

## The Importance of a Correct Diet in Preventing Osteoporosis

Pavone V\*, Testa G, Alberghina F, Lucenti L and Sessa G

Clinica Ortopedica e Traumatologica, Azienda Ospedaliero Universitaria Policlinico Vittorio Emanuele Catania, Catania, 95125, Italy

### Abstract

Osteoporosis is a very common bone disease characterized by low bone mass and micro architectural deterioration of bone tissue. It may result in high risk of bone fractures with impaired quality of life. The prevention of this disorder is based on an appropriate lifestyle and a proper diet. The correct supply of Vitamins D and K, protein, fatty acids and dietary components are notable factors that help maintain healthy bone structure. The effects of these elements have been briefly discussed and they have been shown to correlate to bone health.

**Keywords:** Diet; Osteoporosis; Prevention; Bone mineral density

### Introduction

Osteoporosis is very common—it is likely the most common bone disease [1]. It is characterized by low bone mass and mineral density that causes bone fragility and a subsequent increase in susceptibility to fracture [2]. The disorder occurs more frequently in the elderly and women, but may result in substantial morbidity and mortality in men [3]. In the European Union, twenty-two million women and 5.5 million men were estimated to have osteoporosis. Annually, there are 3.5 million new fragility fractures including 610,000 hip fractures, 520,000 vertebral fractures, 560,000 forearm fractures and 1,800,000 other fractures (i.e. fractures of the pelvis, rib, humerus, tibia, fibula, clavicle, scapula, sternum and other femoral fractures). The economic burden of these injuries and prior injuries is estimated to be €37 billion. Incident fractures represented 66% of this cost, long-term fracture care 29% and pharmacological prevention 5%. Previous and incident fractures also accounted for 1,180,000 quality-adjusted life years lost during 2010. The costs are expected to increase by 25% in 2025 [4].

Lifestyle and correct diet are the first elements to prevent the risks of bone fractures—nutrients and food ingredients have a positive or negative impact on bone health (5). The poor dietary supply of these elements or due to pathology leads to bone weakness and osteoporosis [5]. These dietary factors range from inorganic minerals (e.g., calcium, magnesium, phosphorus, sodium, potassium, and various trace elements) and vitamins (vitamins A, D, E, K, C, and certain B vitamins) to macronutrients such as protein and fatty acids [6].

The goal of this review is to describe the dietary factors involved in bone metabolism and their role in the prevention of osteoporosis.

### Calcium

Dietary calcium is present as salts or associated with other dietary constituents in the form of calcium ion complexes ( $\text{Ca}^{2+}$ ) absorbed in the proximal small intestine.

There are two routes of absorption: the first consist is via an active transcellular vitamin D dependent pathway (especially when intake is low). This includes entry of luminal  $\text{Ca}^{2+}$  across the microvillar membrane into the enterocyte via an electrochemical gradient via specific ion channels such as CAT1 (calcium transport protein 1). The second route is a non-vitamin D dependent paracellular pathway (especially when intake is high). Here, passive calcium is transported through the tight junctions between mucosal cells. It is non-saturated and is essentially independent of nutritional and physiological regulation as well as concentration [7].

An intake at which the calcium excretion is zero (intake and absorption are equal) is a minimal requirement. Though considerable differences exist between the results obtained from different researchers, a WHO expert panel has concluded that the correct value is 520 mg per day for adults eating a Western diet. Calcium is lost via urine, feces and other routes. In adults, the minimum urinary loss is up to 140 mg/day. When the amount of calcium introduced by diet is less than 200 mg/day, the net intestinal absorption is practically zero because approximately the same quantity is secreted daily into the gastrointestinal lumen and is lost with the feces [7].

There are many food constituents that have been suggested to be potential enhancers of calcium absorption including lactose, lactulose, and casein phosphopeptides. These common milk components play a considerable role particularly in postmenopausal women as well in reducing incident fractures in individuals who had previously suffered fractures and a reduction in bone mass [8]. Non-digestible oligosaccharides may also improve calcium absorption as well some legumes (soybean, peas, beans, lentils) that contain a large amount of calcium. Cooked spinach containing oxalic acid may interfere in the absorption of calcium and actually increase excretion [9]. Calcium supplementation in healthy postmenopausal women is not risk-free. Bolland et al. (10) studied 1471 healthy postmenopausal women on a calcium supplementation diet consisting of 1 g of elemental calcium daily as the citrate or identical placebo. Relevant complications such as myocardial infarction, stroke and sudden death were observed for five years in the treated group. The researchers noted significantly increased rates of adverse cardiovascular events. Therefore calcium supplementation has benefits on bone mass structure, but these should be balanced against potential detrimental effects [10].

The effectiveness of calcium supplementation has been showed to have a significant effect in reducing or blocking bone loss [11]. This effect has been mainly demonstrated in postmenopausal women (12). The influence of calcium supplementation on fracture risk is still a

\*Corresponding author: Vito Pavone, Clinica Ortopedica e Traumatologica, Azienda Ospedaliero Universitaria Policlinico Vittorio Emanuele Catania, Catania, 95125, Italy, Tel: +39-095-7435240; E-mail: [vitopavone@hotmail.com](mailto:vitopavone@hotmail.com)

Received July 28, 2015; Accepted September 14, 2015; Published September 21, 2015

Citation: Pavone V, Testa G, Alberghina F, Lucenti L, Sessa G (2015) The Importance of a Correct Diet in Preventing Osteoporosis. J Osteopor Phys Act 3: 160. doi:10.4172/2329-9509.1000160

Copyright: © 2015 : Pavone V, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

matter of debate, having reported in literature only a few studies that have addressed fracture endpoints [12].

### Vitamin D

Vitamin D consists of a group of sterol elements that act on bone modulation. Among these elements, only two show a nutritional relevant importance: ergocalciferol (vitamin D<sub>2</sub>) and cholecalciferol (vitamin D<sub>3</sub>), which are produced via ultraviolet radiation on 7-dehydrocholesterol and ergosterol, respectively. Vitamin D has an important role in bone metabolism in that it increases plasma levels of calcium and phosphorus, regulates osteoblast and osteoclast activity, and prevents PTH hyper secretion. Thus it has a critical role in regulating bone formation and preventing/treating osteoporosis.

Skin exposure to sunlight is an important source of endogenous Vitamin D. This is much more important than food intake; it is not affected by serum calcitriol levels.

Vitamin D is mainly found in fish that feed on plankton with limited quantities in other foods. Human milk contains Vitamin D levels sufficient for newborns; bovine milk has low levels.

The daily adult requirement of vitamin D, in absence of sun exposure, is between 600–800 IU per day but varies according to age and conditions such as gestation or lactation [9]. Cod liver oil, fatty fish and fortified foods (milk and cereals) are recommended supplements in the event of low sun exposure [13].

According to Lips [13] the 24,25(OH)<sub>2</sub>-D decreases fracture risk by stimulating osteoblast activity and bone formation. It has instead been shown that 1,25 (OH)<sub>2</sub>-D decreases activity of osteoclasts and slows the process of bone demineralization directly by inhibiting the synthesis and secretion of parathormone PTH[11]. This hormone is secreted when serum levels of calcium fall and stimulates activity of 1 $\alpha$ -hydroxylase in the kidney enhancing production of 1, 25 (OH)<sub>2</sub>-D.

Contraindications have been reported in the intake of high dose of oral vitamin D. In fact, Sanders et al. [14] reported that individuals receiving high-dose cholecalciferol within one year experienced 15 % more falls and 26 % more fractures (some not directly associated with a fall) versus the placebo group.

### Sodium and potassium intake

Sodium intake in adult age ranges from 2,990 to 4,600 mg/day. Animal and human studies have showed that the excretion of calcium in the urine increases as dietary sodium intake increases. In postmenopausal women, the intake of 1,768 mg/day of calcium or urinary sodium excretion of 2,110 mg/day did not result in hip bone loss [15]. The increased urine calcium excretion is estimated at approximately 26 mg per gram of salt ingested. An excess of sodium reabsorption at the proximal tubule and at Henle's loop is related to an increased salt intake, which reduces calcium levels. This is then eliminated in the urine to decrease decreasing the calcium pool.

Potassium is plentiful in food particularly in fruit and vegetables. Dietary consumption varies considerably with a recommended intake of 1-2 mEq/Kg. The intestines absorb approximately 90 % of the ingested potassium.

The link between dietary potassium and bicarbonate and the bone metabolism has been recently investigated by Hanley et al. [16], that reported in a review article that high intake of potassium with an increased feed with fruit and vegetables decreased the urinary calcium excretion through two possible mechanisms of action: 1) high fiber

content of the diet that minimizes calcium absorption, or 2) a reduction of the "acid load" that conserves calcium for bone retention [16].

### Protein intake

Protein provides the structural matrix of bone, optimizes IGF-1 levels, and increases urinary calcium and intestinal calcium absorption [17].

As protein intake increases, there is a major elimination of urinary calcium with most subjects developing negative calcium balance. It is estimated that there is a 50% increase in urinary calcium associated with doubling protein intake or roughly 1 mg urinary calcium for every gram of dietary protein [18]. However, the increase in urinary calcium observed with purified proteins or aminoacid infusions is not readily observed with food sources of protein [18].

In healthy adults, the increased protein intake from 0.7 to 2.1 g/kg/day increases urinary calcium elimination, but also increases intestinal absorption [19]. Increased calciuria is not necessarily correlated to calcium loss, negative calcium balance, or reduced bone density. On the contrary, several studies have demonstrated a positive association between dietary protein intake and increased bone mineral content or decreased risk of fracture [18].

Moreover, intake of high-quality protein is notable on the maintenance of adequate muscle mass and function and consequently in the quality of bone strength and density especially in the elderly population [20].

Sellmeyer et al. [21], in a prospective study performed in elderly women submitted to a diet with high ratio of animal to vegetable protein intake, showed a more rapid femoral neck bone loss and a greater risk of hip fracture than do those with a low ratio. This suggests that an increase in vegetable protein intake and a decrease in animal protein intake may decrease bone loss and the risk of hip fracture.

The relationship between protein intake and bone structure is further correlated to the potential negative effects of overall dietary acid-base balance. Urinary calcium increases with acid-forming foods such as meat, fish, eggs, and cereal, but decreases with plant foods. It is likely determined by the acid-base status of the entire diet. Bone loss may be attributable to the mobilization of skeletal salts to balance the endogenous acids generated from acid-forming foods. The meat intake and bone loss may, in fact, be related to an inadequate intake of fruits and vegetables than the overconsumption of meat [18].

### Vitamin K

Vitamin K is a coenzyme for glutamate carboxylase, which mediates the conversion of glutamate (Glu) to gamma-carboxyglutamate (Gla). At least 3 Gla proteins are found in bone tissue (osteocalcin 5 bone Gla Protein, matrix Gla protein, and protein S). The most plentiful and best known is osteocalcin (OC), which is released into the blood and is used as a marker of bone formation. The synthesis of functionally active OC is both vitamin D and vitamin K dependent. Vitamin K deficiency results in inadequate carboxylation of OC and leads to high serum levels of undercarboxylated (Glu) OC with low biologic activity [22]. Booth et al. [23] reported on a cross-sectional associations study between self-reported dietary vitamin K intake and BMD of the hip and spine performed in men and women of age range from 29-86 years. This study has been performed measuring BMD at the hip and spine in 1112 men and 1479 women who participated in the Framingham Heart Study. They obtained these following results: no significant associations were found between dietary vitamin K intake and BMD

in men; conversely, low dietary vitamin K intake was associated with a decrease in Body Mass Index (BMI) and an increase in fracture risk in women [23].

There are two types of vitamin K: K1 (phytonadione) and K2 (menatetrenone, menaquinone). Vitamin K1 is the form used to fortify foods and as a medication. Vitamin K2 is a group of compounds called menaquinones, which are produced by intestinal bacteria. Vitamin K deficiency is not related to low dietary intake—it may be secondary to the wide use of broad-spectrum antibiotics that interferes with vitamin K1-producing bacteria resulting in low levels of vitamin K. There is uncertainty regarding the relative importance of intestinally-produced vitamin K2. However vitamin K2, derived from intestinal bacterial metabolism is more important than green vegetable vitamin K1 in preventing fracture [24].

A daily adequate intake (AI) of vitamin K for women is around 90 g [25]. Vitamin K is contained in egg yolk, butter, liver and the kidneys of beef, pork, cereals as well as dark green leafy vegetables such as spinach and kale. Vitamin K seems to play an important role in preventing osteoporosis with increasing age but the mechanism of action in bone is not clear. Prolonged intake of *Natto* diets containing menaquinone-7 (total, 18.8 mg/100g diet) is thought to prevent osteoporosis [25].

### Fatty acids

There are two classes of essential fatty acids (EFAs): omega-3 and omega-6. Humans (like all mammals) are unable to synthesize EFAs so this must be provided in the diet. The parent compound in the omega-6 fatty acid family is linolenic acid (LA), while the parent compound of the omega-3 fatty acid family is  $\alpha$ -linolenic acid (ALA).

Essential FAs are necessary for maximal vitamin D-dependent calcium absorption [26]. Orchard et al. [27] reported that when EFAs were given alone (excluding the concomitant administration with calcium and vitamins), no beneficial effects were noted. In contrast, a diet with abundant ALA upon addition of walnuts, walnut oil and flaxseed oil versus an average American diet decreased NTx—a marker of bone resorption. In addition, a mixture of plant and marine sources of n-3 FAs, with ALA in highest quantity, was used to fortify dairy products with a resulting decrease in u-Dpyr—a urinary marker of bone resorption [27].

Recent research highlights the role of FA in inflammatory regulation of bone remodeling via cellular pathways. Emerging research suggests significant roles for FA in reducing bone and muscle loss with aging; however, findings are conflicted for FA and fracture risk. A relationship between higher omega-3 FA and better muscle/bone in older adults was advanced by Mangano et al. [28].

Fatty fish are the major source of EPA and DHA in the U.S. diet, while vegetable oils, especially soybean and canola oils are the primary sources of ALA.

Nuts, seeds, vegetables, some fruit, as well as egg yolk, poultry, and meat offer small amounts of omega-3 fatty acids to the diet. Factors known to inhibit fatty acid desaturation are aging, smoking, diabetes, high sodium intake, and biotin deficiency [29].

### Magnesium, zinc, copper

Minerals such as magnesium, zinc and copper are all essential for health. They help promote strong bones and are involved in the interaction of more than 300 enzyme reactions. These minerals are also necessary for the transmission of nerve impulses, temperature

regulation, detoxification and the formation of healthy bones and teeth [30].

Magnesium deficiency can affect bone health through several mechanisms. Low magnesium alters the structure of apatite crystals. Indeed, osteoporotic women with demonstrated magnesium deficiency have larger organized crystals in trabecular bone than healthy women and larger crystals because bones do not bear a normal load. In addition, magnesium deficiency is associated with a reduction in PTH levels and thus decreases in vitamin D. The recommended adult daily intake of magnesium is 310 mg/day [31].

Zinc, as a trace element, is essential for function of several critical enzymes in osteoblasts that are essential for collagen synthesis and other products. In addition, alkaline phosphatase requires zinc for osteoblast activity. The recommended adult daily intake is 8mg/day [32].

Until recently, the physiological role of copper in bone homeostasis has remained unclear. It has shown that copper is needed for the enzyme that increases crosslinking of collagen and elastin molecules. It may also have roles in other enzymes of bone cells. Bone mineralization may also be reduced via the changes induced in the two matrix proteins by low copper intakes [33]. Daily intake of copper in adults is 900 mg/day.

### Coffee

Hallström et al. [34] reported that high coffee consumption (four or more cups per day) plus high calcium intake (more than 1200 mg/day) did not interfere with BMI versus with those who had high consumption of coffee and low (< 600 Mg/Day) Or Intermediate Intake (600-1200 Mg/Day) Of Calcium. It Is Still Questionable If Caffeine Plays A Role In Reducing The Risk Of Fractures [34].

### Conclusion

As reported by the large literature studies, adequate nutrition is a contributing factor in preventing osteoporosis—a multifactorial disorder that affects many people worldwide. Prevention should start in childhood because that is when bone formation is very intense and the achievement of optimal peak bone mass is a useful requirement for optimal bone density in older age. The optimal intake of calcium, vitamins D and K, fatty acids, and others is a relevant factor in primary as well as in secondary prevention of osteoporosis. Literature studies reported that it is important to exclude large amounts of salt and caffeine, which may interfere with bone metabolism. Clearly, other factors besides these nutrients are important including avoiding smoking, high alcohol intake and sedentary lifestyle. These all contribute to reduced risk of fracture.

### References

1. Burge R, Dawson-Hughes B, Solomon DH, Wong JB, King A, et al. (2007) Incidence and economic burden of osteoporosis-related fractures in the United States, 2005–2025. *J Bone Miner Res* 22: 465–475.
2. Ray N, Chan J, Thamer M et Melton LJ (1997) Medical expenditures for the treatment of osteoporotic fractures in the United States in 1995: report from the National Osteoporosis Foundation. *J Bone Miner Res* 12: 24–35.
3. Halling A, Persson GR, Berglund J, Johansson O, Renvert S (2005) Comparison between the Klemetti index and heel DXA BMD measurements in the diagnosis of reduced skeletal bone mineral density in the elderly. *Osteoporos Int* 16: 999–1003.
4. Herlund E, Svedbom A, Ivergård M, Compston J, Cooper C, et al. (2013) Osteoporosis in the European Union: medical management, epidemiology and economic burden. A report prepared in collaboration with the International Osteoporosis Foundation (IOF) and the European Federation of Pharmaceutical Industry Associations (EFPIA). *Arch Osteoporos* 8: 136.

5. Harter DL, Busnello FM, Dibi RP, Stein AT, Kato SK, et al. (2013) Association between low bone mass and calcium and caffeine intake among perimenopausal women in Southern Brazil: cross-sectional study. *Sao Paulo Med J* 131: 315-322.
6. Cashman KD (2004) Diet and control of osteoporosis. In: Remacle C, Reusens B, editors. *Functional foods, ageing and degenerative disease*. Cambridge, UK: Woodhead Publishing Limited; p. 83–114.
7. Keller J, Schinke T (2013) The role of the gastrointestinal tract in calcium homeostasis and bone remodeling. *Osteoporos Int* 24: 2737-2748.
8. Christakos S (2012) Recent advances in our understanding of 1, 25-dihydroxyvitamin D (3) regulation of intestinal calcium absorption. *Arch Biochem Biophys* 523: 73-76.
9. Rossini M, Bianchi G, Di Munno O, Giannini S, Minisola S, et al. (2006) Determinants of adherence to osteoporosis treatment in clinical practice. *Osteoporos Int* 2006; 17: 914-921.
10. Bolland MJ, Barber PA, Doughty RN, Mason B, Horne A, et al (2008) Vascular events in healthy older women receiving calcium supplementation: randomised controlled trial. *BMJ* 2;336:262-266.
11. Meier C, Kränzlin ME (2011) Calcium supplementation, osteoporosis and cardiovascular disease. *Swiss Med Wkly* 141:w13260.
12. Shea B, Wells G, Cranney A, Zytaruk N, Robinson V, et al (2002) Meta-analyses of therapies for postmenopausal osteoporosis. VII. Meta-analysis of calcium supplementation for the prevention of postmenopausal osteoporosis. *Endocr Rev* 23:552–559.
13. Lips P (2006) Vitamin D physiology. *Prog Biophys Mol Biol* 92: 4-8.
14. Sanders KM, Stuart AL, Williamson EJ, Simpson JA, Kotowicz MA, et al. (2010) Annual high-dose oral vitamin D and falls and fractures in older women: a randomized controlled trial. *JAMA* 12; 303:1815-1822.
15. Harrington M, Bennett T, Jakobsen J, Ovesen L, Brot C, et al. (2004) The effect of a high-protein, high-sodium diet on calcium and bone metabolism in postmenopausal women and its interaction with vitamin D receptor genotype. *Br J Nutr* 91: 41-51.
16. Hanley DA, Whiting SJ (2013) Does a high dietary acid content cause bone loss, and can bone loss be prevented with an alkaline diet? *J Clin Densitom* 16: 420-425.
17. Noakes M, Keogh J, Foster P, Clifton P (2005) Effect of an energy-restricted, high-protein, low-fat diet relative to a conventional high-carbohydrate, low-fat diet on weight loss, body composition, nutritional status, and markers of cardiovascular health in obese women. *Am J Clin Nutr* 81: 1298–1306.
18. Heaney RP, Layman DK (2008) Amount and type of protein influences bone health. *Am J Clin Nutr* 87(suppl): 1567S-1570S.
19. Nuttall F, Schweim K, Hoover H, Gannon M (2006) Metabolic effect of a LoBAG30 diet in men with type 2 diabetes. *Am J Physiol Endocrinol Metab* 291: E786–791.
20. Rondanelli M, Guido D, Opizzi A, Faliva MA, Perna S, et al. (2014) A path model of sarcopenia on bone mass loss in elderly subjects. *J Nutr Health Aging* 18: 15-21.
21. Sellmeyer D, Stone K, Sebastian A, Cummings S (2001) A high ratio of dietary animal to vegetable protein increases the rate of bone loss and the risk of fracture in postmenopausal women. *Am J Clin Nutr* 73:118–122.
22. Shea MK, Booth SL (2008) Update on the role of vitamin K in skeletal health. *Nutr Rev* 66: 549-557.
23. Booth SL, Broe KE, Gagnon DR, Tucker KL, Hannan MT, et al. (2003) Vitamin K intake and bone mineral density in women and men. *J Clin Nutr* 77:512-516.
24. Iwamoto J (2014) Vitamin K2 Therapy for Postmenopausal Osteoporosis. *Nutrients* 6: 1971–1980.
25. Yamaguchi M, Kakuda H, Gao YH, Tsukamoto Y (2000) Prolonged intake of fermented soybean (natto) diets containing vitamin K2 (menaquinone-7) prevents bone loss in ovariectomized rats. *J Bone Miner Metab* 18: 71-76.
26. Haag M, Magada ON, Claassen N, Böhmer LH, Kruger MC (2003) Omega-3 fatty acids modulate ATPases involved in duodenal Ca absorption. *Prostaglandins, Leukotrienes and Essential Fatty Acids* 68: 423–429.
27. Orchard TS, Pan X, Cheek F, Ing SW, Jackson RD (2012) A systematic review of omega-3 fatty acids and osteoporosis. *Br J Nutr* 107 Suppl 2: S253-260.
28. Mangano KM, Sahni S, Kerstetter JE, Kenny AM, Hannan MT (2013) Polyunsaturated fatty acids and their relation with bone and muscle health in adults. *Curr Osteoporos Rep* 11:203-212.
29. Kruger MC, Horrobin DF (1997) Calcium metabolism, osteoporosis and essential fatty acids: a review. *Prog Lipid Res* 36: 131-151.
30. Gür A1, Colpan L, Nas K, Cevik R, Saraç J, et al. (2002) The role of trace minerals in the pathogenesis of postmenopausal osteoporosis and a new effect of calcitonin. *J Bone Miner Metab* 20:39–43.
31. Castiglioni S, Cazzaniga A, Albiseti W, Maier M (2013) Magnesium and Osteoporosis: Current State of Knowledge and Future Research Directions. *Nutrients* 5:3022–3033.
32. Sadighi A, Roshan MM, Moradi A, Ostadrahimi A (2008) The effects of zinc supplementation on serum zinc, alkaline phosphatase activity and fracture healing of bones. *Saudi Medical Journal* 29:1276–1279.
33. John J (2012) Nutrition and bone health. In: Mahan LK, editor. *Escott-Stumos Krauses food. Nutrition and diet therapy*. 12th ed. Philadelphia: W.B. Saunders Co; p. 300.
34. Hallström H, Wolk A, Glynn A, Michaëlsson K (2006) Coffee, tea and caffeine consumption in relation to osteoporotic fracture risk in a cohort of Swedish women. *Osteoporos Int* 17: 1055-1064.

**Citation:** Pavone V, Testa G, Alberghina F, Lucenti L, Sessa G (2015) The Importance of a Correct Diet in Preventing Osteoporosis. *J Osteopor Phys Act* 3: 160. doi:[10.4172/2329-9509.1000160](https://doi.org/10.4172/2329-9509.1000160)

### OMICS International: Publication Benefits & Features

#### Unique features:

- Increased global visibility of articles through worldwide distribution and indexing
- Showcasing recent research output in a timely and updated manner
- Special issues on the current trends of scientific research

#### Special features:

- 700 Open Access Journals
- 50,000 editorial team
- Rapid review process
- Quality and quick editorial, review and publication processing
- Indexing at PubMed (partial), Scopus, EBSCO, Index Copernicus and Google Scholar etc
- Sharing Option: Social Networking Enabled
- Authors, Reviewers and Editors rewarded with online Scientific Credits
- Better discount for your subsequent articles

Submit your manuscript at: <http://www.omicsonline.org/submit>