

P3.053: Ambient PM2.5 Exposure and EGFR Mutation Status Among NSCLC Patients in a US Real-World Cohort

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INTRODUCTION

- Ambient fine particulate matter (PM2.5) is an established lung carcinogen¹ and has been associated with worse lung cancer outcome in some settings²
- Emerging evidence suggests that PM2.5 may promote tumorigenesis in cells harboring pre-existing oncogenic alterations, including EGFR-driven lung cancer^{3,4}
- To extend this growing literature, we evaluated the association between residential PM2.5 exposure and EGFR mutation status in a US real-world cohort

DISCUSSION AND CONCLUSION

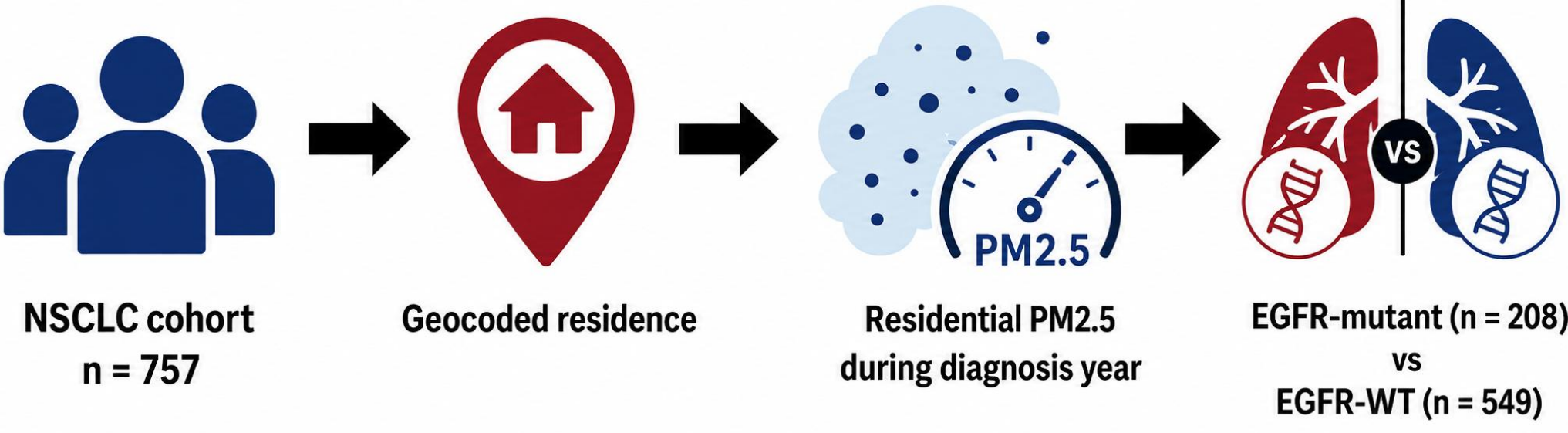
Conclusion

- PM2.5 exposure was associated with EGFR status among patients with NSCLC, with higher exposure observed in those with EGFR-WT tumors. Validation in larger longitudinal cohorts is needed

Limitation

- Single-center, retrospective design
- Modeled exposure at the diagnosis-year residence may not reflect personal or cumulative exposure
- Different ascertainment periods for the EGFR-mutant (2009–2024) and EGFR-WT (2009–2015) cohorts may introduce calendar-time bias

METHODS



- Retrospective study from MECDC cohort: EGFR-mutant (2009–2024) vs EGFR-WT NSCLC (2009–2015)
- Eligibility required documented EGFR status and linked residential PM2.5 data; squamous histology or missing PM2.5 were excluded.
- PM2.5 during diagnosis year was modeled from geocoded residences using monitor-calibrated satellite/GEOS-Chem data²
- PM2.5 comparisons used Wilcoxon and χ^2 tests, stratified by smoking and gender
- Logistic regression assessed the association between PM2.5 and EGFR mutation status, adjusting for smoking and sex

REFERENCES & ACKNOWLEDGEMENTS

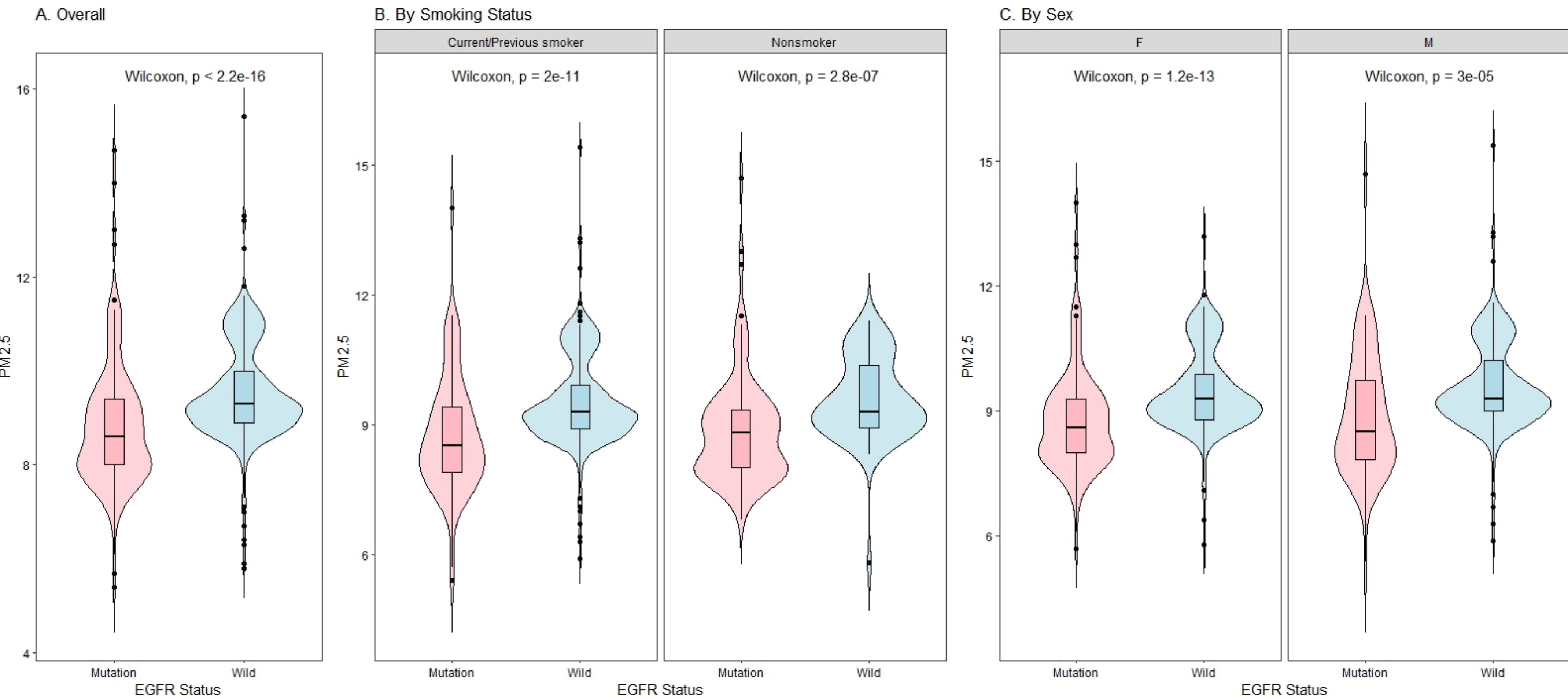
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AI was used to assist with language, formatting, and image editing; all content was independently reviewed and verified by the authors

RESULTS



Characteristic	EGFR-mut (n=208)	EGFR-WT (n=549)	P
PM2.5, $\mu\text{g}/\text{m}^3$ (median [IQR])	8.60 [8.00, 9.40]	9.30 [8.90, 10.00]	<.001
Age, mean (SD)	68.8 (12.5)	66.3 (11.03)	.009
Female gender, n (%)	153 (73.6)	291 (53.0)	<.001
Current or previous smoker, n (%)	96 (46.2)	479 (87.2)	<.001
Stage IV, n (%) [*]	164 (79.6)	279 (51.4)	<.001
BMI, kg/m^2 median [IQR]	24.70 [20.88, 28.09]	24.45 [21.29, 28.89]	.450
Histology [†]			.282 [‡]
Adenocarcinoma	178 (86.0)	397 (72.3)	
Other adenocarcinoma subtypes	17 (8.2)	69 (12.6)	
NSCLC/carcinoma, NOS	7 (3.4)	62 (11.3)	
Other mixed or rare histology	5 (2.4)	21 (3.8)	
Common EGFR mutation (19 del or L858R), n (%)	171 (82.2)	0 (0.0)	
Uncommon EGFR mutation, n (%)	37 (17.8)	0 (0.0)	

^{*} Stage available for 206/208 mutant and 543/549 wild-type cases; percentages use nonmissing denominators. [†] Histology available for 207/208 mutant and 549/549 wild-type cases. [‡] P=.282 is from the original 28-level histology variable; categories are grouped here for display.

- Median residential PM2.5 exposure during the year of diagnosis was 9.20 $\mu\text{g}/\text{m}^3$ (IQR, 8.70–9.80)
- Median PM2.5 exposure was higher in EGFR-WT than EGFR-mutant patients
- Higher PM2.5 exposure among EGFR-wild-type patients was consistent across sex and smoking subgroups
- Association remained significant when PM2.5 was dichotomized at the US EPA cutoff of 9 $\mu\text{g}/\text{m}^3$ (χ^2 P<.0001)
- Each 1 $\mu\text{g}/\text{m}^3$ higher PM2.5 level was associated with greater odds of EGFR-WT status (aOR, 1.94; P<0.001; adjusted for smoking and sex)