherence behaviour based on the mean rate of attempted use and critical technique errors. The clinical and psychosocial characteristics of each cluster were examined. The rate of all-cause mortality and healthcare use at 12 months was recorded. Survival analysis was used to evaluate the time to first event across adherence groups. Adherence data was available for 195 patients. We identified four patterns of Adherence behaviour: (1) Regular habit of use and good technique (28%); (2) Regular habit of use and poor technique (21%); (3) Poor habit of use and good technique (33%); (4) Poor habit of use and poor technique (19%). The overall event rate was lowest in Cluster 1, 5.46/person/year. Cluster 2 had the lowest annual rate of hospital presentation, but accounted for the majority of community prescriptions for antibiotics and steroids, mean 4.6/person/

year. In an adjusted Cox regression model, Cluster 3 had an increased risk of any adverse outcome compared to Cluster 1, Hazard Ratio 1.8 (1.1–2.9), $p=0.02$. This group were notable for high anxiety scores and mild cognitive impairment. There was a stepwise increase in mortality across groups, from 11% in Cluster 1 to 33% in Cluster 4, $p<0.001$. Cluster 4 was older, female, with higher co-morbidity and cognitive impairment. We have identified four clusters of adherence behaviour. There is an association between adherence patterns and clinical outcomes. Each cluster also exhibits distinct clinical and psychosocial traits which may act as drivers of their behaviour. Personalised interventions targeting these specific adherence behaviour patterns may prove a cost-effective strategy to curtail COPD-related healthcare costs.

P269 DESCRIBING ADHERENCE DATA IN A CLINICAL EFFECTIVENESS TRIAL: THE SALFORD LUNG STUDY IN COPD (SLS COPD)

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Background Adherence to inhaled therapy is key to effective COPD management; poor adherence is associated with suboptimal outcomes. While adherence may be more accurately measured in traditional double-blind randomised controlled trials (RCTs) than in effectiveness trials conducted in everyday clinical practice, the latter may more closely reflect “typical” patient adherence.

Aim To describe adherence in SLS COPD, a 12 month open-label effectiveness RCT that evaluated initiating fluticasone furoate/vilanterol (FF/VI) vs continuing usual care (UC) in COPD patients in UK primary care.

Methods Adherence was estimated by proportion of days covered (PDC; based on study medication prescribing data from patients’ electronic case report forms [eCRFs]/electronic health records [EHRs] during the study) and the MARS-A 9 questionnaire. Selected outcomes were descriptively analysed by PDC <80% or ≥80%.

Results Mean PDC during the study was similar for FF/VI and UC; the proportion of patients with PDC ≥80% was high in both groups (Table). Summary statistics showed little change in MARS-A 9 during the study and similar responses in both treatment arms. Clear relationships between PDC category and exacerbations, healthcare resource utilisation, and COPD Assessment Test were not observed.

Conclusions In SLS COPD, adherence estimated by PDC was high and there was no clear association between PDC and outcomes. Limitations include adherence based on patients’ self-reporting and eCRF/EHR prescribing data.

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Please refer to page A260 for declarations of interest in relation to abstract P269.

Abstract P269 Table 1 Adherence by treatment group (ITT analysis)[†]

	FF/VI (n=1396)	UC (n=1403)
PDC [†] n	1378	1361
Mean (SD), % <80%, n (%)	84.99 (22.29) 346	82.40 (23.11) 447
≥80%, n (%)	(25)	(33)
	1032 (75)	914 (67)
MARS-A 9 score	1393	1402
Baseline (randomisation) n	4.42 (0.677) 1318	4.44 (0.666) 1324
Mean (SD)	4.61 (0.563) 1315	4.44 (0.664) 1324
Study end [‡] n	0.20 (0.730)	0.00 (0.750)
Mean (SD)		
Difference (baseline → study end [‡]) n		
Mean (SD)		
	PDC<80% (n=793)	PDC≥80% (n=1946)
Outcomes in patients with		
PDC<80% and≥80% by treatment arm		
(ITT analysis)^{*†}	FF/VI (n=346)	UC (n=447)
	FF/VI (n=1032)	UC (n=914)
LS mean annual rate of moderate/severe exacerbations, n (PEA population) [‡]	n=282	n=364
	n=839	n=738
Rate ratio (95% CI)	1.68	1.87
	0.90 (0.78–1.04)	0.92 (0.84–1.01)
HRU		
LS mean COPD-related primary care contacts, n	2.13	2.46
	2.51	2.45
Rate ratio (95% CI)	0.87 (0.76–0.98)	1.03 (0.95–1.11)
LS mean COPD-related secondary care contacts, n	1.31	1.24
	1.64	1.53
Rate ratio (95% CI)	1.06 (0.75–1.49)	1.07 (0.87–1.33)
CAT score [§] n	345	447
	1031	913
Baseline score, mean (SD) n at endpoint	22.9	22.4
	21.1	21.6
Responder, n (%)	(9.10)	(9.02)
	(8.78) 990	(8.57)
Non-responder, n (%)	312	415
	456 (46)	876
	133 (43)	165 (40)
	534 (54)	304 (35)
	179 (57)	250 (60)
		572 (65)
Adjusted odds ratio (95% CI)	1.07 (0.78–1.46)	1.71 (1.40–2.07)

CAT, COPD Assessment Test; CI, confidence interval; HRU, healthcare resource utilisation; ITT, intent-to-treat; LS, least squares; MARS-A 9; Medication Adherence Report Scale for Asthma (modified for use in COPD, 9-item questionnaire used [omitting score for Question ‘Before doing something’]; higher MARS-A 9 scores indicate better adherence); PEA, primary effectiveness analysis; SD, standard deviation. [†]Based on a total of 2739 patients in the ITT population with available treatment adherence data, unless otherwise stated. ^{††}PDC determined at study end, using all prescriptions during the study treatment period. [‡]Based on last available MARS-A 9 measurement post-randomisation. ^{‡‡}Based on a total of 2223 patients in the PEA population with available treatment adherence data. [§]Post-hoc summary/analysis of CAT data by PDC category; all other summaries/analyses were pre-specified. CAT responder is defined as a change from baseline of ≤ −2 at endpoint (last available on-treatment measurement).