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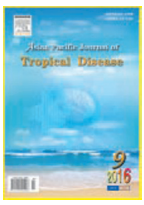

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Anti-diabetes and anti-obesity: A meta-analysis of different compounds

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ABSTRACT

Diabetes and obesity are chief health crisis and mainly age-related metabolic disorders. Diabetes is the main cause of morbidity and mortality worldwide. Obesity is linked with the progression of diabetes mellitus. High levels of glycerol, fatty acids, enzymes, pro-inflammatory markers and other obese entities build up insulin resistance in obese persons. The pathology of diabetes involves the dysfunction of β-islet cells of pancreas leading to deficient management of blood glucose level. This study demonstrates the anti-diabetic and anti-obesity effect of plant derived chemicals, some agonists, nutraceuticals, pharmaceutical drugs, some proteins and other compounds from 1998 to 2015. Plant derived from chemicals and other compounds were concluded to control diabetes and obesity by increasing number of glucose transporters, β-islet cells of pancreas, and lipid metabolism enzymes. In this way, the impairment of β-islet cells of pancreas is restored and the serum cholesterol and glucose levels of individuals are lowered. Further approaches are also needed to handle and cure diabetes and obesity.

1. Introduction

Sugar has been considered the essential component of human diet with most primitive rumor of its consumption from India and China since ancient times. High ingestion of sugar was linked with high risk situations such as cardiovascular diseases, dental caries, and obesity for decades[1]. Diabetes mellitus is a metabolic disorder which is connected with high levels of blood sugar throughout the world[2]. According to International Diabetes Federation, about 95% people are affected by diabetes out of 380 million. Diabetes (type 1 and 2) caused by impaired glucose homeostasis is due to insufficient production of insulin by pancreatic β-cell[3]. As communicable diseases, the status change is viewed in low and middle income countries. Chronic malady is more in high income countries. The most commonly examined condition is diabetes-obesity- hypertension nexus[4]. Diabetes mellitus is affecting many countries and its range is increasing vigorously. Depression risk in diabetes type 2 patients increases with the increase of obesity[5]. Diabetes mellitus and obesity are the main causes of diseases and death in developing countries. In North American countries, diabetes mellitus reached about 10.2% in 2010 and will reach about 12.1% up till 2030, and also is rising in other countries[6]. The use of sugar beverages was associated with elevated incidence of type 2 diabetes[7]. Type 2 diabetes patients (60%–90%) were found obese by estimation. Insulin resistances and deficiency are the two main factors in obesity and diabetes. These factors are strongly connected with bi-fold dictatorial cycle. Ultimately, hyperglycemia excites insulin secretion and lowers the rise in glycemia. Both insulin confrontation and insulin deficiency might depict genetic action in the development of obese diabetes[8]. Body mass index and cardiovascular malfunctions associated death have been expansively premeditated[9]. Study was conducted to diagnose the reduction of body mass index and obesity with diabetes mellitus (type 2) extensively in rural areas as compared to urban areas[10]. Majority of people are affected by obesity and diabetes caused by over nutrition. Physical laziness also causes cardiovascular diseases[11]. High utilization of sugar and fat diet are considered as the main cause of obesity and diabetes. More than 50 kinds of rare sugars are present. D-allulose among these sugars has been studied to show reduced energy density exhibiting about zero calories[12].

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Endoplasmic reticulum is extended by protein rejoinder and become active in various condition. Hypothalamic and hepatic endoplasmic reticulum cause irritation and emerging steatosis due to detachment of insulin fusion and death of β-cells[13]. Protein convertase activates inactive pro-peptides to active peptides. Its two relatives of proprotein convertase subtilisine/kenin type 1 and type 2 are articulated in neuroendocrine tissues having prohormones like pro-opio-melano-cortin, thyroid-releasing hormone and gonadotropin releasing hormone. Its deficiency has been linked with hyperinsulinemia, malabsorptive diarrhea, incomplete vital imperfections in adrenal and thyroid glands, stern obesity hypogonadotropic hypogonadism[14]. Type 2 diabetes can also cause risk of macro-vascular complications and affect adipose tissues, liver, muscles and pancreas, which can cause irritation by the ingestion of macrophages[15]. The metabolism of glucose related to β-cells supports the production of normal glucose. But when it's not responded, normal glucose concentration would lower[16]. Metabolic syndrome caused by blood pressure, abnormal fasting, atherogenic dyslipidemia can cause cardiovascular diseases and obesity along with endothelinB receptor antagonists by endothelin-1 human vasculature involvement[17]. Obesity is rapidly becoming more common worldwide during pregnancy and considered the major medical concern. Gestational diabetes mellitus and maternal obesity are linked with modifications in the expression and activity of placental nutrient carrier[18]. Pregnant women metabolic state is essential in the offspring adiposity extension. Placenta passes on substrates modified in gestational diabetes mellitus such as amino acids and lipids, which ultimately lead to improve fetal weight, enzymes and proteins associated to placental metabolism of fatty acids. Placental composition might be changed and its association with diabetes mellitus and obesity will be revealed[19]. Obesity linked with co-morbidities like diabetes and cardiovascular diseases causes morbidity and death[20]. Various clinical conditions such as hypertension, insulin confrontation and dyslipidemia are related with central obesity[21]. Childhood obesity is under the concerns of US which causes chronic health problem in children (basically type-2 diabetes). Dental diseases and other oral problems are also concerned with the basic health problems. These increasing problems are in Native Americans and in native People[22]. Obesity is the major cause of colorectal cancer progression. Colorectal cancer prognosis after diagnosis might be affected by obesity[23]. The disorder of diabetes is caused by chronic hyperglycemia and other complications like atherosclerosis, coronary heart disease, macro- and micro-diseases[24]. The literature showed that plasma non-esterifies fatty acid called free fatty acids. Insulin suppressed the mobilization of fatty acids from adipose tissues[25]. The crowd of excessive amount of adipose tissue in body causes obesity measured by body mass index scale at a range of 18.8–24.9 kg/m^2[26]. Atherosclerosis is the major cause of transience and morbidity in type 1 diabetes mellitus. Cardiovascular disease related with type 1 diabetes mellitus risk factors recognition is very significant[27]. Many reports predict that omega-3 poly unsaturated fatty acid has no or little action on metabolic control, while lowering hypertriglyceridemia in these patients, erythrocyte docosahexaenoic acid with eicosapentaenoic acid treatment significantly lowers the liver fat proportion in nonalcoholic fatty liver disease patients[28].

2. Anti-diabetic and anti-obesity agents

2.1. Effect of some agonists against diabetes and obesity

Anti-diabetic outcome of β3-adrenergic agonist CL observation was accomplished in overweight Zucker diabetic fatty rats (ZDF)[29]. ZDF-rats aged 7 weeks were directed with CL at a dose of 1 mg/kg/day for 14 days with the help of osmotic minipumps. Intravenous glucose tolerance tests were carried out for 13 days after beginning of disodium salt (CL-316243) treatment in mindful and 3 h-fasted rats. Glucose consumption determination is accomplished by glucose metabolic index using [2-^{3}H] deoxy glucose method. Then plasma levels of glucose with glucose analyzer, free fatty acids with a non esterified fatty acid kit, and insulin with radioimmunoassay were verified. Hyperinsulinemic-euglycemic clamps were done in aware, uncontrolled, unagitated rats[30]. At the end, they evaluated the facts obtained statistically. They winded up that in obese ZDF rats, CL handling normalizes the glycemia and increases insulin sensitivity[31]. β3-agonists increase the defective mitochondrial oxidation due to the increase of energy expenditure and fat oxidation which reduce free fatty acid level in plasma. Glucose consumption by skeletal muscles is accomplished via glucose fatty acid cycle. Thus, this agonist (β3/CL-316243) was found useful for type 2 diabetes and obesity treatment[32]. The β-adrenergic receptors agonistic action of SWR-0342SA in rats was studied by using segregated tissues and its anti-diabetic and anti-obesity consequences were studied in KK-Ay mice and C57BL mice[33-35]. They were provided with SWR-0342SA suspended in distilled water, then assessed body weight with food ingestion and blood glucose level with glucose B-test wok kit adjusted according glucose oxidase method and serum insulin level with Lbis mouse insulin enzyme immunoassay kit using streptavidin biotin method at regular intervals. Then they analyzed the values obtained statistically. They finished off that SWR-0342SA is a discerning β3-adrenergic receptor agonist and owns more anti- diabetic activity than anti-obesity activity (Table 1). Although its mechanism is not obvious, it described that in white adipose tissue of the obese mice, β3 adrenergic receptor agonists (*i.e.* BRL 26830A and CL-216347) increased the insulin receptors and returned the expression of glucose transporter type 4 (GLUT4)[36]. The reaction of tissue lipoprotein lipase to resupplying of food were described after delayed (4 h) fasting in weak and overweight Zucker rats. Lipoprotein lipase activity was studied in muscle and adipose tissues in fasted and fed conditions at various intervals along with or without propranolol during re-nourishing. They concluded that in lean rats, β-adrenergic pathway was activated by re-feeding after delayed fasting. This β-adrenergic pathway works against lipoprotein lipase modulation by insulin mediation. β-adrenergic pathway was enhanced by insulin mediated modulation, while in obese Zucker rats, the pathway was not activated by re-feeding in adipose tissues and muscles[37]. Linoleic acid was experimented for isomer-specific anti-diabetic activities. Lean and male Zucker diabetic fatty rats were taken at 6 weeks of age. Monitoring of animals was accomplished for glycemia after 7 days acclimation phase and allocated to dietary treatments. Fatty rat's weight was higher than lean rats initially but not diverse in different treatments. Intra-peritoneal glucose tolerance tests were accomplished on Day 11 to a subset of animals from