

P3.001: Impact of Cooking Oil Fume Exposure on Disease Characteristics in EGFR-Mutated Lung Cancer: Korean EGFR Registry

Kang-Mo Gu^{1,2}, Kyoung Min Moon^{1,2}, Hae In Jung^{1,2}, Myeong Geun Choi³, Korean EGFR Registry Investigators

¹ Department of Internal Medicine, Chung-Ang University College of Medicine; ² Division of Pulmonary and Allergy Medicine, Department of Internal Medicine, Chung-Ang University Hospital, Seoul, Korea; ³ Department of Pulmonary and Critical Care Medicine, Asan Medical Center, University of Ulsan College of Medicine, Seoul, Korea

Correspondence: Kang-Mo Gu (9kangmo@gmail.com)

INTRODUCTION

- Cooking oil fumes**
 - Contain polycyclic aromatic hydrocarbons and aldehydes
 - Established risk factor → Lung cancer in women who have never smoked in East Asia
- EGFR-mutated lung cancer**
 - Well-established targeted therapies
 - But, a subset shows **primary resistance** to first-line EGFR-TKI
 - Whether cooking oil fume exposure influences the course of EGFR-mutated lung cancer is unknown.
- Objective**
 - To evaluate the association between cooking oil fume exposure and disease characteristics, metastatic patterns, and response to first-line EGFR-TKI.

METHODS

- Design**
 - Korean EGFR Registry — multicenter retrospective cohort of patients with EGFR-mutated lung cancer across institutions in South Korea.
- Exposure definition**
 - Self-reported frequency of cooking with oil.
 - Exposed = ≥3 days/week
 - Unexposed = <3 days/week or never.



- Endpoints**
 - Primary:** Best overall response to first-line EGFR-TKI
 - Secondary
 - Organ-specific metastasis at diagnosis, clinical and pathologic stage, and new metastasis during EGFR-TKI therapy
- Statistics**
 - Chi-square and Fisher's exact tests for categorical variables; t-tests for continuous variables.

DISCUSSION & CONCLUSION

- Key findings**
 - Exposed patients presented at an **earlier stage** yet showed a markedly higher rate of primary progressive disease on first-line EGFR-TKI.
 - The metastatic pattern differed: less liver but more pleural involvement.
 - EGFR mutation subtypes and PD-L1 expression were comparable, so intrinsic resistance is not explained by conventional mutation profile.
- Actionable take-away message**
 - A cooking oil fume exposure history may flag patients at higher risk of primary TKI resistance and warrants closer early response assessment.
- Limitations**
 - Retrospective design and self-reported exposure. The exposed group was strongly enriched for women with no smoking history, so sex and smoking history are potential confounders.
- Future direction**
 - Prospective studies adjusting for sex and smoking history, with molecular profiling to define mechanisms of intrinsic resistance.

REFERENCES & ACKNOWLEDGEMENTS

References

- Berg CD, Schiller JH, Boffetta P, et al. Air pollution and lung cancer: a review by the IASLC Early Detection and Screening Committee. J Thorac Oncol. 2023;18(10):1277-1289.
- IARC. Household use of solid fuels and high-temperature frying. IARC Monogr Eval Carcinog Risks Hum. Vol 95. Lyon: IARC; 2010.
- Yu ITS, et al. Wok-cooking and lung cancer in non-smoking Chinese women. Cancer Res. 2006;66:4961–7.

Acknowledgements

We thank the Korean EGFR Registry investigators and the patients who participated in this study.

- Artificial Intelligence (AI) Use Disclosure**
- Disclosure for this poster:** Anthropic Claude was used for language editing and layout preparation. It was not used to generate, analyse, or interpret study data. All scientific content was created and verified by the authors.

RESULTS

Table 1. Baseline and tumour characteristics

Variable	Unexposed (n=1,732)	Exposed (n=210)	p
Age, yr, median [IQR]	68 [61–76]	65 [57–74]	<0.001
Female, n (%)	994 (57.4)	173 (82.4)	<0.001
Never smoked, n (%)	1,086 (63.0)	160 (76.6)	<0.001
Pack-years, mean (SD)	10.0 (18.0)	5.1 (12.2)	<0.001
Secondhand smoke, n (%)	588 (33.9)	88 (41.9)	0.027
Severe kitchen fume, n (%)	10 (0.6)	21 (10.0)	<0.001
ECOG PS 0–1, n (%)	1,449 (93.1)	175 (93.1)	1.000
Adenocarcinoma, n (%)	1,680 (97.0)	201 (95.7)	0.345
EGFR Ex19del, n (%)	830 (47.9)	101 (48.1)	1.000
EGFR L858R, n (%)	794 (45.8)	93 (44.3)	0.723
PD-L1 ≥50%, n (%)	201 (11.6)	23 (11.0)	0.869

Exposed: cooking oil use ≥3 days/week. Bold p-values indicate p<0.05.

Figure 2. Organ-specific metastasis at diagnosis (stage IV, N=1,108)

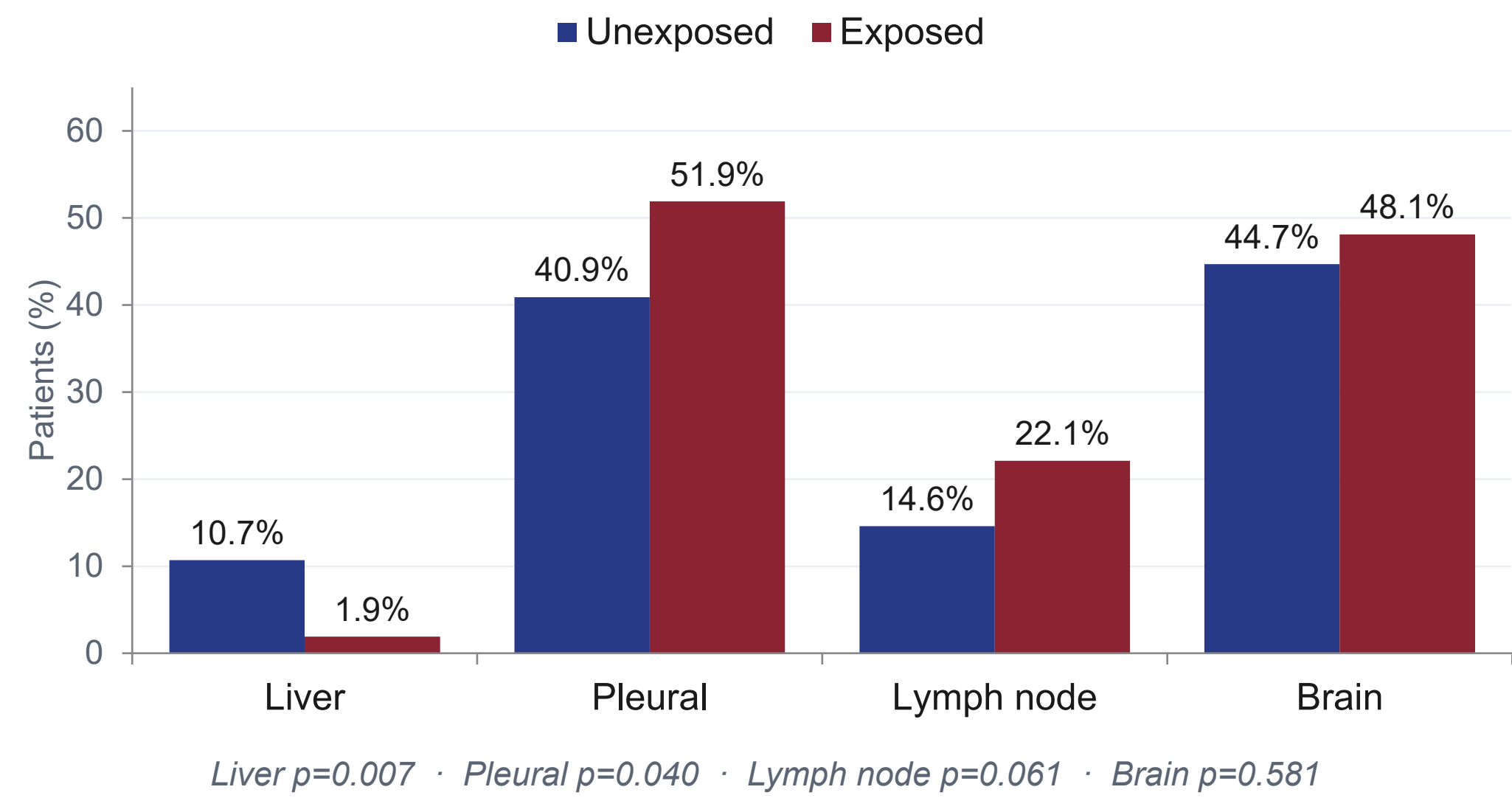


Figure 1. Earlier stage at diagnosis in the exposed group

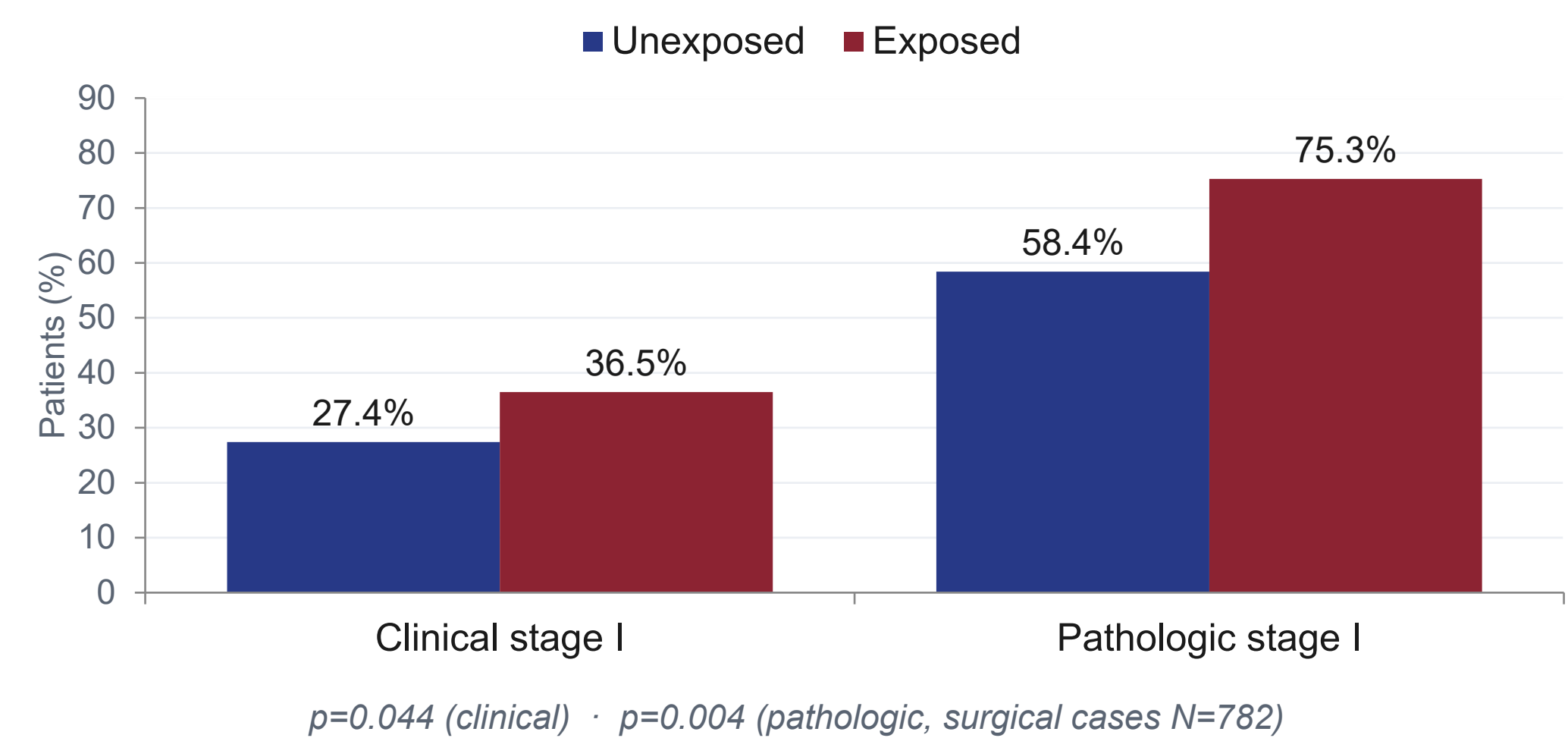
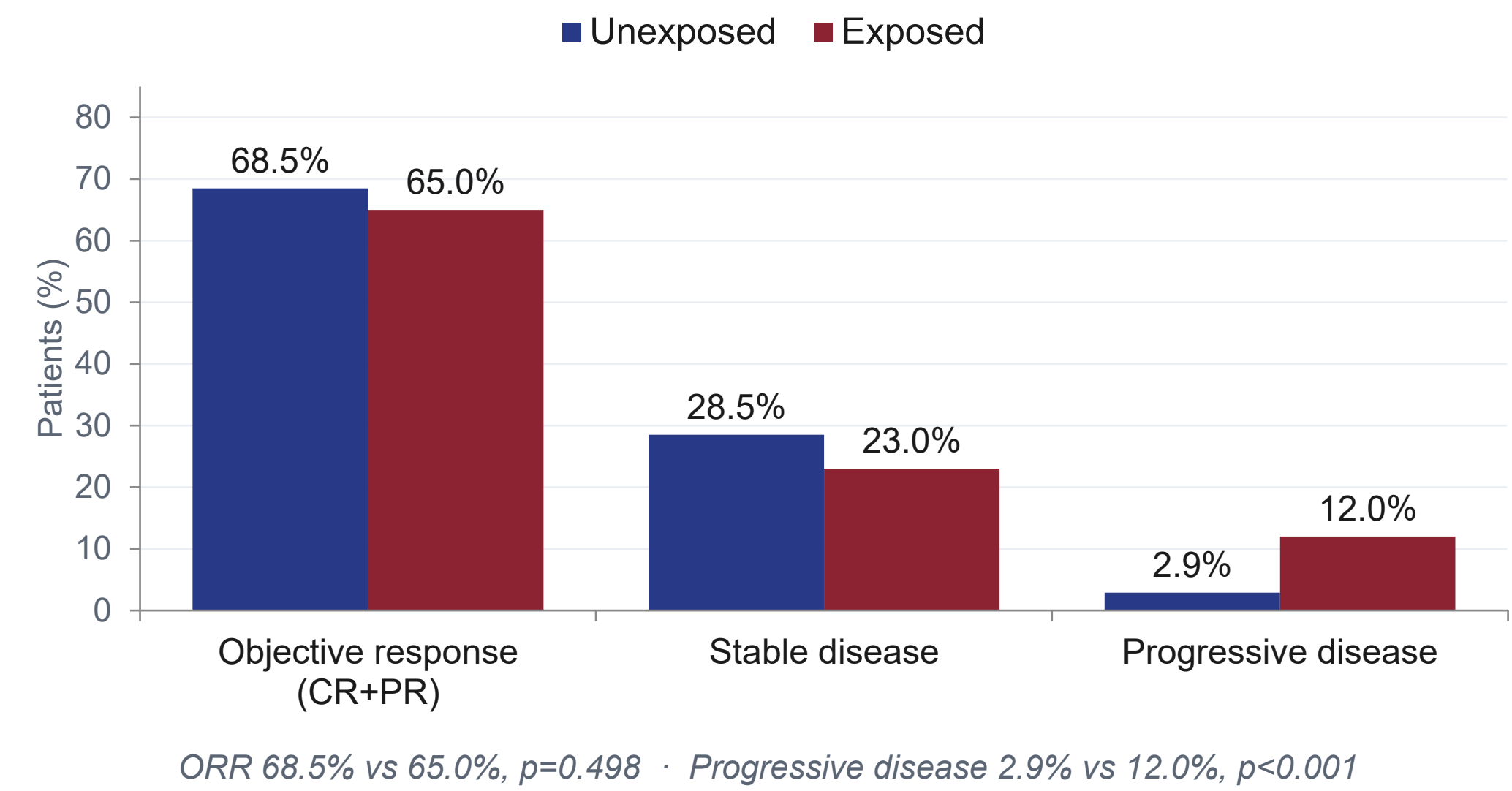


Figure 3. Best response to first-line EGFR-TKI (N=1,025)



New metastasis during first-line EGFR-TKI therapy

Event	Unexposed	Exposed	p
New CNS metastasis (N=597)	39/542 (7.2%)	6/55 (10.9%)	0.468
New bone metastasis (N=585)	26/526 (4.9%)	0/59 (0.0%)	0.157

KEY MESSAGE

Frequent cooking oil fume exposure was associated with an **earlier stage at diagnosis** but a **four-fold higher rate of primary progressive disease** on first-line EGFR-TKI (12.0% vs 2.9%, p<0.001), despite identical EGFR mutation profiles.

- Interpretation**
 - The exposed group was enriched for women with no smoking history and low cumulative tobacco exposure, and was diagnosed at an earlier stage, yet showed a substantially higher rate of primary progression on first-line EGFR-TKI.
 - Because EGFR mutation subtypes and PD-L1 expression were similar between groups, the difference is not explained by conventional mutation profiling; the distinct metastatic distribution points to a biologically different disease.
 - Prospective studies adjusting for sex and smoking history are needed to validate these observations.