not piecemeal necrosis grade, bile duct paucity and cholangitis, or immunological laboratory data, were associated with smoking history. Multiple logistic regression analysis identified smoking intensity, years of passive smoking and significant necroinflammatory histological activity as independent risk factors of advanced liver fibrosis (F3–F4 stage) at diagnosis, adjusted for age, gender, BMI and alcohol consumption. For every pack-year increase in smoking intensity there was a 3.2 times higher likelihood of advanced fibrosis (95% CI: 2.018–6.294).

**Conclusion** Our study results confirm the previously reported link between smoking history and the risk of advanced liver fibrosis at diagnosis in PBC. The mechanism by which smoking may accelerate the histological progression of PBC is unknown and larger studies are needed to define it.

**Disclosure of Interest** None Declared.

**REFERENCES**

1 The Health Survey for England 2007

**Disclosure of Interest** None Declared.