IN VITRO ANTI-DIABETIC ACTIVITY OF MICROENCAPSULATED AND NON-ENCAPSULATED ASTAXANTHIN

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ABSTRACT

Objective: Diabetes is a long term condition which indicates the high blood pressure. The symptoms indicates, polyuria (frequent urination), they will become increasingly thirsty (polydipsia) and hungry (polyphagia). Many drugs has been discovered for curing diabetes. Recent studies reported that the administration of astaxanthin reduces the blood pressure in the diabetic patient. Astaxanthin is a powerful antioxidant found in wide variety of aquatic living organism which has wide applications in pharmaceutical studies.

Methods: In vitro anti-diabetic study of both encapsulated and non-encapsulated astaxanthin such as DNSA method, starch-iodine color assay method and a glycosidase enzymes assay was carried out.

Results: The results of the present study indicated that both encapsulated and non-encapsulated astaxanthin shows higher antidiabetic activity in all the method. Each test samples possess the best activity when compared to standard drug acarbose.

Conclusion: The present study, it is concluded that both non-encapsulated and encapsulated astaxanthin exhibit good anti-diabetic activity.

Keywords: Astaxanthin, Anti-diabetic, DNSA, Starch-iodine, Acarbose, α glycosidase

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INTRODUCTION

Diabetes is one of the major causes of premature death worldwide. Every ten second a person dies from diabetes related causes mainly from cardiovascular complications. Metabolic disease, including dyslipidemia and diabetes, constitutes a major emerging health crisis in the world. The WHO, in the 2009 report, states that high blood plasma ranked first in the list of leading global risks for mortality and accounted for 7.5 million deaths in the world in 2004 [1]. According to reports, 415 million people worldwide were diabetic in 2015, most of them suffering from Type II diabetes [2].

Diabetes, which is diagnosed based on blood plasma hyperglycemia, has been linked to lipid overload and abdominal obesity and may synergize with these conditions to promote negative clinical outcomes [3, 4]. Although the symptoms and clinical pathology and physiology of these conditions are well understood, the question of pharmacologic treatment of dyslipidemia and diabetes remains unresolved well. The marine world, due to its phenomenal biodiversity, is a rich natural resource of many biologically active compounds such as polyunsaturated fatty acids (PUFAs), sterols, proteins, polysaccharides, antioxidants and pigments. People worldwide know that marine foods participate in human health promotion. A diet rich in marine products is considered to result in a lower incidence of diabetes, cancer and obesity. To date, many of reports have also showed that bioactive compounds from marine organisms, including Fucosterol, Astaxanthin, Marine Collagen Peptides, Diecikol and Krill Oil, exert a positive influence on metabolic dysfunction (diabetes and obesity) [1].

Astaxanthin, a red-orange carotenoid pigment, is a biological antioxidant that naturally found in a wide variety of aquatic living organisms, such as shrimp, crab, and salmon [5]. The green microalgae Haematococcus pluvialis and the red yeast Phaffia rhodozyma are common sources of natural astaxanthin [5]. Astaxanthin has shown various pharmacological activities, including anti-inflammatory [6, 7] and antidiabetic activities [8], as well as antioxidative effects [9-12]. Diabetes mellitus is strongly associated with oxidative stress, which can be a consequence of increased free radical production, reduced antioxidant defences or both [13].

Oxidative stress induced by hyperglycemia possibly causes the dysfunction of pancreatic b-cells and various forms of tissue damage in patients with diabetes mellitus. It was found that astaxanthin could diminish the oxidative stress caused by hyperglycemia in the pancreatic β cells, significantly improve glucose tolerance, increase serum insulin levels, and decrease blood glucose levels, indicating that astaxanthin might exert beneficial effects on pancreatic b-cell function and could protect pancreatic b-cells against glucose toxicity by preventing the progressive destruction of these cells [8]. The main objective of the present study is to investigate the antidiabetic activity for both encapsulated and non-encapsulated astaxanthin.

MATERIALS AND METHODS

Microencapsulation of astaxanthin using different agents

Astaxanthin purchased from Rudra Bio ventures Pvt Ltd, Bangalore was encapsulated using four different agents by ionotropic gelation method. In the first method, microencapsulated astaxanthin was prepared by using sodium alginate and calcium chloride [14, 15, 40]. In the second method, microencapsulated astaxanthin was prepared using sodium alginate and chitosan [16]. In the third method, chitosan-Tripolyphosphate was used to produce microencapsulated astaxanthin [17, 18]. In the fourth method, liposome encapsulated astaxanthin was carried out by the method [19]. These test samples (Both encapsulated and non-encapsulated astaxanthin) were used to study the antidiabetic activity using four different methods.

In vitro anti-diabetic activity

α amylase enzyme assay (DNSA method)

Starch solution (0.1% w/v) was prepared by stirring 0.1g of starch in 100 ml of 16 mmol of sodium acetate buffer. The enzyme solution was prepared by mixing 27.5 mg of alpha-amylase in 100 ml of distilled water. The colorimetric reagent was prepared by mixing the sodium potassium tartrate solution and 3, 5 Di-nitro salicylic acid solution at 96 mmol concentration [20, 21]. The control tube contains an only reagent and the test sample in the range of 100–500 µg/ml was prepared. From this, 500 µl of sample was mixed with 500 µl of starch solution and 500 µl of alpha-amylase solution.
which is incubated at 37 °C for 10 min. The reaction was stopped by the addition of 1 ml of 3, 5 Di-nitro salicylic acids and incubated in boiling water bath for 5 min, cooled at room temperature. The reaction mixture was then diluted by adding 10 ml of distilled water. The absorbance was measured at 540 nm [22, 23, 24]. Control was tested by replacing test sample with DMSO. The similar procedure were also followed for the standard drug Acarbose. The percentage of inhibition was calculated using the formula:

\[
\% \text{ inhibition} = \left(\frac{\text{O.D. of control} - \text{O.D. of test sample}}{\text{O.D. of control}}\right) \times 100
\]

α amylase enzyme assay (Starch-Iodine color assay method)

Screening of test samples for α-amylase inhibitors was carried out according to [25, 26] with slight modification based on the starch-iodine test. Test samples of varied concentrations in 500 μL were added to 500 μL of 0.02 M sodium phosphate buffer (pH6.9 containing 6 mmol sodium chloride) containing 0.04 units of the α-amylase solution and were incubated at 37 °C for 10 min.

Then 500 μL soluble starch (1%, w/v) was added to each reaction well and incubated at 37 °C for 15 min. 1 M HCl (20 μL) was added to stop the enzymatic reaction, followed by the addition of 100 μL of iodine reagent (5 mmol I2 and 5 mmol KI). The color change was noted and the absorbance was read at 620 nm on a microplate reader. The control reaction representing 100% enzyme activity were taken. Inhibition of enzyme activity was calculated as:

\[
\% \text{ inhibition} = \left(\frac{\text{O.D. of control} - \text{O.D. of test sample}}{\text{O.D. of control}}\right) \times 100
\]

α glucosidase enzyme assay

The inhibitory activity of α-glucosidase enzyme was determined by 1 ml solution of starch substrate (2% w/v maltose or sucrose) with 0.2 M Tris buffer pH 8.0 and 1 ml of test samples in the range of 100-500 μg/ml were incubated separately for 5 min at 37 °C. The reaction was initiated by adding 1 ml of alpha-glucosidase enzyme (1U/ml) to it followed by incubation for 40 min at 35 °C. Then the reaction was terminated by the addition of 2 ml of 6N HCl. Control was noted and the absorbance was read at 620 nm on a microplate reader. The control reaction containing 100% enzyme activity were taken. Inhibition of enzyme activity was calculated as:

\[
\% \text{ inhibition} = \left(\frac{\text{O.D. of control} - \text{O.D. of test sample}}{\text{O.D. of control}}\right) \times 100
\]

The inhibitory activity of α-glucosidase enzyme was determined according to a modified procedure [31, 32]. Briefly, 50 μl of 0.1M potassium phosphate buffer (pH6.9) was pre-incubated with 50 μl of reduce algluthathione (1 mg/ml). 20 μl α-glucosidase (1 U/ml) was added to the phosphate buffer, pH 6.9 and 20 μl of test samples at 37 °C for 10 min. After the incubation, 20 μl pNPG was added and the mixture was further incubated at 37 °C for 30 min. The reaction was terminated by adding 1 ml of 0.1M sodium carbonate. The absorbance of the samples and control were taken at 405 nm against a blank devoid of pNPG and sample. The control reaction (with 100% enzyme activity) contained buffer or DMSO instead of the respective samples while acarbose was used as a positive control. The inhibitory activity was calculated by using the following Equation:

\[
\% \text{ inhibition} = \left(\frac{\text{O.D. of control} - \text{O.D. of test sample}}{\text{O.D. of control}}\right) \times 100
\]

Statistical analysis

The statistical analyses for all the experiments were done using Excel 2013 through the statistical formula. Experimental data were expressed as mean±SD and IC 50 values were calculated. The experiment was performed in triplicates for all the test samples.

RESULTS

In vitro anti-diabetic activity

In vitro Alpha-amylase inhibitory activity (DNSA method).

In the present study, astaxanthin was encapsulated using four different methods were investigated for their potential to inhibit α-amylase activity and α-glucosidase activity. Five different concentrations viz., 250, 500, 750, 1000 and 1250 μg/ml of test samples were separately tested for the inhibition of α-amylase activity and α-glucosidase activity along with standard acarbose. The IC 50 values were also calculated from the percentage of inhibition by each samples. The IC 50 values of standard drug acarbose was 690.830 μg/ml which is compared with test samples. Five different concentrations viz., 250 μg/ml, 500 μg/ml, 750 μg/ml, 1000 μg/ml and 1250 μg/ml were incubated separately for 5 min at 37 °C. The reaction was stopped by adding 1 ml of 3, 5 Di-nitro salicylic acids and incubated in boiling water bath for 5 min. The absorbance was measured at 540 nm [22, 23, 24]. The percentage of inhibition was calculated using the formula:

\[
\% \text{ inhibition} = \left(\frac{\text{O.D. of control} - \text{O.D. of test sample}}{\text{O.D. of control}}\right) \times 100
\]

**Table 1:** In vitro Alpha-amylase inhibitory activity (DNSA method) of standard drug acarbose

| Content | Concentration (μg/ml) | mean±SD percentage | IC 50 values |
|---------|----------------------|--------------------|--------------|
| Blank   | -                    | 0.00               | 690.830      |
| S1      | 250                  | 32.3±0.100         | 669.129      |
| S2      | 500                  | 51.1±0.058         | 669.129      |
| S3      | 750                  | 66.2±0.053         | 669.129      |
| S4      | 1000                 | 82.9±0.100         | 669.129      |
| S5      | 1250                 | 99.2±0.058         | 669.129      |

**Table 2:** In vitro Alpha-amylase inhibitory activity (DNSA method) percentage for different concentration of test samples

| Concentration (μg/ml) | Non-encapsulated astaxanthin mean±SD percentage | ME 1 mean±SD percentage | ME 2 mean±SD percentage | ME 3 mean±SD percentage | ME 4 mean±SD percentage |
|----------------------|-------------------------------------------------|-------------------------|-------------------------|-------------------------|-------------------------|
| 250                  | 19.53±0.115                                    | 19.3±0.100              | 19.7±0.058              | 19.9±0.058              | 20.3±0.100              |
| 500                  | 35.0±0.100                                     | 34.8±0.153              | 35.3±0.058              | 35.5±0.058              | 36.0±0.115              |
| 750                  | 56.4±0.059                                     | 56.3±0.058              | 56.8±0.100              | 57.1±0.115              | 57.5±0.115              |
| 1000                 | 73.7±0.100                                     | 73.5±0.058              | 74.3±0.153              | 74.6±0.058              | 75.0±0.100              |
| 1250                 | 87.9±0.100                                     | 87.8±0.153              | 88.9±0.058              | 89.0±0.058              | 98.8±0.115              |
| IC 50 Values         | 685.169                                         | 687.703                 | 679.168                 | 675.168                 | 669.129                 |

Among all the test samples (both encapsulated and non-encapsulated astaxanthin) the ME 4 at 1250 μg/ml concentration, had the highest amylase inhibition of 89.82% followed by ME 2 and ME 3 with inhibition of 88.92% and 89.08% respectively. ME 1 and non-encapsulated astaxanthin showed the inhibition of 87.81% and 87.97% at concentration 1250 μg/ml. The standard drug acarbose showed the percentage of inhibition 87.55% at concentration 1250 μg/ml when compared with the test samples. The graph was represented in Graph 1 and Graph 2.

The IC 50 values were also calculated from the percentage of inhibition by each samples. The IC 50 values of standard drug acarbose was 690.830 μg/ml which is compared with test samples such as non-encapsulated astaxanthin, ME 1, ME 2, ME 3 and ME 4 that possessed 685.169 μg/ml, 687.703 μg/ml, 679.168 μg/ml, 675.168 μg/ml and 669.129 μg/ml respectively (table 1 and table 2). Thus, all the test samples showed highest α-amylase inhibition at different concentration (250 μg/ml to 1250 μg/ml) than standard drug acarbose.
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Graph 1: In vitro Alpha-amylase inhibitory activity (DNSA method) standard drug acarbose

Graph 2: In vitro Alpha-amylase inhibitory activity (DNSA method) for different concentration of test samples

Table 3: In vitro Alpha-amylase inhibitory activity (Starch-Iodine color assay method) of standard drug acarbose

| Content  | Concentration (µg/ml) | mean±SD percentage | IC 50 values |
|----------|-----------------------|--------------------|--------------|
| Blank    | -                     | 0.00               | 658.755      |
| S1       | 250                   | 19.84±0.211        |              |
| S2       | 500                   | 35.01±0.037        |              |
| S3       | 750                   | 60.07±0.091        |              |
| S4       | 1000                  | 75.26±0.042        |              |
| S5       | 1250                  | 94.34±0.103        |              |

Table 4: In vitro Alpha-amylase inhibitory activity (Starch-Iodine color assay method) percentage for different concentration of test samples

| Concentration (µg/ml) | Non-encapsulated astaxanthin mean±SD percentage | ME 1 mean±SD percentage | ME 2 mean±SD percentage | ME 3 mean±SD percentage | ME 4 mean±SD percentage |
|-----------------------|-------------------------------------------------|-------------------------|-------------------------|-------------------------|-------------------------|
| 250                   | 18.37±0.115                                     | 19.20±0.093             | 20.53±0.133             | 19.84±0.141             | 21.17±0.191             |
| 500                   | 32.55±0.132                                     | 36.93±0.051             | 34.12±0.121             | 33.23±0.072             | 35.40±0.116             |
| 750                   | 55.10±0.051                                     | 55.64±0.003             | 57.61±0.213             | 56.23±0.063             | 57.56±0.053             |
| 1000                  | 71.54±0.033                                     | 72.82±0.001             | 72.43±0.115             | 71.54±0.041             | 73.36±0.051             |
| 1250                  | 84.64±0.001                                     | 85.57±0.012             | 85.92±0.043             | 86.66±0.004             | 88.08±0.158             |

In vitro Alpha-amylase inhibitory activity (Starch-Iodine color assay method)

We investigated the encapsulated and non-encapsulated astaxanthin as well as standard drug acarbose for their α-amylase inhibitory activities using starch iodine color assay. The OD values were noted in table 3 and table 4.

When we compared the nonencapsulated and encapsulated astaxanthin, the maximum activity at 1250 µg/ml was exhibited by ME 4 (88.08%) followed by ME 3 (86.66%) and ME 2 (85.92%). Non-encapsulated astaxanthin and ME 1 test sample exhibit 84.64% and 85.57% which is similar to other test samples. Standard drug acarbose possessed very high inhibition of above 94.34% at concentration 1250 µg/ml which is greater than that of the test samples. At concentration 250 µg/ml the test samples such as non-encapsulated astaxanthin, ME 1, ME 2, ME 3 and ME 4 possessed 18.37%, 19.20%, 20.53%, 19.84% and 21.17% respectively. The standard exhibit maximum of 19.84% when compared with all other test samples (Graph 3 and Graph 4). The IC 50 values of standard drug, non-encapsulated astaxanthin and encapsulated astaxanthin was 658.755 µg/ml, 714.438 µg/ml, 690.224 µg/ml, 689.056 µg/ml, 699.113 µg/ml and 675.573 µg/ml respectively.

Graph 3: In vitro Alpha-amylase inhibitory activity (Starch-Iodine color assay method) of standard drug acarbose.

Graph 4: In vitro Alpha-amylase inhibitory activity (Starch-Iodine color assay method) for different concentration of test samples.
**In vitro Alpha-glucosidase inhibitory activity**

Table 5: *In vitro* Alpha-glucosidase inhibitory activity of standard drug ascorbose

| Content | Concentration (µg/ml) | mean±SD percentage | IC 50 values |
|---------|-----------------------|--------------------|-------------|
| Blank   | -                     | 0.00               | 674.687     |
| S1      | 250                   | 16.67±0.158        |             |
| S2      | 500                   | 38.02±0.058        |             |
| S3      | 750                   | 61.98±0.208        |             |
| S4      | 1000                  | 71.30±0.238        |             |
| S5      | 1250                  | 88.76±0.118        |             |

The results of the α-glucosidase are summarized in table 5 and table 6. All the test samples showed the varying effect on α-glucosidase activity. The standard drug showed maximum inhibition with the highest value of 88.76% seen at 1250 µg/ml concentration. Among the test samples, the highest value was obtained by ME 474.30% and ME 3 70.83% at concentration 1250 µg/ml. Compared to these test samples non-encapsulated astaxanthin, ME 1 and ME 2 possessed 68.96%, 64.93% and 67.46% (Graph 5 and Graph 6) respectively.

Table 6: *In vitro* Alpha-glucosidase inhibitory activity percentage for different concentration of test samples

| Concentration (µg/ml) | Non-encapsulated astaxanthin mean±SD percentage | ME 1 mean±SD percentage | ME 2 mean±SD percentage | ME 3 mean±SD percentage | ME 4 mean±SD percentage |
|-----------------------|-----------------------------------------------|--------------------------|--------------------------|--------------------------|--------------------------|
| 250                   | 14.79±0.178                                   | 13.44±0.113              | 14.37±0.167              | 14.79±0.153              | 15.22±0.123              |
| 500                   | 25.61±0.143                                   | 25.28±0.134              | 26.45±0.124              | 28.89±0.146              | 30.23±0.126              |
| 750                   | 46.54±0.243                                   | 45.60±0.145              | 49.20±0.153              | 50.23±0.126              | 50.23±0.126              |
| 1000                  | 59.93±0.232                                   | 58.38±0.178              | 62.41±0.156              | 65.26±0.174              | 65.26±0.174              |
| 1250                  | 68.96±0.134                                   | 67.46±0.103              | 70.83±0.183              | 74.30±0.126              | 74.30±0.126              |
| IC 50 Values          | 869.760                                       | 889.683                  | 838.895                  | 802.093                  |

The IC 50 values were also evaluated from the percentage of inhibition by each test samples. Test samples such as non-encapsulated astaxanthin, ME 1, ME 2, ME 3 and ME 4 showed IC 50 values of 869.760, 923.286, 889.683, 838.895 and 802.093 µg/ml along with standard drug acarbose that possessed 674.687 µg/ml.

**In vitro** Alpha-glucosidase inhibitory activity (Alternative method)

The result of *In vitro* Alpha-glucosidase inhibitory activity (Alternative method) was given in table 7 to table 8. All samples showed maximum inhibition at 1250 µg/ml and least inhibition at 250 µg/ml. At concentration 1250 µg/ml the test samples such as non-encapsulated astaxanthin, ME 1, ME 2, ME 3 and ME 4 produced 88.68%, 83.11%, 85.53%, 84.37% and 89.94% along with standard i.e. 90.21%. The least inhibition at 250 µg/ml of concentration were recorded by test samples i.e. 18.96%, 17.61%, 16.98%, 18.15% and 19.95%. The standard drug also exhibits similar percentage of inhibition 19.32%. The graph was plotted against the percentage of inhibition and concentration for both standard and test samples indicated in Graph 7 and Graph 8.

Table 7: *In vitro* Alpha-glucosidase inhibitory activity (Alternative method) of standard drug acarbose

| Content | Concentration (µg/ml) | mean±SD percentage | IC 50 values |
|---------|-----------------------|--------------------|-------------|
| BLANK   | 250                   | 19.32±0.141        | 650.436     |
| S1      | 500                   | 39.71±0.017        |             |
| S2      | 750                   | 58.22±0.043        |             |
| S3      | 1000                  | 75.83±0.221        |             |
| S4      | 1250                  | 90.21±0.107        |             |
Table 8: *In vitro* Alpha-glucosidase inhibitory activity (Alternative method) percentage for different concentration of test samples

| Concentration (µg/ml) | Non-encapsulated astaxanthin mean±SD percentage | ME 1 mean±SD percentage | ME 2 mean±SD percentage | ME 3 mean±SD percentage | ME 4 mean±SD percentage |
|-----------------------|-----------------------------------------------|-------------------------|-------------------------|-------------------------|-------------------------|
| 250                   | 18.96±0.189                                   | 17.61±0.043             | 35.22±0.057             | 36.62±0.117             | 36.66±0.145             |
| 500                   | 37.35±0.119                                   | 53.46±0.061             | 68.55±0.113             | 68.46±0.134             | 69.72±0.029             |
| 750                   | 55.71±0.113                                   | 68.11±0.182             | 83.11±0.182             | 85.53±0.165             | 84.37±0.075             |
| 1000                  | 71.97±0.210                                   | 72.5.011                | 719.134                | 710.211                | 672.294                |
| IC 50 Values          | 684.879                                       |                         |                         |                         |                         |

DISCUSSION

Lack of insulin affects the metabolism of carbohydrates, proteins, fat and causes significant disturbance of water and electrolyte homeostasis [33]. Recent advances in understanding the activity of intestinal enzymes (α-amylase and α-glucosidase both are important in carbohydrate digestion and glucose absorption) have led to the development of newer pharmacological agents. A high postprandial blood glucose response is associated with micro-and macro-vascular complications in diabetes and is more strongly associated with the risk for cardiovascular diseases than are fasting blood glucose. α-Glucosidase enzymes in the intestinal lumen and in the brush border membrane play main roles in carbohydrate digestion to degrade starch and oligosaccharides to monosaccharides before they can be absorbed. It was proposed that suppression of the activity of such digestive enzymes would delay the degradation of starch and oligosaccharides, which would, in turn, cause a decrease in the absorption of glucose and consequently the reduction of postprandial blood glucose level elevation [34].

Alpha-glucosidase inhibitor retards the digestion of carbohydrates and slows down the absorption. Acarbose and miglitol are competitive inhibitors of α-glucosidases and reduces absorption of starch and disaccharides [35]. The α-amylase inhibitors act as an anti-nutrient that obstructs the digestion and absorption of carbohydrates. Acarbose is complex oligosaccharides that delay the digestion of carbohydrates. It inhibits the action of pancreatic amylase in the breakdown of starch. Synthetic inhibitor causes side effect such as abdominal pain, diarrhoea and soft faeces in the colon.

The present study reveals that both encapsulated and non-encapsulated astaxanthin effectively inhibit both α-amylase and α-glucosidase enzymes. Our findings were compared with other research articles. The mechanisms were investigated underlying the insulin sensitivity effects of ASX in a non-genetic insulin resistant animal model. The results showed that ASX improved insulin sensitivity by activating the post-receptor insulin signalling, i.e. enhancing the auto phosphorylation of insulin receptor-b (IR-b), IRS-1 associated PI3-kinase step, phospho-Akt/Akt ratio and GLUT-4 translocation in skeletal muscle [37].

Oxidative stress induced by hyperglycemia possibly causes the dysfunction of pancreatic β-cells and various forms of tissue damage in patients with diabetes mellitus. It was found that astaxanthin could diminish the oxidative stress caused by hyperglycemia in the pancreatic β-cells, significantly improve glucose tolerance, increase serum insulin levels, and decrease blood glucose levels [8]. Recently, α-amylase and α-glucosidases reduced the high postprandial (PP) blood glucose peaks in diabetes [36]. Acarbose and Miglitol are competitive inhibitors of α-glucosidases and reduces absorption of starch and disaccharides [35]. The α-amylase inhibitors act as an anti-nutrient that obstructs the digestion and absorption of carbohydrates. Acarbose is complex oligosaccharides that delay the digestion of carbohydrates. It inhibits the action of pancreatic amylase in the breakdown of starch. Synthetic inhibitor causes side effect such as abdominal pain, diarrhoea and soft faeces in the colon.
[38] demonstrated that astaxanthin could substantially improve insulin sensitivity through abolishing significant elevation in both glucose and insulin levels induced by a high fat plus high fructose diet in mice.

But beyond that, the effects of astaxanthin in a metabolic syndrome animal model of spontaneously hypertensive corpu lent rats, the results showed that astaxanthin markedly decreased the levels of blood glucose, triglycerides and non-esterified fatty acids, and significantly increased the levels of high-density lipoprotein cholesterol and adiponectin. It is suggested that astaxanthin ameliorates insulin resistance and improve insulin sensitivity by increasing glucose uptake, and by modulating the levels of circulating adiponectin and blood lipids [39].

CONCLUSION

To date, more and more metabolic diseases have influenced in human’s health and quality of life. In the last few years, there has been a growing interest in the herbal medicine in care and management of diabetes both in developing and developed countries, due to their natural origin and less side effects.

The adverse effects of current drug treatment are not always satisfactory in maintaining normal levels of blood Glucose. Hence there is continuous thrust towards discovering or identification of bioactive compounds derived from plants and marine sources with potent antidiabetic activity. In the present study, the astaxanthin in free form and encapsulated form has been found to exhibit better antidiabetic potential by inhibition of Amylase and Glucosidase. Our other studies also proved their antioxidant, radical scavenging and anti-inflammatory potential. However, further research should go in this direction in order to show new preventive and potential therapeutic strategies against diabetes and associated disorders.

CONFLICT OF INTERESTS

Declare none

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