

# Influence of parental body mass index on offspring body mass index in a Spanish population

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A number of studies suggest that genetic factors may play a role in the obesity epidemic via an increased rate of coincidental mating between obese individuals. We have analyzed the correlation between parental and offspring BMI and spouses' BMI, at present and when the parents were 20 years old.

Four hundred and eight Spanish children and adolescents (174 obese) and their parents were enrolled. The children's BMI was measured by trained personnel. The parental BMI values were calculated from self-reported data. Conditional logistic regression was applied to estimate the childhood obesity risk associated to parental obesity. Spouse resemblance in BMI was assessed using the Spearman correlation coefficient and the linear trend test.

We found that parents of children with extreme BMI values (highest or lowest) have greater BMI resemblance than those of 'medium range BMI' children. We have also observed a significant linear association between parental BMI quintiles after years of cohabitation, but there was no such association at age 20.

In conclusion, the present study corroborates the existence of a positive correlation

between parental and offspring BMI values. This is an important aspect that nutritional educators should consider in the management of obesity. However, it seems that there is no coincidental mating for obesity in this adult Spanish population.

**Key words:** *Coincidental mating. Obesity. Children. Family.*

### Influencia del índice de masa corporal de los progenitores sobre el de sus descendientes en una población española

Existen estudios que señalan que factores genéticos podrían desempeñar un papel en la epidemia de obesidad a través de una proporción aumentada de emparejamiento selectivo entre individuos obesos. Hemos analizado la correlación entre los índices de masa corporal de padres e hijos y de los padres entre sí, considerando los datos actuales y los correspondientes a cuando los padres tenían 20 años de edad.

Se enrolaron en el estudio 408 niños y adolescentes españoles (174 de ellos obesos) y sus padres. Los IMC de los niños y adolescentes fueron determinados por personal entrenado; los de los padres se calcularon

a partir de datos comunicados por ellos mismos. Se aplicaron procedimientos de regresión logística condicionada para estimar el riesgo de obesidad infantil asociado a la obesidad parental. La similaridad de IMC entre los progenitores se evaluó mediante el coeficiente de correlación de Spearman y pruebas de tendencia lineal.

Encontramos que los padres de niños con IMC extremos (valores más altos y más bajos) mostraban mayor similaridad de IMC que los padres de niños con IMC "intermedios". También hemos observado una asociación lineal significativa entre quintiles de IMC entre los esposos al cabo de años de convivencia, asociación que no se daba cuando tenían 20 años de edad.

En conclusión, nuestros resultados corroboran la existencia de una correlación positiva entre los IMC de padres e hijos. Éste es un aspecto importante que los educadores nutricionales deberían tener en cuenta en el tratamiento de la obesidad. Sin embargo, no parece existir un emparejamiento selectivo en cuanto a obesidad en esta población adulta española.

**Palabras clave:** *Emparejamiento selectivo. Obesidad. Niños. Familia.*

## INTRODUCTION

Over the last two decades, the obesity incidence rate has increased dramatically in children, adolescents and adults worldwide<sup>(1,2)</sup>. In Navarra, the PECNA study disclosed an obesity prevalence of 14.7%<sup>(3,4)</sup>. As obesity is assumed to be a multifactorial disease caused by genetic and environmental factors and their interactions<sup>(5-8)</sup>, some authors have sought to explain the current obesity epidemic through the change in lifestyle factors in Western societies, which leads to an increased energy intake and a reduced level of physical activity.

However, studies performed in twins have shown that obesity is highly inheritable, with estimates in the range of 84-88%<sup>(9,10)</sup> giving evidence for the role of genes in body fatness. Parental obesity is associated with obesity in the offspring<sup>(11,12)</sup>. Data show that, for a child up to 5 years of age, having two obese parents represents a much greater (>10 times) risk for later obesity than being obese as a child [odds ratio (O.R.) <5]<sup>(10)</sup>. However, with increasing age, the weight status of the child seems to become a strong predictor of adulthood obesity regardless of parental obesity.

It is difficult to establish a relationship between obesity incidence increase rates and genetic factors. Some studies have reported that genetic factors might be involved in the obesity epidemic via an increased rate of coincidental mating between obese individuals<sup>(13-15)</sup>. Such coincidental mating might increase homozygosity at *loci* associated to higher obesity risk. With similar rates of coincidental mating over generations, a growing prevalence of obesity in the offspring of obese parents might be expected<sup>(10,16)</sup>. This might even worsen if coincidental mating for BMI should still increase in the coming generations.

The aim of the present study was to analyze, in a Spanish children and adolescent population, if there is any association between parental and offspring BMI and also between spouses' BMI (both current and when the spouses were 20 years old).

## METHODS

The study population, recruited from the Paediatrics Departments at the "Virgen del Camino" Hospital, the University of Navarra Clinic and other Primary Care Centers, included 408 Spanish children and adolescents (5-19 years of age) and their biological parents (currently 34-55 years old) ( $N = 1224$ ).

Children and adolescents were recruited while attending Primary Care Centers for routine medical examinations or vaccinations or to be treated for obesity or growth delay. Ex-

clusion criteria were exposure to hormonal treatment, development of secondary obesity due to endocrine disease, or serious coincident illness.

The "offspring" population consisted of 174 obese and 234 non-obese subjects. Obesity in children was defined as a body mass index (BMI) over the 97<sup>th</sup> percentile of the Spanish BMI reference data for age and gender<sup>(17-19)</sup>. The same criteria were used to calculate the BMI standard deviation score (or *z*-score) (SDS). The study was approved by the Ethics Committee of the University of Navarra. All parents and children over 12 years old gave their informed consent for study participation in writing.

Anthropometric measurements were performed in a medical environment by trained researchers using standard procedures<sup>(20)</sup>. Height was measured to the nearest centimeter and weight to the nearest 100g with a digital scale (TBF-300A Body Composition Analyzer/Scale, TANITA<sup>®</sup>, Tokyo, Japan). Body fat percentages were determined by bioelectrical impedance (TBF-300A Body Composition Analyzer/Scale, TANITA<sup>®</sup>, Tokyo, Japan).

In a personal interview the parents were asked about their current weight (if they reported a recent weight loss, the highest weight was computed), their weight when they were 20 years old, and their height. From these data, the BMI was calculated. Parental obesity was defined as a BMI >30 kg/m<sup>2</sup> in either the father or the mother. Data were collected over the period from February 2006 to March 2007.

## Statistical analyses

Descriptive values are reported as means and standard error (SE). To test the relationship between parental BMI and offspring BMI-SDS, the Spearman correlation coefficient was used due to the nonparametric data distribution. Age- and gender-adjusted conditional logistic regression was used for estimating childhood obesity risk associated to parental obesity.

Inter-spouse BMI resemblance was also assessed using the Spearman correlation coefficient, and the linear trend test was used to evaluate the spouse' resemblance uniformity across the entire BMI range (gender-specific BMI quintiles were computed). The offspring were distributed into quintiles based on BMI-SDS values so as to study the correlation with the spouses' BMI across the offspring BMI-SDS range.

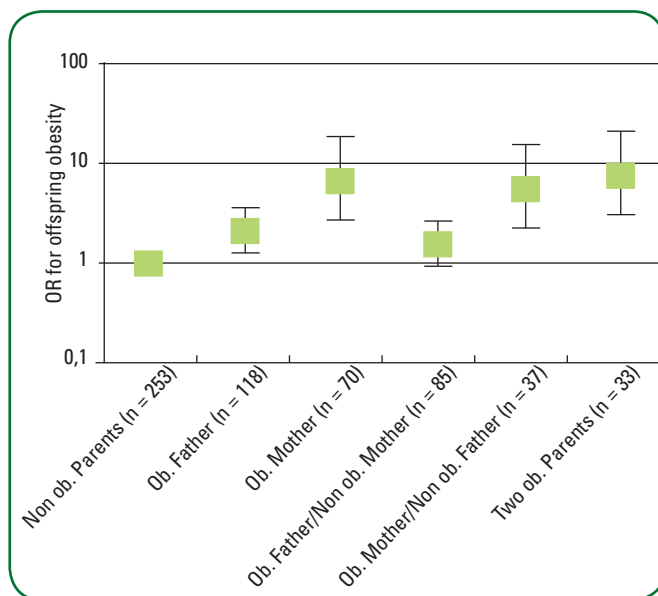
The statistical analyses were carried out using the SPSS 15.0.1 (Statistical Package Software Solutions, Chicago, USA) for Windows statistical package. A *P* value lower than 0.05 was considered statistically significant.

Table 1. **ANTHROPOMETRICAL CHARACTERISTICS (MEAN, SD) OF 408 OBESE AND NON-OBESE CHILDREN AND ADOLESCENTS (5-19 YEARS OLD)**

	Offspring	
	Non-obese	Obese
<b>N</b>	174	234
<b>Age (y)</b>	12.6 (0.28)	11.6 (0.18)
<b>% Males</b>	38.8%	51.3%
<b>BMI (kg/m<sup>2</sup>)</b>	20.1 (0.67)	27.6 (0.30)
<b>BMI-SDS</b>	0.43 (0.08)	3.70 (0.09)
<b>% Body fat</b>	20.2 (0.25)	34.9 (0.50)
<b>Mother's BMI at present (kg/m<sup>2</sup>)</b>	23.9 (0.29)	27.0 (0.41)
<b>Father's BMI at present (kg/m<sup>2</sup>)</b>	27.5 (0.55)	31.0 (0.88)
<b>Mother's BMI (20 years old) (kg/m<sup>2</sup>)</b>	20.4 (0.19)	22.4 (0.26)
<b>Father's BMI (20 years old) (kg/m<sup>2</sup>)</b>	23.7 (9.43)	26.5 (0.71)
<b>% Obese mother at present</b>	5.4%	35.7%
<b>% Obese father at present</b>	20.7%	24.7%

## RESULTS

Four hundred and eight children and adolescents and their parents were enrolled. The anthropometrical data of the children are summarized in **Table 1**. Among the children / adolescents,



**Figure 1.** Association between parental obesity and offspring obesity risk. The model was adjusted for offspring age and gender. Error bars represent 95% confidence intervals.

Table 2. **CORRELATION DATA OF BMI IN FAMILIES IN 408 FAMILIES WITH OBESE AND NON OBESE OFFSPRING**

	At present		At age 20	
	r	p	r	p
<b>Mother-son</b>	0.340	<0.001	0.234	0.001
<b>Mother-daughter</b>	0.389	<0.001	0.343	<0.001
<b>Father-son</b>	0.257	<0.001	0.202	<0.006
<b>Father-daughter</b>	0.273	<0.001	0.246	<0.001
<b>Mother-father</b>	0.217	<0.001	0.016	0.755

*Spearman correlation data*

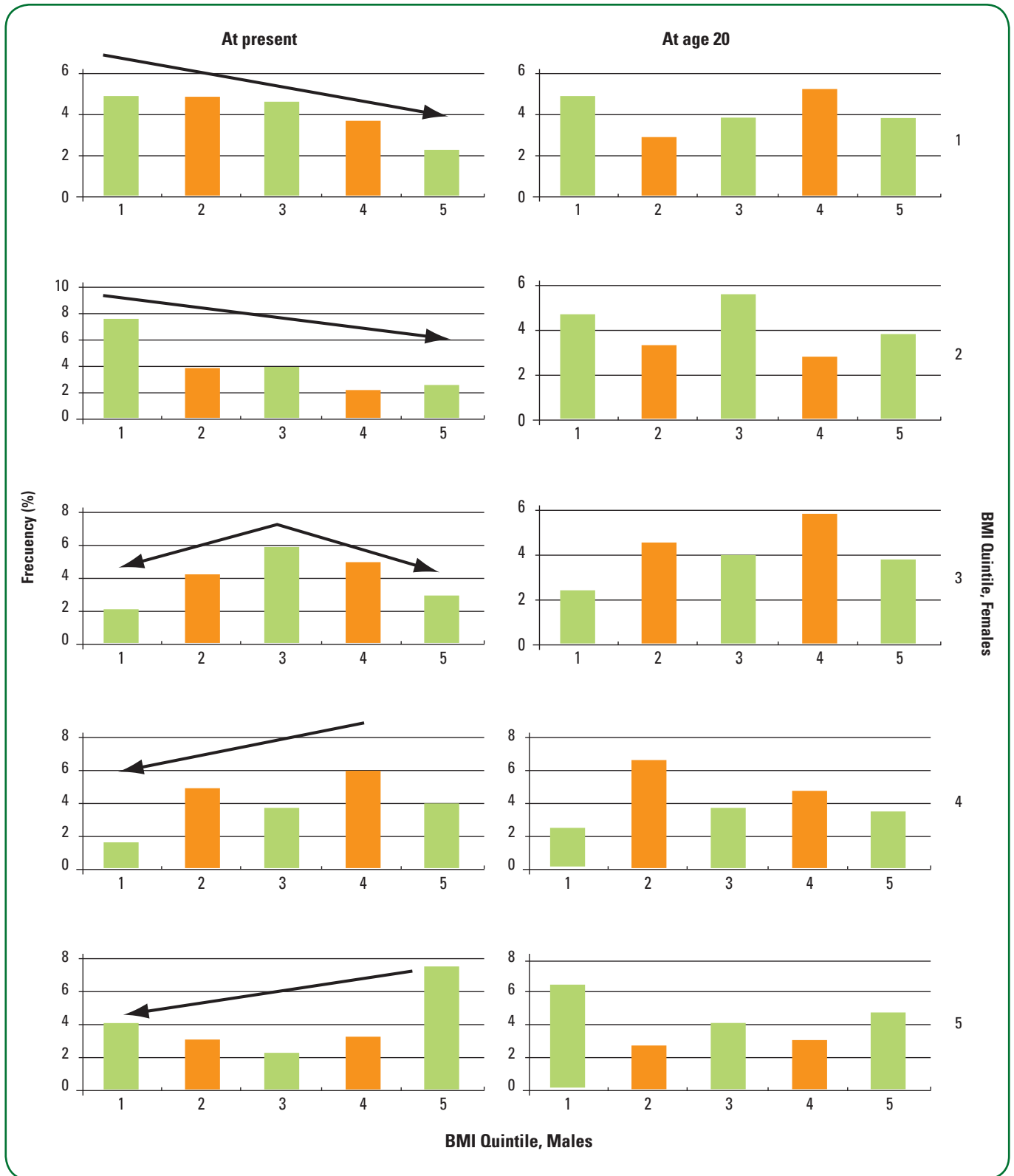
30.4% were less than 10 years old, 42.3% were between 10 and 14 and 25.6% were older than 14 years. The mean paternal BMI was 29.7 (0.55) kg/m<sup>2</sup> and the maternal one was 25.8 (0.26) kg/m<sup>2</sup> (current value); and 25.3 (0.45) kg/m<sup>2</sup> and 21.5 (0.17) kg/m<sup>2</sup> respectively, when they were 20. The paternal BMI of the fathers of obese children was higher than that of the fathers of non-obese children (27.5 vs. 31.0;  $P < 0.001$ ).

### Parent-offspring resemblance in BMI

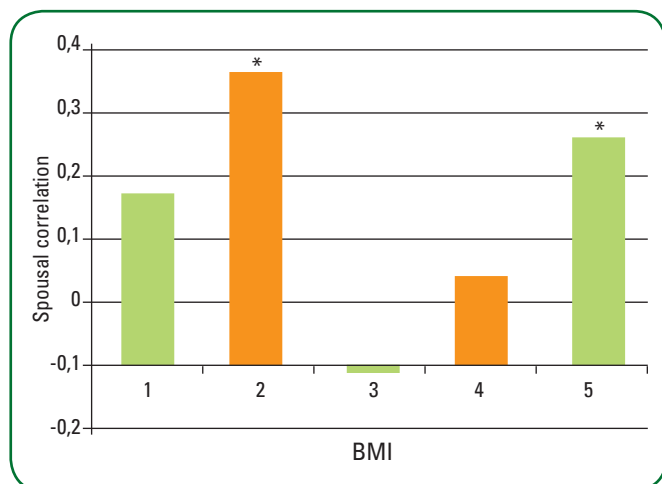
The parental BMI value correlated with offspring BMI-SDS (**Table 2**). The correlations were slightly stronger with the parental present BMI than with the BMI at age 20, and they were also higher for daughters as compared to sons. There were no statistically significant differences in correlation data for obese and non-obese people.

A logistic regression model was used to study the association between present parental obesity and childhood obesity risk in the offspring (**Figure 1**). Children with an obese father (and a normal-weight or overweight mother) had twice the risk of becoming obese as compared to subjects without an obese parent (OR = 2.11; 95% confidence interval [95% CI] = 1.25-3.56). When children had an obese mother the obesity risk was 7.66 (95% CI = 2.69-20.46), and children with two obese parents had an obesity risk of 8.05 (95% CI = 2.72-23.80) after adjustment for gender and age. A lower, but also statistically significant, risk of obesity occurs when the father (OR = 1.90; 95% CI = 1.19-3.05) or the mother (OR = 3.05; 95% CI = 2.60-4.63) are overweight.

Children with an obese mother and a non-obese father had an odds ratio for obesity risk = 5.29 (95% CI = 2.00-13.97). However, children having an obese father and non-obese mother did not evidence an increased risk of obesity (OR = 1.44 [95% CI = 0.87-2.40]).



**Figure 2.** Distribution of BMI quintiles among 408 spouse pairs. Frequencies higher than 4% (the expected frequency given a random mating) indicated non-random assortment.



**Figure 3.** Correlations for spouses BMI at present according to the BMI-SDS quintile of their offspring (\*  $p$ -value < 0,05).

When parental BMI at age 20 was examined, it was found that having a mother who had been obese already at age 20 increased obesity risk 8.9-fold (95% CI = 1.15-69.10;  $n = 14$ ) and children with a father who had been obese at age 20 had an OR for obesity of 4.33 (95% CI = 1.63-11.5;  $n = 34$ ) (data not shown).

### Spouse resemblance in BMI

The mother-to-father current BMI correlation coefficient was 0.217 ( $P < 0.001$ ); however, there was no statistically significant correlation between the parental BMI values at age 20 ( $r = 0.016$ ,  $P = 0.755$ ).

The resemblance between spouses' BMI was studied by computing the frequency of BMI quintiles of males and females in pairs (Figure 2). For the present BMI, the quintile-concordant pairs had a higher frequency than discordant pairs. This means that there was a higher frequency of subjects paired with people in the same current BMI quintile than spouses in different BMI quintiles. Indeed, a linear trend was found between BMI quintiles in males and females ( $P$ -value in the linear trend test < 0.001). However, this linear trend was not observed for BMI quintiles at age 20 ( $P = 0.612$ ).

We also examined the correlation between inter-spouse BMI and the current BMI-SDS quintiles of the offspring (Figure 3). A higher inter-spousal resemblance was found among parents of either lean or obese offspring (extreme BMI values) than among parents of "intermediate BMI-SDS" ('U' shaped distribution).

## DISCUSSION

In the present study we have found a correlation between parental and offspring BMI considering parental BMI both at present and when the parents were 20 years old. There are studies on familial resemblance and adiposity-related parameters with similar results<sup>(21,22)</sup>.

One limitation of this study is sample size, and another one is that the analysis is based on parental BMI calculated from self-reported data. Only the anthropometric data for the 408 offspring were directly measured by trained personnel.

Nowadays, it is clear that there is a parent-offspring resemblance in obesity degree, and that subjects with obese parents have a high obesity risk due to the effects of both genetic and environmental factors<sup>(12,23-25)</sup>. Our results suggest that obesity in mothers increases the offspring obesity risk more than obesity in fathers. One possible explanation for this effect is that in this particular Spanish population mothers probably play a greater role in educating and feeding children than fathers. On the other hand, it has been shown that maternal obesity might lead to obesity in the offspring via prenatal factors<sup>(26,27)</sup>. In the study of Kivimaki *et al.*<sup>(10)</sup>, maternal BMI was also more strongly associated with offspring birth weight than paternal BMI.

In our study, we found a significant linear association between BMI quintiles in spouses at present, but this association in BMI quintile frequencies was not significant when the spouses were 20 years old. This might indicate that there is no coincidental mating for obesity in this Spanish adult population; however, spouses probably change their habits with cohabitation and time, as when the offspring are 5-19 years old a correlation between spouses' BMI arises.

This could be an important issue in the development of nutritional education programs. Nutritional educators should endeavour to modify not only the habits of the children but also the parental lifestyle and alimentary habits. The key ingredient for a successful obese children treatment program is probably the participation and implication of both parents. Hebebrand *et al.*<sup>(28)</sup> found coincidental mating among parents of extremely obese children and adolescents, and Jacobson *et al.*<sup>(13)</sup> showed that spouse correlations in BMI were strongest among couples with the shortest duration of cohabitation; however, we have not seen a similar effect in our study population of parents of obese and non-obese children. In contrast, other studies have found spouse similarity in BMI to be due, at least partly, to the shared household environment<sup>(29,30)</sup>.

Our results show that parents of obese or lean children have a higher resemblance in BMI than parents of "average" children. This effect was also observed by Katzmarzyk *et al.*<sup>(30)</sup> in a study of 1341 families, and is also in line with the findings of Hebebrand *et al.*<sup>(12)</sup>. The U-shaped distribution pattern could lead to a population with higher prevalence of extremely lean and extremely obese subjects.

To our knowledge, this is the first time that this phenomenon is reported in a Spanish population. It is also confirmed that obese parents should be considered as a serious risk for obesity in the offspring.

In summary, the present study corroborates the correlation between parental and offspring BMI, although it seems that there is no coincidental mating for obesity in this Spanish adult population. This finding has important implications in the context of the obesity epidemic.

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