Among The Plant Seeds, Oil Producing Seeds Can Be A Promising Source Of Lead Compounds For The Palliative Treatment Of Non-Communicable Diseases: A Review

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Abstract: Once, infectious diseases were major cause of death. Discovery and application of vaccines as well as bioactive natural compounds from fungus, plants and other natural sources put spectacular contribution in victory against infection. Currently, non-communicable diseases, which can be called metabolic disorder related disease, are at the top of the top ten causes of death. It is anticipated that the trend will continue in the forth coming decades. Unlike the infectious diseases, every non-communicable disease is a collection of disorders in the physiological processes. It takes long time to reach at the phase of noticeable clinical symptom of a non-communicable disease. With the expunction of knowledge, it is becoming clearer that disorder of fat metabolism is at the center of the most non-communicable diseases. Food behavior, foods' oil chemistry (in terms of unsaturated fatty acid, unsaponifiable matter of oil), chronic infection or toxicity, and life style put corresponding role in initiation and complication of non-communicable diseases. So, the best ways to keep away of non-communicable diseases are to go behind safe and sound etiquette regarding food behavior and life style through ought the life.

Keywords: Non-communicable disease, Poly unsaturated fatty acids, Obesity, Infectious diseases

I. INTRODUCTION

In ancient time people suffered not only from shortage of food but also from infectious diseases (Earla, 2014). With the agricultural and industrial revolutions the deficiency of food was conquer but infection remained as major problem for human mortality and morbidity even after 1900s. The global drug and chemical innovation that took place in 1940s put profound contribution for improving the life-expectancy from 46.7 Y to 61.13 Y by 1980 (Mishra, 2016). As the major struggle against infectious disease is over other than the problem of resistant organisms and opportunistic infections, at this moment the main challenge ahead of medical science is non-communicable disease are disorders in the different biochemical pathways in physiological system. An interesting similarity between plant and animal kingdom is energy source. Seeds utilize stored carbohydrates, fats and proteins as energy source at the time of germination where as human beings and lot of other animals use it throughout the life. The main objective of the article is shed light on the view that seed derived small molecule and various fatty acids can be a promising source of lead molecules against noncommunicable diseases as well as the ideals that food behavior and life style plays decisive role in incidence and progression of the non-communicable diseases.

II. CHANGEOVER OF LEADING CAUSE OF DEATH: INFECTIOUS TO NON-COMMUNICABLE DISEASE

Although all infections are not equally contagious, the diseases, once known as contagious or communicable are now known as infectious diseases (Nelson and Willams, 2014). Long after the invention of microscope (1683), germ theory was established by Pasteur in 1857; subsequently, causative microorganisms of different types of infectious diseases were discovered (leprosy: 1847, Cholera: 1883, Plagues: 1894). Later on success in discovery of vaccines, growing public awareness, and implementation of rules and policies of public health cut down the death toll to one sixteenth (1/16) from the previous century (Armstrong et al., 2009). As civilization forwarded the expunction of business and urbanization got new pace. So, the risk of infection prevails. Yet to 1940, infectious diseases remained as the major cause of death (Gordon, 1953). Although penicillin was discovered in 1928, its full-fledged success story started in 1942 with its successful application in the treatment of a woman named Anne Miller, and others (Bennett and Chung, 2001). By 1960, approximately all the major practical evils for dealing with infectious disease had been deciphered (Brachman, 2003). Currently, non-communicable diseases (Ischemic Heart Disease, Stroke, hundreds of different types cancers, diabetes etc.) are major cause of death particularly in developed and developing countries (WHO, 2005; Anderson & Chu, 2007). Surprisingly, in some way all most all of the noncommunicable diseases are linked with disorder or deregulation of metabolic path ways.

III. FAT'S UNSATURATED BOND AND METABOLIC DISORDER

The total amount of stored triacylglycerol in adult human weighing 70kg is around 15 kg (Snyder, 1975), and glycogen is around 0.5kg (Wasserman, 2009). Although the endogenous triacylglycerols is the main fuel reserve in our body (Horowitz and Klein, 2000) and acts as the central pool of supplying the necessary raw materials of cell membrane, Mammals lack the enzymes to introduce double bonds at carbon atoms beyond C9 (Bagchi and Khurana, 2013). Hence, all fatty acids containing a double bond at positions beyond C9 have to be supp lied in the diet. These are called Essential fatty acids (EFA). Other unsaturated fatty acids such as arachidonic acid $(20:4\Delta 5,8,11,14)$ are derived from these EFA. Eicosanoids, which are broadly classified as prostaglandins, thromaoxanes, and leukotrienes (Henderson, 1994) play pivotal role in numerous biochemical pathways of inflammation and immune system. That's why change in the ratio of saturated and unsaturated fat or that of different types of unsaturated fats causes a lot of metabolic disorders leading to chronic diseases or illness.

IV. OBESITY AND DIABETES

Progression of age, lack of exercise and obesity are primary risk factors for insulin resistance and it leads to type-2

diabetes (Colditz et al., 1990; Helmrich et al., 1991). Particularly abdominal obesity is a risk factor for type-2 diabetes (Vessby, 2000). Ingestion of foods with a high proportion of fat raises the risk of obesity (Astrup et al., 1997; West and York, 1998), and throughout the world over a billion of adults are thought to be obese people (Gnacińska et al., 2009) There are evidences that support the notion that FFAs are important regulators of glucose metabolism and elevated FFAs are associated with insulin resistance (Boden, 1999). The adipocyte is the site of synthesis of particular family of proteins (TNF- α , IL-1 β , IL-6, IL-8, IL-10) involved in inflammation (Hotamisligil, 2010). Out of them IL-6 expression in adipose tissue and serum concentration are positively correlated with both obesity and insulin resistance. Surprisingly, about 30 % of circulating IL-6 comes from adipose tissue and (Kershaw, 2004).

V. ATHEROSCLEROSIS, FATTY ACID COMPOSITION AND INFLAMMATION

In atherosclerosis, plaque made up of fat, cholesterol, calcium, and other substances found in the blood blocks the blood flow and oxygen supply proportionately the to the tissues reliant to it. Depilation of oxygen supply in tissue creates hypoxic condition. There are reports that hypoxia incites inflammation (Eltzschig et al., 2011) and both share similar basic mechanisms involving the adhesion of leukocytes to the vascular endothelium in their early phases (Gnacińska et al., 2009). The endothelium is a highly metabolically active organ that is involved in many physiological processes (Aird, 2005) in inflammatory situation different types of poly unsaturated fatty acids (PUFA) and their enzymatic take part in the process to normalization or healing process of the tissue. So, the presence of different types of PUFA like ALA, LA, AA, n-3 and n6 fatty acid is vital for the maintenance good health of different tissues and cardiac muscle.

VI. FAT METABOLISM AND CANCER

Including eiconasanoid metabolism, cellular proliferation, alteration of gene expression, and numerous mechanisms for the carcinogenic effect of dietary fat have been anticipated (Birt et al., 1999). There are reports of relationship between animal fat or monounsaturated fatty acid intake and the risk of advanced prostate cancer (Giovannucci et al., 1993). Observational studies suggest strong correlations among dietary composition and incidence and mortality rates of cancer (Armstrong and Doll, 1975). Whereas epidemiological studies have shown that fish consumption reduces the risk of breast and colorectal cancer (Caygill et al., 1996; Sasaki et al., 1993). Increased intakes of energy, total fat, n -6 polyunsaturated fatty acids, cholesterol, sugars, protein, and some amino acids have been thought to increase the risk of various cancers, whereas intakes of n-3 fatty acids, dietary fiber, and physical activity are thought to be protective against cancer. Some n-3 fatty acids, like- docosahexaenoic acid and eicosapentaenoic acid, have been shown to restrain neoplastic

alteration (Takahashi et al., 1992), slow down cell growth and propagation (Grammatikos et al., 1994), induce apoptosis (Calviello et al., 1998), and hinder angiogenesis (Rose and Connolly, 2000), which suppose to happen by suppressing n-6 fatty acid eicosanoid production.

VII. SECONDARY METABOLITES ARE SPECIAL MOLECULES OF PLANT

Difference between secondary and primary metabolite is hedging. The metabolites that are not directly involved in normal growth, development, and reproduction are called secondary metabolites (Anulika et al., 2016). Surprisingly, most of the secondary metabolites have biological functions (Wink, 1988). To date, structures of about 100, 000 different types of plant derived natural compounds have been established (Ribera and Zuñiga, 2012), and majority of API available in pharmaceutical marked are either natural compounds or inspired by them (Jesse and Vederas, 2009). The major families of secondary metabolites are alkaloids, Glycosides, Flavonoids, and Lignans. Lignan is dimar of monolignol whereas lignin, that constitutes about 30% body mass of higher order plants, is polymer of mono-lignols. There are reports that some fatty acids modulate gene expression (Schlotz et al., 2012). So, Seconadry metabolites can be thought of as special molecules that are species specific, tissue specific, environment specific or even cell specific (Yazdani et al., 2011), and they have profound effect in the life cycle of the organism.

VIII. SEEDS CAN BE PROMISING SOURCE OF LEAD COMPOUND AGAINST NON-COMMUNICABLE DISEASE

In an adult plant main biomass is Cellulose and lignin (Sun and Cheng, 2002), and the stored nutrient in the seed or fruits are proteins starch and fats (Duranti 2006; Wojciech 2009; Singh). In human or mammals, main biomass is proteins and fats, and vast of it comes as staples like cereals, pulse, vegetable oils of plant origin. In case of plants, cells are not only stationary (Lloyd, 1994) it creates an outer lignocellulosic wall around the cell membrane. So, cell to cell signal is transmitted mainly by hormones or smaller molecules which are synthesized inside the cells, and subsequently secreted and transported outside to act upon other cells. In lipids of seeds, there are some secondary metabolites that put profound effect on the metabolic path way of lipid, and have influence on cell differentiation, proliferation, induction of apoptosis. The smaller signaling molecule that controls the biosynthesis and metabolic steps can be promising source of novel molecule of metabolic disorder related disease like CVD, diabetes, different types of cancers. In some cases fatty acids acts as cell signaling molecule and modify expression of specific genes involved in lipid and carbohydrate metabolism (Jump and Clarke, 1999; Sessler and Ntambi, 1998). Fatty acids may themselves be ligands for, or serve as precursors for, the synthesis of unknown endogenous ligands for nuclear peroxisome proliferator activating receptors (Kliewer et al., 1997). These receptors are important regulators of adipogenesis, inflammation, insulin action, and neurological function. Linoleic acid is the precursor to arachidonic acid. Arachidonic acid and other unsaturated fatty acids are involved with regulation of gene expression resulting in decreased expression of proteins that regulate the enzymes involved with fatty acid synthesis (Ou et al., 2001).

IX. CONCLUSION

As metabolism can be thought of changes in molecular level, inflammation and repair can be thought of changes in cellular to tissue levels: where replacement of faulty cells with healthy cells, or exclusion of foreign matters takes place. Lipids are not merely the main component of cell membrane; some important poly unsaturated fatty acid moieties of lipids play vital role in inflammation and repair. The PUFA that are not synthesized in the human body is known as EFA. If there is a chronic shortage or imbalance in essential fatty acids because of ingested food quality, life style, chronic inflammation - risk of illnesses like CVD, Stroke, Diabetes, Cancers etc. increases. Injection of vegetable, fruits and fish can reduce the risks of metabolic disorder related disease. Although different types of fatty acids and smaller molecules originated from oil seeds can be a promising source of lead compound against metabolic disorder related diseases, life is like a following river-there are lot of turns and twists. At different turns the direction of current is different- the effort to control the river with a handful sand is very little So, the best ways of maintaining healthy life are to have: a) fresh and diversified vegetables, fruits, vegetable oils, fish oils rich in n-3 and n-6 fatty acids, b) regular exercise to burn out the saturated fatty acids, and c) healthy life style.

REFERENCES

- [1] Astrup, A., Toubro, S., Raben A., Skov, A.R., 1997. The role of low-fat diets and fat substitutes in body weight management: What have we learned from clinical studies? J Am Diet Assoc 97, S82–S87.
- [2] Anderson, G. F., Chu, E., 2007. Expanding priorities confronting chronic disease in countries with low income. The New England Journal of Medicine 356, 209–211.
- [3] Armstrong, G. L., Conn, L. A., Pinner, R. W., 1999. Trends in in fectious disease mortality in the United States during the 20th century. Journal of the American Medical Association 281, 61–66.
- [4] Anulika, P.N., Ignatius, E.O., Raymond, E.S., Osasere O-I., Abiola, A.H., 2016. The Chemistry of Natural Product: Plant Secondary Metabolites. International journal of technology enhancements and emerging engineering research 4(8), 1-8. ISSN 2347-4289
- [5] Aird, W.C., 2005. Spatial and temporal dynamics of the endothelium. Journal of Thrombosis and Haemostasis 3, 1392–1406.
- [6] Bagchi, G., Khurana, S.M.P., 2013. Mega health benefits of omega fats. Science Reporter SR 50(9), 38-41.

- [7] Bennett, J.W., Chung, K-T., 2001. Alexander Fleming and the Discovery of Penicillin. Advances In Applied Microbiology 49,163-184.
- [8] Birt D.F., Shull J.D., Yaktine A.L., 1999. Chemoprevention of cancer. In: Shils ME, Olson JA, Shike M, Ross AC, eds. Modern Nutrition in Health and Disease, 9th ed. Baltimore, MD: Williams and Wilkins. Pp. 1263–1295.
- [9] Boden G., 1999. Free fatty acids, insulin resistance, and type 2 diabetes mellitus. Proc Assoc Am Physicians. 111, 241–8.
- [10] Brachman, P.S., 2003. Infectious diseases—past, present, and future. International Journal of Epidemiology. 32(5), 684–686. DOI: 10.1093/ije/dyg282
- [11] Calviello, G., Palozza, P., Piccioni, E., Maggiano, N., Frattucci, A., Franceschelli, P., Baroli, G.M., 1998. Dietary supplementation with eicosapentaenoic and docosahexaenoic acid inhibits growth of Morris hepatocarcinoma 3924A in rats: Effects onproliferation and apoptosis. Int J Cancer 75:699–705.
- [12] Caygill, C.P.J., Charlett, A., Hill, M.J., 1996. Fat, fish, fish oil and cancer. Br J Cancer 74:159–164.
- [13] Colditz, G.A., Willet, t W.C., Stampfer, M.J., Manson, J.E., Hennekens, C.H., Arky, R.A., Speizer, F.E., 1990. Weight as a risk factor for clinical diabetes in women. Am J Epidemiol 132:501–513.
- [14] Duranti, M., 2006. Grain legume proteins and nutraceutical properties. Fitoterapia 77, 67 – 82. www.elsevier.com/locate/fitote
- [15] Eltzschig, H.K., Carmeliet, P., 2011. Hypoxia and Inflammation. N Engl J Med. 364(7), 656–665. doi:10.1056/NEJMra0910283
- [16] Earla, P., 2014. Ancient Diseases-Microbial Impact. J Anc Dis Prev Rem. 2(1) R1-001. http://dx.doi.org/10.4172/2329-8731.R1-001
- [17] Giovannucci, E., Rimm, E.B., Colditz, G.A., Stampfer, M.J., Ascherio, A., Chute, C.C., Willett, W.C., 1993. A prospective study of dietary fat and risk of prostate cancer. J Natl Cancer Inst 85:1571–1579.
- [18] Gnacińska, M., Małgorzewicz, S., Stojek, M., Łysiak-Szydłowska, W., Sworczak, K., 2009. Role of adipokines in complications related to obesity: A review. Advances in Medical Sciences. 54(2), 150-157. DOI: 10.2478/v10039-009-0035-2
- [19] Gordon, T., 1953. Mortality in the United States, 1900-1950. Public Health Reports. 68(4), 441-444.
- [20] Grammatikos S.I., Subbaiah, P.V., Victor, T.A., Miller, W.M., 1994. n-3 And n-6 fatty acid processing and growth effects in neoplastic and non-cancerous human mam-mary epithelial cell lines. Br J Cancer 70:219–227.
- [21] Helmrich, S.P., Ragland, D.R., Leung, R.W., Paffenbarger, R.S., 1991. Physical activity and reduced occurrence of non-insulin-dependent diabetes mellitus. N Engl J Med 325:147–152.
- [22] Henderson, W.R., 1994. The Role of Leukotrienes in Inflammation. Basic Science Review. Annals of Internal Medicin 121(9), 684-697.
- [23] Horowitz, J.F., Klein, S., 2000. Exercise and lipid metabolism. The American Journal of Clinical Nutrition. 72(suppl), 558S–63S.

- [24] Hotamisligil, G.S., 2010. Endoplasmic Reticulum Stress and the Inflammatory Basis of Metabolic Disease. Cell. 140(6), 900–917. doi:10.1016/j.cell.2010.02.034.
- [25] Jesse, W.-H., Li., Vederas, J.C., 2009. Drug Discovery and Natral Products: End of an Era or an Endless Frontier? Science 325, 161-165.
- [26] Jump, D.B., Clarke S.D., 1999. Regulation of gene expression by dietary fat. Annu Rev Nutr 19:63–90.
- [27] Kliewer, S.A., Sundseth, S.S., Jones, S.A., Brown, P.J., Wisely, G.B., Koble, C.S., Devchand, P., Wahli, W., Willson, T.M., Lenhard, J.M., Lehmann, J.M., 1997. Fatty acids and eicosanoids regulate gene expression through direct interactions with peroxisome proliferator-activated receptors α and γ . Proc Natl Acad USA 94:4318–4323.
- [28] Kershaw, E.E., Flier. J.S., 2004. Adipose tissue as an endocrine organ. J Clin Endocrinol Metab 89(6), 2548-56.
- [29] Lloyd, C., 1994. Why Should Stationary Plant Cells Have Such Dynamic Microtubules? Molecular Biology of the Cell. 5, 1277-1280.
- [30] Mishra, S., 2016. Does modern medicine increase lifeexpectancy: Quest for the Moon Rabbit? Indian heart journal 68(1) 19–27. http://dx.doi.org/10.1016/j.ihj.2016. 01.003
- [31] Ou, J., Tu, H., Luk, A., DeBose-Boyd, R.A., Bashmakov, Y., Goldstein, J.L., Brown, M.S., 2001. Unsaturated fatty acids inhibit transcription of the sterol regulatory elementbinding protein-1c (SREBP-1c) gene by antagonizing ligand-dependent activa-tion of the LXR. Proc Natl Acad Sci USA 98:6027–6032.
- [32] Ribera, A.E., Zuñiga, G., 2012. Induced plant secondary metabolites for phytopathogenic fungi control: a review. Journal of Soil Science and Plant Nutrition, 12(4), 893-911.
- [33] Sasaki S, Horacsek M, Kesteloot H. 1993. An ecological study of the relationship between dietary fat intake and breast cancer mortality. Prev Med 22:187–202.
- [34] Schlotz, N., SØensen, J.G., Martin-Creuzburg, D., 2012. The potential of dietary polyunsaturated fatty acids to modulate eicosanoid synthesis and reproduction in Daphnia magna: A gene expression approach. Comparatives Biochemistry and physiology: A, Molecular and Integrative Physiology. 162(4-S), 449-454.
- [35] Sessler, A.M., Ntambi, J.M., 1998. Polyunsaturated fatty acid regulation of gene expression. J Nutr 128:923–926.
- [36] Snyder, W.S., 1975. Report of the task force on reference man, Pergamon Press for the International Commission on Radiological Protection, Oxford
- [37] Sun, Y., Cheng, J., 2002. Hydrolysis of lignocellulosic materials for ethanol production: A review. Bioresource Technology 83(1), 1-11.
- [38] Takahashi, M., Przetakiewicz, M., Ong, A., Borek, C., Lowenstein, J.M., 1992. Effect of omega 3 and omega 6 fatty acids on transformation of cultured cells by irradiation and transfection. Cancer Res 52:154–162.
- [39] Vessby, B., 2000. Dietary fat and insulin action in humans. Br J Nutr 83:S91–S96.
- [40] Wasserman, D.H., 2009. Four grams of glucose. Am J Physiol Endocrinol Metab 296, E11–E21, doi:10.1152/ajpendo.90563.2008. http://www.ajpendo.org

- [41] West, D.B., York, B., 1998. Dietary fat, genetic predisposition, and obesity: Lessons from animal models. Am J Clin Nutr 67, 505S–512S.
- [42] Wink, M., 1988. Theoretical and Applied Genetics 75, 225-233.
- [43] World Health Organization, 2005. Preventing Chronic Diseases: A Vital Investment. WHO, Geneva.
- [44] Yazdani, D., Tan, Y.H., Zainal Abidin, M.A., Jaganath, I.B., 2011. A review on bioactive compounds isolated from plants against plant pathogenic fungi. Journal of Medicinal Plants Research. 5(30), 6584-6589.

