FP09.05 Driver Oncogenic Alterations and Indoor Radon in NSCLC Patients From the IFCT Biomarker Cohort: Bioradon France Study

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and 11+ drinks per week (Table). Those with FH of LC were more likely to be obese regardless of demographic factors, and less likely to exercise 2+ times per week (Table). Those with FH of LC were more likely to have received 10+ imaging scans and to have been exposed to hydrocarbons compared to those without FH (Table). There were no reported differences in any other occupational or home exposures, including asbestos and radon in those with v. without FH of LC (Table). **Conclusion:** FH of LC is associated with higher-risk behaviors that increase the likelihood of developing both lung and other cancers. As such, FH can be used in cancer prevention and control programs to identify higher-risk patients and educate patients that FH of lung cancer is associated with risk-increasing behaviors that are nonetheless modifiable. **Keywords:** Family History, Risky Behaviors, Prevention

FP09.05

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Introduction: Radon is a radioactive gas, considered as the leading cause of lung cancer in non-smokers. In a previous work, we studied the correlation between the estimation of radon concentration from the French Indoor Radon Map (*Institut de Radioprotection et de Sûreté Nucléaire,* IRSN) and the regional prevalence of driver alterations in a cohort of 116.424 NSCLC patients in France (*Mezquita et al, WCLC 2018*). The prevalence of driver oncogene alterations was significantly higher in high-radon areas but clinical data were not available. We aim to confirm this hypothesis in an annotated database of NSCLC patients with matched molecular data available for adjustment (*Barlesi et al, Lancet 2016).* **Methods:** Retrospective assessment of patients with NSCLC tested for *EGFR/BRAF/HER2/KRAS* mutations (m) and *ALK* fusion from the 28 Platform led by the National Cancer Institute between Apr.2012 and Apr.2013, and included in the Biomarkers France dataset. We studied the association between the prevalence of driver oncogenic alterations (*EGFR/ALK/BRAF/HER2/KRAS*) and the radon mean concentration in the area where the patient was born according to the IRSN Map. Adjustment on age, gender and smoking was performed. **Results:** Out of 17664 patients, we analyzed 3994 with birthplace available: 63% males, 82% smokers, with a median age of 64 years [18-94]. Lung cancer tumors were mostly adenocarcinoma (76%), followed by other histologies (18%) and squamous (6%). By molecular alterations: 468 tumors harbor *EGFRm* (12%), 129 *ALK* (3%), 89 *BRAFm* (2%), 32 *HER2m* (1%), 985 *KRASm* (25%); 2273 *wildtype* or harbor other non-driver alterations (control; 57%). Adenocarcinoma histology (83.7% vs. 80.2%, p=0.0034), and non-smoker habit (19.5% vs. 16.5%, p=0.0251) were more common in radon high-risk group (comparatively at the low risk group). The mean radon concentration by birthplace was 74.36 Bq/m^3 ±53.28SD [range 16.6-622.3], and by molecular groups: *EGFRm* 72.49 Bq/m^3 ± 47.98 SD [16.6-461.4], *ALK* 80.24 Bq/m^3 ±55.22SD [19.3-384.7], *BRAFm* 73.22 Bq/m^3 ±47.86SD [19.3-319.3], *HER2m* 72.74 Bq/m^3 ±39.51SD Bq/m^3 [27.8-231.3], *KRASm* 71.79 Bq/m^3 ±53.32SD [16.6-576.8] and control group 75.67 Bq/m^3 ± 54.5SD [16.6-622.3] (p=0.20). The prevalence of driver alterations was higher in high-radon areas (table 1; *p=0.0472*); but no significant difference was observed after adjustment on age, gender and smoking. **Conclusion:** We observed a higher prevalence of driver oncogenic alterations in NSCLC patients born in high radon areas; but no significant difference was observed after adjustment on age, gender and smoker. This study warrants further research on radon gas and driver oncogenes. **Keywords:** radon, lung cancer, driver oncogenic alterations

FP10 SMALL CELL LUNG CANCER/NET

FP10.01

Survival in Advanced SCLC: Projected Impact of Immuno-Oncology-Associated Durable Response on Population Health Gains in US

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			Low radon <50 Bq/m3	High radon ≥50 Bq/m3	P value	P value adjusted*
EGFR	*Mutation*	N (%)	155 (11.4%)	313 (13.2%)	0.1218	0.3024
	Control (1)	N (%)	1199 (88.6)	2059 (86.8)		
ALK	***Fusion***	**N (%)**	**42 (3.4%)**	**87 (4.1%)**	0.3272	0.4708
	Control (1)	N (%)	1199 (96.6)	2059 (95.9)		
BRAF	***Mutation***	**N (%)**	**32 (2.6%)**	**57 (2.7%)**	0.8708	0.9865
	Control (1)	N (%)	1199 (97.4)	2059 (97.3)		
HER2	***Mutation***	**N (%)**	**7 (0.6%)**	**25 (1.2%)**	0.0880	0.1781
	Control (1)	N (%)	1199 (99.4)	2059 (98.8)		
KRAS	***Mutation***	**N (%)**	**375 (31.3%)**	**610 (29.6%)**	0.3227	0.3478
	Control (2)	N (%)	824 (68.7)	1449 (70.4)		
DRIVER	***Positive***	**N (%)**	**236 (16.4%)**	**482 (19%)**	**0.0472**	0.2128
	Control (1)	N (%)	1199 (83.6)	2059 (81)		

(1) population no EGFRm, noBRAFm, no HER2m and no ALKr. (2) population no EGFRm, noBRAFm, no HER2m, no ALKr and no KRASm. *adjustment on age, gender and smoking