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Full Length Research Paper

Isolation and screening of α-glucosidase enzyme inhibitor producing marine actinobacteria

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α-Glucosidase enzymes are responsible for hydrolyses of carbohydrates thereby converting dietary disaccharides into absorbable monosaccharide. Inhibitors of such enzymes can be used as clinical tool for treating diabetes. Conventionally such inhibitors are reported from plant and microbial sources. However, microbial inhibitors are considered to be the most important as they are capable of producing micro molecular compounds. Hence the present study was conducted to test actinobacteria for their ability to produce yeast and rat α-glucosidase inhibitors. α-glucosidase inhibitor producing ability of 30 strains of marine actinobacteria was characterized. The strains showed significant inhibitory activity against yeast and mammalian α-glucosidase. Of the 30 strains, only four strains showed significant yeast α-glucosidase inhibitory activity. Among the four, PSG-22 showed 90% significant inhibitory activity. Similarly, six strains showed above 50% of inhibition of mammalian α-glucosidase enzyme in the rat system. Among the six strains, PSG-22 showed significant activity of about 80% inhibition activity. Interestingly strain PSG-22 was looking like an indigenous marine form as it required seawater for its growth.

Key words: Actinobacteria, α-glucosidase inhibitor, marine, enzyme inhibitor.

INTRODUCTION

Marine actinobacteria are representative of terrestrial micro organisms and usually are isolated from soils of varied origin, compared to terrestrial actinobacteria, only little work has been conducted on marine actinobacteria (Imada, 2005). Actinobacteria in marine and estuarine sediments have not been investigated extensively although their ubiquitous presence in marine sediments has been well documented (Takizawa et al., 1993; Moran et al., 1995; Sivakumar et al., 2005). Actinobacteria contains the most economically significant prokaryotes, which are producing over half of the bioactive compounds in the antibiotic literature database (Lazzarini et al., 2000). However, studies documenting the diversity, distribution and ecology of actinobacteria in the oceans are scarce (Stach et al., 2003). But marine environment is considered to be the best source of novel actinobacteria, many of which produce bioactive compounds (Fiedler et al., 2005; Jensen et al., 2005).

α-glucosidases is a very important enzyme responsible for the hydrolysis of dietary disaccharides into absorbable monosaccharide in microbial system and in small intestine of animal digestive system .Glucosidases are not only essential for carbohydrate digestion but it is also very important for processing of glycoproteins and glycolipids and are also involved in a variety of metabolic disorders and other diseases such as diabetes (Jenkins et al., 1981). α-Glucosidase inhibitors are useful for the control of carbohydrates – dependent diseases such as diabetes and obesity (Bio-linn et al., 1982). Studies on glycosidase inhibitors proved that they are the prospective therapeutic agents for the disorders caused by glucosidases (Winchester et al., 1992). When compared to the enzyme inhibitors from animal and plants, the microbial inhibitors possess low molecular

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abbreviations: cpcsea, committee for the purpose of control and supervision of experiment on animals; **PNPG,** ρ-nitrophenyl-α-D-glucoside; **AIA,** actinomycetes isolation agar.

Table 1. Acinobacterial population density recorded in different media and samples.

Samples	AIA (CFU/g)	Kuster agar (CFU/g)
I	1.6×10^4	1.5×10^4
II	20×10^4	7.3×10^4
III	9.5×10^4	21×10^4
IV	17×10^4	2.2×10^4
V	30×10^4	14×10^4
VI	41×10^4	2.3×10^4
VII	1.9×10^4	2.4×10^4
VIII	13×10^4	21×10^4
IX	3.6×10^4	37×10^4
X	4.3×10^4	8.5×10^4

weight compounds derived from hydrolysis of macro-molecular substances (Imada et al., 2004). The terrestrial sources of new enzyme inhibitors seem to be rare and different species of microorganisms may produce structurally identical inhibitors (Umezawa et al., 1970; Hamato et al., 1992) and thereby provides limited options for enzyme inhibition activities. Imada (2005) has reported the α-amylase enzyme inhibitor producing marine *S. corichorusii* subsp. *rhodomarinus* subsp. nov and incidentally it is the only enzyme inhibitor report from marine actinobacteria. As, α-glucosidase enzymes extracted from rat intestine closely mimics the mammalian system (Ohta et al., 2002) and therefore it may be a better model to identify, design and develop antihyperglycaemic agents particularly for the management of postprandial Hyperglycemia in diabetes. Considering the importance of α-glucosidase enzyme inhibitors especially of microbial origin, the present study was attempted to evaluate the α-glucosidase enzyme inhibitor activity of marine actinobacteria.

MATERIALS AND METHODS

Sediment samples were collected from Parangipettai coast, east coast of India at the depth of 10 m. Sediment samples are shade dried, grinded and mixed with equal weight of $CaCO_3$ (1:1) and incubated for ten days in an inverted petri plates with saturated disk of filter paper, at room temperature (El-Nakeeb and Lechevalier, 1963). Pre-treated samples (1 g) were serially diluted (Jensen et al., 1991) and spread on Actinomycetes Isolation agar (AIA, Hi-Media, Mumbai) and Kuster's agar (Kuster and Williams., 1964) by spread plate method. The plates were incubated at 30°C for 14 days. Based on the white powdery colony and good growth performances, 30 strains were selected and tested for their ability to produce α-glucosidase inhibitor activity. To determine requirement for seawater, each strains were grown on nutrient agar prepared with distilled water and seawater.

Culture for production α-glucosidase inhibitor of selected actinobacteria were initially grown in 20 ml of R2YE seed medium (Imada and Simidu, 1992) prepared in distilled water as well 25% and 50% seawater. The production cultures were incubated for 7 days at 28°C, under constant agitation of 150 rpm. An aliquot of 1.5 ml of culture broth was sampled, after which the supernatants were separated via centrifugation (Hyun et al., 2005). The fermentation media was mixed with equal volume of methanol to extract the α-glucosidase inhibitors and the resultant extract was concentrated by lyophilisation (Mahmud et al., 1999). The lyophilised powder was dissolved in the distilled water (1 mg/ml) for the assay of *in vitro* α-glucosidase inhibitory activity.

Yeast and mammalian α-glucosidase inhibitory activity assay

Yeast α-glucosidase inhibitory activity was evaluated by following Dahlqvist method (Dahlqvist, 1970), slightly modified version of the ρ-nitrophenyl-α-D-glucoside (PNPG) method. The mammalian α-glucosidase was assayed by following the modified ρ-nitrophenyl-α-D-glucoside method (Dahlqvist and Asp, 1967). The animal model experiment for the α-glucosidase inhibitor assay was carried out using male albino wistar rats weighing 150-200 g (Goto et al., 1995). Animals were obtained and maintained in Central Animal House, Rajah Muthiah medical college and Hospital, Annamalai University, Tamil Nadu, India. The experiments were carried out according to the guidelines of the Committee for the Purpose of Control and Supervision of Experiment on Animals (CPCSEA), New Delhi, India, and approved by the Animal Ethical Committee of Annamalai University.

The experimental animals were divided into VI groups, each group containing 6 rats. Group: I rats were oral fed with 1 ml of distilled water (negative control); Group II: rats oral fed with only 1 ml of 500 mg sucrose solution (Positive control); Group III : rats oral fed with 1ml of sucrose solution and 0.03g /kg of inhibitor; Group IV: rats oral fed with 1 ml of sucrose solution and 0.06 g/kg of inhibitor; Group V: rats oral fed with 1 ml of sucrose solution and 0.1 g/kg of inhibitor and Group VI: Normal rats fed with only 0.03 g/kg of inhibitor.

Determination of blood glucose and residual gut sucrose content: Blood samples were obtained from the tail vein before (0 min) and after 30, 60 and 120 min of sucrose administration. Some of the rats were sacrificed before as well as 30, 60 and 120 min after sucrose loading. The gastrointestinal tract was excised and divided into the following 6 segments: the stomach, upper, middle, and lower 20 cm of the small intestine, the cecum and the large intestine. Each segment was washed out with ice-cold saline, acidified with 2 ml of 2 NH_2SO_4 and centrifuged at 3000 rpm for 10 min. The supernatant thus obtained was boiled for 2 h to hydrolyze the sucrose and then neutralized with 1 N NaOH. Glucose was estimated using modified O-toluidine reagent method (Sasaki et al., 1972) and protein concentration of the inhibitors was tested by following Lowry's method (Lowry et al., 1953) using Bovine serum albumin as standard.

RESULTS

In the present study, actinobacteria isolated by using two different media showed varied results; actinomycetes isolation agar (AIA) recorded the minimum of 1.6×10^4 CFU/g in sample I and the maximum of 41×10^4 CFU/g was recorded in sample VI. In case of Kuster's Agar Medium the minimum of 1.5×10^4 CFU/g was recorded in sample I and the maximum of 37×10^4 CFU/g was recorded in sample IX (Table 1). Totally 90 strains were selected from the plates based on the colony morphology and are subcultured. The strains are designated as PSG1-PSG 90. Among the 90 strains, 30 strains were selected based on colony morphological similarity and aerial mycelium with white powder mass colour for further

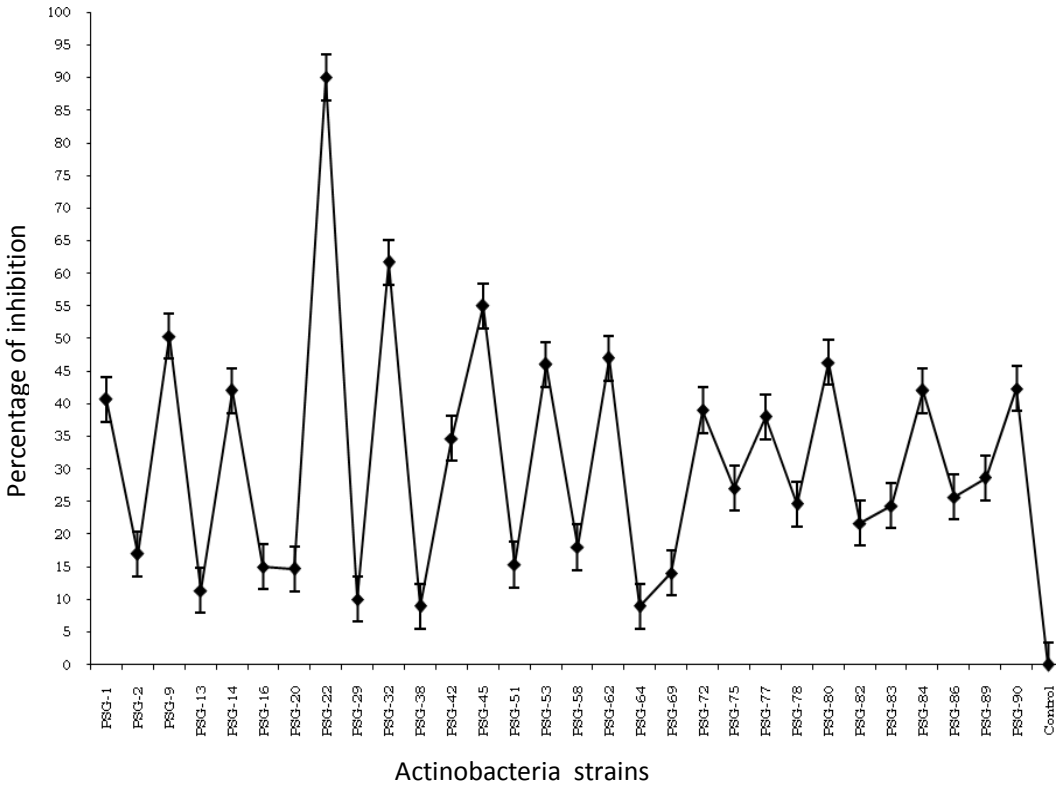

Figure 1. Yeast α-glucosidase inhibitory activity of actinobacteria.

work.

These 30 strains were tested for the requirement of sea water for their growth. Out of 30 strains, 8 strains (26.6%) grow only in seawater and failed to grow in distilled water. The remaining 22 strains (73.3%) grow both in seawater at varied concentration and also in distilled water. The actinobacteria reported in this study represent only a portion of the total population, because of the selective culture conditions and media composition used for the experiments. When compared to control, strain PSG-22 showed very significant activity of about 90% inhibition and strains PSG-32, PSG-45 and PSG- 9 showed 61, 66, 55 and 50.33% of inhibition respectively (Figure 1).

The homogenate prepared from rat intestine contains, the enzyme activity (α-glucosidase) of about 0.45 units/ml, and the amount of protein in homogenate was around 1.25 mg/ml. Of the 30 strains tested only six strains (23%) showed significant inhibitory activity of above 50% of inhibition of α-glucosidase enzyme. Among the seven strains, PSG-22 showed significant activity of about 77.6% inhibition and strains PSG-1, PSG-2, PSG-32, PSG-58, PSG- 69 and PSG-82 recorded 60, 55.3, 60.3, 62.4, 57.5 and 54.5% of inhibition respectively (PSG-22 > PSG-58 > PSG-32 > PSG-1 > PSG-69 > PSG-2 and PSG-69) (Figure 2), and the remaining 23 strains shows inhibitory activity of less than 50%.

From the above result, the extracts of strain PSG-22 showed maximum inhibition compared with other strains and hence the extract of that strain was used for the animal experiments. Changes in blood glucose level of the experimental groups are listed in Table 2. The oral administration of α-glucosidase inhibitors to the test groups with 500 mg of sucrose significantly reduced the rise of blood glucose level Figures 3 and 4.

The residual sucrose content of the intestine of the animal administrated with α-glucosidase inhibitors also reveals the blocking of sucrose conversion in intestine and resulted in low glucose level in blood. There was no residual sucrose in Group I, as the group administrated with no sucrose. The blood glucose level of this group ranged from maximum of 81.33 ± 1.52 mg/dl at 0 h and minimum of 70.66 ± 1.52 mg/dl at the end of 2 h.

In Group II, the sucrose content in the stomach after one hour of administration was estimated as 62 µg/ml of intestinal fluid and 16 µg/ml of intestinal fluid in upper intestine, and after 2 h there was no sucrose residual in all the intestinal parts. This reveals the sucrose was actively digested in the stomach and upper intestine. The blood glucose of the rats of this group ranged from minimum of 83.33 ± 2.08 mg/dl at 0 h and sudden increase in blood glucose after sucrose administration which reached the maximum value of 275.66 ± 4.50 mg/dl at 30 min and started to decrease gradually to169.33 ± 4.04 mg/dl at the end of 2 h.

In Group III, the residual sucrose content of the various parts of digestive tract after one hour was ranged from 92

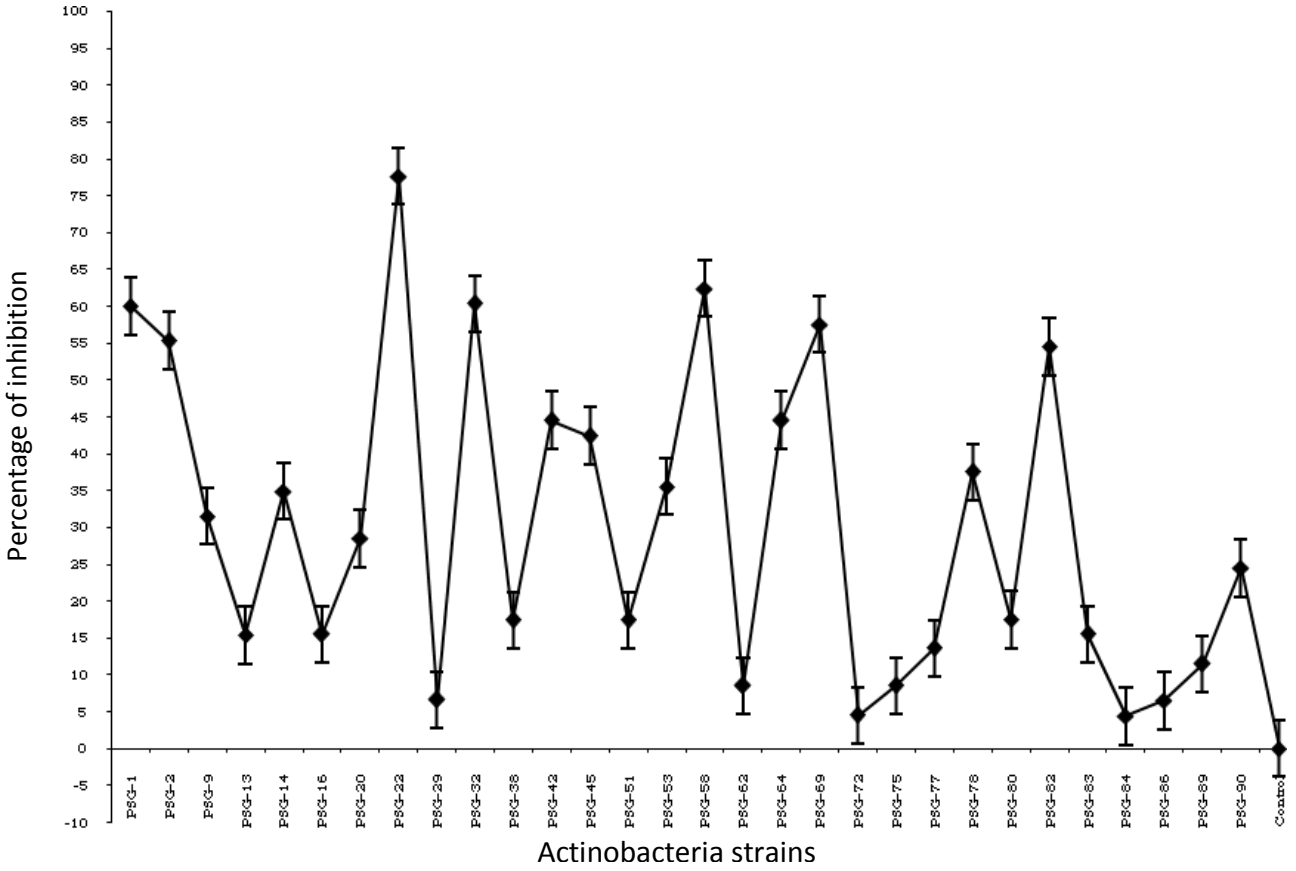

Figure 2. Mammalian α-glucosidase inhibitory of actinobacteria.

Table 2. Changes in blood glucose level in the test groups.

S/N	Groups	Blood glucose in mg/deciliter of blood at various time intervals (Min)			
		0	30	60	120
1	I	81.33±1.52[a]*	77.00±2.64[a]	74.33±2.08[a]	70.66±1.52[a]
2	II	83.33±2.08[a]	275.66±4.50[b]	217.66±2.51[b]	169.33±4.04[b]
3	III	84.33±3.05[a]	219.66±5.50[c]	159.00±4.58[c]	149.66±14.22[c]
4	IV	80.33±3.05[b]	191.33±1.52[d]	147.33±3.05[d]	131.66±3.51[d]
5	V	76.33±1.52[b]	182.66±3.51[e]	133.00±4.58[e]	130.00±3.60[e]
6	VI	76.00±3.60[b]	66.00±2.00[f]	66.00±4.00[f]	63.33±3.51[a]

(Values are mean ± SD) *Values not sharing a common superscript letter differ significantly at $p < 0.05$ (DMRT).

µg/ml in stomach and 24 µg/ml in upper, 52 µg/ml in middle and 3 µg/ml in lower intestines. The residual sucrose after 2 h was ranged from 8 µg/ml in upper intestine, 48 µg/ml in middle intestine and 16 µg/ml in lower intestine revealed the transformation of undigested sucrose to the next parts in due course of time. The blood glucose of the rats of this group ranged from minimum of 84.33 ± 3.05 mg/dl at 0 h and sudden increase in blood glucose after sucrose administration and reached the maximum value of 219.66 ± 5.50 mg/dl at 30 min and started to decrease gradually to 149.66 ± 14.22 mg/dl at the end of 2 h.

In Group IV, the residual sucrose content of the various parts of digestive tract after one hour was ranged from 123 µg/ml in stomach and 30 µg/ml in upper, 61 µg/ml in middle and 11 µg/ml in lower intestines. The residual sucrose after 2 h was ranged from 42 µg/ml in upper intestine, 53 µg/ml in middle intestine and 28 µg/ml in lower intestine, 59 µg/ml in caecum and 2 µg/ml in large intestine revealed the transformation of undigested sucrose to the next parts in due course of time. The blood glucose of the rats of this group ranged from minimum of

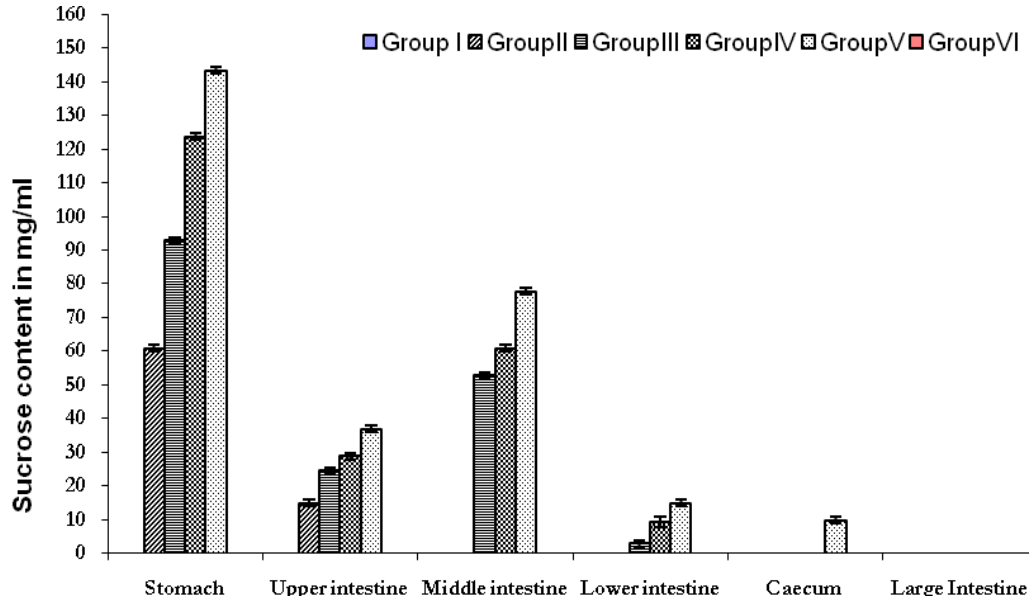

Figure 3. Residual sucrose content of intestinal fluid after 1 h.

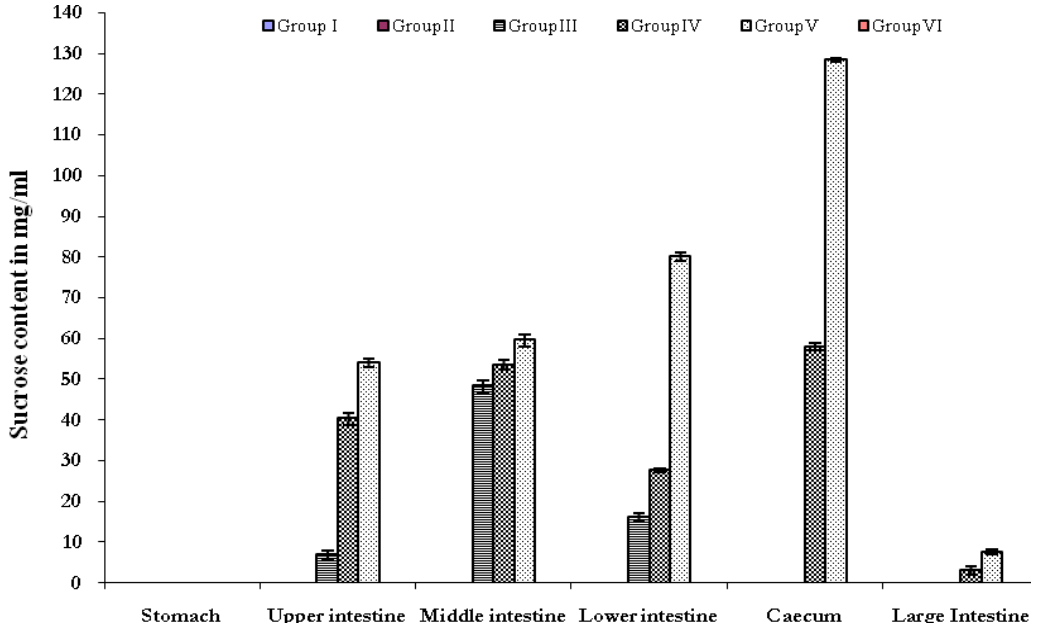

Figure 4. Residual sucrose content of the intestinal fluid after 2 h.

80.33 ± 3.05 mg/dl at 0 h and sudden increase in blood glucose after sucrose administration which reached the maximum value of 191.33 ± 1.52 mg/dl at 30 min and started to decrease gradually to 131.66 ± 3.51 mg/dl at the end of 2 h.

In Group V, the residual sucrose content of the various parts of digestive tract after one hour was ranged from 143 µg/ml in stomach and 38 µg/ml in upper 79 µg/ml in middle and 16 µg/ml in lower intestines. The residual sucrose after 2 h was ranged from 54 µg/ml in upper, 60 µg/ml in middle and 81 µg/ml in lower intestines, 128 µg/ml in caecum and 8 µg/ml in large intestine revealed the transformation of undigested sucrose to the next parts in due course of time. The faecal matter of this group contain sucrose of about 2 mg/g this evidenced the inhibition by α-glucosidase inhibitors, that blocks the conversion of sucrose and leads to excrete the undigested sucrose through faecal matter. The blood

glucose of the rats of this group ranged from minimum of 76.33 ± 1.52 mg/dl at 0 h and sudden increase in blood glucose after sucrose administration which reached the maximum value of 182.66 ± 3.51 mg/dl at 30 min and started to decrease gradually to 130.00 ± 3.60 mg/dl at the end of 2 h.

There is no residual sucrose in Group VI, where no sucrose was administrated except α-glucosidase inhibitors. The blood glucose of the rats of this group ranged from minimum of 76.00 ± 3.60 mg/dl at 0 h and gradually decreased and reach the value of 63.33 ± 3.51 mg/dl at the end of 2 h.

DISCUSSION

It has long been known that actinobacteria can be isolated from marine sediments (Weyland, 1969; Jensen et al., 1991) raising the possibility that these bacteria, like their terrestrial counter parts in soils, play important role in the decomposition of recalcitrant organic matter in the sea floor. In the present study, population density of actinobacteria was ranged from 1.6×10^4 to 41×10^4 CFU/g in actinomycetes isolation agar and from 1.5×10^4 to 37×10^4 CFU/g in Kuster's agar. This population yielded 90 strains of morphological different colonies. Actinobacteria represents a small component of the total bacterial population in marine sediments (Weyland, 1969; Goodfellow and Williams, 1983) and their role in the marine environment is difficult to assess.

It has been proposed that most actinobacteria isolated from marine source of terrestrial origin, reside in the sea as spore or resting propagules (Goodfellow and Williams., 1983). Goodfellow and Haynes (1984) support this that actinobacteria isolated from North Atlantic sediments, includes Streptomyces, Micromonospora and *Rhodococcus* sp. have no specific seawater requirements and growing equally well on media prepared either distilled water or seawater. The actinobacteria are common soil bacteria, produce resistant spores and are known to be salt tolerant (Okazaki and Okami., 1975). This concept is supported by the observations of Goodfellow and Williams (1983) who stated that actinobacteria are less common in marine sediments relative to terrestrial soils. However, Macleod (1965) in the opinion that requirement of seawater for growth is a well-defined marine adaptation.

In the present investigation 5 strains (16.65%) showed seawater requirement for growth. The remaining 25 strains (83.25%) are able to grow in the absence of seawater but better growth was observed in the media containing seawater. This is in agreement with the results of Weyland (1969) for streptomycetes isolated from deep-sea sediments. Okami and Okazaki (1974) conclude that many actinobacteria can survive for a period of time in the sea and some can grow or survive in shallow sea bottoms. Therefore, shallow sea sites represent an interesting source of special actinobacteria which can survive and live under both terrestrial and marine environment.

Recently, marine derived actinobacteria have been considered as a source of novel antibiotics and anticancer agents (Faulkner, 2002; Mincer et al., 2002) suggesting that they represent a new resource for natural product drug discovery (Bull et al., 2000; Jensen et al., 2000). The prior evidence of existence of indigenous marine actinobacteria populations were reported earlier (Jensen et al., 1991; Takizawa et al., 1993; Colquhoun et al., 1998) and *in situ* metabolic activity was also reported (Moran et al., 1995). About two-third of the naturally-derived antibiotics in current use, and most of the important bioactive metabolites are synthesized by these gram-positive soil bacteria (Kim et al.,1998; Yoo et al., 2002; Weber et al., 2003). As the result of the complete sequencing of two genomes, *Streptomyces coelicolor* and *Streptomyces avermitilis,* was made the identification of a staggering number of gene clusters wholly devoted to the synthesis of secondary metabolites (Bentley et al., 2002; Ikeda et al., 2003). Bentley et al. (2002) counted 23 such clusters, while attempting to locate genes typical for secondary metabolism. The C7 N-aminocyclitol family is a relatively newly-discovered class of natural products, and is gaining increasing recognition, due to the significant biomedical and agricultural applications with which it has been associated.

Out of the 30 strains tested for the yeast α-glucosidase inhibitory activity, only 4 strains (13%) has showed inhibitory activity of above 50% while 10 (35%) strains showed inhibitory activity of less than 50% while the remaining 16 strains (52%) were not having any activity. Like in the case of yeast α-glucosidase inhibitors producing actinobacteria, only 7 strains were good in inhibiting rat α-glycosidase while other 24 strains were less effective. Interestingly the strain PSG-22 which showed maximum yeast α-glucosidase inhibition activity also showed maximum rat α-glucosidase inhibition. The strain PSG-22 which showed maximum inhibition of 90% was showed requirement of seawater for its growth. This is again one of the strong points that marine forms are of good source of secondary metabolites. The inhibitory effect of the actinobacteria against yeast and rat α-glucosidase showed the potential of actinobacteria to produce antidiabetic agents. *In vitro* yeast and rat α-glucosidase inhibitory study showed that only 4 strains and 6 strains having the ability to produce the yeast and rat α-glucosidase inhibitor compounds respectively. The 90% inhibition of yeast α-glucosidase and 78% inhibition of rat α-glucosidase shown by the strain PSG-22 was taken as the promising strain for further characterization using the present culture conditions. Though, there is no α- glucosidase inhibitor was reported other inhibitor activity was reported from marine strains (Imada and Simidu, 1992). The low and no inhibitory activity by the other strains might be their inability to produce

α-glucosidase inhibitor under present culture condition.

Kim et al. (1999) opinioned that the inhibition test carried out in *in vitro* using purified α-amylase, was not a suitable comparison for what occurs in the *in vivo.* Hence in the present study production of α-glucosidase inhibitors by the strain PSG-22 was confirmed by administrating the inhibitor in rat model. The oral administration of inhibitor solution followed by the administration of sucrose solution to the animals reveals the Hyperglycemic effect of the inhibitor solution.

The actinobacteria secondary metabolites shows pronounced inhibitory effect on a number of α-glycosidases of bacterial, fungal (Truscheit et al., 1981) and mammalian origin, including human intestinal α-glucosidases such as sucrase, maltase and glucoamylase (Caspary and Graf., 1979; Puls et al., 1980).

Alpha-glucosidase inhibitors interfere with enzymatic action in the brush border of the small bowel, slowing the breakdown of polysaccharides and disaccharides to glucose and thereby delaying glucose absorption and decreasing postprandial plasma glucose levels (Lebovitz, 1998). Due of this only the α-glucosidase inhibitors lowers the postprandial and not fasting plasma glucose levels (Bell, 2004). To date, most α-glucosidase inhibitors of microbial origin have been isolated from terrestrial actinobacteria and there are no reports on α-glucosidase inhibitor producing marine microbes except of Imada (2005) who reported the ability of the Streptomyces strains in production of inhibitors in presence of salt.

Alpha-glucosidase inhibitors can prolong the processes of carbohydrate absorption along the entire intestine, length; and flatten the blood glucose concentration over time curve (Bischoff., 1993). In this experiment lowering of postprandial blood glucose level after the administration of sucrose solution was strongly suppressed by the α-glucosidase inhibitor produced by the strain PSG- 22 and the percentage of inhibition is increased with the increase in dose for Group III and Group IV. However this dose dependency was not recorded in Group V, which receives 0.1 g/kg of inhibitors, and also the blood sugar value was not much lowered remain same as group IV. It showed that the effective dose of inhibitor for rat was around 0.06 g/kg. Such dose dependent blood glucose changes was well established by the Duncans analysis (Table 2) which showed three different levels of blood glucose levels in all the three experimental groups provided with different doses of inhibitors. The dose dependent inhibition of sucrose conversion was also observed by Goto et al. (1995) and Ye et al. (2002) with their respective glucosidase inhibitors. The inhibition of rapid rise in blood glucose levels by the administration as compared with the control 275.66 ± 4.50> 219.66 ± 5.50> 191.33 ± 1.52> 182.66 ± 3.51 mg/dl of 0.03, 0.06 and 1 g/kg of inhibitors respectively showed that the α- glucosidase inhibitor produced by the strain PSG- 22. Their α- glucosidase inhibitory activity can be used as first line drug in treatment of type-2 diabetes that is not controlled through diet alone.

The control of postprandial blood glucose was only through α-glucosidase inhibitors and this was confirmed by the residual sucrose in the digestive tract of the rat. The maximum residual sucrose in digestive tract is observed in the Group V, and also the residual sucrose was available in the faecal matter of this Group V (2 mg/gm). This was confirmed by the transformation of undigested sucrose to the next part of the digestive tract. The upper part of the small intestine was actively involved in the digestion of the sucrose, evidenced by the very low level of residual sucrose in that part and also by the non availability of sucrose in middle intestine in Group II after one hour.

The action of the actinobacteria inhibitor might be dependent on delaying rather than preventing sucrose absorption. This was confirmed by the sucrose content in 6 segments of the rat digestive tract after an oral sucrose load. The lower dose of inhibition increased the sucrose content of the upper and middle small intestine after 1 h, as well as that of the middle and lower small intestine after 2 h. Very low quantity of sucrose was present in the cecum or large intestine even after 2 h. These findings suggested that low dose of α-glucosidase inhibitor produced by the strain PSG-22 delayed but not suppressed sucrose absorption. On the other hand, the higher dose increased the sucrose content of the cecum and large intestine after 2 h. The differences in the gastrointestinal transit of sucrose between the lower and higher doses might be due to differences in the grade of α- glucosidase inhibition.

Conclusion

The findings of the present study suggest that, α-glucosidase inhibitor produced by the strain PSG- 22 can delay sucrose absorption without suppressing it at certain dose levels. In other words, a certain optimal dose of α-glucosidase inhibitor produced by the strain PSG- 22 inhibits rapid digestion and absorption of carbohydrate at the small intestine and un-digested carbohydrate could be digested and absorbed beyond these parts completely before reaching the cecum. From the results, it is confirmed that the actinobacteria strain PSG- 22 is the potential α-glucosidase inhibitor producers which can be explored further for characterization of α-glucosidase inhibitor compounds.

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