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Review

Vitamin E protection in the biochemical adaptation of marine organisms to cold-water environments

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ABSTRACT

Vitamin E is one of the most important lipid-soluble antioxidants to occur in plants and animals for cellular protection against lipid peroxidation. An essential adaptation to low temperature is the elaboration of high levels of unsaturated fatty acids in the composition of cellular membranes, which is necessary to maintain functional membrane fluidity. Increasing the content of lipid unsaturation, however, occurs at the expense of enhancing the vulnerability of cellular membranes to oxidative damage. First isolated from salmon eggs, cold-water marine organisms were found to produce, or acquire, a specific vitamin E homologue, named "marine-derived tocopherol" (MDT), having an unusual methylene unsaturation at its isoprenoid-chain terminus. In this overview we compare the antioxidant composition of tropical, temperate and polar fishes, present provisional evidence that MDT is produced at the primary food chain, and provide empirical confirmation that the enhanced reactivity of MDT at low temperature is attributed to its greater rate of diffusion in viscous lipids at low temperatures. This claim of biochemical adaptation is supported by a unique model of diffusion-limited reactivity that mimics changes in the ratio of the MDT/α-tocopherol rate constants at diminishing levels of radical flux in viscous media at low temperature. We offer in conclusion future outlooks to research on antioxidant protection in cold-water ectotherms.

Contents

1. Introduction

Cold-adapted organisms have successfully evolved key genotypic or phenotypic traits to enable growth at low temperatures. Ectothermic organisms inhabiting cold environments (psychrophiles) are adapted by altering the molecular composition of their cells and tissues, by having metabolic functions and physiological features suited to low temperature performance and by adopting behaviors appropriate for survival at cold temperatures (Hazel, 1993). Marine organisms are prevalent in cold-water habitats and, like ectotherms inhabiting cold terrestrial climes, have evolved special adaptations for existence at low temperatures (Hochachka and Somero, 2002). An essential feature of cold temperature adaptation is the biochemical regulation of membrane bilayer fluidity that is critical to cellular function at low temperatures (Sinensky, 1974). Membrane homeostasis at low temperature is attained by the production of high levels

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of low molecular weight, unsaturated and branched-chain fatty acids in the lipid composition of cellular membranes (reviewed in: Hazel and Williams, 1990; Crockett, 2008), which in fish importantly includes low-temperature compensation to brain synaptic membranes for neural function (Logue et al., 2000) and to retina neural membranes for vision (Cunningham and Hyde, 1995). Having high levels of lipid unsaturation, however, incurs risk of enhancing the vulnerability of cellular membranes to oxidative damage, which, if not prevented or repaired, can impair normal cellular function (Slater, 1984). In compensation, cold-water ectotherms elaborate high levels of biochemical defenses, including the expression of antioxidant enzymes and the production or sequestration of small-molecule antioxidants, which is aptly demonstrated by a greater nutritional need for dietary vitamin E by cold-acclimated fish (Cowey et al., 1984). In one such examination, we discovered a novel vitamin E constituent in the eggs of the Pacific chum salmon, *Oncorhynchus keta* (Yamamoto et al., 1999). This vitamin E homolog was identified as an α-tocomonoenol having an unusual methylene unsaturation at the isoprenoid-chain terminus of the tocopherol molecule. Given that this vitamin E constituent is structurally distinct from the isomeric plant α-tocomonoenol of terrestrial origin (Matsumoto et al., 1995), we have assigned the salmon egg homolog as "marine-derived tocopherol" (MDT) in designation of its unique marine provenance.

MDT has since been detected in the tissues and organs of all fish examined, albeit at remarkably low concentration in tropical marine fish of the Great Barrier Reef. The predominance of high levels of MDT occurring in fishes inhabiting cold-water environments suggests that it evolved as a particular vitamin E homolog capable of efficient antioxidant performance at low temperatures (Yamamoto et al., 2001) befitting of its wide distribution in cold-water marine biota, including the marine food chain of Antarctica. Herein we review evidence that the biosynthetic origin of MDT is derived from primary algal producers, that MDT is accumulated in the pelagic food chain at higher trophic levels, and that MDT provides enhanced antioxidant protection in marine organisms adapted to cold-water environments.

2. Oxidative stress and biochemical protection in cold-water ectotherms

Temperature is a major determinant of habitat suitability and is the most significant abiotic factor to affect the complex metabolic processes and cellular integrity of living organisms (Hochachka and Somero, 2002). At low-temperature physiological states, the metabolic enzymes of ectothermal organisms are adapted to function with reduced activation energy (E_a) (Vetter and Bucholz, 1998), yet this may not fully apply to the antioxidant enzyme activities of polar fish, which are essential for protection against oxidative damage (Speers-Roesch and Ballantyne, 2005). A physical trait of cold aquatic environments is having high saturation levels of dissolved oxygen in well-mixed surface waters. Indeed, the solubility of oxygen in seawater increases by 40% between 15° and 0°C. Although intracellular oxygen diffusion rates are constrained by higher cytosolic viscosities at low temperatures (Sidell, 1998), high oxygen concentrations are usual in the tissues of cold-water ectotherms (reviewed in: Abele and Puntarulo, 2004). A particular feature of cold acclimation in fish is a profound proliferation of mitochondria in high oxygen demand tissues to increase aerobic capacities at low temperatures (Guderley and St-Pierre, 2002). Mitochondria are the proximal source of deleterious reactive oxygen species (ROS) generated during normal respiration (Turrens, 2003), most notably in Complexes I and III of the electron transport chain in which ROS production is directly dependent on the magnitude of the mitochondrial membrane potential (Korshunov et al., 1997). Not only are mitochondrial densities increased, but they are often (with notable exception of Egginton and Sidell, 1989) peripherally located in cells of cold-adapted ecotherms to reduce the diffusion distance, and thus oxygen gradients, within the cytoplasm. Long diffusion distances in the cytoplasm would not only limit mitochondrial performance, but by restricting oxygen supply to distantly located mitochondria could potentially enhance intracellular ROS production as a usual cellular response to hypoxic stress (Johnston et al., 1998). Although respiration rates are typically depressed at cold temperatures, such high densities of mitochondria combined with high tissue pO_2 levels implicate the need for effective antioxidant defenses in cold-adapted fishes to mitigate the toxic effects of mitochondrial ROS production (Filho, 2007).

An essential characteristic of all organisms adapted to cold environments is the requirement for biochemical modification of their cellular membranes so as to operate within a narrow physiological range of lipid bilayer viscosity (homoviscous adaptation) (Hazel, 1995) and the strict confines of a dynamic liquid-crystalline phase (homeophasic adaptation) (McElhaney, 1984). Processes of membrane mass transport, molecular signaling and interactions within the lipid bilayer and importantly the metabolic electron transport chains of mitochondria are reliant on the physicochemical structure of cellular membranes. Ectothermic organisms regulate their membrane properties in response to temperature by altering the lipid composition of their membranes. Cold adaptation or acclimation is achieved by regulating a greater content of low molecular weight, unsaturated and branched-chain fatty acids in the lipid composition of their cell membranes (Hazel and Williams, 1990). This biochemical restructuring of cellular membranes in adaptation to cold environments, however, renders cold-adapted ectotherms more vulnerable to oxidative damage since membrane unsaturated lipids, particularly poly-unsaturated fatty acids, are a primary target for ROS in the initiation of radical-chain lipid peroxidation. In the initiation step {1} an α-unsaturated (allylic) lipid proton is abstracted by an initiating ROS, such as a hydroxyl (HO•) or hydroperoxyl radical (HOO•), to produce a conjugated lipid radical (L•). The lipid radical (L•) being highly reactive combines readily with molecular oxygen to form a lipid peroxyl radical (LOO•) to complete the initiation step {2}. The lipid peroxyl radical (LOO•) abstracts an α-unsaturated proton from another lipid substrate to form an additional lipid radical (L•) and the product lipid hydroperoxide (LOOH) in the propagation step {2}, and this cycle of radical-chain propagation {2}→{3} continues until the reaction is terminated, usually on reduction by lipid-phase antioxidants such as the vitamin E constituent tocopherols or reduced coenzyme Q (ubiquinols), that reside within cellular membranes. Lipid hydroperoxides are unstable and their decomposition products, such as malondialdehyde, can damage vital cellular components, including DNA, which becomes toxic if not removed or repaired (Marnett, 1999).