Association Between Obesity, Flow Rate of Whole Saliva, and Dental Caries in Adolescents

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In a cross-sectional study design, we test the hypothesis whether childhood obesity is associated with reduced flow rate of stimulated whole saliva and dental caries. Obese adolescents (*n* = 65) with a mean age of 14.5 years and normal weight subjects (*n* = 65) with a mean age of 14.2 years were clinically examined with respect to dental caries, visible plaque accumulation (visible plaque index (VPI%)), gingival inflammation in terms of bleeding on probing (BOP%) as well as answered a questionnaire concerning medical history, medication, oral hygiene habits, smoking habits, and sociodemographic background. The flow rate of stimulated whole saliva (ml/min) was determined. BMI was calculated and adjusted for age and gender (BMI-sds). The obese subjects exhibited higher number of decayed surfaces (DS), 0.7 vs. 0.1 (*P* = 0.008) and lower flow rate of stimulated whole saliva 1.2 vs. 2.0 ml/min (*P* < 0.001). Of obese patients, 17 subjects had VPI% >25 and 21 had BOP% >25, both compared to only 5 subjects of the normal weight with *P* values of 0.005 and <0.001, respectively. In a multivariate logistic regression model BMI-sds was significantly associated with the flow rate of stimulated whole saliva less than the median value 1.5 ml/min (*P* < 0.001; odds ratio (OR) 1.36) as well as with DS (DS >0) (*P* = 0.002; OR 1.31) and the associations were not found to be confounded by any of the studied variables. The results indicate that childhood obesity is associated with reduced flow rate of stimulated whole saliva and dental caries and further strengthens obesity's negative effect on children's oral health.

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INTRODUCTION

An increasing prevalence of obesity is well documented in all ages and ethnicities worldwide (1–3). Obesity and in particular abdominal obesity, commonly accompanied by elements of the metabolic syndrome, including insulin resistance, hypertension, and dyslipidemia, is associated with increased risk of chronic inflammatory diseases such as type 2 diabetes, atherosclerosis, cancer, and respiratory disorders (4–6). In addition, several cross-sectional studies have demonstrated that obesity is associated with chronic periodontitis in adults (7–9) and we recently reported that the link between childhood obesity and periodontal disease is demonstrated already during adolescence (10).

Both dental caries and obesity are diseases with multifactor etiology related to dietary habits but also closely correlated with sociodemographic background of the individuals. Most of the studies regarding association between obesity and dental caries are based on clinical data expressing caries experience reflecting not only the actual caries situation but also previous accumulation of caries and filled surfaces. Although there are clinical studies demonstrating a relationship between obesity and dental caries (11–15) contradictory results are present (16,17). Recently, however, it was demonstrated that the number of proximal caries lesions were more frequently diagnosed in teenagers of 15 years with overweight (ISO-BMI >25) compared to normal-weight individuals (11).

Although very low saliva flow rate has been demonstrated to be associated with BMI >25 in a cohort of adults <50 years of age (18) there is limited knowledge concerning saliva flow rate in obese subjects in relation to healthy controls. Some studies based on limited number of obese subjects report normal salivary pattern compared with normal-weight individuals (19,20).

Obesity is linked to chronic inflammation and a number of adipose-related proinflammatory cytokines, so called adipokines, are enhanced in plasma from obese subjects contributing to enhanced inflammatory response in many body organs (21). The immune system modulates central nervous system function particular by cytokines and the hypothalamic–pituitary–adrenal axis is reported to be dysregulated in subjects with abdominal obesity (22). Altered function of the hypothalamic–pituitary–adrenal axis may affect the neuroendocrine regulation of salivary

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Table 1 Clinical, oral hygiene, and sociodemographic variables of the subjects

	Obesity (n = 65)	Control (n = 65)	
Variables	Mean (s.d.)	Mean (s.d.)	P value
BMI (kg/m^2)	36.8 (5.8)	19.7 (2.4)	<0.001[a]
BMI-sds[b]	5.7 (1.1)	0.3 (1.0)	<0.001[a]
Chronic disease (yes/no)[d]	15/65	6/65	0.032[c]
Asthma	5	4	
Diabetes type 1	0	1	
Diabetes type 2	6	0	
Thyroid dysfunctions	5	0	
Epilepsy	0	1	
Neuropsychological	2	0	
Polycystic ovaries	1	0	
Medication (yes/no)[e]	18/65	10/65	0.088[c]
Subjects treated with one or more drugs affecting saliva production[f]	9/65	8/65	0.795[c]
VPI (%)			
0–25	48/65	60/65	
>25	17/65	5/65	0.005[c]
BOP (%)			
0–25	44/65	60/65	
>25	21/65	5/65	<0.001[c]
DFT	2.2 (2.5)	2.1 (2.7)	0.839[a]
DMFS	2.2 (2.8)	2.6 (3.8)	0.510[a]
DS	0.7 (1.6)	0.1 (0.4)	0.008[a]
Flow rate of stimulated whole saliva (ml/min)	1.2 (0.5)	2.0 (0.9)	<0.001[a]
Country of birth[g]			
Two parents born in Sweden	27	25	
One parent born abroad	10	16	
Two parents born abroad	26	23	0.441[c]
Education[h]			
Two parents ≤12 years	44	38	
One parent ≤12 years	11	14	
Two parents >12 years	9	8	0.694[c]
Tooth-brushing			
In the morning			
Daily	34/65	52/65	
Not daily	31/65	13/65	<0.001[c]
In the evening			
Daily	35/65	53/65	
Not daily	30/65	12/65	<0.001[c]
Flossing			
Daily	3/65	0/65	
Not daily	62/65	65/65	0.447[c]
Last dental visit			
≤12 months	46/65	16/65	
>12 months	19/65	49/65	<0.001[c]

BOP, bleeding on probing; DS, decayed surfaces; VPI, visible plaque index.
[a]ANOVA as statistical method. [b]Roland–Cacherra. [c]χ^2 as statistical method. [d]Three subjects had multiple diagnoses. [e]Three obese subjects and four controls used prescribed allergy medication when needed. [f]β_2-agonists, methylphenidate, anti-histamine, lamotrigine, carbamazepine, fluoxetin. Other drugs not affecting saliva production: metformin, budesonide, levothyroxine, mometason furoat monohydrate. [g]In three cases the father's place of birth was unknown. [h]In six cases the parent's educational level was unknown.

Table 2 Bivariate logistic regression analysis with decayed surfaces (DS >0) as dependent variable

Variable	Wald	*df*	*P* value	OR	95% CI
Age (years)	0.24	1	0.623	1.06	0.83–1.36
Gender (male)	0.79	1	0.375	1.48	0.62–3.55
BMI-sds (kg/m^2)	9.93	1	0.002	1.31	1.11–1.56
Chronic disease	0.14	1	0.708	1.24	0.41–3.74
Medication	0.18	1	0.669	0.79	0.27–2.32
Flow rate of stimulated whole saliva (ml/min)	3.89	1	0.049	0.52	0.28–1.00
VPI (>25%)	3.72	1	0.054	2.68	0.99–7.28
BOP (>25%)	8.40	1	0.004	4.03	1.57–10.36
Tooth-brushing					
In the evening (not daily)	3.79	1	0.052	2.37	0.99–5.65
In the morning (not daily)	3.04	1	0.081	2.16	0.91–5.12
Electric toothbrush (not daily)	0.00	1	0.993	1.01	0.34–2.99
Dental floss (not daily)	0.01	1	0.927	1.06	0.32–3.47
Food intake (>5 times/day)	3.08	1	0.079	2.29	0.91–5.78
Last dental visit (≤12 months)	1.80	1	0.180	1.80	0.76–4.26
Country of birth					
Two parents born abroad	3.34	2	0.188	1.00	
One parent born abroad	2.52	1	0.112	0.28	0.06–1.35
Educational level					
Two parents ≤12 years	0.84	2	0.658	1.00	
One parent ≤12 years	0.00	1	0.957	0.58	0.34–3.17
Two parents >12 years	0.81	1	0.367	1.72	0.53–5.58

BOP, bleeding on probing; CI, confidence interval; DS, decayed surfaces; OR, odds ratio; VPI, visible plaque index.

glands which seems to be the case in Sjögren's syndrome, characterized by diminished salivary gland secretion (23). In light of these observations, we were interested to investigate whether the salivary function is affected by obesity and test the hypothesis that childhood obesity is associated with reduced flow rate of stimulated whole saliva and dental caries.

METHODS AND PROCEDURES

The present study was conducted on 65 obese subjects (mean age 14.5, range 10.3–17.2 years) and 65 normal-weight controls (mean age 14.2, range 11.0–18.3 years). The obese subjects were treated at the National Childhood Obesity Center Karolinska University Hospital Huddinge and consecutively referred to the Division of Pediatric Dentistry in order to study the oral comorbidity among obese children and adolescents. All subjects had a BMI within the obesity range for age (ISO-BMI >30) (24). The control patients with ISO-BMI <25 were recruited from the Division of Pediatric Dentistry, Department of Dental Medicine, Karolinska Institutet and consisted of individuals receiving their regular dental treatment at the department. The normal-weight controls were matched with respect to age and gender and the following exclusion criteria were used; any antibiotic treatment last 3 months, daily smoking and ongoing orthodontic treatment. The ethics committee at Karolinska University Hospital Huddinge approved the study and all subjects gave oral consent before participating in the study.

Body weight (kg) and height (m) of the subjects were determined and BMI (kg/m^2) as well as BMI adjusted for age and sex (BMI-sds) (25) was calculated for each subject.

Questionnaire

All the subjects answered a questionnaire regarding, sociodemographic situation, education, country of birth, oral hygiene habits, food intake, as well as medical conditions, and use of prescript medication. The subject's medical conditions were categorized for statistic analyzes into with or without a chronic disease as well as taking or not taking prescript medication with special reference to drugs with reduced salivary production as potential side effect. In case subjects did not understand the Swedish language an interpreter assisted. Parent's country of birth was categorized into "born in Sweden" or "born abroad." The educational level of the parents was stratified according to "two parents ≤12 years," "one parent ≤12 years" (one of two), and "two parents >12 years."

Clinical examination

Dental caries. The number of decayed (D), missing (M), and filled (F) teeth (T)/surfaces (S) was registered and expressed as DMFT/S indexes. Manifest caries lesions were registered on smooth surfaces that were verified as a cavity detectable by probing and in fissures as a catch of the probe under slight pressure. The clinical examination was supplemented by two bitewing radiographs. Manifest caries was diagnosed on proximal surfaces when the lesion clearly extended into the dentine.

Saliva. The saliva collection procedure was performed both in the morning as well as in the afternoon and no food restriction was given to the patients. Stimulated whole saliva was collected by asking the subject chewing 1 g of paraffin wax for 5 min. Before the saliva collection, the subjects rinsed the mouth with water. The subjects spitted the saliva into a test tube. The amount of saliva was determined after collection and the saliva secretion rate was expressed as ml/min.

Dental plaque and gingival inflammation. Dental plaque on tooth surfaces were recorded when clearly visible and expressed as the visible plaque index (VPI) (26). Gingival inflammation was based on bleeding on probing (BOP) of the gingival sulcus of all teeth (wisdom teeth excluded) at six points. The proportion of surfaces (%) with dental plaque or bleeding on probing, respectively, was calculated for each individual.

Statistics

Data analysis was carried out using the statistical software package SPSS, version 16.0 (SPSS, Chicago, IL). For analyzing the data, frequency tables, cross tables, and logistic regression were used. χ^2-tests were carried out to assess the significance of differences in proportions and independent *t*-test was used to assess the significance of differences in means. Bivariate analyses of associations were carried out between the dependent variables "decayed surfaces, DS (>0)" or "flow rate of stimulated whole saliva less than the median value (<1.5 ml/min)," and the potential independent variables by logistic regression binary model. In the multivariate logistic regression with, "decayed surfaces, DS (>0)," or "flow rate of stimulated whole saliva (<1.5 ml/min)" as dependent variables, the independent variable BMI-sds, was adjusted

Table 3 Multiple logistic regression analysis with flow rate of stimulated whole saliva (<1.5 ml/min) as dependent variable and BMI-sds as independent variable, adjusted for potential confounders

Variable	β-Coefficient	Wald	d*f*	*P* value	OR	95% CI
BMI-sds						
Not adjusted	0.31	19.72	1	<0.001	1.36	1.19–1.56
Adjusted for						
Age	0.32	20.86	1	<0.001	1.38	1.20–1.59
Gender	0.31	19.69	1	<0.001	1.37	1.19–1.57
Chronic disease	0.31	18.57	1	<0.001	1.36	1.18–1.56
Medication	0.32	19.81	1	<0.001	1.38	1.20–1.59
Age, gender, chronic disease and medication	0.32	19.36	1	<0.001	1.38	1.20–1.59
BMI-sds						
Adjusted for						
BOP (> 25%)	0.29	16.36	1	<0.001	1.34	1.03–1.45
VPI (> 25%)	0.29	16.57	1	<0.001	1.34	1.16–1.54
DS (>0)	0.32	19.22	1	<0.001	1.37	1.19–1.58
Not daily tooth-brushing (evening)	0.32	18.36	1	<0.001	1.37	1.19–1.59
Not daily tooth-brushing (morning)	0.36	20.51	1	<0.001	1.43	1.22–1.67
BOP (>25%), VPI (>25%), DS (>0), and not daily tooth-brushing (evening and morning)	0.34	16.93	1	<0.001	1.41	1.20–1.66
BMI-sds						
Adjusted for						
Parental country of birth	0.32	18.84	1	<0.001	1.37	1.19–1.58
Parental educational level	0.29	16.62	1	<0.001	1.33	1.16–1.53
Parental country of birth, and educational level	0.30	16.39	1	<0.001	1.34	1.17–1.55

BOP, bleeding on probing; CI, confidence interval; DS, decayed surfaces; OR, odds ratio; VPI, visible plaque index.

for potential confounders. The odds ratio (OR) and confidence interval (95% confidence interval) was calculated and the level of significance was accepted at *P* values <0.05. In addition, the sensitivity and specificity for BMI-sds as a discriminator of "flow rate of stimulated whole saliva (<1.5 ml/min)" and "decayed surfaces (DS >0)," respectively, were also determined.

RESULTS

Clinical, oral hygiene, sociodemographic and general health variables of the subjects are presented in **Table 1**. The frequency of subjects with chronic diseases was significantly higher ($P = 0.032$) among the obese subjects compared with the control whereas the use of prescribed medication including drugs known to affect salivary production did not differ significantly between the two groups. On the contrary, the obese subjects demonstrate significantly higher number of DS ($P = 0.008$) compared to the controls as well as higher VPI% ($P = 0.005$) and BOP% ($P < 0.001$). There was no significant difference between the groups with respect to DFT or DMFS indexes. The obese subjects reported more frequently previous dental visit performed ≤1 year compared with the controls ($P < 0.001$). The average of flow rate of stimulated whole saliva (ml/min) was significantly lower in obese subjects compared with the controls ($P < 0.001$). The mean value of stimulated whole saliva secretion was 2.0 ml/min of the controls compared with 1.2 ml/min of obese subjects (**Table 1**).

A bivariate logistic analysis was performed with DS (>0) as dependent variable and following variables were significantly associated with dental caries; BMI-sds ($P = 0.002$, OR = 1.31), flow rate of whole saliva (ml/min) ($P = 0.049$, OR = 0.52), and BOP (>25%) ($P = 0.004$, OR = 4.03) (**Table 2**). A bivariate logistic regression analysis with flow rate of whole of whole saliva (<1.5 ml/min) as dependent variable was also tested (data not shown).The OR of the independent variable medication was 0.95 ($P = 0.907$) and after adjustment for obesity 0.58 ($P = 0.260$).

In a multivariate model, BMI-sds was tested in relation to flow rate of whole saliva (<1.5 ml/min), both unadjusted as well as adjusted with respect to different potential confounders (**Table 3**). The association between BMI-sds and flow rate of stimulated whole saliva (<1.5 ml/min) was not confounded by any of the studied variables. One unit increase in BMI-sds (unadjusted) implies an increase of OR of flow rate (<1.5 ml/min) by 1.36. The sensitivity of BMI-sds as a discriminator of flow rate of whole saliva (<1.5 ml) was estimated to 0.72 and the specificity was 0.74.

In next multivariate model the variable BMI-sds was also tested in relation to dental caries (DS >0) both unadjusted as well as adjusted with respect to various potential confounders such as age, gender, chronic disease, medication, salivary flow, sociodemographic factors, oral hygiene variables, BOP%, and VPI% (**Table 4**). Obesity in terms of BMI-sds as continuous variable

Table 4 Multiple logistic regression analysis with decayed surfaces (DS >0) as dependent variable and BMI-sds as independent variable, adjusted for potential confounders

Variable	β-Coefficient	Wald	*df*	*P* value	OR	95% CI
BMI-sds						
Not adjusted	0.27	9.93	1	0.002	1.31	1.11–1.56
Adjusted for						
Age	0.27	9.65	1	0.002	1.31	1.11–1.56
Gender	0.27	9.93	1	0.002	1.32	1.11–1.56
Chronic disease	0.28	9.86	1	0.002	1.32	1.11–1.56
Medication	0.29	10.55	1	0.001	1.33	1.12–1.59
Flow rate of stimulated whole saliva (ml/min)	0.24	6.99	1	0.008	1.28	1.07–1.53
Age, gender, chronic disease, medication and salivary flow	0.26	7.32	1	0.007	1.30	1.08–1.58
BMI-sds						
Adjusted for						
BOP (> 25%)	0.23	6.73	1	0.009	1.26	1.06–1.51
VPI (> 25%)	0.25	7.97	1	0.005	1.29	1.08–1.53
Not daily tooth-brushing (evening)	0.25	7.85	1	0.005	1.29	1.08–1.53
Not daily tooth-brushing (morning)	0.25	7.89	1	0.005	1.29	1.08–1.54
BOP (>25%), VPI (>25%) and not daily tooth-brushing (evening and morning)	0.22	5.11	1	0.024	1.24	1.03–1.49
BMI-sds						
Adjusted for						
Parental country of birth	0.28	9.72	1	0.002	1.32	1.11–1.58
Parental educational level	0.29	10.16	1	0.001	1.34	1.12–1.60
Parental country of birth, and educational level	0.30	10.12	1	0.001	1.36	1.12–1.64

BOP, bleeding on probing; CI, confidence interval; DS, decayed surfaces; OR, odds ratio; VPI, visible plaque index.

was significantly associated with the decayed surface (DS >0) with OR of 1.31 (unadjusted) and the association was not found to be confounded by any of the studied variables. Furthermore, the sensitivity of BMI-sds as a discriminator of decayed surfaces (DS >0) was estimated to 0.31 and the specificity was 0.89.

DISCUSSION

We here demonstrate that childhood obesity is associated with reduced flow rate of stimulated whole saliva and dental caries. In addition, the finding that obese subjects have more gingival inflammation is in line with our previous report (10).

The BMI-sds is used in the statistical analysis instead of regular BMI to minimize bias effects. In addition, we also adjusted for a number of potential confounders including gender, age, chronic disease, medication, sociodemographic variables, gingival inflammation, and oral hygiene variables when analyzing the association between obesity and saliva flow rate as well as dental caries. In light of the close relationship between sociodemographic factors and dental caries it is important to consider that there is no significant difference in sociodemographic profile of the subjects between the two groups.

Our findings that obesity is linked to dental caries is in agreement with a number of clinical studies (11–14), although conflicting results are presented (16,17). We here report that there is an association between obesity and dental caries even after adjustment of sociodemographic background, a strong predictor of dental caries among children (27). According to Marshall *et al.* (15) caries and obesity coexists in children of low socioeconomic status. One possible explanation to different results concerning association between dental caries and obesity is the fact that the authors do not always control for confounders such as sociodemographic determinants but also that the association between obesity and dental caries might be weak (14) and vary over time as well as between different regions due to different preventive fluoride program addressed to the children. One weakness of our study is that the amount of preventive care given to our patients is not possible to analyze but according to the multiple logistic regression analysis, the variables "not daily tooth-brushing evening or morning," did not confound the link between obesity and dental caries. That is important because tooth-brushing procedure includes use of fluoride in toothpaste which exhibits a significant anti-caries effect. However, one cannot rule out that prevention care differ between the groups which may affect the strength of the link between obesity and dental caries. In this study, we did not demonstrate any association between caries experience in terms of DFT and obesity which probably is due to the fact that DFT also includes fillings and thereby reflect previous caries experience. Based on our data it seems more appropriate to use decayed surface as outcome variable. We did not register

the duration of obesity of the subjects which probably also, in addition to the severity of obesity, is of importance when looking upon the association between obesity and dental caries.

Many researchers have postulated dietary habits as a possible cause to enhanced caries prevalence among obesity subjects. However, we here demonstrate that the number of food intake per day did not differ between the two groups indicating other factors of importance behind the link between obesity and dental caries. However, we cannot rule out that the obese subjects, compared to controls, exhibit dietary habits that are more favorable for caries development nor can we rule out that the dietary habits of the obese patients have been altered after they commenced treatment at the National Childhood Obesity Center Karolinska University Hospital Huddinge.

The novel finding that obese adolescent's exhibit a lower flow rate of stimulated whole saliva compared to normal-weight controls is interesting and may add new information explaining a possible link between obesity and dental caries. Noticeable, the number of subjects taking one or more drugs with a potential to negatively affect salivary production was similar both among the obese subjects and the controls. In a multivariate logistic model, the association between obesity and flow rate of stimulated whole saliva (<1.5 ml/min) was confirmed and the magnitude of OR of sds-BMI among obese subjects for having flow rate of whole saliva below the median value differed from 1.33 to 1.43. The median value of flow rate of stimulated whole saliva was used as the cutoff value in the multiple logistic regression analysis because there is lack of valid reference value concerning flow rate of whole saliva in adolescents. The difference in β-coefficient of the independent variables in the multiple logistic regression analysis with flow rate of stimulated saliva as dependent variable was low indicating that none of the variables including chronic disease or medication confounds the link between obesity and flow rate of stimulated saliva. Furthermore, based on the data of sensitivity (0.72) and specificity (0.74) of obesity, BMI-sds may be a potential factor to discriminate subjects with reduced flow rate of stimulated whole saliva. The link between obesity and flow rate is well compatible with the finding of an association between overweight and prevalence of hyposalivation recently reported among adults (18). On the contrary, our finding is not in agreement with older data based on limited number of subjects indicating no difference between obese subjects and healthy controls concerning flow rate of saliva (19,20).

There is a limitation of our study because saliva collection procedures were taken place at various time points on the day and therefore to some extent influence the results due to circadian rhythm of salivary flow (18). Due to marked difference in the mean value of saliva secretion rate between the two groups, the lower flow rate of whole saliva secretion among the obese subjects reflects presumably an effect by adiposity rather than difference in sampling time of saliva collection. However, clinical studies using standardized saliva collection procedure are needed to confirm our observation.

The regulation of saliva production is complex involving both autonomic nervous system and endocrine/paracrine regulation. Whether there is a direct or indirect effect of obesity that affects negatively the flow rate is unclear. Interestingly, obese subjects have been reported to exhibit a significant enlargement of parotid glands probably by an enhanced storage of adipocytes in the parotid parenchyma whereas the submandibular glands seem to remain unaffected (28,29). Based on that, proinflammatory cytokines derived from adipocytes as well as macrophages, accumulated in adipose tissue (30) may negatively affect the function of salivary glands due to chronic low-grade inflammation in the gland. Moreover, we have reported enhanced levels of proinflammatory cytokines tumor necrosis factor–α, interleukin-1, and interleukin-8 in crevicular fluid in obese adolescents compared with normal-weight subjects (10,31) indicating a hyperinflammatory reaction in the periodontal tissue as well. In Sjögren's syndrome there is a lymphocytic infiltrate in the salivary gland parenchyma (32) and enhanced level of eicosanoids like prostaglandin E_2 and thromboxane B2 is reported in saliva (33). Altogether, suggesting a hypothesis that inflammatory mediators play a role in the hypofunction of salivary glands among obese subjects. However, the negative effect of obesity on salivary glands may be related to stress hormones linked to hypothalamic–pituitary–adrenal axis (34) that may negatively influence the function of saliva glands as well (23).

In conclusion, the novel finding is that childhood obesity is associated with reduced flow rate of stimulated whole saliva and dental caries which further strengthens obesity's negative effect on children's oral health.

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DISCLOSURE

The authors declared no conflict of interest.

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