10.1148/radiol.2021203531

Appendix E1

Supplemental Methods

Cohort Description

Both the COPDGene and ECLIPSE study cohorts have been described in detail previously and details regarding the cohorts as well as lists of investigators and prior publications describing other analyses of these cohorts are available in Appendices 3–6 (11,12). Briefly, the COPDGene Study is a multicenter longitudinal observational investigation of smokers focused on the epidemiologic and genetic factors associated with COPD (12). The majority of the participants are current and former smokers with and without COPD, and the study includes baseline 5 year and 10 year follow-up visits. The baseline and 5-year follow-up visits are complete. The 10-year follow-up visits are still ongoing, and thus for this current work those participants who completed both the baseline and 5-year follow-up visits were included. Mortality was assessed through the COPDGene long-term follow-up program and from the social security death index (13). Cause specific mortality information was obtained and adjudicated by the COPDGene death adjudication committee (41). Imaging was performed between November 2007 and July 2017 for COPDGene and the COPDGene specific datasets used for this study include the phase 1 and phase 2 dataset from March 2019 and the mortality dataset from October 2018.

The ECLIPSE study was a 3-year multicenter longitudinal study comprised of participants with COPD and a smaller number of smoking and never-smoking controls, with aims that included the identification of biomarkers that correlated with COPD subtypes and predicted disease progression. Assessments of disease severity, including pulmonary function and CT imaging were obtained at multiple time points, the latter specifically at baseline, one year and three years of follow-up. Longitudinal follow-up included assessment of all-cause mortality for a total of 8 years (5 years after the 3-year follow-up visit) (11). Imaging and clinical assessments were performed between January 2006 and March 2010 and the specific dataset used for this study was generated April 2015.

All participants from both cohorts who had complete longitudinal and mortality follow-up data as described above as well as complete baseline data with regard to the covariates for the multivariable analyses below were included in this current study with the exception of never-smokers, who were excluded other than for establishing the progression thresholds described below.

Image Acquisition

For COPDGene participants, volumetric CT scans of the chest were performed at both maximal inflation and relaxed exhalation without the administration of intravenous contrast. Images were acquired with the following CT protocol: for General Electric (GE) LightSpeed-16, GE VCT-64, Siemens Sensation-16 and-64, and Philips 40-and 60-slice scanners with 120kVp, 200mAs, and 0.5s rotation time. Images were reconstructed in an axial plane using a standard algorithm at 0.625 mm slice thickness and 0.625 mm intervals for GE scanners; using a B31f algorithm at 0.625 (Sensation-16) or 0.75 mm slice thickness and 0.5 mm intervals for Siemens scanners; and

using a B algorithm at 0.9 mm slice thickness and 0.45 mm intervals for Philips scanners. For ECLIPSE participants, volumetric CT scans of the chest were performed at full inspiration without the administration of intravenous contrast (120 kV peak, 40mA) and reconstructed in an axial plan with 1.00 (Siemens) or 1.25 mm (GE) contiguous slices and a low spatial frequency reconstruction algorithm (GE: Standard, Siemens b35f).

Emphysema Quantification and Definition

Multiple approaches have been taken to quantifying and defining the presence of emphysema on CT (7,9,16,26). For this study, we defined a participant as having emphysema at baseline if they had more than the "normal" amount of low attenuation area (LAA) on Chest CT (3). More specifically, we classified participants as having emphysema at baseline if they had greater than the upper limit of normal amount of tissue with a density of less than-950 Hounsfield units (HU) as defined by work from the Multi-Ethnic Study of Atherosclerosis (MESA) cohort (16).

Similarly, multiple approaches have been used to measure and define emphysema progression using CT (7,9,42). Recently, extensive work by the Quantitative Imaging Biomarkers Alliance (QIBA), a group organized by the Radiological Society of North America (RSNA) (qibawiki.rsna.org), has led to a clear approach that enables reliable, repeatable of quantitative measures of lung density and emphysema outside of the research setting (17). Based on this work, we quantified emphysema for the purposes of measuring progression using a volume adjusted percentile density method: the volume adjusted lung density measured at the 15th percentile of the CT lung density histogram (Lung Density Perc15) (Fig 1 in the primary manuscript) (17).

To calculate Lung Density Perc15, the lung attenuation for each CT voxel measured in Hounsfield units, is first translated into a more general measure of density, grams per liter (g/L) using a simple formula:

$$Density\left(grams\ per\ liter\right) = Attenuation\left(Hounsfield\ Units\right) + 1000\,.$$

The frequency of lung density measures for the entire lung, ie, the lung density histogram, is then used to identify the 15th percentile of lung density (Fig 1 in the primary manuscript). Because lung volume is a critical determinant of lung density this number is then adjusted for lung volume using the sponge model (17,18):

$$Volume\ adjusted\ lung\ denisty\ Perc15 = Measured\ lung\ density\ Perc15 \cdot \left(\frac{Measured\ CT\ Lung\ Volume}{Predicted\ CT\ Lung\ Volume}\right),$$

where "measured lung density Perc15" is the raw value measured from the histogram, "measured CT lung volume" is the total volume of the voxels in the lungs after segmentation from the surrounding structures, and "predicted CT lung volume" is that predicted from equations generated from normal individuals with no smoking history (16–18). Using this approach, increases in emphysema are expressed as decreases in density (ie, a lower Lung Density Perc15 indicates more emphysema) (Fig 1 in the primary manuscript).

Emphysema progression was analyzed using two general approaches: For the primary analyses, the annualized rate of change in Lung Density Perc15 was analyzed as a continuous predictor. Thus, the effects shown are for the association between a 1 g per liter per year faster rate of decline in density (increase in emphysema). Secondary, dichotomized analyses were then

performed comparing “progressors” with “non-progressors.” For these analyses, progression was defined in one of two ways. The first was based on having a decrease in density more than the test-to-test variability, ie, the repeatability coefficient of Lung Density Perc15. The aforementioned work by QIBA on emphysema quantification found that a decrease of at least 11 g/L in lung density was required for the detection of an increase in the extent of emphysema with 95% probability (17). Thus, those individuals referred to as “progressors” for the first part of the dichotomized analyses are those who had a decrease in the Lung Density Perc15 of at least 11 g/L (the repeatability coefficient), and those who are “non-progressors” are the remainder of the individuals in the cohort. The second definition of progression for the dichotomized analyses was based on the minimum clinically important difference (MCID) derived from individuals who have never smoked and who have no lung disease, termed “never-smoking normals.” For this definition, we pooled never-smoking normal participants who had longitudinal imaging data from the COPDGene and ECLIPSE cohorts and calculated a distribution based MCID, ie, the rate of decline in Lung Density Perc15 that is 1/2 standard deviation faster than the mean rate of decline for normal individuals (-1.22 g per liter per year) (43). Thus, those individuals referred to as “progressors” for the second portion of the dichotomized analyses are those who had a rate of decline in their Lung Density Perc15 of at least-1.22 g per liter per year, and those who are “non-progressors” are the remainder of the individuals in the cohort.

Statistical Analysis

Differences in the annualized rate of decline in Lung Density Perc15 were analyzed using *t* tests. The association between emphysema progression and mortality, was analyzed using multivariable Cox regression with adjustments made for both baseline and longitudinal variables including race, gender, baseline age, baseline smoking status, baseline pack years, baseline forced expiratory volume in one second (FEV1), baseline six minute walk distance, baseline Lung Density Perc15, change in smoking status, annualized rate of change in FEV1 and annualized rate of change in six minute walk distance (16,17,19–22). All of the covariates were assessed using the Schoenfeld residuals method and none were found to violate the proportional hazards assumption (44). To visualize the survival of progressors compared with nonprogressors adjusted survival curves were created using the Cox models and the corrected group prognosis method (45–47). To determine the relationship between emphysema progression and respiratory mortality, cause specific mortality analyses were performed in the COPDGene cohort, using the Fine and Gray method of accounting for competing risk (48). Due to a smaller number of events and to avoid overfitting, respiratory mortality was analyzed using multivariable Cox regression adjusted for a limited number of covariates (age, gender and race).

Finally, to determine whether the addition of longitudinal imaging data improved the prediction of subsequent mortality, we compared the model fit of multivariable Cox models containing all of the demographic and exposure covariates from the primary analyses and combinations of: baseline Lung Density Perc15 and/or FEV1, and longitudinal Lung Density Perc15 and/or FEV1. Model fit and parsimony were quantified using Akaike Information Criteria (AIC) while differences in model fit for the nonnested models, such as those comparing the relative performance of baseline imaging to baseline spirometry were assessed using partial likelihood ratio tests, and differences in model fit for nested models, such as those comparing the relative performance of baseline imaging to longitudinal imaging (the latter of which includes baseline measures) were assessed using likelihood ratio tests (23–25).

All statistical tests were two sided and *P* values of less than 0.05 were taken to indicate statistical significance. All analyses were performed in R (version 4.0.3, R Foundation for Statistical Computing, Vienna, Austria), implemented using RStudio (Version 1.3.1093, Boston, MA) and using the tidyverse, survival, survminer, cmprsk, and nonnestcox packages.

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Table E1A: Characteristics of those with Emphysema in the COPDGene Cohort

	Subgroup	Visit 1	Visit 2
Demographic and other Characteristics			
n		2097	2097
Age (Years) (mean (SD))		60 (9)	66 (9)
Gender (%)	Male	985 (47.0)	985 (47.0)
	Female	1112 (53.0)	1112 (53.0)
Race (%)	White	1365 (65.1)	1365 (65.1)
	Black	732 (34.9)	732 (34.9)
Smoking Status (%)	Former Smoker	817 (39.0)	1138 (54.3)
	Current Smoker	1280 (61.0)	959 (45.7)
Pack Years (mean (SD))		46.4 (25.1)	48.2 (25.3)
Time between CT and PFTs (Days) (mean (SD))		1.2 (16.9)	0.9 (14.1)
Time between Visit 1 and Visit 2 CTs (Years) (mean (SD))			5.5 (0.8)
Pulmonary Function			
Post Bronchodilator FEV1 (L) (mean (SD))		2.0 (0.9)	1.8 (0.9)
Post Bronchodilator FVC (L) (mean (SD))		3.3 (1.0)	3.0 (1.0)
Gain or Loss of FEV1 (%)	Gained FEV1		351 (16.7)
	Lost FEV1		1746 (83.3)

Baseline Quantitative CT Measures			
Total Lung Capacity by CT (L) (mean (SD))		5.8 (1.4)	5.6 (1.5)
Total Lung Capacity by CT (% Predicted) (mean (SD))		112.4 (15.9)	112.4 (17.4)
Volume Adjusted Lung Density measured at the 15th percentile of the lung density histogram (Lung Density Perc15) (grams per liter) (mean (SD))		73.2 (25.1)	74.6 (28.2)
Longitudinal Change in Quantitative CT Measures			
Gain or Loss of Emphysema based on the Change in Lung Density Perc15 Relative to Repeatability Coefficient (RC) (%)	Gained Emphysema by Lung Density Perc15 (absolute change > RC)		387 (18.5)
	No Change or Loss in Emphysema by Lung Density Perc15 (absolute change < RC)		1710 (81.5)
Rate of Emphysema Progression Relative to Distribution Based Minimum Clinically Important Difference (%)	Rate of progression faster than never-smoking normals		617 (29.4)
	Rate of progression not faster than never-smoking normals		1480 (70.6)
Mortality			
Mortality Rate (%)	Alive	1921 (91.6)	
	Dead	176 (8.4)	
Survival Duration from Enrollment (Years) (mean (SD))		8.2 (1.2)	

Table E1B: Characteristics of those with Emphysema in the ECLIPSE Cohort

	Subgroup	Screening	1 Year	3 Years
Demographic and other Characteristics				
n		1179	1124	1179
Age (Years) (mean (SD))		63 (7)	63 (7.1)	65 (7)
Gender (%)	Female	453 (38.4)	430 (38.3)	453 (38.4)
	Male	726 (61.6)	694 (61.7)	726 (61.6)
Race (%)	African American/African Heritage	25 (2.1)	24 (2.1)	25 (2.1)
	American Indian or Alaskan Native	1 (0.1)	1 (0.1)	1 (0.1)
	Asian-Central/South Asian Heritage	1 (0.1)	1 (0.1)	1 (0.1)
	Asian-Japanese Heritage	1 (0.1)	1 (0.1)	1 (0.1)
	Mixed race	2 (0.2)	2 (0.2)	2 (0.2)
	White-Arabic/North African Heritage	2 (0.2)	2 (0.2)	2 (0.2)
	White-White/Caucasian/European Heritage	1147 (97.3)	1093 (97.2)	1147 (97.3)
Clinical Group (%)	COPD Subjects	1101 (93.4)	1050 (93.4)	1101 (93.4)
	Smoker Controls	78 (6.6)	74 (6.6)	78 (6.6)
Pack Years (mean (SD))		46.8 (25.7)	46.7 (25.3)	46.8 (25.7)
Time between CT and PFTs (Days) (mean (SD))		9.6 (39.1)	6.4 (22.0)	3.4 (35.0)
Time from Baseline CT (Years) (mean (SD))		0.0 (0.0)	1.0 (0.1)	3.0 (0.2)
Pulmonary Function				
Post Bronchodilator FEV1 (L) (mean (SD))		1.5 (0.7)	1.4 (0.7)	1.4 (0.7)
Post Bronchodilator FVC (L) (mean (SD))		3.2 (0.9)	3.2 (0.9)	3.0 (0.9)
Gain or Loss of FEV1 (%)	Gained		510 (45.4)	362 (30.8)
	Lost		613 (54.6)	815 (69.2)
Baseline Quantitative CT Measures				

Total Lung Capacity by CT (L) (mean (SD))		6.2 (1.4)	6.2 (1.4)	6.1 (1.4)
Total Lung Capacity by CT (% Predicted) (mean (SD))		113.0 (17.4)	112.2 (18.2)	111.6 (19.1)
Volume Adjusted Lung Density measured at the 15th percentile of the lung density histogram (Lung Density Perc15) (grams per liter) (mean (SD))		53.9 (24.0)	53.1 (23.4)	50.7 (23.8)
Longitudinal Change in Quantitative CT Measures				
Gain or Loss of Emphysema based on the Change in Lung Density Perc15 Relative to Repeatability Coefficient (RC) (%)	Gained Emphysema by Lung Density Perc15 (absolute change > RC)			173 (14.7)
	No Change or Loss of Emphysema by Lung Density Perc15 (absolute change < RC)			1006 (85.3)
Rate of Emphysema Progression Relative to Distribution Based Minimum Clinically Important Difference (%)	Rate of progression faster than never-smoking normals			606 (51.4)
	Rate of progression not faster than never-smoking normals			573 (48.6)
Mortality				
Mortality Rate (%)	Alive	915 (77.6)		
	Dead	264 (22.4)		
Survival Duration from Enrollment (Years) (mean (SD))		6.8 (1.9)		

Table E2: Association between Rate of Change in Emphysema Progression and Mortality

		Confidence Interval		
Cohort	**Hazard Ratio**	**Lower**	**Upper**	***P***
COPDGene	1.06	1.00	1.12	0.0499
ECLIPSE	1.07	1.02	1.12	0.01

[1] Effects expressed as change in the risk of all-cause mortality per 1 g/L/year faster rate of change in density.

[2] Mortality assessed as time since follow-up visit.

[3] All models adjusted for: a) Race and gender; b) Baseline age, smoking status, pack years, forced expiratory volume in one second, six minute walk distance and volume adjusted lung density measured at the 15th percentile of the CT lung density histogram (Lung Density Perc15); c) Change in smoking status, rate of change in forced expiratory volume in one second and rate of change in six minute walk distance.

[4] Results shown are in those with and without emphysema at baseline (all ever smokers).

Table E3: Association between Emphysema Progression and Mortality (Responder Analysis)

		Confidence Interval		
Cohort	**Hazard Ratio**	**Lower**	**Upper**	***P***
Loss of Density more than the Repeatability Coefficient				
COPDGene	1.47	1.05	2.07	0.03
ECLIPSE	1.32	0.91	1.93	0.15
Rate of Loss of Density Faster than the Minimum Clinically Important Difference' Individuals				
COPDGene	1.52	1.14	2.02	0.004
ECLIPSE	1.61	1.19	2.18	0.002

[1] For 'Loss of Density more than the Repeatability Coefficient' the effects are expressed as those with decrease in density (Lung Density Perc15) more than the repeatability coefficient compared with those

who had a decrease in density less than or equal to the repeatability coefficient (Lung Density Perc15), ie, progressors versus nonprogressors.

[2] For 'Rate of Loss of Density Faster than that in 'Never-smoking Normal' Individuals' the effects are expressed as those with a rate of decrease in density (Lung Density Perc15) faster than the minimum clinically important difference as defined by the rate in 'never smoking normals.'

[3] Mortality assessed as time since follow-up visit.

[4] All models adjusted for: a) Race and gender; b) Baseline age, smoking status, pack years, forced expiratory volume in one second, six minute walk distance and volume adjusted lung density measured at the 15th percentile of the CT lung density histogram (Lung Density Perc15); c) Change in smoking status, rate of change in forced expiratory volume in one second and rate of change in six minute walk distance.

[5] Results shown are in those with and without emphysema at baseline (all ever smokers).

Table 4A: Comparison of Model Performance in the COPDGene Cohort

	Akaike Information Criterion		p	
Comparison	**AIC of First Model**	**AIC of Second Model**	**First Model vs. Second Model (non-nested only)**	**Second Model vs. First Model**
Baseline vs. Baseline				
Baseline Spirometry vs. Baseline Imaging	3318.28	3349.52	0.04	0.96
Baseline Spirometry vs. Baseline Spirometry + Baseline Imaging	3318.28	3308.96	N/A	< 0.001
Baseline Imaging vs. Baseline Spirometry + Baseline Imaging	3349.52	3308.96	N/A	< 0.001
Longitudinal vs. Longitudinal				
Longitudinal Spirometry vs. Longitudinal Imaging	3294.12	3335.99	0.02	0.98
Longitudinal Spirometry vs. Longitudinal Spirometry + Longitudinal Imaging	3294.12	3287.60	N/A	0.01
Longitudinal Imaging vs. Longitudinal Spirometry + Longitudinal Imaging	3335.99	3287.60	N/A	< 0.001
Baseline vs. Longitudinal				
Baseline Spirometry vs. Longitudinal Spirometry	3318.28	3294.12	N/A	< 0.001

	Akaike Information Criterion		p	
Comparison	**AIC of First Model**	**AIC of Second Model**	**First Model vs. Second Model (non-nested only)**	**Second Model vs. First Model**
Baseline Imaging vs. Longitudinal Imaging	3349.52	3335.99	N/A	< 0.001
Baseline Spirometry + Baseline Imaging vs. Longitudinal Spirometry + Longitudinal Imaging	3308.96	3287.60	N/A	< 0.001

[1] All models adjusted for: a) Race and gender; b) Baseline age, smoking status and pack years. All models with longitudinal data also adjusted for change in smoking status, rate of change in forced expiratory volume in one second and rate of change in six minute walk distance.
[2] Baseline models include baseline forced expiratory volume in one second and/or baseline volume adjusted lung density
[3] Longitudinal models include baseline and rate of change in forced expiratory volume in one second and/or baseline volume adjusted lung density
[4] For non-nested models, the p value given for each comparison is the p value for partial likelihood ratio test with the null hypothesis that the model fits are equally close to the true model. For example, for baseline spirometry vs. baseline imaging comparison, the first p value given is for the alternative hypothesis that the baseline spirometry model fits better than the baseline imaging model, and the second p value given is for the alternative hypothesis that the baseline imaging model fits better than the baseline spirometry model. (24, 25)
[5] For the nested models, the p value given is for the likelihood ratio test with the null hypothesis that both models fit equally well and the alternative hypothesis that the second (larger) model fits better than the first (smaller) model. (24, 25)
[6] Abbreviations: Akaike information criterion (AIC). (26)
[7] Results shown are in those with and without emphysema at baseline (all ever smokers)

Table 4B: Comparison of Model Performance in the ECLIPSE Cohort

	Akaike Information Criterion		p	
Comparison	**AIC of First Model**	**AIC of Second Model**	**First Model vs. Second Model (non-nested only)**	**Second Model vs. First Model**
Baseline vs. Baseline				
Baseline Spirometry vs. Baseline Imaging	3002.63	3023.15	0.11	0.89
Baseline Spirometry vs. Baseline Spirometry + Baseline Imaging	3002.63	2986.94	N/A	< 0.001
Baseline Imaging vs. Baseline Spirometry + Baseline Imaging	3023.15	2986.94	N/A	< 0.001

	Akaike Information Criterion		p	
Comparison	**AIC of First Model**	**AIC of Second Model**	**First Model vs. Second Model (non-nested only)**	**Second Model vs. First Model**
Longitudinal vs. Longitudinal				
Longitudinal Spirometry vs. Longitudinal Imaging	2985.00	3005.98	0.13	0.87
Longitudinal Spirometry vs. Longitudinal Spirometry + Longitudinal Imaging	2985.00	2969.20	N/A	< 0.001
Longitudinal Imaging vs. Longitudinal Spirometry + Longitudinal Imaging	3005.98	2969.20	N/A	< 0.001
Baseline vs. Longitudinal				
Baseline Spirometry vs. Longitudinal Spirometry	3002.63	2985.00	N/A	< 0.001
Baseline Imaging vs. Longitudinal Imaging	3023.15	3005.98	N/A	< 0.001
Baseline Spirometry + Baseline Imaging vs. Longitudinal Spirometry + Longitudinal Imaging	2986.94	2969.20	N/A	< 0.001

[1] All models adjusted for: a) Race and gender; b) Baseline age, smoking status and pack years. All models with longitudinal data also adjusted for change in smoking status, rate of change in forced expiratory volume in one second and rate of change in six minute walk distance.
[2] Baseline models include baseline forced expiratory volume in one second and/or baseline volume adjusted lung density
[3] Longitudinal models include baseline and rate of change in forced expiratory volume in one second and/or baseline volume adjusted lung density
[4] For non-nested models, the p value given for each comparison is the p value for partial likelihood ratio test with the null hypothesis that the model fits are equally close to the true model. For example, for baseline spirometry vs. baseline imaging comparison, the first p value given is for the alternative hypothesis that the baseline spirometry model fits better than the baseline imaging model, and the second p value given is for the alternative hypothesis that the baseline imaging model fits better than the baseline spirometry model. (24, 25)
[5] For the nested models, the p value given is for the likelihood ratio test with the null hypothesis that both models fit equally well and the alternative hypothesis that the second (larger) model fits better than the first (smaller) model. (24, 25)
[6] Abbreviations: Akaike information criterion (AIC). (26)
[7] Results shown are in those with and without emphysema at baseline (all ever smokers)

Appendix E2

COPGene Site Institutional Review Boards

Clinical Center	Institution Title	Protocol Number
National Jewish Health	National Jewish IRB	HS-1883a
Brigham and Women's Hospital	Partners Human Research Committee	2007-P-000554/2; BWH
Baylor College of Medicine	Institutional Review Board for Baylor	H-22209
	College of Medicine and Affiliated Hospitals	
Michael E. DeBakey VAMC	Institutional Review Board for Baylor College of Medicine and Affiliated Hospitals	H-22202
Columbia University Medical Center	Columbia University Medical Center IRB	IRB-AAAC9324
Duke University Medical Center	The Duke University Health System Institutional Review Board for Clinical Investigations (DUHS IRB)	Pro00004464
Johns Hopkins University	Johns Hopkins Medicine Institutional Review Boards (JHM IRB)	NA_00011524
Los Angeles Biomedical Research Institute	The John F. Wolf, MD Human Subjects Committee of Harbor-UCLA Medical Center	12756–01
Morehouse School of Medicine	Morehouse School of Medicine Institutional Review Board	07–1029
Temple University	Temple University Office for Human Subjects Protections Institutional Review Board	11369
University of Alabama at Birmingham	The University of Alabama at Birmingham Institutional Review Board for Human Use	FO70712014
University of California, San Diego	University of California, San Diego Human Research Protections Program	070876
University of Iowa	The University of Iowa Human Subjects Office	200710717
Ann Arbor VA	VA Ann Arbor Health care System IRB	PCC 2008–110732
University of Minnesota	University of Minnesota Research Subjects' Protection Programs (RSPP)	0801M24949
University of Pittsburgh	University of Pittsburgh Institutional Review Board	PRO07120059
University of Texas Health Sciences Center at San Antonio	UT Health Science Center San Antonio Institutional Review Board	HSC20070644H
Health Partners Research Foundation	Health Partners Research Foundation Institutional Review Board	07–127
University of Michigan	Medical School Institutional Review Board (IRBMED)	HUM00014973
Minneapolis VA Medical Center	Minneapolis VAMC IRB	4128-A
Fallon Clinic	Institutional Review Board/Research Review Committee Saint Vincent Hospital–Fallon Clinic–Fallon Community Health Plan	1143

Appendix E3

ECLIPSE Site Institutional Review Boards

Inv/Site No.	Institution & Address	IEC/IRB Committee
027904/023622	Asthma Centre Ivan Vazov Street Nr 31 PO Box 1018 Pleven, 5800 Bulgaria	Ethics Committee for Multicentre Trials 8, Damyan Gruev str., Sofia 1303 Bulgaria
076209/023623	Military Medical Academy Georgi Sofiiski 3 str Sofia 1606 Bulgaria	Ethics Committee for Multicentre Trials 8, Damyan Gruev str., Sofia 1303 Bulgaria
006269/023794	Montreal Chest Institute 3650 St-Urbain, Room K307 Montreal, QC H2× 2P4 Canada	McGill University Health Center Research Ethics Board 3650 St Urbain Montreal, QC H2× 2P4
006610/023668	The Lung Center 2775 Laurel St., seventh Floor Vancouver, BC V5Z 1M9 Canada	The University of British Columbia Office of Research Services Clinical Research Ethics Board Room210, 828 West 10th Ave Vancouver, BC V5Z 1 L8 Canada
004970/023483	Queen Elizabeth II Health Sciences Centre Halifax Infirmary 1796 Summer St., Room 5452 Halifax, NS B3H 3A7 Canada	Capital Health Research Ethics Board Centre for Clinical Research Building 118–5790 University Avenue Halifax, NS B3H 1 V7 Canada

029347/023796	McMaster University, Health Sciences Center 1200 Main St. West, Room 3U25 Hamilton, ON L8N 3Z5 Canada	Hamilton Health Sciences/Faculty of Health Sciences Research Ethics Board 293 Wellington St N, Suite 102 Hamilton, Ontario L8 L 8E7 Canada
035031/023795	Pacific Lung Health Center 1081 Burrard Street 8B Providence Wing Vancouver, BC V6Z 1Y6 Canada	Office of Research Services (ORS) Providence Health Care Research Institute Room 1125, 11th Floor 1190 Hornby Street c/o 1081 Burrard Street Vancouver, BC V6Z 1Y6
006193/023547	Hopital Laval, Recherche Clinic Centre de Pneumologie 2725 Chemin Sainte Foy Pavillion U, Locale U 1751 Sainte Foy, QC G1 V 4G5 Canada	Comité d'éthique de la recherche Institut Universitaire de Cardiologie et de Pneumologie de Québec (IUCPQ) 2725, chemin Ste-Foy Quebec, Qc Canada G1 V 4G5
004395/02409 4	Kingston General Hospital Richardson House 102 Stuart Street Kingtson, ON K7 L 2 V6 Canada	Queens University Office of Research Services Fleming Hall, Jemmett Wing, Room 301 Queens University Kingston, ON, Canada
000309/024204	SPLiN s.r.o. Oddeleni TRN Cimicka 37/446 Praha 8 18200 Czech Republic	Multicentric Ethics Committee Fakultni nemocnice v Motole V Uvalu 84 Prague 5 ZIP: 150 06 Czech Republic
000683/023960	H:S Hvidovre Hospital Hjerte-Lungemedicinsk afdeling Kettegaard Alle 30 Opgang 1 Hvidovre 2650 Denmark	Den videnskabsetiske komité for region hovedstaden Regionsgaarden Kongensvænge 2 3400 Hillerød
001687/024403	Astmacentrum Hornerheide Hornerheide 1 Horn 6085 NM Netherlands	METC Zuidwest-Holland M.H.H.A. Kirkels-Breukers P.O Box 5011 2600 GA Delft The Netherlands

Appendix E4

COPDGene Publications

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Note that publications of secondary analyses of the COPDGene cohort are ongoing and additional detail can be found at http://www.copdgene.org/publications.

Appendix E5

ECLIPSE Publications

Population

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Note that publications of secondary analyses of the ECLIPSE cohort are ongoing and additional detail can be found at *http://www.eclipse-copd.com/*.

Appendix E6

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