

Fatty Acid Oxidation Assay ab217602

12 References 2 Images

Overview

Product name	Fatty Acid Oxidation Assay
Detection method	Fluorescent
Sample type	Adherent cells, Suspension cells
Assay type	Cell-based (quantitative)
Product overview	<p>Fatty Acid Oxidation Assay (ab217602) allows the detection of Fatty Acid Oxidation (FAO) in live cells when used in combination with our <u>Extracellular Oxygen Consumption Assay (ab197243)</u>.</p> <p>The assay uses the 18C unsaturated fatty acid Oleate as substrate, and includes two FAO modulators, etomoxir and FCCP. Etomoxir, an inhibitor of the carnitine transporter CPT1, prevents Oleate import and thereby limits the supply of reducing equivalents to the ETC, reducing oxygen consumption in turn. The remaining ETC (electron transport chain) activity is driven by non-long chain FAO. FCCP treatment induces maximal ETC activity by dissipating the mitochondrial membrane potential, while the increased demand for reducing equivalents causes a concomitant increase in the FAO activity. If exogenous long-chain fatty acid is unavailable or import is inhibited, FAO activity will be limited.</p>
Notes	<p>Learn more about the full range of <u>assays to measure glycolysis, oxygen consumption, fatty acid oxidation and metabolic flux</u> in live cells.</p> <p>Or review the full <u>metabolism assay guide</u> for other assays for metabolites, metabolic enzymes, mitochondrial function, and oxidative stress.</p>
Platform	Microplate reader

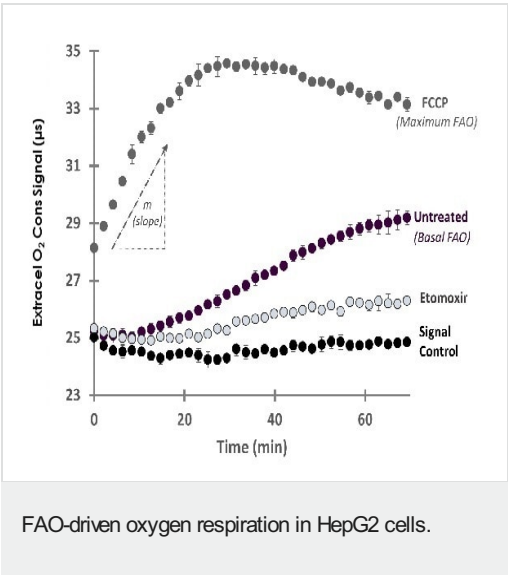
Properties

Storage instructions Please refer to protocols.

Components	55 tests
Etomoxir	1 vial
FAO Conjugate	1 x 1ml

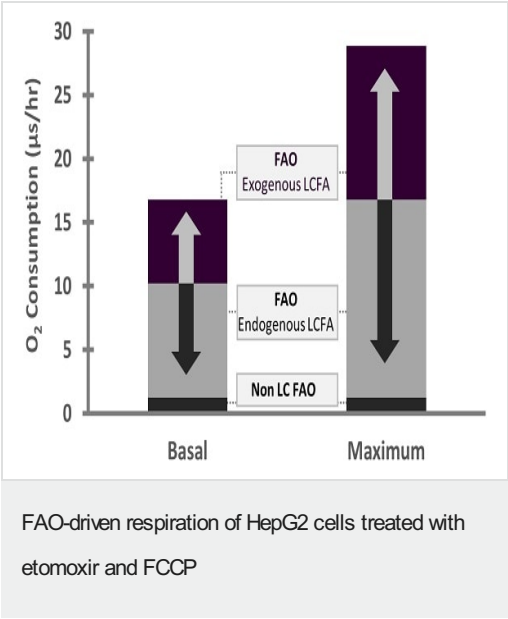
Components	55 tests
FAO Control	1 x 500µl
FAO Tablet	1 tablet
FCCP	1 vial
L-Carnitine	1 vial

Images



FAO-driven oxygen respiration in HepG2 cells treated with the CPT-1 inhibitor Etomoxir (white) and uncoupler FCCP (gray).

Untreated cells (basal FAO) curve shows a steady increase of the Extracellular O₂ Consumption Reagent signal reflecting ETC-driven oxygen consumption. **Signal Control** shows probe signal in the absence of cell respiration. **Etomoxir treatment** prevents oleate import, resulting in reduced availability of reducing equivalents and a resultant decrease in ETC activity. The remaining ETC activity (difference between Etomoxir treatment and Signal Control) is driven by metabolic activity other than long chain FAO. **FCCP treatment** induces maximal ETC activity by dissipating the mitochondrial membrane potential. Increased demand for reducing equivalents causes a concomitant increase in FAO as indicated by the rapid increase in Extracellular O₂ Consumption Reagent signal. This strong increase in ETC activity is not observed where exogenous LCFA is unavailable or where import is inhibited.



The figure summarizes the balance between these parameters under “Basal” and “Maximum” (FCCP treated) conditions. The increased energy demand imposed by FCCP treatment is met by increased FAO.

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