Assessing the effect of 24 weeks Fenofibrate therapy on markers of AAA severity and growth.

Final findings of the FAME2 trial

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1. Background: Fenofibrate limits AAA severity in mouse models by reducing aortic osteopontin (OPN), kallistatin and proteolytic remodelling [1]. We hypothesised that fenofibrate may block AAA pathogenesis and progression in patients.

Aim: Test the effect of 24 weeks fenofibrate therapy in patients with small AAAs.

Outcomes: Compare 1) serum OPN, kallistatin and MMP9 concentrations; and 2) AAA diameter between patients allocated fenofibrate or placebo

2. Methods: Double-blind, placebo controlled RCT [2]

Sample size: 140 patients required to detect a 30% reduction in circulating OPN and kallistatin (β 0.9; 2-tailed α: 0.025).

Statistics: Linear mixed effects modelling assessing interaction between time and treatment (intention to treat analysis).

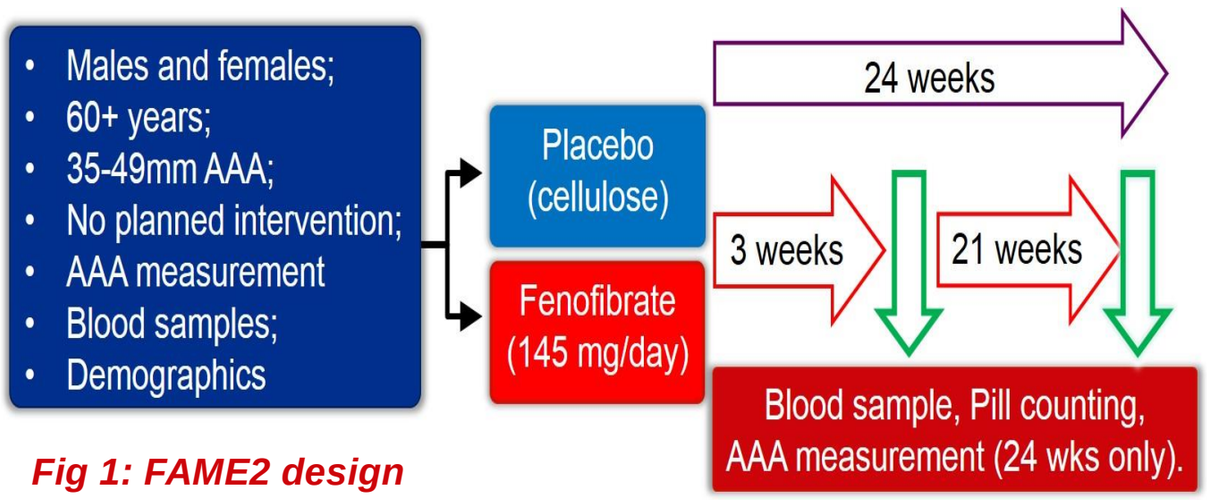


Fig 1: FAME2 design

3. Results (Fig. 2)

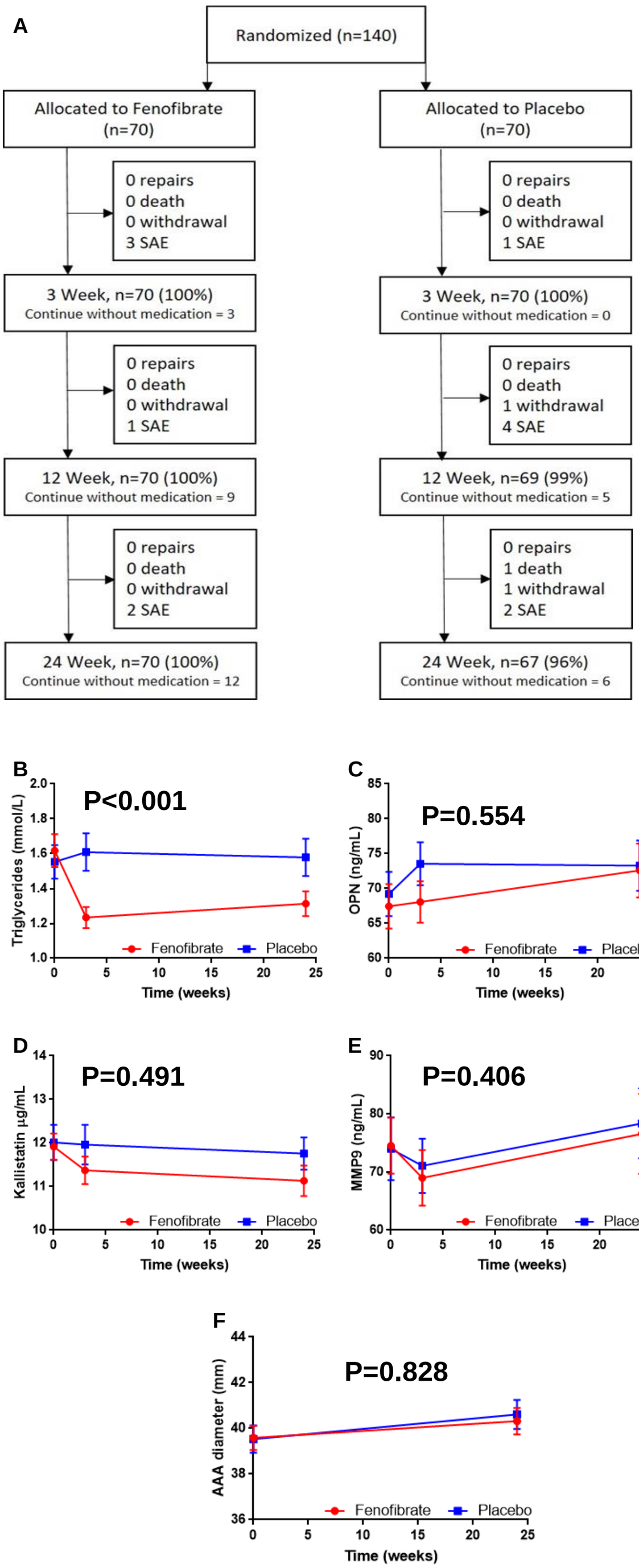
- Patient workflow: 140 patients were recruited (n=70/group); 97.9% followed to completion and 85% compliant with medication **(A)**.
- Serum triglycerides significantly lower in patients allocated fenofibrate **(B)**.
- No differences in serum OPN, kallistatin or MMP9 between groups **(C-E)**.
- AAA growth rates similar between groups **(F)**.

4. Conclusion: Data suggest that 24 weeks fenofibrate therapy does not alter AAA biomarkers or growth, and is unlikely to influence clinical management [3].

5. References:

1. *Krishna et al. Am J Pathol 2012; 18(1):706-718.*
2. *Rowbotham et al. Int J Clin Trials 2016; 3:217-224.*
3. *Pinchbeck et al. JAHA (in press).*

Fig 2: FAME2 results



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