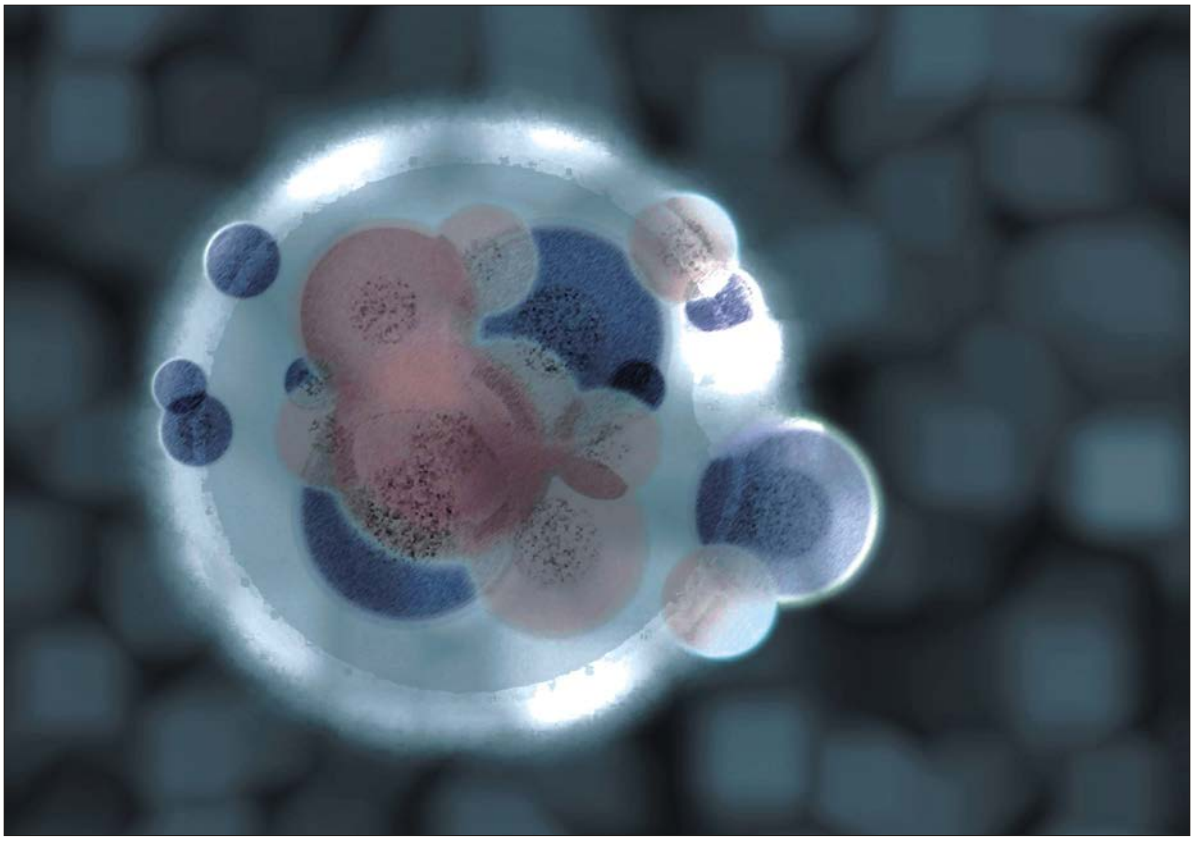

Journal of Biological Sciences

ISSN 1727-3048

science alert

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Journal of Biological Sciences 7 (7): 1201-1207, 2007
ISSN 1727-3048

The Effects of Some Over-The-Counter Dietary Weight Loss Supplements on Growth, Hepatic Glycogen Stores, Liver Lipid Profile, Pancreatic Protein and the Gastrointestinal Tract of Rats

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Abstract: The high prevalence of obesity has spawned a multimillion dollar industry marketing quick-fix supplements for weight loss. The supplements together with calorie restriction are mainly used by adolescents who wrongly perceive themselves as overweight. Calorie restriction and the supplements may have adverse effects at a critical growth stage. The short term effects of 3 classes of weight loss supplements on growth, the viscera, the exocrine pancreas, hepatic lipids and glycogen, of rats on restricted feed intake was investigated. Forty eight male Sprague Dawley rats were fed standard rat feed *ad libitum* or restricted to 10% body mass and supplemented with a weight-loss product (carbohydrate blocker, fat blocker or, metabolism booster) for 14 days. Feed restriction significantly ($p<0.01$) decreased body mass gain. Restricted feed intake and the weight loss supplements did not significantly ($p>0.05$) affect GIT morphology, hepatic lipid profile and hepatic glycogen stores, compared to *ad libitum* feeding. The rats supplemented with carbohydrate blocker had significantly larger testes than the others. The fat blocker significantly increased ($p<0.05$) soluble pancreatic proteins. The reduction in body mass gain was due to the feed restriction as opposed to the weight loss products. The fat blocker and carbohydrate blocker potentially have adverse effects in growing male rats.

Key words: Phaseolus, chitosan, kelp, gastrointestinal tract, growth

INTRODUCTION

Obesity is a problem of epidemic proportions worldwide, with more than half of the US adult population being classified as obese or overweight. The high prevalence of obesity has resulted in the need for weight loss becoming an important and topical issue (Kruger *et al*., 2004; Orzano and Scott, 2004; Schoeller and Buchholz, 2005). Most people wishing to lose weight tend to opt for quick and effortless weight loss strategies. The consequence of which has been the establishment and growth of a multi-million dollar industry marketing quick-fix weight loss measures and products (Blanck *et al*., 2001; Egger *et al*., 1999; Saper *et al*., 2004). Many consumers use these products without accurate knowledge of their full effects (Woods *et al*., 2004). Several studies have shown that these weight loss products are mainly used by adolescents, who incorrectly perceive themselves as overweight (Kant *et al*., 2002; Whisenhunt *et al*., 2003). Since these young people are still at a critical growth stage within their lives, the misuse of weight loss products could have adverse effects on their overall growth and also affect visceral organs such as the gastrointestinal tract (GIT).

The gastrointestinal tract plays an important role in the regulation of energy homeostasis within the body ultimately controlling body weight. The GIT relays various satiety and adiposity signals to the hypothalamus. The information is then used to determine the type and amount of food which should be eaten in order to restore the body's energy balance to an optimal level (Wilding, 1997; Woods *et al*., 2004; Wynne *et al*., 2005). Hormonal and paracrine substances which serve as peripheral signals released from enteroendocrine cells in the GIT in response to the different physical and chemical properties of the ingested food which passes along the gastrointestinal tract include; ghrelin, leptin, oxyntomodulin and cholecystokinin (Strader and Woods, 2005; Wynne *et al*., 2005). Since most of the weight loss products are taken orally, the gastrointestinal tract is the first point of contact for these products. When taking into account how the weight loss products are thought to

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bring about their weight loss effects, it is possible that the products could alter the release or passage of some of the regulatory signals from the GIT, thus upsetting the overall energy balance of the body and resulting in potentially adverse effects concerning various growth processes as well as the general functioning of the gastrointestinal tract.

Classes of weight loss products that are readily available fall into one of several broad categories based on their mode of action i.e., fat blockers which inhibit the digestion/absorption of fat, carbohydrate blockers which inhibit the digestion/absorption of carbohydrates and metabolic boosters which increase the basal metabolic rate so as to burn more calories and appetite suppressants (Bjorvell and Rossner, 1987; Kruger *et al*., 2004; Pittler and Ernst, 2004; Udani *et al*., 2004).

Most studies on weight-loss products generally target obese, adult individuals. There is a dearth of information on studies focussing on non-obese growing individuals. The aim of the present study was to investigate the effects of three classes of weight loss supplements on growth performance, glycogen storage in the liver, profile of lipids in the liver, pancreatic proteins and on the gastrointestinal tract of growing rats on restricted food intake as a model of the growing adolescent human.

MATERIALS AND METHODS

The study was approved by the Animal Ethics Screening Committee (AESC) of the University of the Witwatersrand, South Africa (AESC approval number: 2005/17/4). The study was undertaken in the Central Animal Services animal unit at the University of the Witwatersrand, in 2005-2006.

Animals: Forty eight male Sprague Dawley rats (166±2 g) were used in the study. The Rats were housed individually in solid-bottom cages, with wood shavings for bedding. The ambient temperature was set at ~22°C and lighting was on a 12 h light-dark cycle, lights on from 07:00. Drinking water was provided *ad libitum*. Rats were habituated to the housing conditions and experimental interventions (handling, weighing and feeding regime) for three days prior to starting the experimental protocol.

Feeding: The rats were randomly divided into eight groups of six rats each. Group 1 served as a control, receiving standard rat chow (Table 1) *ad libitum* and plain gelatine cubes. Groups 2-7 were fed standard rat chow restricted to 10% of their body mass (to mimick the calorie restriction practised by humans when taking weight loss supplements) and supplemented with one of three weight loss products mixed in flavoured gelatine cubes. Group 8 also received standard rat chow restricted to 10% body weight however the gelatine cubes did not contain any weight loss products. Two doses (low or high) of weight loss products were administered to the rats. The groups were identified as follows:

Table 1: Proximate analysis of the of standard rat feed used in the experiment (data provided by manufacturer)

Ingredient	Values
Protein (g kg^{-1})	180.0
Fat (g kg^{-1})	25.0
Energy (MJ kg^{-1})	18.7
Calcium (g kg^{-1})	18.0
Phosphorus (g kg^{-1})	7.0
Fibre (g kg^{-1})	25.0

Group 1 = Control *ad libitum* feed (AL)
Group 2 and 3 = Carbohydrate blocker, low (CL) and high (CH) dose
Group 4 and 5 = Fat absorption inhibitor, low (FL) and high (FH) dose
Group 6 and 7 = Kelp, low (KL) and high (KH) dose
Group 8 = Control Restricted (R)

The rats were weighed and fed standard rat chow (Epol, South Africa) together with one gelatine cube on a daily basis for 14 consecutive days. The rats' weight and food intake was recorded daily. Feed intake was determined by calculating the difference in the amount of feed given to each rat daily and measuring the leftovers daily. Food conversion efficiency was determined by dividing the weight gain of the rats by the total food intake over the study period and expressed as a percentage.

Weight-loss products: Three different weight loss products were used namely, EVOX™ Carb Block, NutriLife™ Fat Ban and Vital™ Kelp. The composition of the products is shown in Table 2. The doses administered were based on the manufacturer's recommendation for humans. The high dose was three times that of the low dose. The doses administered were as follows; Carb Block low dose 60 mg kg^{-1}, Carb Block, high dose 180 mg kg^{-1}, Fat blocker, low dose 40 mg kg^{-1}, Fat blocker high dose 120 mg kg^{-1}, Kelp low dose 50 mg kg^{-1} and Kelp high dose 150 mg kg^{-1}.

The weight loss products were incorporated in flavoured gelatine cubes. The gelatine cubes were prepared as described by Kamerman *et al*. (2004). The cubes were made to a volume of 2 mL.

Visceral measurements and histology: After the 14 day feeding trial, the rats were killed by anaesthetic

Table 2: Composition of weight-loss supplements used in the experiment. The composition is given for each tablet according to the manufacturers information

Evox™ Carb Block	NutriLife™ Fat Ban	Vital ™ Kelp
Magnesium (50 mg)	Chitosan (750 mg)	Norwegian Kelp (415 mg) Containing iodine (200 μg)
Phaseolus extract (750 mg)	Lemon micronutrients (75 mg)	
Gymnema extract (100 mg)		
Alpha lipoic acid (25 mg)		
Chromium picolinate (150 μg)		
Chromium poliniconate (50 μg)		
Vanadium (100 μg)		
Grape fruit extract (150 mg)		

overdose of Pentobarbitone (Eutha-naze, Centaur labs, Johannesburg, South Africa). The viscera were removed and weighed. The intestines were gently stretched out on a board for length measurements, the intestinal content was gently removed and the intestines weighed. Segments of small intestine for histology were collected and placed in 10% buffered formal-saline and then processed and stained using haematoxylin and eosin. Villus height and crypt depth were measured under light microscopy using a micrometer.

The livers were weighed and then frozen (at -5°C) for later analysis of lipids and glycogen. The pancreas was meticulously dissected out, weighed and then frozen for later determination of soluble proteins.

Liver lipid analysis: The sample lipids were extracted using chloroform:methanol (2:1), essentially as per Bligh and Dyer (1959). A 1 mL aliquot of each extract was then used to determine the lipid dry weight and a further aliquot, approximating to 20 mg of lipid, transmethylated using 10% boron trifluoride in methanol incubated at 100°C to prepare methyl esters of the fatty acids. These were then extracted into hexane and the methyl esters separated using a Varian 3400 gas chromatograph, with a 10% SP2330 6′×⅛ packed column, run at 190°C and with FID detection. The peaks were quantitated using a Varian 4270 integrator and identified by comparison with authentic FAME standards.

Liver glycogen: Liver glycogen was determined indirectly by hydrolysis to glucose and measurement of glucose (Passonneau and Lauderdale, 1974). In summary 0.1 g of liver was placed in 1 mL of 0.03 M HCL and homogenised. To hydrolyse the glycogen 1 mL of 1M HCl was added and the samples were placed sealed tubes in a boiling water bath for 2 h. One milliliter of 1 M NaOH was then added to neutralise the samples before glucose determination. Glucose concentration of the hydrosylate was determined with a glucose (glucose oxidase) assay kit (Sigma Catalogue, GAGO-20) and a spectrophotometer (LKB Ultrospec II, LKB Biochrom Ltd., England).

Pancreatic soluble proteins: The pancreata were homogenised in 0.2 M TRIS-HCl buffer + 0.05 M $CaCl_2$ pH 7.8 (1:10w/v) in an ultra turrex homogeniser for 20 sec and then centrifuged at 15,000 x g for 20 min at 4°C and the supernatant was used to determine the concentration of soluble proteins using the Lowry *et al.* (1951) method modified to be performed on microplates with bovine serum albumin (Sigma, St. Louis, USA) as a standard.

Data analysis: All data are expressed as mean±SD. An analysis of variance (ANOVA) was used to assess the effects of the various weight loss products on the parameters measured. Appropriate post hoc tests were performed (i.e., For pair wise comparisons, the Student Newman Keuls test was carried out and when comparing all groups to controls, Dunnett's post hoc test was used). In all statistical tests values of $p<0.05$ were considered significant. All statistical analysis was performed using Prism 403 for Microsoft Windows (Graphpad Software, USA).

RESULTS

Average weight gain and food conversion: A significantly lower weight gain was observed (Table 3) in all the groups compared to the rats that consumed the rat chow *ad libitum* (ANOVA; $p<0.001$) however no significant differences in average weight gain were observed amongst the groups on the restricted diet (ANOVA; $p>0.05$).

The food conversion efficiency was significantly lower (ANOVA; $p<0.05$) in both the group on restricted feed without weight loss supplements and that on the high dose of fat absorption inhibitor compared to the rats fed standard rat chow *ad libitum* (Table 3).

Mass of viscera and histological observations: Differences in absolute organ weights observed for the large intestines and the liver weights in *ad lib* fed rats compared to those on restricted feed (Table 4).

When considering the organs in relation to overall body mass (Table 4), a significantly higher stomach weight as a percentage of total body weight was observed

Table 3: Effect of weight loss supplements on average body mass gain (g) and food conversion efficiency (%) over the 14 day feeding trial (n = 6 rats per group)

Parameters	AL	CR	CL	CH	FL	FH	KL	KH
Initial body mass (g)	212.8±30.4	209.3±20.2	212.3±22.9	216.7±26.5	208.2±21.9	216.9±23.8	201.0±29.3	209.0±26.6
Average body mass gain (g)	125.0±6.90	62.2±10.9*	68.3±12.8*	76.3±8.10*	70.3±6.50*	63.4±10.9*	76.2±10.4*	71.6±13.7*
Feed conversion efficiency (%)	29.1±2.20	22.0±4.30*	23.3±4.70	25.1±3.20	24.0±6.70	22.1±4.10*	25.0±5.40	24.1±4.80

All data are represented as mean±SD. * p<0.001 versus the *ad libitum* group. R = Restricted, CL = Carbohydrate blocker low dose, CH = Carbohydrate blocker high dose, FL = Fat blocker low dose, FH = Fat blocker high dose, KL = Kelp low dose, KH = Kelp high dose and AL = *ad libitum*

Table 4: Effect of weight loss supplements on the absolute mass (g) and relative mass (% body mass) of the viscera on growing rats (n = 6 rats per group)

		Groups							
Organs	Units	R	CL	CH	FL	FH	KL	KH	AL
Large intesine	(g)	1.58±0.11**	1.65±0.13*	1.60±0.11*	1.80±0.23	1.66±0.24*	1.69±0.14	1.56±0.32**	2.12±0.44
	(%)	0.65±0.06	0.66±0.05	0.62±0.06	0.71±0.08	0.69±0.12	0.66±0.03	0.61±0.12	0.68±0.13
Small intesine	(g)	7.57±0.21	7.92±0.87	8.00±0.65	7.91±0.70	7.91±1.02	8.18±1.32	7.92±0.76	9.00±0.51
	(%)	3.13±0.16	3.16±0.21	3.10±0.21	3.10±0.21	3.26±0.32	3.16±0.36	3.11±0.36	2.91±0.13
Stomach	(g)	1.82±0.16	1.75±0.14	1.71±0.13	1.99±0.19	1.83±0.28	1.88±0.08	1.86±0.15	1.86±0.13
	(%)	0.75±0.09**	0.70±0.05	0.66±0.04	0.78±0.05***	0.75±0.09**	0.73±0.05*	0.73±0.06*	0.60±0.03
Heart	(g)	0.97±0.13	1.02±0.11	0.99±0.04	1.00±0.0.5	0.94±0.10	1.03±0.12	1.01±0.10	1.08±0.05
	(%)	0.40±0.04	0.41±0.05	0.39±0.03	0.40±0.03	0.39±0.03	0.40±0.03	0.40±0.04	0.35±0.02
Testes	(g)	2.89±0.10	2.99±0.09	2.95±0.27	3.14±0.36	3.08±0.28	3.06±0.41	3.05±0.23	3.12±0.29
	(%)	1.19±0.09	1.20±0.07	1.14±0.07	1.23±0.13*	1.27±0.09**	1.19±0.15	1.20±0.14	1.01±0.11
Liver	(g)	10.25±0.66***	10.65±1.13***	11.24±0.65**	11.42±1.50**	9.99±1.04***	10.74±1.55***	10.53±1.16	13.90±0.69
	(%)	4.23±0.16	4.25±0.28	4.36±0.23	4.47±0.40	4.12±0.28	4.14±0.37	4.12±0.32	4.50±0.17
Pancreas	(g)	0.93±0.34	1.09±0.22	1.13±0.26	0.95±0.20	1.01±0.14	1.21±0.21	1.17±0.26	1.17±0.26
	(%)	0.39±0.13	0.43±0.07	0.45±0.12	0.37±0.08	0.42±0.06	0.47±0.08	0.46±0.09	0.37±0.08
Kidneys	(g)	2.02±0.09	2.11±0.15	2.16±0.12	2.27±0.16	2.08±1.15	2.18±0.16	2.20±0.09	2.31±0.13
	(%)	0.83±0.05	0.85±0.08*	0.84±0.04	0.89±0.06***	0.86±0.05*	0.85±0.03*	0.86±0.05*	0.75±0.03
Spleen	(g)	0.76±0.07	0.83±0.13	0.88±0.15	0.84±0.12	0.76±0.09	0.85±0.17	0.84±0.13	0.98±0.07
	(%)	0.31±0.03	0.33±0.05	0.34±0.05	0.33±0.04	0.31±0.03	0.33±0.05	0.33±0.05	0.32±0.02
Cecum	(g)	1.21±0.22	1.28±0.26	1.22±0.12	1.33±0.08	1.22±0.37	1.11±0.12	1.48±0.75	1.23±0.26
	(%)	0.50±0.07	0.51±0.11	0.47±0.05	0.52±0.06	0.51±0.15	0.43±0.05	0.58±0.28	0.40±0.08

*: p<0.05, **: p<0.01 and ***: p<0.001, Data are represented as mean±SD

Table 5: Effect of weight loss supplements on the profile of lipids in the liver, glycogen storage in the liver and soluble proteins in the exocrine pancreas of growing rats (n = 6 rats per group)

Liver	AL	CR	CL	CH	FL	FH	KL	KH
TSFA (%)	40.9±2.6	36.4±2.9	37.9±1.6	38.9±0.9	41.6±9.9	39.4±1.2	38.4±1.4	37.4±1.9
TMUFA (%)	16.7±1.3	16.9±2.1	16.7±3.9	17.7±3.3	15.8±5.9	15.2±3.1	17.3±3.5	15.1±2.5
Tn6PUFA (%)	26.9±1.0	30.5±2.7	30.4±2.5	29.2±2.4	28.0±5.8	30.2±3.1	30.3±2.9	29.8±2.9
Tn3PUFA (%)	13.6±2.2	13.5±3.1	13.6±1.9	12.8±1.6	13.3±1.7	14.2±1.4	12.4±0.9	15.5±1.5
Glucose as glycogen equivalents (mg g^{-1})	15.8±1.2	15.6±2.9	16.8±4.1	15.3±4.7	15.3±2.6	14.4±4.2	14.2±3.8	14.6±2.9
Pancreas								
Soluble protein (mg g^{-1})	65.7±6.6	60.7±6.8	61.7±6.4	64.4±5.7	93.7±5.9*	73.7±8.1	81.4±9.9	88.7±9.9

Data are represented as mean±SD. TSFA = Total Saturated Fatty Acids, TMUFA = Total monounsaturated fatty acids, Tn6PUFA = Total n6 polyunsaturated fatty acids and Tn3PUFA = Total n3 polyunsaturated fatty acids. * Soluble protein significantly different (p<0.05) to all other groups except those on kelp

in the restricted group and the rats fed fat blocker and kelp compared to those on *ad libitum* feed (ANOVA; p = 0.0002). The rats on fat blocker had significantly increased relative testes mass compared to the *ad libitum* group (ANOVA; p = 0.0132). The relative weight of the kidneys was also affected significantly in some groups (ANOVA; p = 0.0028).

The various dietary treatments had no significant effect on the small intestinal villus height and crypt depth (ANOVA; p>0.05). Data not shown.

Profile of lipids in the liver and liver glycogen stores: There were no significant differences (ANOVA; p>0.05) observed (Table 5). There was also no significant difference in the total lipid concentrations in the livers from the different groups (data not shown).

Pancreatic protein: The fat blocker at a low dose significantly (p<0.05) increased the concentration of soluble pancreatic proteins (Table 5).

DISCUSSION

The present study focused on the effects of three different classes of weight loss products on the viscera of growing rats as a model for the current use of over-the-counter weight loss dietary supplements by growing, human adolescents. According to the manufacturers, the EVOX™ Carb Block brings about weight loss by reducing carbohydrate absorption and significantly lowering the storage of fat. The Fat blocker (NutriLife™ Fat Ban) causes weight loss by preventing the absorption of dietary fats, with accelerated fat burning as well as an

overall increase in the basal metabolic rate. The Kelp supplement on the other hand is supposed to cause weight loss through the action of iodine, which influences thyroid gland function, which in turn regulates the body's metabolic rate.

The higher average gain in body mass for the rats which consumed the standard rat chow *ad libitum* compared to those that were fed standard rat chow restricted to 10% of their body weight and the insignificant difference in the body mass gain of all the groups on the restricted diet made us conclude that the weight loss supplements were not directly responsible for the reduced body mass gain in the rats on restricted food intake. Physical activity could impinge on the body masses of the rats, we however did not determine physical activity in the different groups of rats and furthermore given the insignificant differences in body mass gain of the groups of rats on restricted feeding regime that we observed it is unlikely that any differences in physical activity may have impacted on the present study.

The differences in the absolute organ weights (g) observed in the restricted group, the carbohydrate blocker groups, as well as in high dose fat blocker and kelp groups were indicative of the significantly reduced overall body mass of the rats on the restricted diet compared to the rats that consumed the standard rat chow *ad libitum*. On examination of these organs as a percentage of total body weight, no significant differences between the groups were observed, thus indicating that both the large intestine and the liver weights were reduced in proportion to the overall body size of the rats in these groups. Although the large intestine and liver weights did not differ in relation to overall body size, significant differences in stomach weight, testicular weight as well as kidney weight per rat were observed in relation to overall body size in a number of the rat groups. These observed effects could either be ascribed to the specific organ having already reached its growth plateau at the commencement of the study or result from the weight loss products.

The non-significant results observed for intestinal morphology and hepatic glycogen and lipids may have been influenced by certain drawbacks within the study design. It is possible that the supplements were administered for too short a period. However, in rats, changes in intestinal epithelial structure and proliferation have been observed to be established as soon as 9 days following dietary changes (Brunsgaard and Eggum, 1995). The method of administration of the weight loss products may have also affected their effectiveness.

Keeping in mind the active ingredients of the various weight loss products, together with their respective weight loss actions, we expected the administration of these weight loss products together with the hypocaloric diet to result in a significantly reduced average weight gain (g) over the 14 day study period compared to the rats on the restricted diet. However this was not the case showing the weight loss products to be ineffective. Egger *et al.* (1999) and Saper *et al.* (2004) reported that the efficacy of some of the active ingredients contained within the weight loss products used, which included chromium picolinate (an active ingredients in the EVOX™ Carb Block) and chitosan (an active ingredients in the NutriLife™ Fat Ban) were ineffective with respect to aiding weight loss. Bjorvell and Rossner (1987) also concluded that kelp was relatively ineffective in aiding weight loss. Present results are in support of these findings. Nevertheless, despite being ineffective as a weight loss supplement, iodine containing supplements should be taken cautiously as there have been reports of hyperthyroidism occurring (Shilo and Hirsch, 1986).

Despite all three weight loss products being ineffective in reducing the average weight gain over the 14 day study period, the high dose of Fat blocker significantly reduced the average percentage food conversion compared to the group that consumed the standard rat chow *ad libitum*. The Carb Blocker and Kelp did not affect food conversion, whereas feed restriction alone reduced food conversion efficiency. The cause of which is uncertain however it is possible that the weight loss products may have been of some nutritive value, partially supplementing the restricted food intake. Thus, in the absence of the supplemented weight loss products, the rats in the restricted group had less nutrients to convert into appropriate weight gain.

Although the rats consuming the restricted diet in the absence of any weight loss products had a significantly lower percentage food conversion compared to the group that consumed the standard rat chow *ad libitum*, the restricted diet did not adversely affect any of viscera. A significantly higher stomach weight as a percentage of total body weight was observed in the restricted group and both the fat blocker and kelp groups. Since the restricted group also displayed the higher stomach weight in relation to overall body size it can not be attributed to the administration of the fat blocker and kelp products; instead it is most likely due to the stomach having already reached its growth plateau at that particular rat age. Administration of all three weight loss products affected the combined kidney weight per rat, an effect which could be due to the weight loss products themselves, as a similar increased combined kidney weight was not observed in the restricted group. However, since kidney function and histology were not assessed the exact cause

of the apparent increase in kidney weight as a percentage of total body weight is unclear. It recommended that renal function be examined in future studies.

Previous studies have expressed concern for the development of renal failure following long term administration of chromium picolinate (Cerulli *et al*., 1998), thus it should not be ruled out as a possibility.

The Fat Blocker also resulted in a significantly greater testicular mass compared to the rats that consumed the standard rat chow *ad libitum*. This effect could also be a direct effect of the fat blocker itself as no similar effects were observed in any of the other rat groups. Nevertheless, like with the kidneys, testicular function and histology were not examined, thus we can not be certain of the exact cause of the increased testicular mass.

Pittler and Ernst (2004) and Saper *et al*. (2004) concluded that the administration of both chromium picolinate and chitosan were not associated with any adverse effects (Pittler and Ernst, 2004; Saper *et al*., 2004). Apart from the relatively increased kidney and testes mass which can not be directly attributed to the weight loss products with any amount of certainty, we did not find any adverse effects associated with the use of weight loss products containing these ingredients.

Although Garber *et al*. (1993), reported no significant adverse clinical effects associated with the administration of iodine-enriched eggs (produced by chickens that are fed a diet rich in kelp) in human subjects, Shilo and Hirsch (1986) found that the ingestion of kelp tablets was associated with the development of hyperthyroidism which resolved upon cessation of the tablet use. We did not find any gross adverse effects from the kelp however, we did not assess function of the thyroid in the rats.

The weight loss supplements did not significantly affect the small intestinal morphology. We expected the administration of the EVOX™ Carb Block (containing *Phaseolus* extract) to cause potentially detrimental effects within the gastrointestinal tract as previous studies have reported that the phytohaemagglutinins contained within *Phaseolus* extracts induced maturational changes in the gastrointestinal tract (Kruszewska *et al*., 2003) and enhanced small intestine growth and increased the number of crypt cells (Linderoth *et al*., 2005). In contrast, Udani *et al*. (2004) demonstrated that the administration of a weight loss product containing *Phaseolus* extract was not associated with any adverse effects.

The weight loss products did not significantly alter the liver lipid profiles. The Carb Blocker supplements are supposed to aid weight loss by reducing carbohydrate absorption and significantly decreasing fat storage. As a result of the reduced fat storage an increased mobilisation of fat stores would result and thus an increased liver fat content would be observed. Present results are not in support of this as the liver lipid content was not significantly altered by the administration of the carbohydrate blocker product. The fat blocker should also have resulted in a increased liver lipid content as by sufficiently blocking the absorption of dietary fat, fat storage is reduced resulting in increased fat mobilisation and elevated liver lipid levels.

Gades and Stern (2005) found that the overall amount of dietary fat trapped in association with the administration of a chitosan supplement was clinically insignificant. Studies with chitosan use in rats have shown that it was only when included at 3-4% w/w of the diet that fat digestibility was affected. These are relatively high amounts in comparison to what was given to the rats in present study. The percentage of fat in the rat feed was also relatively low (2.5%) compared to what humans normally eat.

Dietary supplementation of chromium picolinate and chitosan (the active ingredients in the EVOX™ Carb Block and the NutriLife™ Fat Ban, respectively) has previously been shown not to be associated with significant weight loss (Egger *et al*., 1999; Saper *et al*., 2004; Pittler and Ernst, 2004). Since both these active ingredients are supposed to affect fat storage, but were proved to be ineffective, the liver lipid profiles were likely to have been unaltered, thus supporting present results.

The rate of secretion and composition of pancreatic juice adapt to changes in diet composition, in rats the proteases and carbohydrases adapt to a greater magnitude than do lipases (Buddington and Lepine, 1999). The soluble pancreatic proteins are an indirect measure of exocrine pancreatic zymogen concentration. We however did not assay for specific enzyme activities. In our study it is only the low dose of fat blocker that had a significant effect on the concentration of soluble pancreatic proteins. This needs further investigation.

Most studies with weight loss supplements do not directly assesses the effects of the weight loss products on the gastrointestinal tract itself and the majority of studies are performed in obese humans. The present study focussed on the effects of the weight loss products in non-obese, growing animals. From this study, we can conclude that the hypocaloric diets which are recommended when taking the weight loss products are in fact more efficient in reducing weight gain than the weight loss products themselves. Also, the short term administration of the weight loss products together with a hypocaloric diet did not significantly affect the gastrointestinal tract morphology, pancreatic proteins or the liver lipid profiles and liver glycogen stores. However, potentially adverse side effects associated with the use of these weight loss products should not be totally dismissed and further, long-term research in this area of interest should be conducted.

ACKNOWLEDGMENTS

We thank the staff of the Central Animal Service (University of the Witwatersrand, South Africa) for provision and care of the animals and Ms Margaret Badenhorst and Mrs Avonne Pickering for their technical assistance. Ms T. Mdluli, Ms J. Kuhlase, Ms T Hleza and Ms Annie Seabela for glycogen assay. The research was funded by the University of the Witwatersrand, the Medical Faculty Research Endowment Fund and the National Research Foundation, South Africa.

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