

## How *fdhF* Deletion Affects Ion Transport and ATPase Activity in *E. coli*?

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### ABSTRACT

Formate dehydrogenase-H plays a key role in anaerobic metabolism, particularly in formate metabolism during fermentation. To assess its glucose-dependent role, proton ( $J_{H^+}$ ) and potassium ( $J_{K^+}$ ) fluxes, along with  $F_0F_1$ -ATPase activity, were examined in wild-type and *fdhF* mutant grown under low ( $2 \text{ g L}^{-1}$ ) and high ( $8 \text{ g L}^{-1}$ ) glucose. When cells were grown in the presence of low glucose and during assays similar amount was added mutant showed 40% higher  $J_{H^+}$  but 30% lower  $F_0F_1$ -conditioned flux, consistent with a 20% reduction in ATPase activity upon potassium supplementation, but there were no differences when extra formate, or potassium and formate together were supplemented. At the same time total  $J_{K^+}$  was lower compared to wild type, meanwhile DCCD does not affect the flux. Under high glucose, mutants displayed a 50% increase in total and DCCD-sensitive  $J_{H^+}$  fluxes, a 35% decrease in  $J_{K^+}$ , and a 50% drop in  $F_0F_1$ -conditioned flux. When cells were grown in a presence of high glucose and during assays similar amount was supplemented total  $J_{H^+}$  was similar in mutant and wild type, meanwhile DCCD-sensitive flux decreased by 25-35%, conversely total  $J_{K^+}$  increased by 35% and with DCCD potassium outflux was observed. Potassium had no effect on ATPase activity, but formate increased it by 25%. These findings suggest that under low glucose, accumulated formate stimulates proton efflux while inhibiting ATPase, whereas under high glucose, formate regulates ATPase via potassium, highlighting a glucose-dependent shift in formate's control of membrane bioenergetics.

**Keywords:** formate dehydrogenase H,  $F_0F_1$ -ATPase,  $J_{H^+}$ ,  $J_{K^+}$ , glucose concentration

### References:

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