Electron transport chain mechanism pdf

The introduction of oxidation and the role of oxygen oxidation is defined in chemistry as the removal of electrons or the reduction in the number of oxidation of the element. Since these distant electrons should end up on some other oxidation elements one compound is always accompanied by the abbreviation of the other. The very word oxidation occurs etymologically from oxide, a compound containing oxygen. As we will see soon, oxygen is not really required for oxidation as defined above to take place. Oxygen is an excellent oxidimable agent; he likes to get electrons. The proximity of the element to electrons is expressed by the electro-health of the element. Oxygen has one of the highest electronegavitations of all elements; in fact it is second only to fluoride. This means that the transfer of electrons to oxygen, including the formation of bonds with oxygen, is thermodynamically favorable, i.e. releases energy. The only reason we don't spontaneously burn oxygen in our 20% oxygen atmosphere is the kinetic barrier created by the limitations of dioxygen triplet rotation (see Subchapter 2/4). Organisms living in a highly oxidative environment (such as ours) quickly found a way to harness the energy available from the transfer of electrons from less electronegate elements to oxygen. The often used metaphor for oxidative metabolism is burning nutrients. When we light a campfire organic polymers in wood are oxidized by oxygen dioxide and water, and we enjoy the energy emitted by this redox reaction as soothing heat and shimmering light that make bonfires so much fun. In a similar (but rather different) way, our cells oxidize the organic chemicals we get in the diet to CO2 and H2O, consuming oxygen and producing energy. How exactly do they do it? Unlike a campfire, in our cells the transfer of electrons from an organic molecule to oxygen is divided into many stages. In addition, most of the energy generated by these reactions is not released as heat and light, but is stored as a chemical potential. For didactic purposes, we can divide oxidative metabolism into two phases: 1) Substrate oxidation with reduction of coenzymes 2) Oxygen reduction of reduced coenzymes. Take glucose as an example. Six carbon molecules (C6H12O6) are gradually oxidized in glycolysis to produce two molecules of acetylcoinzyme A, acetyl-CoA) and two CO2 molecules. In the process, four molecules of redox coenzyme nicotinamide adenine dinucleotide (NADH) are reduced to nadh, each of which receives two electrons (often referred to as hydride anion, H-). Each acetyl-coA goes into the cycle of citric acid and additionally oxidizes up to two CO2 molecules and four reduced coenzymes (three NADH and one flavin adenin dinucleotide, FADH2). In general, oxidation glucose gives 12 reduced coenzymes and (like (as (as six CO2 molecules. Note that this process does not require molecular oxygen (O2) - the missing oxygen atoms are provided by water. Water contains oxygen already in a reduced form (O-II), so in these reactions oxygen is not part of the transmission of electrons. Reducing coenzyme must be reoxidised in order to keep this metabolic pathway going. Moreover, as we mentioned above, oxygen reduction promises to release a significant amount of energy. Reoxidation occurs in the mitochondria. Mitochondrial electronic transport chain Mitochondria Mitochondria are complex organelles with two membranes (external and internal) that enclose the space of the intermembrane and the mitochondrial matrix respectively. While the outer membrane is freely permeable for small molecules (zlt; 5000 Da), the inner membrane is almost entirely impervious to polar molecules (except for a select few, which have dedicated transporters). The mitochondrial matrix is a thick protein gel containing enzymes that catalyze reactions in critical metabolic pathways, such as the cycle of citric acid or beta-oxidation of fatty acids. The electronic transport chain Reduced comes, coming from the cytoplasm (through a special shuttle system) and from reactions occurring in the matrix, are reoxed to the inner mitochondrial membrane by a set of enzymes called the electronic transport chain (ETC, or respiratory chain). Mitochondrial ETC consists of four enzyme complexes called Complex I-IV. Electrons from NADH enter Complex I and are transmitted through Complex III and IV to oxygen. The electrons from FADH2 are extracted by Complex II and other enzymes (bypassing Complex I) and then follow the same path. Complex I (NADH dehydrogenase or NADH:ubiquinone oxidoreductase) Complex I catalyzes the oxidation of NADH into NADH and the transfer of two electrons to coenzyme or ubiquinone. The exact structure of the mitochondrial complex I is still unknown, but we know that it contains more than 40 units (450,000 Da), one molecule of flavin mononucleotide (FMN) and several iron atoms complex in iron-sulfur clusters (FeS). The electrons released from the nadha bind to fmn and then cascade one from one FeS cluster to the next until they arrive at ubihinone and lower it to ubiquinol. Ubiquinol (Co'G2 or UZ2) is a reduced form of ubiquinone (Cohen or ULTRASOUND). Coenzyme (an umbrella term for both forms of redox) is an extremely hydrophobic molecule due to its very long isoprenoid side chain (ten five-carbon isoprenoid units in mammals Co10), which reliably blocks it in the non-polar nucleus of the inner mitochondrial membrane. There Cohen acts as a mobile carrier of electrons from complexes I and II (and some others To Complex III. Complex II Complex ii catalyzes the oxidation of succinat for fumarate and as such is an integral part of the acid cycle (succinate dehydrogenase or succinata: ubiquinone oxidoreductase). The electrons derived from this oxidation are first transferred to FAD, bound by the enzyme, and then through a chain of three feS and cytochrome b to ubiquinone. Complex III (cytochrome c-redukaza or ubiquinol: cytochrome with oxidoredukaza) Complex III takes electrons from the reduced Coen basin and transmits them (through two cytochromes and a cluster of FeS) to another mobile electron carrier, Cytochrome c. Cytochrome C is a small hemoprotein attached to the outer surface of the inner mitochondrial membrane, i.e. in space with an intermbran. Unlike previously discussed coenzyme cytochrome C can carry only one electron per molecule (reducing its heme gland from ferric to ferros). Ubiquinol electrons are thus transmitted one by one in a complex process called a cycle. Complex IV (cytochrome c oxidase or cytochrome c:dioxygen oxidoreductase) Complex IV is the final member of the ETC, which takes electrons from reduced cytochrome C and through two cytochromes A and 3 copper atoms and deposits them to the final electronic intake, oxygen. The potential of Redox In order for the mitochondrial ETC to function as described above, there must be strength, pushing electrons from NADH to O2 along the chain. In the case of wood burning, we talked about the electronity of elementary oxygen. The associated measure of affinity for electrons is the potential of redox. In Subchapter 2/4 we discussed the potential of the electrode created by immersing the bar of pure metal in the solution of its ions (i.e. its oxidized shape). We noted that if we separate the two semi-refocias present in each redox reaction - a reduction in semi-readephation and semi-readephization of oxidation - we can determine the standard electrode potentials for these reactions occurring under standard conditions. Depending on the direction of these reactions, we often refer to such potentials as standard oxidation or standard reduction potentials. The umbrella term is the potential of redox, which is usually in the direction of reduction. The reason why electrons flow from NADH to oxygen through ETC complexes and mobile electron carriers in the correct order is that all individual stops along the way have increased redox (more specifically reducing) potentials. This means that as we go along the chain, things get easier and easier to shrink. Oxygen at the end is definitely the easiest to reduce - that's why it's such a big oxidizing agent! Where's the energy? When we started discussing oxidative metabolism, we mentioned that the advantage of using oxygen as a final electronic receiver is the amount of energy that can be provided. So far we have followed the electrons on their way to being alone with their but so far it has not been mentioned on how our cells take advantage of this. Significant increase the potential between NADH (or FADH2) and oxygen and the corresponding difference in free energy (GG) is really not wasted. It is used to pump protons (H) from the mitochondrial matrix into the intermembrane space. The flow of electrons through complexes I, III and IV is accompanied by pumping a certain number of protons into an electronic pair; Complex II does not pump anything. Since the inner mitochondrial membrane is very impervious to protons, proton potential is built through it - more protons are stermran in space than in the matrix. Higher concentration of protons means lower pH and positive electrical potential. Thus, the intermembrane space in the creative mitochondria is more acidic and more positive than the matrix. The potential of the mitochondrial membrane is usually expressed as tension. Since the gravitational potential energy of tons of water stored in the reservoir can be used at hydroelectric power plants to generate electricity, our mitochondria use the energy stored in the proton gradient through the inner membrane to make another kind of energy- chemical energy stored in adenosine-5'-triphosphate (ATP). ATP synthesis is catalyzed by an enzyme called F1. FO-ATP synthase. The FO division forms a channel in the inner mitochondrial membrane that allows protons from the intermembrane space to flow back into the matrix. This flow of protons along the electrochemical gradient is used to rotate part of the enzyme just as the water flowing through the dam causes the turbine to rotate. The rotation is then transferred to the central stem of the F1 unit, which pushes onto external units held by the immobile peripheral stems and makes them phosphorylate adenosine-5'-diphosphate (ADP) in ATD. Since the F1 division has three ATP synthesis sites, the total turnover of the enzyme is three ATP molecules. Newly synthesized ATP molecules are then transported from the matrix to the cytoplasm via a special transporter (adenin nucleotide translocator, ANT) in exchange for ADP. Stoichiometry All the processes described above are primarily about getting energy from the substrate in order to do some useful work. So it is clear that we would like to know how much useful energy our mitochondria can extract, for example, from glucose molecules or palmitic acid. Although this topic is usually dealt with fairly in a number of textbooks, it is far from trivial. We discussed above the number of electrons removed (as a decrease in coenzymes) in glucose metabolism; similar numbers can be calculated for other substrates. Thus, the question of mitochondrial stoigiometry can be formulated as follows: how many ATP molecules can be synthesized by ETC on a certain number of transported electrons. It's possible Two subs: 1) How many protons are transported through the membrane per single Couple? 2) How many protons should be moved back to the matrix to make one ATP molecule? The current consensus answer to question 1) is 10 protons per two electrons. Complexes III and IV transport six protons together and Complex I is likely to transport four protons to an electronic pair. Since the mechanism of pumping at the molecular level is still understood only partially, these figures may change in the future. The answer to question b) is even more complicated. Based on current theoretical models of the F1 function. The Fo-ATP syntasis H'/ATP ratio appears to be 4.33, in other words 13 translocation protons to make three ATP molecules (10 F1 translocation protons). FO-ATP synthases, an additional three are used to import ADP and phosphates and export ATP through ANT). The final answer to this question lies in the future. At the same time, it is important to understand that these figures are the maximum values achievable in ideal conditions, when all ETC components work flawlessly and the inner mitochondrial membrane is completely impervious to protons. This, however, is usually not the case. The disconnection of the Energy Transduction Mechanism between ETC and ATP synthesis can be disconnected, allowing protons to flow from the intermembrane space back into the matrix. This inefficient scattering of the proton gradient converts the energy stored there into the least useful form of energy: heat. Despite the heat, usually much less useful than, for example, ATP, there is one situation in which the ability to produce heat can be life-saving, namely when it is cold and you have just realized that you have left a coat, sweater, thermal underwear ... actually all your clothes, at home. The well-known heat production mechanism we (and other mammals) use tremors that creates heat through inefficiency in energy transduction muscle contraction. Newborns and many animals have a different method - mitochondrial delululuce. Mitochondrial separation to produce heat occurs in a special tissue called brown adipose tissue. Brown fat (unlike regular white fat) is brown because it is full of mitochondria. Brown fat tissue 2x - The cell contains more fat drops of different sizes. While white fat exists to store energy, the role of brown fat (in those with it) is to waste it. Mitochondria in brown fat contain a special protein that forms a channel in the inner mitochondrial membrane for translocation of protons - a heating protein-1 (UCP-1). When the brown fat tissue is activated by norepinephrine (through the 3-adrenergic receptor), it hydrolyses its triglycerides and the resulting free fatty acids provide energy for ETC and activate UCP-1. There are other subtypes of UCP (UCP-2 - UCP-5) expressed in other tissues, but their physiological still not clear. Clear. Author: Jan Trnak Trnak electron transport chain mechanism ppt. electron transport chain mechanism slideshare. electron transport chain mechanism pdf. etc electron transport chain mechanism. mitochondrial electron transport chain mechanism. role and mechanism of atp synthase in electron transport chain. mechanism of energy transfer in the electron transport chain. structure and mechanism of mitochondrial electron transport chain