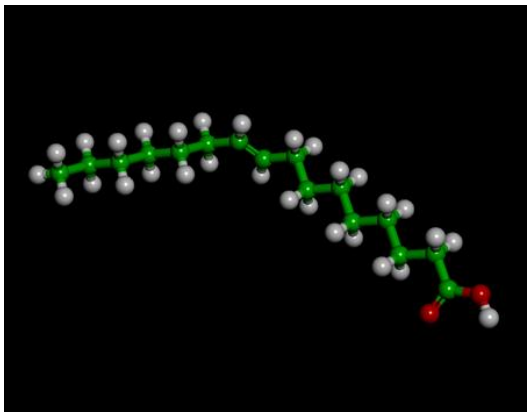
Cancer Association of South Africa (CANSA)

Research • Educate • Support

Fact Sheet
on
Palmitoleic Acid (Omega-7)

Introduction

Most people are aware of the following polyunsaturated fatty acids (PUFAS), namely Omega-3, Omega-6, Omega-9 and Omega-12. Most people are also aware of the wide-ranging benefits of Omega-3. There is, however, another category of fatty acid called Omega-7 in which the site of unsaturation is seven carbon atoms from the end of the carbon chain. The two most common omega-7 fatty acids in nature are palmitoleic acid and vaccenic acid. Whereas Omega-3, -6, -9, and -12 are polyunsaturated fatty acids (PUFAS), Omega-7 (Palmitoleic Acid) is a monounsaturated fatty acid (MUFA).



[Picture Credit: Palmitoleic Acid]

Palmitoleic acid (Omega-7) can be abbreviated as $16{:}1\Delta^{9}$. Dietary sources of palmitoleic acid include a variety of animal oils, vegetable oils, and marine oils. Macadamia oil (*Macadamia integrifolia*) and sea buckthorn oil (*Hippophaë rhamnoides*) are botanical sources with high concentrations, containing 17% and 19% (minimum) to 29% (maximum) of palmitoleic acid, respectively.

Palmitoleic acid has the formula $CH_3(CH_2)_5CH{=}CH(CH_2)_7COOH$.

Souza, C.O., Teixeira, A.A.S., Biondo, L.A., Silveira, L.S., de Souza Breda, C.N., Braga, T.T., Camara, N.O.S., Belchior, T., Festuccia, W.T., Diniz, T.A., Ferreira, G.M., Hirata, M.H., Chaves-Filho, A.B., Yoshinaga, M.Y., Miyamoto, S., Calder, P.C., Sethi, J.K. & Rosa Neto, J.C. 2020.

"Palmitoleic acid (POA, 16:1n-7) is a lipokine that has potential nutraceutical use to treat non-alcoholic fatty liver disease. We tested the effects of POA supplementation (daily oral gavage, 300 mg/Kg, 15 days) on murine liver inflammation induced by a high fat diet (HFD, 59% fat, 12 weeks). In HFD-fed mice, POA supplementation reduced serum insulin and improved insulin tolerance compared with oleic acid (OA, 300 mg/Kg). The livers of POA-treated mice exhibited less steatosis and inflammation than those of OA-treated mice with lower inflammatory cytokine levels and reduced toll-like receptor 4 protein content. The anti-inflammatory effects of POA in the liver were accompanied by a reduction in liver macrophages (LM, $CD11c^{+}$; $F4/80^{+}$; $CD86^{+}$), an effect that could be triggered by peroxisome proliferator activated receptor (PPAR)-γ, a lipogenic transcription factor upregulated in livers of POA-treated mice. We also used HFD-fed mice with selective deletion of

Researched and Authored by Prof Michael C Herbst
[D Litt et Phil (Health Studies); D N Ed; M Art et Scien; B A Cur; Dip Occupational Health; Dip Genetic Counselling; Diagnostic Radiographer; Dip Audiometry and Noise Measurement; Medical Ethicist]
Approved by Ms Elize Joubert, Chief Executive Officer [BA Social Work (cum laude); MA Social Work]

PPAR-γ in myeloid cells (PPAR-γ $KO^{LyzCre+}$) to test whether the beneficial anti-inflammatory effects of POA are dependent on macrophages PPAR-γ. POA-mediated improvement of insulin tolerance was tightly dependent on myeloid PPAR-γ, while POA anti-inflammatory actions including the reduction in liver inflammatory cytokines were preserved in mice bearing myeloid cells deficient in PPAR-γ. This overlapped with increased $CD206^+$ (M2a) cells and downregulation of $CD86^+$ and $CD11c^+$ liver macrophages. Moreover, POA supplementation increased hepatic AMPK activity and decreased expression of the fatty acid binding scavenger receptor, CD36. We conclude that POA controls liver inflammation triggered by fat accumulation through induction of M2a macrophages independently of myeloid cell PPAR-γ."

Good Sources of Palmitoleic Acid

A good source of Palmitoleic Acid is obtained from the oil of the Macadamia plant (*Macadamia integrifolia)*. Macadamias are large, spreading evergreen trees reaching 10 to 15 metres high and almost as wide. Macadamias are considered to be among the finest table nuts in the world. It contains high quantities of oil, and are, therefore, very fattening.

[Picture Credit: Macadamia]

Another excellent source of Palmitoleic Acid is obtained from oil from the seeds of the Sea Buckthorn plant.

[Picture Credit: Sea Buckthorn]

Sea Buckthorn (*Hipophaë rhamnoides*) is an arborescent armed, deciduous shrub or tree sometimes reaching up to18 metres. Its crown is irregular in shape with spiny, grey branches. The fruit is edible and has a tart, bittersweet taste. Its fruit is rich in Vitamins C, E, K, B_1 and B_2, as well as niacinamide, pantothenic acid, carotenoids and other substances such as oil, sugar, malic acid, amino acids and pectin.

The plant is considered a general panacea (a solution or remedy for all difficulties or diseases) and extensive use is made of its roots, stems, leaves, flowers, fruits and seed. Oil from the fruit acts as an antioxidant and is traditionally used to treat wounds, frost bite and pathological problems of the alimentary mucous membranes. Serotonin (5-hydroxy-tryptamine) extracted from sea buckthorn possesses anti-tumour capabilities.

Omega-7 is quoted to be high in *palmitoleic acid* (not present in Omega-3, -6 or -9) which is effective against a range of life-threatening disorders – including cancer. Omega-7, in its natural source (i.e. macadamia nuts and sea buckthorn), is quoted to be a double-edged sword as it also contains high levels of *palmitic acid. Palmitic acid is a thick, gooey palm oil*, which in turn raises the risk of certain life-threatening disorders. It is essential when acquiring Omega-7 (palmitoleic acid) to ascertain that it has been 'purified' of palmitic acid.

Palmitoleic Acid (Omega-7) Fights the Factors of Metabolic Syndrome
Metabolic syndrome is a major contributor to the following:

- Elevated glucose and insulin resistance
- Lipid disturbances [causing high triglycerides and low High Density Lipoprotein (HDL)]
- High blood pressure
- Central obesity ('apple shape') – a well-known contributing factor to the increase in the risk of certain cancers like cancer of the prostate, kidney, breast, ovaries, colon, pancreas, cervix, thyroid and endometrium
- Chronic Inflammation which is also known to increase the risk for certain cancers

If one has metabolic syndrome, it means that one is possibly already along the road to heart disease, diabetes, certain cancers and other life-threatening disorders.

González-Becerra, K., Ramos-Lopez, O., Barrón-Cabrera, E., Riezu-Boj, J.I., Milagro, F.I., Martínez-López, E. & Martínez, J.A. 2019.
Background: Chronic illnesses like obesity, type 2 diabetes (T2D) and cardiovascular diseases, are worldwide major causes of morbidity and mortality. These pathological conditions involve interactions between environmental, genetic, and epigenetic factors. Recent advances in nutriepigenomics are contributing to clarify the role of some nutritional factors, including dietary fatty acids in gene expression regulation. This systematic review assesses currently available information concerning the role of the different fatty acids on epigenetic mechanisms that affect the development of chronic diseases or induce protective effects on metabolic alterations.
Methods: A targeted search was conducted in the PubMed/Medline databases using the keywords "fatty acids and epigenetic". The data were analyzed according to the PRISMA-P guidelines.
Results: Consumption fatty acids like n-3 PUFA: EPA and DHA, and MUFA: oleic and palmitoleic acid was associated with an improvement of metabolic alterations. On the other hand, fatty acids that have been associated with the presence or development of obesity, T2D, pro-inflammatory profile, atherosclerosis and IR were n-6 PUFA, saturated fatty acids (stearic and palmitic), and trans fatty acids (elaidic), have been also linked with epigenetic changes.
Conclusions: Fatty acids can regulate gene expression by modifying epigenetic mechanisms and consequently result in positive or negative impacts on metabolic outcomes.
Omega-7 works in five distinct ways to reduce most of metabolic syndrome's harmful effects on one's health:

- It reduces insulin resistance and lowers blood glucose
- It suppresses fat production and accumulation
- It normalises abnormal lipid profiles (including raising beneficial HDL-cholesterol)
- It fights obesity
- It powerfully suppresses the inflammation that drives metabolic syndrome

The following table shows how different drugs compare to Omega-7 in fighting metabolic syndrome:

Metabolic Syndrome Parameter	Statins (Lipitor and others)	Fibrates (Lopid and others)	Glitzones (Actos and others)	Sulfonylureas (Glipizide and others)	Palmitoleic Acid (Omega-7)
LDL ('bad' cholesterol)	Reduce	Reduce	Increase	No effect	Reduce
HDL ('good' cholesterol)	Little effect May decrease	Increase	Increase	Decrease	Increase
Blood sugar	May increase	No effect	Reduce	Reduce Increases insulin	Reduce
Insulin Resistance	May worsen	No effect	Reduce	May improve	Reduce
Body weight/ Composition	Increase weight Decrease fat-free mass	Mazy increase weight and fat mass	Decrease fat	Increase	Reduce appetite
Inflammation	May reduce	May reduce	Reduce	No effect	Reduce
Side effects	Muscle pain (myalgia), may increase risk of diabetes	Gallstones, muscle pain	May increase risk of cardiovascular death	Increased risk of cardiovascular death	None known

(Yang, Miyahara & Hatnaka, 2011; Stefan, et al., 2010; Experimental Animal Laboratory, 2008; Green, 2012; Martinez, 2013).

Acosta-Montaño, P., Rodríguez-Velázquez, E., Ibarra-López, E., Frayde-Gómez, H., Mas-Oliva, J., Delgado-Coello, B., Rivero, I.A., Alatorre-Meda, M., Aguilera, J., Guevara-Olaya L, & García-González, V. 2019.

"Metabolic overload by saturated fatty acids (SFA), which comprises β-cell function, and impaired glucose-stimulated insulin secretion are frequently observed in patients suffering from obesity and type 2 diabetes mellitus. The increase of intracellular Ca^{2+} triggers insulin granule release, therefore several mechanisms regulate Ca^{2+} efflux within the β-cells, among others, the plasma membrane Ca^{2+}-ATPase (PMCA). In this work, we describe that lipotoxicity mediated mainly by the saturated palmitic acid (PA) (16C) is associated with loss of protein homeostasis (proteostasis) and potentially cell viability, a phenomenon that was induced to a lesser extent by stearic (18C), myristic (14C) and lauric (12C) acids. PA was localized on endoplasmic reticulum, activating arms of the unfolded protein response (UPR), as also promoted by lipopolysaccharides (LPS)-endotoxins. In particular, our findings demonstrate an alteration in PMCA1/4 expression caused by PA and LPS which trigger the UPR, affecting not only insulin release and contributing to β-cell mass reduction, but also increasing reactive nitrogen species. Nonetheless, stearic acid (SA) did not show these effects. Remarkably, the proteolytic degradation of PMCA1/4 prompted by PA and LPS was avoided by the action of monounsaturated fatty acids such as oleic and palmitoleic acid. Oleic acid recovered cell viability after treatment with PA/LPS and, more interestingly, relieved endoplasmic reticulum (ER) stress. While palmitoleic acid improved the insulin release, this fatty acid seems to have more relevant effects upon the expression of regulatory pumps of intracellular Ca^{2+}. Therefore, chain length and unsaturation of fatty acids are determinant cues in proteostasis of β-cells and, consequently, on the regulation of calcium and insulin secretion."

Palmitoleic Acid (Omega-7) Fights Inflammation

There is a close connection between fat tissue and the chronic, low-grade inflammation that is associated with metabolic syndrome. The connection may be related to an enzyme known as SCD1 (stearoyl-CoA desaturase 1).

When scientists remove SCD1 activity in laboratory animals, their levels of fat tissue inflammation fall sharply, and their ability to respond to insulin (insulin sensitivity) rises. In the laboratory, adding omega-7 to cultures of fat cells triggers these same benefits by suppressing SCD1 activity.

Animal studies show significantly reduced levels of fat-related inflammatory cytokines (signalling molecules) following administration of omega-7. The livers of supplemented animals show significant reductions in the number of activated inflammatory cells, an effect that may help prevent fatty liver disease. Many of these beneficial anti-inflammatory effects may arise from the ability of omega-7 to deactivate the master inflammatory regulation complex called *NF-kappaB*.

There is now human data on how omega-7 can lower inflammation and reduce the resulting cardiovascular risk. In a pilot trial of adults with high levels of *C-reactive protein* (blood marker for inflammation), supplementation with 210mg a day of *omega-7* resulted in a robust 73% decrease of *C-reactive protein (CRP)*.

Those results were extended in a larger, randomised clinical trial, in which all patients had abnormally high CRP levels (greater than 3 mg/dL). In this study, 30 days of supplementation with 210 mg/day of palmitoleic acid resulted in a significant drop in CRP of 1.9 mg/dL – that is a 43% reduction in a dangerous cardiovascular risk marker. Moreover, by the end of the supplementation period, the average CRP level was reduced from greater than 4 mg/dL to 2.1 mg/dL. The health ramifications of this marked reduction in C-reactive protein are profound, especially in abdominally-obese individuals who often exhibit dangerously elevated levels of this inflammatory indicator (CRP). (NHLBI/AHA Conference Proceedings; Festa, *et al*., 2000; Shah, *et al*., 2008; Liu, *et al*., 2010; Yand, Miyahara & Hatanaka, 2011; Guo, *et al*., 2012; Green, 2012; Martinez, 2013).

De Souza, C.O., Vannice, G.K., Rosa Neto, J.C. & Calder, P.C. 2018.

"Although dietary fatty acids can modulate metabolic and immune responses, the effects of palmitoleic acid (16:1n-7) remain unclear. Since this monounsaturated fatty acid is described as a lipokine, studies with cell culture and rodent models have suggested it enhances whole body insulin sensitivity, stimulates insulin secretion by β cells, increases hepatic fatty acid oxidation, improves the blood lipid profile, and alters macrophage differentiation. However, human studies report elevated blood levels of palmitoleic acid in people with obesity and metabolic syndrome. These findings might be reflection of the level or activity of stearoyl-CoA desaturase-1, which synthesizes palmitoleate and is enhanced in liver and adipose tissue of obese patients. The aim of this review is to describe the immune-metabolic effects of palmitoleic acid observed in cell culture, animal models, and humans to answer the question of whether palmitoleic acid is a plausible nonpharmacological strategy to prevent, control, or ameliorate chronic metabolic and inflammatory disorders. Despite the beneficial effects observed in cell culture and in animal studies, there are insufficient human intervention studies to fully understand the physiological effects of palmitoleic acid. Therefore, more human-based research is needed to identify whether palmitoleic acid meets the promising therapeutic potential suggested by the preclinical research."

Palmitoleic Acid (Omega-7) Helps Manage Body Weight

The reason central or abdominal obesity ('apple shape') is a factor in metabolic syndrome is because it has such strong associations with certain cancers and cardiovascular disease risk. This is due, in large part, to the increased inflammation produced by fat tissue.

Omega-7 helps manage this factor of metabolic syndrome because it signals one's body to stop storing fat.

Animals fed diets rich in omega-7 show significant increases in stomach and intestinal hormones that promote the feeling of fullness (satiety). At the same time, such diets produce decreases in hunger-promoting hormones. The combined effect is a significant reduction in food intake.

Several statin drugs, while lowering cholesterol and triglycerides, also produce increases in body and liver fat deposition. Omega-7 does just the opposite. Omega-7 **reduces** the production of fat in the liver. Increases in liver fat can result in non-alcoholic fatty liver disease (NAFLD), which is considered a major manifestation of the metabolic syndrome - which can eventually lead to liver failure and even cancer.
(Elbassuoni, 2013; Festa, *et al*. 2000; Shah, Mehta & Reilly, 2012; Lu*, et al*., 2012; Yang, Takeo, & Katayama, 2013; Aguirre, *et al*., 2013; Pappachan, 2013; NHLBI/AHA Conference Proceedings; Burns, *et al*., 2012).

Adverse Effects of Palmitoleic Acid (Omega-7)

No significant adverse effects have been reported for Omega-7 fatty acids.
(Yang & Kallio, 2002; Yang, Kalimo, Marrila, *et al*., 1999; Farma Nord).

Medical Disclaimer

This Fact Sheet is intended to provide general information only and, as such, should not be considered as a substitute for advice, medically or otherwise, covering any specific situation. Users should seek appropriate advice before taking or refraining from taking any action in reliance on any information contained in this Fact Sheet. So far as permissible by law, the Cancer Association of South Africa (CANSA) does not accept any liability to any person (or his/her dependants/ estate/heirs) relating to the use of any information contained in this Fact Sheet.

Whilst the Cancer Association of South Africa (CANSA) has taken every precaution in compiling this Fact Sheet, neither it, nor any contributor(s) to this Fact Sheet can be held responsible for any action (or the lack thereof) taken by any person or organisation wherever they shall be based, as a result, direct or otherwise, of information contained in, or accessed through, this Fact Sheet.

Sources and References Consulted or Utilised

Acosta-Montaño, P., Rodríguez-Velázquez, E., Ibarra-López, E., Frayde-Gómez, H., Mas-Oliva, J., Delgado-Coello, B., Rivero, I.A., Alatorre-Meda, M., Aguilera, J., Guevara-Olaya L, & García-González, V. 2019. Fatty acid and lipopolysaccharide effect on beta cells proteostasis and its impact on insulin secretion. *Cells*. 2019 Aug 13;8(8):884. doi: 10.3390/cells8080884.

Aguirre L, Hijona E, Macarulla MT, *et al*. 2013. Several statins increase body and liver fat accumulation in a model of metabolic syndrome. *J Physiol Pharmacol.* 2013 Jun;64(3):281-8.

American Cancer Society
http://www.cancer.org/acs/groups/cid/documents/webcontent/003047-pdf.pdf

American Institute for Cancer Research
http://www.aicr.org/enews/2012/october-2012/enews-1-in-5-pancreatic-cancers-preventable.html

Aspet
http://jpet.aspetjournals.org/content/327/2/316.full

Burns, T.A., Duckett, S.K., Pratt, S.L., & Jenkins, T.C. 2012. Supplemental palmitoleic (C16:1 cis-9) acid reduces lipogenesis and desaturation in bovine adipocyte cultures. *J Anim Sci*. Oct; 90(10):3433-41.

Cancer Research UK
http://www.cancerresearchuk.org/about-cancer/type/pancreatic-cancer/about/pancreatic-cancer-risks-and-causes

De Souza, C.O., Vannice, G.K., Rosa Neto, J.C. & Calder, P.C. 2018. Is Palmitoleic Acid a Plausible Nonpharmacological Strategy to Prevent or Control Chronic Metabolic and Inflammatory Disorders? *Mol Nutr Food Res*. 2018 Jan;62(1). doi: 10.1002/mnfr.201700504. Epub 2017 Dec 11.

De Souza, C.O., Teixeira, A.A.S., Biondo, L.A., Lima Junior, E.A., Batatinha, H.A.P. & Rosa Neto, J.C. 2017. Palmitoleic Acid Improves Metabolic Functions in Fatty Liver by PPARα-Dependent AMPK Activation. *J Cell Physiol*. 2017 Aug;232(8):2168-2177. doi: 10.1002/jcp.25715. Epub 2017 Mar 24.

Elbassuoni, E. 2013. Better association of waist circumference with insulin resistance and some cardiovascular risk factors than body mass index. *Endocr Regul*. 2013 Jan;47(1):3-14.

Experimental Animal Laboratory. 2008. Final report for study on CCO Technologies Oil (CCO-Oil) on the development of atherosclerosis: Department of Cardiovascular Medicine, Cleveland Clinic; 2008.

Farma Nord
http://www.pharmanordnutrients.co.uk/showarticle.asp?aid=94&m=az&al=S

Festa, A., D'Agostino, R. Jr., Howard, G., Mykkänen, L., Tracy, R.P., & Haffner, S.M. 2000. Chronic subclinical inflammation as part of the insulin resistance syndrome: the Insulin Resistance Atherosclerosis Study (IRAS). *Circulation*. 2000 Jul 4;102(1):42-7.

Frigolet, M.E. & Gutiérrez-Aquilar, R. 2017. The Role of the Novel Lipokine Palmitoleic Acid in Health and Disease. *Adv Nutr*. 2017 Jan 17;8(1):173S-181S. doi: 10.3945/an.115.011130. Print 2017 Jan.

González-Becerra, K., Ramos-Lopez, O., Barrón-Cabrera, E., Riezu-Boj, J.I., Milagro, F.I., Martínez-López, E. & Martínez, J.A. 2019. Fatty acids, epigenetic mechanisms and chronic diseases: a systematic review. *Lipids Health Dis*. 2019 Oct 15;18(1):178. doi: 10.1186/s12944-019-1120-6.

Green, J.A. 2012. Effect of two levels of Provinal™ (purified Palmitoleic Acid; C16:1n7; Omega 7) on serum lipid and C-reactive protein(CRP) profiles in humans. Tersus Pharmaceuticals, LLC: 2012.

Guo, X., Li, H., Xu, H., *et al*. 2012. Palmitoleate induces hepatic steatosis but suppresses liver inflammatory response in mice. *PLoS One.* 2012;7(6):e39286.

Lu, X., *et al.* 2012. Postprandial inhibition of gastric ghrelin secretion by long-chain fatty acid through GPR120 in isolated gastric ghrelin cells and mice. *Am J Physiol Gastrointest Liver Physiol.* 2012 Aug 1;303(3):G367-76.

Macadamia
http://ntbg.org/plants/plant_details.php?plantid=7260

Martinez, L. 2013. Provinal (R) in the reduction of CRP: A double blinded, randomized, placebo controlled study. *Provinal purified omega 7.* Vol: Tersus Pharmaceuticals; 2013.

MD Anderson Cancer Center
http://www.mdanderson.org/patient-and-cancer-information/cancer-information/cancer-topics/prevention-and-screening/exercise/flatabs.html

NHLBI/AHA Conference Proceedings
Available at: http://circ.ahajournals.org/content/109/3/433.full.

Omega-7
http://www.amazon.com/Purified-InnovixLabs-Macadamia-Palmitoleic-Capsules/dp/B00GRAAMLY

Palmitoleic Acid
http://www.worldofmolecules.com/foods/palmitoleic_acid.htm

Pappachan, J.M., Antonio, F.A., Edavalath, M., & Mukherjee, A. 2013. Non-alcoholic fatty liver disease: a diabetologist's perspective. *Endocrine.* 2013 Nov 28.

Princeton Longevity Center
http://www.theplc.net/Preventing_Prostate_Cancer.html

Sea Buckthorn
http://newsino.en.hisupplier.com/product-1128444-Hippophae-rhamnoides-Seed-Oil.html

Shah A, Mehta N, Reilly MP. 2008. Adipose inflammation, insulin resistance, and cardiovascular disease. *JPEN J Parenter Enteral Nutr*. 2008 Nov-Dec;32(6):638-44.

Souza, C.O., Teixeira, A.A.S., Biondo, L.A., Silveira, L.S., de Souza Breda, C.N., Braga, T.T., Camara, N.O.S., Belchior, T., Festuccia, W.T., Diniz, T.A., Ferreira, G.M., Hirata, M.H., Chaves-Filho, A.B., Yoshinaga, M.Y., Miyamoto, S., Calder, P.C., Sethi, J.K. & Rosa Neto, J.C. 2020. Palmitoleic acid reduces high fat diet-induced liver inflammation by promoting PPAR-γ-independent M2a polarization of myeloid cells. *Biochim Biophys Acta Mol Cell Biol Lipids*. 2020 Oct;1865(10):158776.

Stefan, N., *et al.* 2010. Circulating palmitoleate strongly and independently predicts insulin sensitivity in humans. *Diabetes Care.* 2010 Feb;33(2):405-7.

WebMD
http://www.webmd.com/ovarian-cancer/ss/slideshow-ovarian-cancer-overview

Weimann, E., Silva, M.B.B., Murata, G.M., Bortolon, J.R., Dermargos, A., Curi, R. & Hatanaka, E. 2018. PLoS One. Topical anti-inflammatory activity of palmitoleic acid improves wound healing. 2018 Oct 11;13(10):e0205338. doi: 10.1371/journal.pone.0205338. eCollection 2018.

Wikipedia
http://en.wikipedia.org/wiki/Omega-7_fatty_acid
http://en.wikipedia.org/wiki/Palmitoleic_acid

Worldagroforestry
http://www.worldagroforestry.org/treedb/AFTPDFS/Hippophae_rhamnoides.pdf

Yang, Z.H., Pryor, M., Noguchi, A., Sampson, M., Johnson, B., Pryor, M., Donkor, K., Amar, M. & Remaley, A.T. 2019. *Mol Nutr Food Res.* 2019 Jun;63(12):e1900120. doi: 10.1002/mnfr.201900120. Epub 2019 Apr 10.PMID: 30921498

Yang, Z.H., Miyahara, H, & Hatanaka, A. 2011. Chronic administration of palmitoleic acid reduces insulin resistance and hepatic lipid accumulation in KK-Ay Mice with genetic type 2 diabetes. *Lipids Health Dis.* 2011;10:120.

Yang ZH, Takeo J, Katayama M. 2013. Oral administration of omega-7 palmitoleic acid induces satiety and the release of appetite-related hormones in male rats. *Appetite.* 2013 Jun;65:1-7.

Yang, B., Kallio, H. 2002 Composition and physiological effects of sea buckthorn lipids. *Trends in Food Sci Technol*; 13: 160-167

Yang, B., Kalimo, K.O., Mattila, L.M., et al. 1999. Effects of dietary supplementation with sea buckthorn seed and pulp oils on atopic dermatitis. *J Nutr Biochem*; 10: 622-630