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Effects of high pressure processing on lipid oxidation: A review

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ABSTRACT

High pressure processing (HPP) is an alternative mild-technology used in the past decades to sterilize and pasteurize food matrices such as meat and seafood. HPP obeys thermodynamic principles, namely Le Chatelier's law of equilibrium and the isostatic rule, both of which account for microbial inactivation. HPP has the advantage of ensuring reduction of pathogens and spoilage in foods, and preserving the organoleptic characteristics of the product that are compromised in traditional heat treatments. However, high pressure changes the thermodynamic equilibrium of chemical reactions. This is the case of lipid oxidation, in which kinetics is accelerated in the presence of high hydrostatic pressure.

In recent years, there has been increasing focus on the response of lipid components to HPP, especially considering the deleterious outcomes that secondary products of oxidation have on the final product. The objective of this work is to review the literature on the effect of this "mild-technology" in the degradation of lipid fraction of foods. We discuss qualitative and quantitative determinations, as well as the thermodynamic and chemical interpretations underlying the phenomenon.

Industrial relevance: In this work we reviewed the literature concerning the effect of high-pressure processing (HPP) on lipid oxidation. Since 1990s HPP has been used as an alternative to thermal treatments to pasteurize and sterilize food products, such as meats and seafood. Many of these raw materials have a high content of lipids (among them trialglycerols and cholesterol-derivative) that are susceptible to oxidation. During the last decade, there has been increasing interest on the response of lipid components to HPP, especially considering the deleterious outcomes that secondary oxidation-derivative molecules have on the final product. This review intends to summarize and discuss the data reported in literature, contextualizing the oxidation within the broad transformation of biological structures due to hydrostatic pressure. A better understanding of the underlying phenomena could lead to the development of predicting models which could be use in food industry.

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1. Introduction

Lipid oxidation is one of the most relevant classes of reaction in the food chemistry. The importance of lipid oxidation is related to biological concerns attributed to oxidation products, and the accumulating evidence that free radicals and reactive oxygen species participate in tissue injury and disease (Frankel, 1991). Studies on the possible pathological significance of lipid oxidation products have developed in three areas of research: lipid peroxides (usually of fatty acids), malondialdehyde, and cholesterol oxidation products (Addis, 1986). Products derived from lipid oxidation are implicated in cancer development (Niki, 2009), disruption of cell membranes (Girotti, 1998; Gutteridge, 1995; Niki, 2009), inactivation of enzymes, and damage of proteins (Gutteridge, 1995). Furthermore, lipid-derived peroxides can act both as modulators of enzymes and as intermediates in biosynthetic processes (Pratt, Tallman, & Porter, 2011).

Lipid oxidation consists of a series of chemical and biochemical reactions which cause changes in the type and concentration of molecular species present in a food (Akoh & Min, 2008; Frankel, 1991). Certain physicochemical characteristics of oxidation products, for example chemical reactivity, dimensions, polarity, interactions, surface activity, partition coefficient, volatility, and thermal stability, can alter the flavor and nutritional quality of foods and produce toxic compounds. As a consequence, lipid oxidation leads to a decrease of food acceptability by consumers (Addis, 1986; Frankel, 1991; Shahidi & Zhong, 2010).

Newly developed food technologies usually focus on preservation while retaining food quality attributes. The expectation is that undesirable micro-organisms and enzymes are inactivated without damage to nutritional and sensory properties resulting normally from thermal treatment. High pressure processing (HPP) is an industrially tested technology that offers a more natural, environmentally friendly alternative for pasteurization or shelf life extension of a wide range of food products (Welti-Chanes et al., 2005). Similar to cold isostatic pressing of metals and ceramics, HPP demands much higher pressures (from 100 to above 800 MPa), faster cycling, high capacity and sanitation (Zimmerman & Bergman, 1993). The effect of high pressure on microorganisms and proteins/enzymes was observed to be similar to that of high temperature. Although it is considered a "mild-technology," the use of HPP in high-fat foods causes a significant increase in oxidative processes. Pressure treatment was found to induce lipid oxidation in food products like turkey thigh muscles (Dissing, Bruun-Jensen, & Skibsted, 1997; Tuboly, Lebovics, Gaál, Mészáros, & Farkas, 2003), chicken breast muscles (Orlien, Hansen, & Skibsted, 2000; Wiggers, Kröger-Ohlsen, & Skibsted, 2004), ham (Andrés, Møller, Adamsen, & Skibsted, 2004; Cava, Ladero, González, Carrasco, & Ramírez, 2009; Clariana & García-Regueiro, 2011), mackerels (Senturk & Alpas, 2012), among others.

This review provides a broad summary of lipid oxidation HPP, considering not only the quantitative aspects, but also the effect of process variables, in order to better understand its impact on food quality and consumer health. To the best of our knowledge, this is the first attempt to organize and discuss the literature on the topic.

2. Free radical autoxidation

2.1. Lipid autoxidation

Lipid peroxidation is the autoxidation of biological lipids, such as fatty acids and steroids. Autoxidation is a free-radical chain process; in the presence of an external source of energy (heat), it is commonly known as thermoxidation. High temperatures (e.g., frying temperatures) have sufficient energy to break covalent C–C or C–H bonds in the acyl backbone to form a variety of lipid alkyl radicals (Frankel, 1991; Schaich, 2005), which then start the radical chains of oxidation. These radicals subsequently react to form lipid oxidation products.

Initiation takes place by loss of a hydrogen radical in the presence of trace materials, light or heat. Production of these radicals is driven catalytically by trace levels of iron, nickel and copper (Schaich, 1992). Metal autoxidation and hydroperoxide decomposition are both very active processes in foods, oils and biological tissues where metals are always present (Schaich, 2005). The resulting carbon-centered alkyl free radicals (R•) react with oxygen to form peroxyl radicals (RO_2•) and other oxygenated compounds. In this propagation process, RO_2• reacts with more RH to from lipid hydroperoxides (RO_2H), which are the fundamental primary products of autoxidation (Frankel, 1984). A general mechanism for oxidation of an unsaturated lipid is shown in Fig. 1.

The primary products of autoxidation are peroxides or hydroperoxides, but these compounds are frequently unstable and undergo scission to form lower molecular-weight secondary oxidation products such as aldehydes, ketones and epoxides (Frankel, 1984; Frankel, 1991; Porter, 2013; Pratt et al., 2011), all of which contribute to flavor and structural deterioration of foods. In fatty acid containing three or more double bonds, the peroxyl radical can react with the additional double bond to form internal or cyclic peroxide (endoperoxide) (Porter, 2013; Schneider, 2009).

Malondialdehyde (MDA) is claimed to be the most important biological breakdown product expected from 5-membered cyclic peroxides of linoleate and linolenate because of its crosslinking ability with amino groups of proteins, enzymes and DNA (Esterbauer, Schaur, & Zollner, 1991). This molecule can be formed in vivo or pre-formed in food products (Addis, 1986) and is rather long lived compared to primary oxidation products. Thiobarbituric acid reactive substances (TBARS) assay is the most frequently used method for the quantification of MDA in foods and biological fluids and tissues (Addis, 1986). Treatment of biological and food samples with TBA under appropriate conditions results in the formation of pink-colored products, which absorb in the 500–550 nm range. However, TBARS has been discussed because MDA-like substances can also be formed during the assay from various precursors produced during lipid peroxidation such as oxidized lipids, 2-alkenals, 2,4-alkadienals, 4-hydroxyalkenals or MDA bound to proteins (Addis, 1986; Esterbauer et al., 1991; Frankel & Neff, 1983).

2.2. Cholesterol autoxidation

As with other lipid molecules, cholesterol is susceptible to oxidation to form cholesterol oxidation products (COPs) or oxysterols by heating (Hur, Park, & Joo, 2007; Medina-Meza, Rodríguez-Estrada, Lercker, Soto-Rodríguez, & García, 2011; Smith, 1987), illumination (Medina-Meza, Rodríguez-Estrada, García, & Lercker, 2012) and enzymatic activity (Björkhem, 2009). COPs are derivatives of cholesterol that contain a second oxygen atom as part of a carbonyl, hydroxyl, ketone or epoxide group on the sterol nucleus and/or a hydroxyl group on the side chain of the molecule. Oxysterols usually occur at low levels, accompanied by a high concentration of the parent cholesterol. Accumulation of oxysterols in the body can occur in several different ways, the major ones being dietary intake and internal chemical and enzymatic oxidation (Ryan, O'Callaghan, & O'Brien, 2005). Oxysterols are present in various foodstuffs, notably cholesterol-rich foods such as dairy products, milk, eggs, dried egg powder, meat products, and dried or stored fish (Hur et al., 2007; Saldanha & Bragagnolo, 2010).

Free radical-mediated oxidation of cholesterol gives 7-hydroperoxycholesterol isomers (7αβ-OOH), 7-hydroxycholesterol isomers (7αβ-OH), 7-ketocholesterol (7-keto), cholesterol-5,6-epoxide isomers (5,6αβ-epoxy), cholestane-3β,5α,6β-triol (triol), and 25-hydroxycholesterol (25-OH) as major products (Fig. 2). Singlet oxygen oxidizes cholesterol to give 5αβ-OOH isomers as a major primary product (Medina-Meza et al., 2011). These compounds have been shown to exert several in vitro and in vivo biochemical activities of both physiologic and pathologic relevance (Poli, Sottero, Gargiulo, & Leonarduzzi, 2009; Ryan et al., 2005), which are mediated by their biophysical effects on membranes and/or stereospecific