**Notes Template**

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Long version

## Regulation of Mitochondrial Structure and Function by the F1F0-ATPase Inhibitor Protein, IF1 (2008)

Campanella, M., Casswell, E., Chong, S., Farah, Z., Wieckowski, M. R., Abramov, A. Y., … Duchen, M. R. (2008). Regulation of Mitochondrial Structure and Function by the F1Fo-ATPase Inhibitor Protein, IF1. *Cell Metabolism*, *8*(1), 13–25. https://doi.org/10.1016/j.cmet.2008.06.001

* Summary
  + Mitochondrial respiratory distress causes the F1F0 ATP synthase enzyme to reverse its role by consuming ATP in order to maintain mitochondrial membrane potential (Ψm).
  + Little is known about the cell biology of IF1 so understanding the functional consequences of IF1 expression is needed.
  + During inhibition of respiration, IF1 conserves ATP @ the expense of Ψm.
  + Overexpression of IF1 is protective against ischemic injury.
  + Expression levels of IF1 vary between cell and tissue types and it mediates the response of cells/tissue to mitochondrial respiratory distress.
  + Overexpression of IF1 leads to increased cristae density.
  + Overexpression of IF1 increases the formation of dimeric ATP Synthase complexes leading to increased FFAS activity.
* Existing Explanations
  + Mitochondrial oxidative phosphorylation[[1]](#footnote-1) is central to the organization of eukaryotic life. Oxidative phosphorylation is the final stage of cellular respiration where Oxygen is the final electron acceptor and splits into two.
* Alternative Explanations
  + What is the paper’s argument
* Methods/Experimental Procedures Short
  + What evidence supports this argument
* Methods/Experimental Procedures Long
  + Comment about the article
* Tools Short
  + Comment about the article
* Tools Long
  + Comment about the article
* Data and Analysis (Methods and Tools)
  + Comment about the article
* Discussion/Future Avenues for Research
  + Comment about the article
* Conclusion
  + Comment about the article

Short version

## IF1 protein regulates mitochondrial structure and function (2008)

Article, *Journal*, Long Citation

* Summary
  + Short summary of the article
* Analysis
  + Comment about the article

Formatting

-use NoteLevels from the ListStyles dropdown; this links the bullet levels to the text styles (I could also make my own to make them easier to see)

-change formatting by placing cursor on the level you want to change, pressing ctrl + shift + s, then modifying the text style (could change this step if I link the notes differently)

-use heading 1 for the weeks and heading 2 for the short article citations

## Section Title

Long version

## The Molecular Mechanism of ATP Synthesis by F1F0-ATP Synthase (2001)

Campanella, M., Casswell, E., Chong, S., Farah, Z., Wieckowski, M. R., Abramov, A. Y., … Duchen, M. R. (2008). Regulation of Mitochondrial Structure and Function by the F1Fo-ATPase Inhibitor Protein, IF1. *Cell Metabolism*, *8*(1), 13–25. https://doi.org/10.1016/j.cmet.2008.06.001

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  + Mitochondrial respiratory distress causes the F1F0 ATP synthase enzyme to reverse its role by consuming ATP in order to maintain mitochondrial membrane potential (Ψm).
  + Little is known about the cell biology of IF1 so understanding the functional consequences of IF1 expression is needed.
  + During inhibition of
* Existing Explanations
  + What does the field look like now
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* Tools Short
  + Comment about the article
* Tools Long
  + Comment about the article
* Data and Analysis (Methods and Tools)
  + Comment about the article
* Discussion/Future Avenues for Research
  + Comment about the article
* Conclusion
  + Comment about the article

Short version

## IF1 protein regulates mitochondrial structure and function (2008)

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As living organisms we require oxygen to live. The reason we need oxygen is for cellular oxidative phosphorylation, the final stage of cellular respiration. Oxidative phosphorylation is made up of two steps which are the ETC and chemiosmosis. In ETC electrons are passed from one molecule to another and energy released in these transfers is used to form an electrochemical gradient. In chemiosmosis, the energy stored in the EC gradient is used to make ATP.

What is oxidative phosphorylation? The final stage of cellular respiration.

What are the two stages of oxidative phosphorylation? ETC and chemiosmosis.

What is ETC? in this stage molecules share electrons leading to the formation of an EC gradient.

What is chemiosmosis? Energy from EC gradient is used to make ATP.

How does oxygen fit into the picture?

Oxygen sits at the end of the ETC where it accepts electrons and picks up protons to form H2O.

Net result of glycolysis is 2 ATP.

Yeast, mice, drosophila, round worm (elegans) and e coli.

Pax6 gene responsible for eye development.

Just like humans have the ability to migrate and survive, so do genes.

1. [Khan Academy explains oxidative phosphorylation.](https://www.khanacademy.org/science/biology/cellular-respiration-and-fermentation/oxidative-phosphorylation/a/oxidative-phosphorylation-etc) [↑](#footnote-ref-1)