Everyone is the age of their heart

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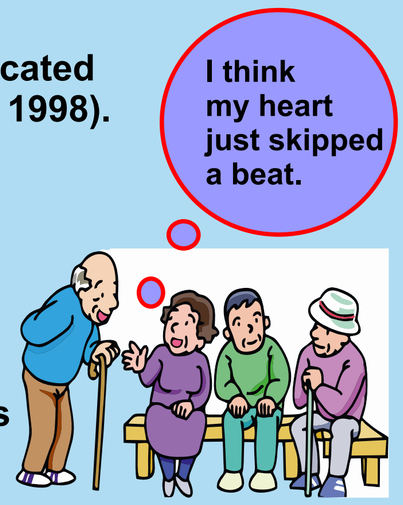
Introduction

- Atrial Fibrillation (AF) is a common heart disease characterised by rapid and irregular heartbeats.
- AF affects 1 - 2% of the total population (Schotten *et al.,* 2011).
- Prevalence of AF increases significantly with age: 6% of the cases are diagnosed in people aged 65+ years and 10% in people aged 80+ years (Hayashi *et al.,* 2002).
- A quarter of all strokes cases are caused by AF (Miyasaka *et al.,* 2005).
- AF is caused by heart muscle cells located in the lung veins (Haissaguerre *et al.,* 1998).
- The prevalence of AF increases as the population gets older.
- Ultrastructural changes of cells within the heart are one of the factors maintaining AF. Changes in the lung vein muscle cells have so far not been characterised..



Aim

- Characterise ultrastructural changes in the lung vein muscle cells during ageing.

Methods

- Lung vein tissue from 3 and 24 month-old mice.
- Imaging using electron microscopy, which uses an electron beam to magnify specimens as much as 20000 times.
- Quantify ultrastructural changes observed.

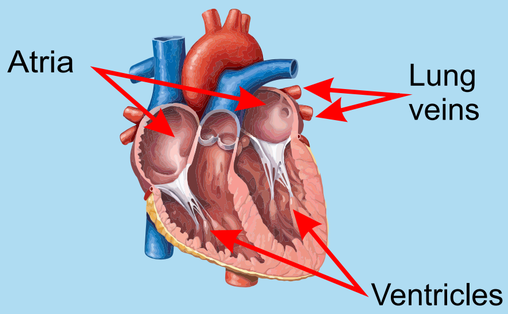


Figure 1: General anatomy of the heart.

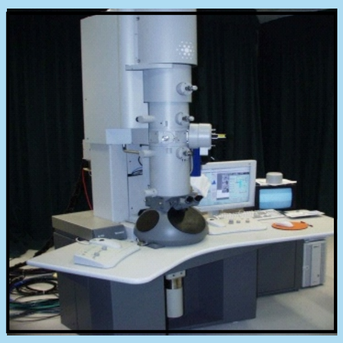


Figure 2: Transmisson electron microscope.

Results

(1) General ultrastructure of lung vein muscle cells.

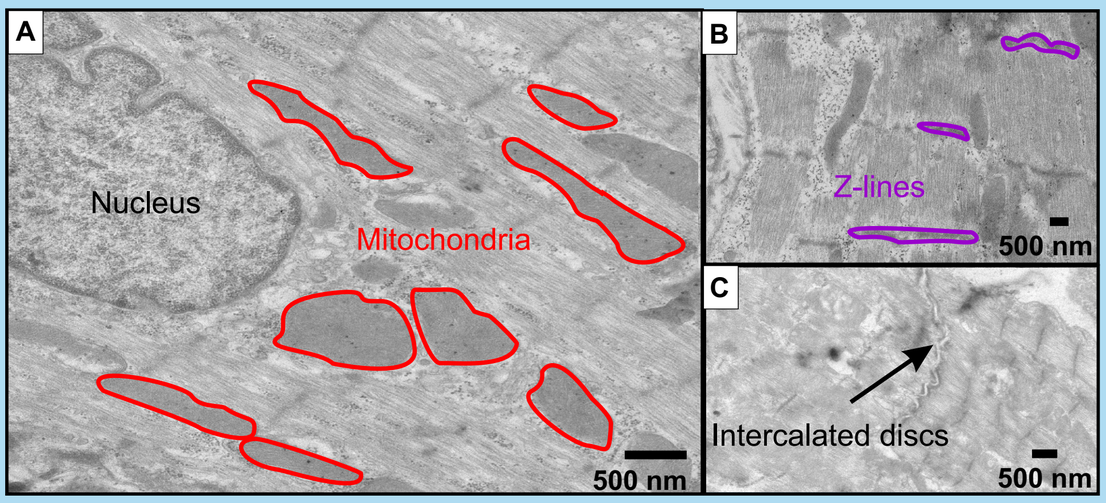


Figure 3: (A) An electron micrograph image showing general ultrastructure of lung vein muscle cells. These cells contain many mitochondria (powerhouses of cells), a nucleus is also seen in the image. (B) A higher magnification electron micrograph showing Z-lines. (C) An electron micrograph showing an intercalated discs connecting the two cells.

(2) Mitochondrial number and size are increased in lung vein muscle cells from 24 compared to 3 month-old mice.

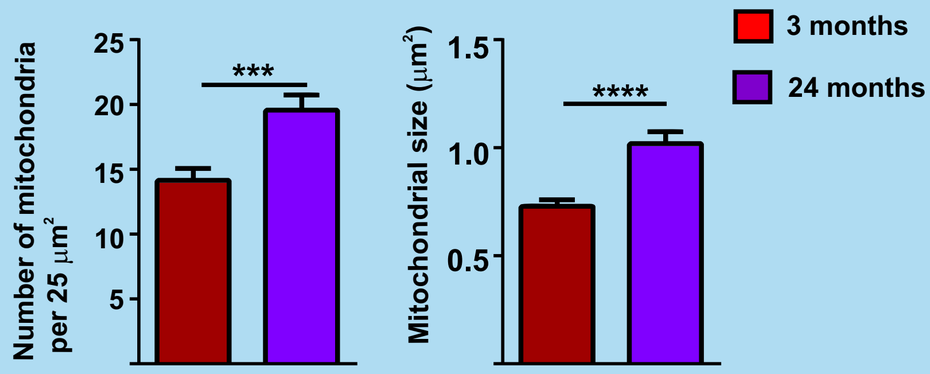


Figure 4: An increase in mitochondrial number and size in 24 month-old animals. (A) shows an increased number and (B) shows an increased size of mitochondria from 24 month-old mice. N = 4 animals per group, $p < 0.05$, unpaired t-test.

(3) An increase in non-degradable materials in 24 month-old mice.

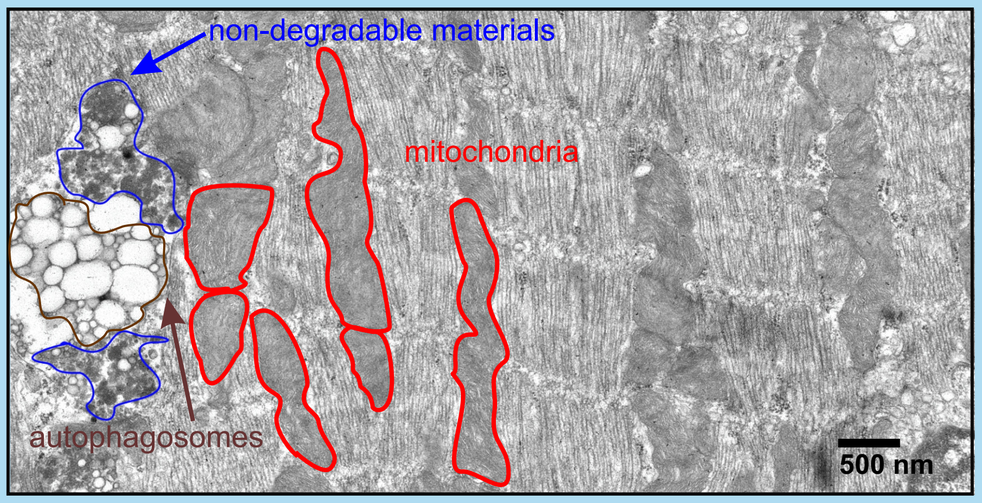


Figure 5: A representative electron micrograph of lung vein muscle cells from 24 month-old mice showing the presence of non-degradable materials (lipofuscin) and general degradation of damaged proteins (autophagosomes). The image also highlights the increase in size and heterogeneity in the shape of mitochondria.

Conclusion

An increase in mitochondrial size and number in 24 month-old mice.

An increase in non-degradable materials in 24 month-old mice.

→ Might interfere with the ability of the heart to beat efficiently.

→ Increase the risk of developing AF.

Acknowledgements

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References

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