Research report

Effect of Caralluma Fimbriata extract on appetite, food intake and anthropometry in adult Indian men and women

Rebecca Kuriyan[a],*, Tony Raj[a], S.K. Srinivas[b], Mario Vaz[a], R. Rajendran[c], Anura V. Kurpad[a]

[a]*Division of Nutrition, Institute of Population Health and Clinical Research, St John's National Academy of Health Sciences, Bangalore 560034, India*
[b]*Gencor Pacific Group, USA*
[c]*Green Chem Limited, Domlur, Bangalore, India*

Abstract

Caralluma fimbriata is an edible cactus, used by tribal Indians to suppress hunger and enhance endurance. The effect of Caralluma extract was assessed in overweight individuals by a placebo controlled randomized trial. Fifty adult men and women (25–60 years) with a body mass index (BMI) greater than 25 kg/m^2 were randomly assigned into a placebo or experimental group; the latter received 1 g of Caralluma extract per day for 60 days. All subjects were given standard advice regarding a weight reducing diet and physical activity. At the end of 30 and 60 days of intervention, blood glucose and lipids, anthropometric measurements, dietary intake and assessment of appetite was performed. Waist circumference and hunger levels over the observation period showed a significant decline in the experimental group when compared to the placebo group. While there was a trend towards a greater decrease in body weight, body mass index, hip circumference, body fat and energy intake between assessment time points in the experimental group, these were not significantly different between experimental and placebo groups. Caralluma extract appears to suppress appetite, and reduce waist circumference when compared to placebo over a 2 month period.
© 2006 Elsevier Ltd. All rights reserved.

Keywords: Caralluma; Appetite; Anthropometry; Food intake

Introduction

Obesity is a major global health problem and a risk factor for several chronic disorders such as diabetes, hyperlipidemia, hypertension and cardiovascular disease. Weight gain and obesity are a result of positive energy balance due to a mismatch between energy intake (EI) and energy expenditure (EE). The EI in turn is subject to a wide range of influences, including appetite, gastro-intestinal signals such as distension of the stomach, chemical signals to the gastric mucosa and blood-borne metabolites such as glucose and fatty acids. Strategies to reduce a positive energy balance have often focused on increasing EE since it was thought that the EI of obese individuals was normal or low and additionally, because of the independent effects of

physical activity on the reduction of risk for many chronic disorders associated with obesity. However, overweight subjects often under-report their food intake (Lissner, 2002; Lissner, Heitmann, & Bengtsson, 2000), and carefully conducted objective measurements have shown that EI is in fact increased in obese subjects (Lichtman et al., 1992; Schoeller, 1990). The complex process of appetite is controlled by several neural, humoral and psychological factors (Bray, 2000), and strategies that suppress appetite are likely to be useful in weight loss and control. Appetite suppressant medications, while effective often have side effects (Haller & Benowitz, 2000).

Traditional health care systems, including herbal medicine are widespread in developing countries (WHO, 2002). Certain herbs, used by native people have also been studied for their appetite suppressing effects. This includes Hoodia gordonii, which is a succulent from the Kalahari desert of South Africa (MacLean & Luo, 2004). In India, Caralluma

*Corresponding author. Tel./fax: 91 80 25532037.
E-mail address: rebecca@iphcr.res.in (R. Kuriyan).

fimbriata, an edible succulent cactus that belongs to the family Asclepiadaceae is also well known as a famine food, appetite suppressant and thirst quencher among tribal populations. It grows wild all over India and is also planted as a roadside shrub and boundary marker in gardens. Native Indian diets over many centuries have included these edible wild succulent cacti, with claims in folklore about its appetite suppressant activity. There has been no previous controlled study on the appetite suppressing effects of Caralluma and this is the first study to examine the effects of Caralluma experimentally. The aim of the present study was to evaluate the effectiveness of Caralluma fimbriata extract on appetite suppression, food choice and anthropometry in overweight and obese individuals who wished to lose weight.

Methods

Subjects

The study was a double blind, placebo controlled, randomized trial. Sixty two healthy volunteers (23 male and 39 female subjects) in the age group of 25–60 years, with a body mass index (BMI) greater than 25 kg/m^2 were recruited into the study. Exclusion criteria were the presence of any chronic disease and the use of any medication for weight loss. The subjects included staff and individuals who visited the Nutrition Clinic of St. John's Medical College Hospital, Bangalore, in order to lose weight. After recruitment, the subjects were randomly assigned into the placebo or experimental group. Twelve subjects dropped out during the study (equal numbers in both groups with no gender bias) and 50 subjects completed the study, 25 subjects each in the placebo and the experimental group. The study was approved by the institutional ethical review committee of St. John's Medical College and an informed consent was obtained from the subjects.

Experimental protocol

The Caralluma extract was made from the aerial parts of the plant with aqueous alcohol which was 40% aqueous (40 parts of alcohol and 60 parts of water). About 12 kg of dried herb was obtained from 100 kg of the fresh plant, which gave a final yield of 1 kg of the extract. It was then purified, granulated and filled in capsules to deliver 500 mg of the extract. Maltodextrin capsules (500 mg) were used as placebo, and both capsules were prepared by Green Chem Limited, Bangalore, India. Prior to the intervention, the subjects underwent baseline investigations which included anthropometric, biochemical, dietary and appetite assessment. The extract was administered as two 500 mg capsules daily (1 g/day) for 60 days, during which the subjects reported weekly to the Nutrition Clinic to record their body weight, collect their weekly capsule supply and report adverse events, if any. The compliance of the subjects to the ingestion of capsules was measured every week when they reported to the Nutrition Clinic. The subjects were provided with a capsule calendar in which they were required to tick mark boxes relating to the daily intake of capsules and also to note down any missed capsule. The calendar and the 'missed' pill count were monitored every week.

Anthropometric measurements

Anthropometric measurements included body weight, height, skinfold thickness and mid-arm, waist and hip circumferences. All the measurements were standardized (Harrison et al., 1988). Skinfold measurements in triplicates were carried out using Holtain skinfold calipers, at four sites (i.e.) biceps, triceps, subscapular and suprailiac. The average sum of four skinfold measurements were used to compute body density using the age and gender specific equations (Durnin & Womersley, 1974) and percent body fat was derived from body density (Siri, 1961). These equations were previously validated in a group of Indian men and women (Kuriyan, Petracchi, Ferro-Luzzi, Shetty, & Kurpad, 1998). The corrected arm muscle area (CAMA) was calculated using the mid-arm circumference and tricep skinfold (Heymsfield, McManus, Smith, Stevens, & Nixon, 1982). The measurements were repeated at day 30 and day 60 of the administration period.

Biochemical measurements

Fasting blood glucose and lipid profile were measured at baseline, day 30 and day 60 of the study period. The blood glucose, triglyceride, total and HDL cholesterol were estimated by automated spectrophotometric assays (Dade Behring Dimension $R \times L$, Newark, USA), while LDL cholesterol was calculated from primary measurements using the empirical formula of Friedewald equation (Friedewald, Levy, & Fredrickson, 1972). All assays were calibrated by use of Dade Dimension human calibrator (Dade Behring Inc, Newark, USA). The analytical coefficient of variation (inter-assay) for total cholesterol, triglycerides and HDL cholesterol were 4.1%, 4.7% and 4.1%, respectively.

Dietary and physical activity assessment

Dietary assessment was carried out using a modified food frequency questionnaire with 129 food items which was developed specifically for the urban south Indian population (Rastogi et al., 2004). This questionnaire was administered at the baseline and the end of the study. A validated physical activity questionnaire (Bharathi, Sandhya, & Vaz, 2000) was used to assess the daily integrated physical activity of the patient at baseline, day 30 and day 60 of the study. All the study subjects were provided with standard health advice on diet and physical activity targeted to achieve a weight loss of about 5–10%

body weight over the study period. The compliance of the subjects to the prescribed diet and physical activity was assessed weekly by asking the subjects to rate their compliance on a scale of 0–100%.

Appetite assessment

The appetite of the subject was assessed at baseline, day 30 and day 60 of the study in the fasted state. Four 100 mm visual analogue scales (VAS, Silverstone, 1981) for 'hunger', 'thoughts of food', 'urge to eat', and 'fullness of stomach' were administered. The scale was administered each time, in triplicate. The mean of the three readings was expressed as a percentage of the scale.

Statistics

The data are presented as Mean ± SD. An independent 't' test analysis was performed to ascertain whether significant differences existed between the physical characteristics of the subjects in the experimental and placebo group at baseline. A repeated measure ANOVA with group as a factor was performed to assess the change over time in the anthropometric, biochemical and visual analog scale parameters between the two groups. The repeated measure ANOVA was then used to assess for significant differences between the various time points in the subjects of both groups independently. The paired 't' test analysis was carried out to ascertain significant differences in the mid-arm circumference, CAMA and in the food intake of subjects belonging to both the groups between the time points. The significance level was set at $p < 0.05$.

Results

The physical characteristics of the subjects in the experimental and placebo groups are summarized in Table 1. The age range of the subjects in the experimental group was 28–53 and 28–52 years in the placebo group. There were no significant differences in the mean age, weight,

Table 1
Physical characteristics of the subjects

Parameter	Experimental group ($n = 25$)	Placebo group ($n = 25$)
Age (yr)	38.6 ± 7.8	38.9 ± 6.1
Body weight (kg)	79.5 ± 16.9	78.2 ± 9.3
Height (cm)	160.9 ± 9.1	162.3 ± 9.4
Body mass index (kg/m^2)	30.6 ± 5.5	29.8 ± 3.9
Waist circumference (cm)	96.9 ± 11.6	95.1 ± 9.6
Hip circumference (cm)	106.3 ± 11.4	107.3 ± 7.2
Percent body fat (%) #	34.6 ± 5.6	34.2 ± 5.4

Mean ± standard deviation (SD).
#—Calculated from the sum of four skinfold measurements and applying the formulae of Durnin and Womersley (1974).
No significant differences were observed between the physical characteristics of the subjects of the two groups (independent 't' test).

height, BMI, waist, hip circumferences and percent body fat between the experimental and placebo groups.

The anthropometric parameters of the subjects in the experimental and placebo groups at various time points of the study are summarized in Table 2. A significant interaction effect was observed between time and the group (repeated measure ANOVA) in the waist circumference. There were no significant differences observed in the change of body weight, BMI, hip circumference and percent body fat over time between the two groups (repeated measure ANOVA). At the end of the study period, significant decreases in body weight, BMI, waist and hip circumferences were observed only in the experimental group, when compared to baseline parameters ($p < 0.01$). The weight loss in the experimental group accounted to 2.5% (2 kg) of the initial body weight. In contrast the placebo group had a non-significant reduction of 1.3% (1 kg) during the study period. In the placebo group, the only significant change observed was in the hip circumference which significantly decreased at month 1 when compared to the baseline values.

The data on appetite assessment from the VAS and on dietary intake assessment are presented in Table 3. The mean 'hunger levels' at baseline of the experimental group tended to be higher (but not significantly) when compared to placebo group, while at day 60 the mean 'hunger levels' of the experimental group was significantly lower than those of the placebo group; this accounts for the significant interaction effect between time and the group (repeated measure ANOVA). There were no significant differences observed in the change of 'thought of food', 'feeling of fullness' and 'urge to eat' over time between the two groups (repeated measure ANOVA). By the end of the study period, the experimental group had a 7.2% increase in the 'feeling of fullness' (NS), a 9.5% decrease in 'urge to eat' (NS) and 19.7% decrease in 'hunger' levels ($p < 0.05$). Corresponding changes in the placebo group were 0.8%, 1.8%, 1.2% (all NS). Changes in 'thoughts of food' were minimal in both groups (1.3%) in experimental group, 1.1% in the placebo group and not significant. Significant reductions ($p < 0.05$) in energy and macronutrient intake at the end of the study period were observed only in the experimental group. This amounted to 188 kcal/day (8.2%) for energy, 20 g (5.2%) for carbohydrate, 4.7 g (8%) for fat and 3.6 g (5.7%) for protein. Further, the intake of cereals, roots and tubers, sugars and sweets, egg and meat products in the experimental group was significantly lower at the end of the study when compared to the baseline, while the intake of fruits, vegetables and fish remained the same. In the placebo group of subjects, there was no change in the intake of nutrients, or in food groups at the end of the study.

The biochemical parameters of the subjects belonging to both the experimental and the placebo group are presented in Table 4. There were no significant differences observed in the change of the biochemical parameters over time between the two groups (repeated measure

Table 2
Anthropometric parameters of the subjects at baseline, day 30 and day 60 of the study

Parameter	Baseline	Day 30	Day 60	F value	p value
Body weight (kg)					
Experimental	79.5 ± 16.9	78.3 ± 16.5[a]	77.5 ± 16.0[a,b]	1.9	0.15
Placebo	78.2 ± 9.3	77.5 ± 8.9	77.2 ± 8.6		
Body mass index (kg/m^2)					
Experimental	30.6 ± 5.5	30.2 ± 5.6[a]	29.9 ± 5.6[a,b]	2.1	0.13
Placebo	29.8 ± 3.9	29.6 ± 4.0	29.5 ± 4.0		
Waist circumference(cm)					
Experimental	96.9 ± 11.6	95.1 ± 12.0[a]	93.9 ± 11.3[a,b]	6.8	<0.001[c]
Placebo	95.1 ± 9.6	94.4 ± 9.43	94.3 ± 9.6		
Hip circumference(cm)					
Experimental	106.3 ± 11.4	105.8 ± 11.5	105.0 ± 11.6[a,b]	2.06	0.13
Placebo	107.2 ± 7.2	106.4 ± 7.9[a]	106.4 ± 7.0		
Percent fat (%) #					
Experimental	34.6 ± 5.6	34.2 ± 5.3	33.4 ± 5.6[a]	2.8	0.07
Placebo	34.2 ± 5.9	34.1 ± 5.5	34.0 ± 5.4		

Mean ± SD.

#—Calculated from the sum of two, three or four skinfold measurements and applying the formulae of Durnin and Womersley (1974)

[a] Mean value was significantly different from that of baseline (repeated measure ANOVA; $p < 0.05$).

[b] Mean value was significantly different from that of Day 30 (repeated measure ANOVA; $p < 0.05$).

[c] Significant interaction between time points and group (repeated measure ANOVA with group as between subjects factor).

Table 3
Appetite and food intake assessment

Parameter	Study group	Baseline (Day 0)	Day 30	Day 60	F value	p value
Thoughts of food (%)	Experimental	34.5 ± 24.3	36.2 ± 21.6	33.2 ± 23.6	1.6	0.21
	Placebo	33.1 ± 20.3	33.6 ± 15.3	32.0 ± 17.5		
Feeling fullness (%)	Experimental	33.3 ± 19.3	41.7 ± 20.5	40.5 ± 21.9	0.84	0.44
	Placebo	37.8 ± 26.9	36.6 ± 17.9	38.6 ± 21.2		
Urge to eat (%)	Experimental	44.0 ± 25.3	39.3 ± 21.5	34.5 ± 21.2	1.78	0.18
	Placebo	35.3 ± 25.3	35.2 ± 17.5	33.5 ± 19.7		
Hunger (%)	Experimental	47.6 ± 22.6	39.2 ± 21.4	27.9 ± 18.8[a]	6.6	<0.001[b]
	Placebo	41.9 ± 24.1	41.6 ± 17.3	40.7 ± 18.9		
Energy intake (kcal/day)[c]	Experimental	2276.5 ± 202.3		2088.8 ± 183.4[d]		
	Placebo	2303.6 ± 107.9		2299.0 ± 109.0		
Fat intake (g/day)[c]	Experimental	59.0 ± 6.4		54.3 ± 3.9[d]		
	Placebo	61.7 ± 2.8		60.9 ± 3.9		
Carbohydrate intake (g/day)[c]	Experimental	360.9 ± 26.1		340.5 ± 23.7[d]		
	Placebo	377.9 ± 21.1		376.8 ± 21.3		
Protein intake (g/day)[c]	Experimental	62.9 ± 6.3		59.3 ± 7.2[d]		
	Placebo	59.1 ± 4.5		59.3 ± 4.8		

Mean values ± Standard deviation. The appetite assessment was carried using Visual Analog Scales and results were expressed as percentage of the scale.

[a] Mean value was significantly different from that of baseline (repeated measure ANOVA; $p < 0.05$).

[b] Significant interaction between time points and group (repeated measure ANOVA with group as between subjects factor). Significant differences between time points were assessed using repeated measure ANOVA with post hoc corrections.

[c] These parameters were measured twice and no interaction term is available. The food intake assessment was carried using food frequency questionnaires at baseline and end of the study and significant differences between time points were assessed using paired 't' test.

[d] Mean value was significantly different from that of baseline.

ANOVA). There were no significant differences observed between the time points in both the experimental and placebo group.

The mean compliance of the subjects in the experimental group to prescribed diet was 72% (55–88%) and 70% (53–88%) to prescribed physical activity, while in the

Table 4
Biochemical parameters of the subjects at baseline, Day 30 and Day 60 of the study

Parameter	Baseline	Day 30	Day 60	*F* value	*p* value
Fasting blood sugar (mg/dl)					
Experimental	89.8 ± 16.8	89.3 ± 10.3	88.9 ± 10.4	0.05	0.95
Placebo	90.5 ± 14.3	89.0 ± 10.9	91.7 ± 11.1		
Post prandial sugar (mg/dl)					
Experimental	110.9 ± 35.3	108.3 ± 25.2	108.6 ± 27.2	0.69	
Placebo	101.9 ± 21.9	106.1 ± 18.1	104.3 ± 25.0		0.50
Total cholesterol (mg/dl)					
Experimental	192.5 ± 27.1	191.0 ± 25.9	191.8 ± 27.0		
Placebo	195.1 ± 36.3	194.8 ± 39.2	196.2 ± 37.3	0.01	0.99
HDL cholesterol (mg/dl)					
Experimental	65.9 ± 11.0	63.6 ± 10.0	64.1 ± 9.95	0.53	0.59
Placebo	56.3 ± 10.8	55.6 ± 13.1	56.1 ± 11.0		
LDL cholesterol (mg/dl)					
Experimental	120.0 ± 39.8	118.0 ± 27.9	115.7 ± 30.3		
Placebo	124.3 ± 38.2	126.3 ± 46.4	128.9 ± 54.6	0.71	0.50
Serum triglycerides (mg/dl)					
Experimental	111.9 ± 52.0	112.1 ± 55.0	110.3 ± 51.5	0.19	0.82
Placebo	101.8 ± 44.3	102.3 ± 37.9	101.3 ± 38.4		

Mean ± SD.
No significant differences in the biochemical parameters at various time points. Significant interaction between time points and group were assessed using repeated measure ANOVA with group as between subjects factor Significant differences between time points were assessed using repeated measure ANOVA with post hoc corrections.

placebo group it was 67% (50–87%) to prescribed diet and 72% (53–88%) to prescribed physical activity. The physical activity level (PAL) of both the experimental and placebo group did not change significantly during the study. The PAL of the experimental group was 1.59 ± 0.03 at the start of the study and 1.58 ± 0.08 at the end of the study period, while in the placebo group, it was 1.59 ± 0.03 at the start of the study and 1.60 ± 0.02 at the end of the study period.

There were no serious adverse events reported by the subjects of the present study. The observed adverse events were minor and limited to initial mild symptoms of the gastrointestinal tract such as abdominal distention, flatulence, constipation and gastritis. Six (24%) of the subjects from the experimental group and five (20%) of the subjects from the placebo group experienced these minor adverse effects. These symptoms were present in both the experimental and placebo group of subjects. The symptoms subsided within a week in all subjects. An animal study (Kurpad et al., unpublished) conducted to determine the LD50 of the extract did not reveal any toxicity and the LD50 was greater than 5g/kg.

Discussion

Food consumption in humans is regulated through a number of complex biological mechanisms which ensures that body weight is relatively constant over long periods. Appetite regulates the body's desire for food through a complex biological process designed to satisfy the body's need for energy, protein, fat, carbohydrates and other nutrients (Beckman et al., 2005). Appetite therefore plays an important role in weight regulation, and obese individuals have been shown to have an increased appetite and eating disorders such as binge eating, night eating or compulsive overeating disorders (Keefe, Wyshogrod, Weinberger, & Agras, 1984; Spitzer et al., 1991). Thus measures to reduce appetite of overweight and obese individuals could help in preventing further weight gain and in enhancing weight reduction.

Herbs contain a wide variety of active phytochemicals, such as flavanoids, terpenoids, lignans, polyphenols, saponins, plant sterols and cartenoids, and there is now a lot of interest in herbs that possess hypolipidemic, antiplatelet, anti-tumour and immune stimulating properties (Craig, 1999). Caralluma fimbriata, which grows widely in India, is associated with folklore of appetite reduction, and it is of interest to verify this effect through controlled studies. There are several varieties of Caralluma that grow in India although these species are botanically and phytochemically similar. The key phytochemical ingredients in Caralluma are pregnane glycosides, flavone glycosides, megastigmane glycosides, bitter principles, saponins and various other flavonoids (Bader, Braca, De Tommasi, & Morelli, 2003). The appetite suppressing action of Caralluma could be attributed to the pregnane glycosides, which are particularly rich in plants belonging to the Asclepiadaceae family (Christiane, Klaus, & Eberhard, 1993). It is unclear as to how pregnane glycosides or its related molecules may suppress appetite, and it is thought that they amplify the signaling of the energy

sensing function in the basal hypothalamus (MacLean & Luo, 2004). A similar appetite suppressing action has been observed in the South African cactus-like plant Hoodia, in which a steroidal glycoside was isolated, which demonstrated anorectic activity in animals (MacLean & Luo, 2004).

In the present study, the hunger levels of the subjects decreased by 20% after the administration period and this could account for the 8% decrease in the EI of the experimental group of subject. However, it should be remembered that food intake could tend to be under/over estimated and there could also be individual variations. The 8% significant calorie deficit (amounting to about 187 kcal/day), if lost as purely fat, would account for a weight loss of about 1.25 kg over the study period. The actual weight loss would have been more if some lean tissue (protein has a lower heat equivalent) was also lost. The observed weight loss in the experimental group of subjects was about 2 kg, while the loss of fat mass was 1.5 kg after 2 months of intervention. This weight loss can therefore be explained by the EI deficit. It seems unlikely that significant amounts of muscle mass may have been lost, since, first, there was a significant decrease of about 3 cm in the waist circumference, second, the mid-arm circumference (which admittedly is measure of both muscle and fat) of the experimental group did not change $(32.1 + 2.1$ cm vs. $32.2 + 2.1$ cm, day 0 and day 60, respectively, $P = 0.87)$. The CAMA also did not change $(56.5 + 7.2$ cm^2 vs. $56.7 + 7.3$ cm^2 between day 0 and day 60, respectively, $P = 0.84)$ and third, the subjects in general followed their physical activity advice with a compliance of about 70%. The decline in waist circumference (3 cm in 2 months) is important since waist circumference is reflective of intra-abdominal fat which is strongly associated with risks of metabolic and cardiovascular disease and risk of other chronic non communicable diseases (Despres, 2006; Despres et al., 1990; Reeder et al., 1997). Abdominal adiposity is also associated with insulin resistance (Carey, Jenkins, Campbell, Freund, & Chisholm, 1996; Kissebah et al., 1982). The waist circumference is a convenient measure of intra-abdominal adipose tissue (Han, van Leer, Seidell, & Lean, 1995; Pouliot et al., 1994), and has been described as a risk factor independent of BMI for cardiovascular disease (Rexrode et al., 1998). It is particularly important in Indians who seem to have a predilection for accumulation of fat in this region (Anjana et al., 2004; Raji, Seely, Arky, & Simonson, 2001; Ramachandran et al., 2001). It is not clear why the waist circumference specifically declined in this study independent of body weight. While one possibility is that this was simply an early indicator over the relatively short intervention, the other possibility is that fat in different depots of the body have different rates of lipolysis during negative energy balance or fasting (Monzon, Basile, Heneghan, Udupi, & Green, 2002), if it can be assumed that there was a negative energy balance induced by the intake of Caralluma extract. Even under lipolytic stimuli like

noradrenaline stimulation for instance, subcutaneous fat in the anterior abdominal wall has a different rates of lipolysis when compared with whole body lipolytic rates (Kurpad et al., 1994).

An additional intriguing feature observed in the experimental group of subjects was a significant reduction in the intake of refined sugars, sweets, cholesterol and saturated fats at the end of the study, while the intake of fruits, vegetables and fish remained unchanged. The consumption of foods such as whole grains, fruits and vegetables has been found to be directly associated with reduction in hunger and increased satiety levels, which could lead to lowered voluntary energy intake (Roberts & Heyman, 2000). Similarly, the reduction in fat intake as a means to reduce EI results in weight loss (Bray & Popkin, 1998). The subjects in the experimental group seemed to have made healthy food choices in their diet after 2 months of the administration period, compared to the placebo group. This interesting aspect of the effect of Caralluma needs more study. We were unable however to explain the lack of any change in the lipid values of the experimental group, despite the reduction in the intake of cholesterol and saturated fats.

The results of the present study suggest that Caralluma fimbriata has a potential appetite suppressing action. This concurs with the anecdotal evidence provided by tribal populations in India. While there was no significant effect of Caralluma on body weight, there was a significant reduction in waist circumference. The effect of appetite suppression translated into a net effect of a reduction in energy and fat intake and was accompanied by a reduction in intake of less desirable food groups, while the intake of desirable (healthy) food groups remained unchanged.

Acknowledgements

References

Anjana, M., Sandeep, S., Deepa, R., Vimaleswaran, K. S., Farooq, S., & Mohan, V. (2004). Visceral and central abdominal fat and anthropometry in relation to diabetes in Asian Indians. *Diabetes Care*, *27*, 2948–2953.

Bader, A., Braca, A., De Tommasi, N., & Morelli, I. (2003). Further constituents from Caralluma negevensis. *Phytochemistry*, *62*, 1277–1281.

Beckman, T., Shi, Q., & Billington, C. (2005). The brain and the biology of obesity. *Minnesota Medicine*, *88*(9), 58–61.

Bharathi, A. V., Sandhya, N., & Vaz, M. (2000). The development and characteristics of a physical activity questionnaire for epidemiological studies in urban middle class Indians. *Indian Journal of Medical Research*, *111*, 95–102.

Bray, G. A., & Popkin, B. M. (1998). Dietary fat intake does affect obesity. *American Journal of Clinical Nutrition*, *68*, 1157–1173.

Carey, D. G., Jenkins, A. B., Campbell, L. V., Freund, J., & Chisholm, D. J. (1996). Abdominal fat and insulin resistance in normal and overweight women: Direct measurements reveal a strong relationship in subjects at both low and high risk of NIDDM. *Diabetes*, *45*, 633–638.

Christiane, S., Klaus, R., & Eberhard, B. (1993). *Liebigs Annual Chemistry*, *10*, 1057.

Craig, W. J. (1999). Health promoting properties of common herbs. *American Journal of Clinical Nutrition*, *70*, 491S–499S.

Despres, J. P. (2006). Intra-abdominal obesity: An untreated risk factor for Type 2 diabetes and cardiovascular disease. *Journal of Endocrinological Investigation*, *29*(3 Suppl), 77–82.

Despres, J. P., Moorjani, S., Lupien, P. J., Tremblay, A., Nadeau, A., & Bouchard, C. (1990). Regional distribution of body fat, plasma lipoproteins, and cardiovascular disease. *Arteriosclerosis*, *10*, 497–511.

Durnin, J. V. G. A., & Womersley, J. (1974). Body fat assessed by total body density and its estimation from skinfold thickness: Measurements on 481 men and women aged 16–72 years. *British Journal of Nutrition*, *32*, 77–97.

Friedewald, W. T., Levy, R. I., & Fredrickson, D. S. (1972). Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clinical Chemistry*, *18*, 499–502.

Haller, C. A., & Benowitz, N. L. (2000). Adverse cardiovascular and central nervous system events associated with dietary supplements containing ephedra alkaloids. *New England Journal of Medicine*, *343*(25), 1833–1838.

Han, T. S., van Leer, E. M., Seidell, J. C., & Lean, M. E. (1995). Waist circumference action levels in the identification of cardiovascular risk factors: Prevalence study in a random sample. *British Medical Journal*, *25*, 1401–1405.

Harrison, G. G., Buskirk, E. R., ECarter, L., Johnston, F. E., Lohman, T. G., Pollock, M. L., et al. (1988). Skinfold thicknesses and measurement technique. In T. G. Lohman, A. F. Roche, & R. Martorell (Eds.), *Anthropometric standardization reference manual* (pp. 55–71). Illinois: Human Kinetics Books.

Heymsfield, S. B., McManus, C., Smith, J., Stevens, V., & Nixon, D. W. (1982). Anthropometric measurement of muscle mass: Revised equations for calculating bone-free arm muscle area. *American Journal of Clinical Nutrition*, *36*, 680–690.

Keefe, P. H., Wyshogrod, D., Weinberger, E., & Agras, W. S. (1984). Binge eating and outcome of behavioral treatment of obesity: A preliminary report. *Behavioral Research Therapy*, *22*, 319–321.

Kissebah, A. H., Vydelingum, N., Murray, R., Evans, D. J., Hartz, A. J., Kalkhoff, R. K., et al. (1982). Relation of body fat distribution to metabolic complications of obesity. *Journal of Clinical Endocrinology and Metabolism*, *54*, 254–260.

Kuriyan, R., Petracchi, C., Ferro-Luzzi, A., Shetty, P. S., & Kurpad, A. V. (1998). Validation of expedient methods for measuring body composition in Indian adults. *Indian Journal of Medical Research*, *107*, 37–45.

Kurpad, A., Khan, K., Calder, A. G., Coppack, S., Frayn, K., Macdonald, I., et al. (1994). Effect of noradrenaline on glycerol turnover and lipolysis in the whole body and subcutaneous adipose tissue in humans in vivo. *Clinical Science(London)*, *86*, 177–184.

Lichtman, S. W., Pisarska, K., Raynes Berman, E. R., Pestone, M., Dowling, H., Offenbacher, E., et al. (1992). Discrepancy between self-reported and actual caloric intake and exercise in obese subjects. *New England Journal of Medicine*, *327*, 1893–1898.

Lissner, L. (2002). Measuring food intake in studies of obesity. *Public Health Nutrition*, *5*(6A), 889–892.

Lissner, L., Heitmann, B. L., & Bengtsson, C. (2000). Population studies of diet and obesity. *British Journal of Nutrition*, *83*(Suppl 1), S21–S24.

MacLean, D. B., & Luo, L. G. (2004). Increased ATP content/production in the hypothalamus may be a signal for energy-sensing of satiety: Studies of the anorectic mechanism of a plant steroidal glycoside. *Brain Research*, *1020*, 1–11.

Monzon, J. R., Basile, R., Heneghan, S., Udupi, V., & Green, A. (2002). Lipolysis in adipocytes isolated from deep and superficial subcutaneous adipose tissue. *Obesity Research*, *10*, 266–269.

Pouliot, M. C., Despres, J. P., Lemieux, S., Moorjani, S., Bouchard, C., Tremblay, A., et al. (1994). Waist circumference and abdominal sagittal diameter: Best simple anthropometric indexes of abdominal visceral adipose tissue accumulation and related cardiovascular risk in men and women. *Amercian Journal of Cardiology*, *73*, 460–468.

Raji, A., Seely, E. W., Arky, R. A., & Simonson, D. C. (2001). Body fat distribution and insulin resistance in healthy Asian Indians and Caucasians. *Journal of Clinical Endocrinolgy and Metabolism*, *86*, 5366–5371.

Ramachandran, A., Snehalatha, C., Kapur, A., Vijay, V., Mohan, V., Das, A. K., et al. (2001). Diabetes Epidemiology Study Group in India (DESI). High prevalence of diabetes and impaired glucose tolerance in India: National Urban Diabetes Survey. *Diabetologia*, *44*, 1094–1101.

Rastogi, T., Reddy, K. S., Vaz, M., Spiegelman, D., Prabhakaran, D., Willett, W. C., et al. (2004). Diet and risk of ischemic heart disease in India. *American Journal of Clinical Nutrition*, *79*, 582–592.

Reeder, B. A., Senthilselvan, A., Despres, J. P., Angel, A., Liu, L., Wang, H., et al. (1997). The association of cardiovascular disease risk factors with abdominal obesity in Canada. Canadian Heart Health Surveys Research Group. *Candian Medical Association Journal*, *1*(Suppl 1), S39–S45.

Rexrode, K. M., Carey, V. J., Hennekens, C. H., Walters, E. E., Colditz, G. A., Stampfer, M. J., et al. (1998). Abdominal adiposity and coronary heart disease in women. *Journal of American Medical Association*, *280*, 1843–1848.

Roberts, S. T., & Heyman, M. B. (2000). Dietary composition and obesity: Do we need to look beyond dietary fat? *Journal of Nutrition*, *130*, 267S.

Schoeller, D. A. (1990). How accurate is self-reported dietary energy intake? *Nutrition Review*, *48*, 373–379.

Silverstone, T. (1981). Techniques for evaluating antiobesity drugs in man. In P. Bjorntorp, M. Cairella, & A. N. Howard (Eds.), *Recent advances in obesity research* (pp. 173–179). London: John Libbey.

Siri, W. E. (1961). Body composition from the fluid spaces and sensity: Analysis of methods. In J. Brozek, & A. Henschel (Eds.), *Techniques for measuring body composition* (pp. 223–244). Washington: National Academy of Sciences NRC.

Spitzer, R. L., Stunkard, A., Yanovski, S., Marcus, M. D., Wadden, T., & Wing, R. etal. (1991). Binge eating disorders: Its further validation in a multisite study. *International Journal of Eating Disorders*, *13*, 137–153.

WHO. (2002). *WHO traditional medicine strategy 2002–2005*. Geneva: World Health Organization.