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# THE EFFECT OF OCCASIONAL ALCOHOL DRINKING ON SEMEN QUALITY AND SPERM MORPHOLOGY AMONG YOUNG AND HEALTHY POLISH MEN

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## ABSTRACT

### Background

Ethanol (EtOH) is an agent that seems to exert an especially harmful effect on male fertility. The impact of high EtOH intake on fertility was demonstrated in numerous researches, with data suggesting that this effect may have been due to decreased semen quality; however, similar negative effects were not identified among occasional EtOH drinkers. There are currently no recommendations for alcohol consumption for men who plan to have a child other than avoiding high EtOH intake. Thus, studies on the effect of moderate and occasional EtOH drinking on semen quality are needed to develop appropriate recommendations for men planning to have a child in the future. The aim of this study was to determine whether changes in semen-quality parameters and sperm morphology occur in healthy young men who occasionally exceed the WHO-recommended weekly dose of EtOH but are not alcohol dependent and do not frequently consume high amounts of EtOH.

### Methods

The study sample consisted of 172 young men residing in urban areas. The semen quality and morphology of men who consumed more than 140 g of ethanol (high-risk group, HR, $n$ = 44) weekly was compared with that of low-risk group members (LR, $n$ = 128) who reported lower alcohol consumption.

**Results**
The only between-group difference in semen characteristics was the identification of a higher percentage of macrocephalic sperm in the HR group ($P = 0.011$). Alcohol intake was the sole factor influencing the percentage of macrocephalic sperm ($\beta = 0.171$, $P = 0.025$, multiple linear regression).

**Conclusions**
We concluded that occasional alcohol consumption did not alter fertility but caused the accumulation of macrocephalic sperm potentially containing damaged DNA. Therefore, we recommend that men who plan to father children stop drinking alcohol at least 3 months before engaging in sexual intercourse that may lead to pregnancy.

**Keywords:** *alcohol intake; semen quality; sperm morphology; macrocephalic sperm*

Infertility is a public health problem that seems to be especially serious in developed countries.[1] As reduced semen quality may play a significant role in infertility,[2] it is important to identify factors underlying this condition. Several environmental pollutants and lifestyle factors that may cause human semen quality to worsen have been identified.[3–5] One agent that seems to exert an especially harmful effect on male fertility is ethanol (EtOH).[6–8]

The impact of high EtOH intake on fertility was confirmed in a recent systematic review and meta-analysis, with data suggesting that this effect may have been due to decreased semen quality; however, similar negative effects were not identified among occasional EtOH drinkers.[9] The authors also indicated that there are currently no recommendations for alcohol consumption for men who plan to have a child other than avoiding high EtOH intake. Such recommendations have been provided only for women who are pregnant, planning to become pregnant, or breastfeeding, and both this group and adolescents are advised to not drink alcohol at all.[10] Thus, studies on the effect of moderate and occasional EtOH drinking on semen quality are needed to develop appropriate recommendations for men planning to have a child in the future.

The aim of this study was to determine whether changes in semen quality parameters and sperm morphology occur in healthy young men who occasionally exceed the WHO-recommended weekly dose of EtOH but are not alcohol dependent and do not frequently consume high amounts of EtOH.

## MATERIALS AND METHODS

### *Participants*

This study examined a homogeneous urban population of young healthy Polish men from the city of Wroclaw (Lower Silesia Region in Southern Poland). Eligible participants were informed about the study via personal communication, flyers and posters distributed on the properties of universities in Wroclaw and their sports clubs in addition to social media applications, such as Facebook, Twitter and Instagram. The volunteers were unpaid. The eligibility criteria were as follows: absence of any known andrologic pathology (past and present), such as hypogonadotropic or hypergonadotropic hypogonadism; absence of urogenital surgery; and absence of drugs that might interfere with the evaluation of semen quality. Approximately 500 volunteers agreed to participate in the study and were subjected to medical interviews and medical record reviews. After the interview, many of the potential respondents refused further participation, perhaps due to religious or cultural reasons. Men who declared alcohol dependence or reported frequent heavy alcohol use were also excluded from the study. The final study sample consisted of 172 Caucasian men aged 18–31 years. The final participants were asked to complete questionnaires covering their medical history and smoking and alcohol drinking habits (7-day recall). Permission to conduct the study was provided by the Ethics Committee of the University School of Physical Education in Wroclaw (no. 36/2.12.2013). All participants provided written informed consent. All procedures involving human subjects were conducted

in compliance with the Declaration of Helsinki and the European Communities Council Directive of 24 November 1986 (86/609/EEC).

### *Alcohol Consumption Estimation*

According to the Polish government's definition and the guidelines for low-risk alcohol consumption proposed by the WHO, each standard drink or unit of alcohol was considered to contain 10 g alcohol.[11,12] To calculate total EtOH intake, all participants were asked to specify the number of bottles of beer (0.5 L = 2.5 units = 25 g EtOH), glasses of wine (0.175 L = 1.68 units = 16.8 g EtOH) and glasses of vodka or other strong alcoholic liquors (0.05 L = 1.6 units = 16 g EtOH) they had consumed during the week prior to the visit to the andrology laboratory. Total alcohol intake was estimated by adding together the mass of EtOH in each drink consumed during the week. For analysis of associations between semen characteristics and alcohol consumption the total EtOH intake was expressed as the mass of alcohol consumed (g) per body mass (kg) of the participant per week (g EtOH/kg body mass/week).

### *Semen Analysis and Estimation of Hormonal Parameters*

Semen-quality parameters as well as sperm morphology were analyzed according to the WHO manual[13] as described elsewhere.[14] Blood samples were collected for hormone assessments on the day of semen collection. Serum sex hormones such as luteinizing hormone (LH), follicle-stimulating hormone (FSH), total testosterone (T), sex hormone-binding globulin and albumin were determined in a government-approved commercial andrology laboratory. Free testosterone (FT) was calculated using a calculator developed by the Hormonology Department, University Hospital of Ghent, Belgium (http://www.issam.ch/freetesto.htm).

### *Statistical Analysis*

The data were analyzed using SigmaPlot, version 13.0 (Systat Software Inc., London, UK). Continuous variables were first tested for normality using the Kolmogorov-Smirnoff test with Lilliefors correction. Biochemical parameters describing the hormonal status of each participant exhibited normal distributions, whereas all semen parameters exhibited non-normal distributions. Untransformed descriptive statistics are presented in Table 1. All values are expressed as the median and 5th and 95th percentiles. Linear simple and multiple regression analyses were performed to evaluate the associations between the percentage of macrocephalic sperm, ethanol intake and hormonal parameters. Dependent variables such as semen volume, sperm concentration and total sperm count were transformed using the Box Cox transformation to obtain normally distributed data. Semen characteristics expressed as percentages (total sperm motility, vitality and numbers of normal and pathological sperm forms) were first converted into proportions and then transformed using arcsine square root transformation. Factors with a $P$ value <0.05 in simple linear regression analysis were included in multiple linear regression analyses. The significance of differences between two proportions was analyzed by the Z-test. A $P$ value <0.05 was considered statistically significant.

## RESULTS

### *Participant characteristics*

Table 1 shows the general characteristics of the studied participants including their hormonal parameters and social and lifestyle characteristic data. All men reported good health status; only 1 (0.6%) man was obese, and 6 (3.5%) men were overweight (data not shown). The participants' hormonal parameters, such as LH, FSH, T, FT, and SHGB, were all within the reference range. All men were urban residents. 55.2% of participants were university students or graduates, 44.8% had completed a secondary level of education. Smoking was not frequently reported among the participants, and only 13.9% of the respondents were current or ex-smokers.

### *Alcohol Intake and Semen Quality Characteristics*

As governmental standard drink definitions and weekly low-risk consumption guidelines for grams of pure EtOH vary significantly between countries, we decided to use the WHO standard drink unit, defined as 10 g of EtOH, and defined the weekly low-risk alcohol consumption for men as 14 units (140 g).[11] We classified the participants into two groups using this method. The low-risk EtOH group (LR) consisted of men whose weekly EtOH intake was no more

**Table 1** Descriptive Characteristics of the Participants ($n$ = 172).

| Variable | All participants $n$ = 172 | LR group $n$ = 128 | HR group $n$ = 44 | LR vs HR $P$ |
|---|---|---|---|---|
| Age (years) | 24.0 (19.6 – 29.0) | 24.0 (20.0 – 29.0) | 23,5 (19.0 – 29.0) | 0.337 |
| BMI (kg/ $m^2$) | 23.8 (19.9 – 29.0) | 24.0 (20.4 – 29.3) | 24.1 (19.4 – 28.7) | 0.573 |
| LH (mIU/ml) | 4.6 (2.3 – 9.4) | 4.5 (2.1 – 9.4) | 4.7 (2.4 – 11.7) | 0.934 |
| FSH (mIU/ml) | 3.3 (1.4 – 9.1) | 3.3 (1.2 – 8.7) | 3.1 (1.3 – 11.3) | 0.757 |
| T (ng/ml) | 5.9 (3.4 – 9.0) | 5.9 (3.7 – 9.4) | 6.0 (3.4 – 10.2) | 0.577 |
| FT (ng/ml) | 0.10 (0.06 – 0.16) | 0.10 (0.06 – 0.16) | 0.11 (0.07 – 0.29) | 0.173 |
| SHBG (nmol/l) | 37.8 (19.3 – 69.7) | 32.4 (17.8 – 69.6) | 39.3 (19.3 – 69.1) | 0.078 |
| Secondary education level ($n$) | 77 (44.8%) | 56 (43.7%) | 21 (47.7%) | 0.776 |
| University students and graduates ($n$) | 96 (55.2%) | 72 (56.2) | 23 (52.3%) | 0.785 |
| Current and ex-smokers ($n$) | 24 (13.9%) | 19 (14.8) | 5 (11.4) | 0.757 |

*Data are medians and 5th – 95th percentiles except three last rows, where participants number and percentage are given. LR, Low-risk alcohol intake ≤ 140 g/week; HR, High-risk EtOH intake > 140 g/week. BMI = body mass index; LH = luteinizing hormone; FSH = follicle-stimulating hormone; T = total testosterone; FT = free testosterone SHBG = sex hormone binding globulin.*

**Table 2** Descriptive Characteristics and Semen Quality Parameters of the Participants Classified According To Ethanol Intake

| Parameter | LR group $n$ = 128 | HR group $n$ = 44 | $P$ |
|---|---|---|---|
| Semen volume (mL) | 2.8 (1.0 – 5.7) | 3 (1.6 – 6.8) | 0.238 |
| pH | 7.9 (7.8 – 8.4) | 7.9 (7.7 – 8.2) | 0.168 |
| Leukocytes ($10^6$/mL) | 0.0 (0.0 – 0.6) | 0 (0.0 – 0.7) | 0.539 |
| Time of liquefaction (min) | 25.0 (15.0 – 45.5) | 25.0 (15.0 – 56.2) | 0.974 |
| Sperm concentration ($10^6$/mL) | 50 (6 – 164) | 52 (6 – 149) | 0.385 |
| Total sperm count ($10^6$/ejaculate) | 132 (15 – 381) | 158 (15 – 745) | 0.127 |
| Progressive motility (grades a + b, %) | 36 (11 -59) | 36 (9 -65) | 0.449 |
| Total motility (grades a + b + c, %) | 55 (23 – 75) | 58 (19 – 80) | 0.263 |
| Vitality (%) | 60 (35 – 80) | 62 (24 – 81) | 0.530 |
| Normal forms (%) | 14.0 (3.4 – 28.0) | 14 (1.7 – 23.7) | 0.563 |

*Data are medians and 5th – 95th percentiles. EtOH = ethanol; LR = low-risk EtOH intake ≤ 140 g/week; HR = high-risk EtOH intake > 140 g EtOH/week.*

than 14 units, and the high-risk EtOH group (HR) comprised men whose weekly alcohol consumption exceeded 14 units. No differences between the two groups were found with regard to anthropometric, lifestyle and hormonal characteristics (Table 1). The comparison of semen-quality characteristics between LR and HR EtOH consumers is shown in Table 2. All tested characteristics were found to be similar in the LR and HR groups, and no statistically significant between-group differences were observed ($P > 0.05$).

The morphological characteristics of the sperm in the samples obtained from the LR and HR groups are shown in Table 3. Almost all pathological sperm forms were present at similar percentages in the LR and HR groups. The only exception was the percentage of macrocephalic sperm; this form was more frequently identified in sperm collected from HR group members. The median values were 4 (1–9.2) and 3 (0–7) in the HR and LR groups, respectively, and the difference between these values was statistically significant ($P = 0.011$).

### *Effects of Gonadotropins, Sex Hormone Levels and Alcohol Intake on the Frequency of Macrocephalic Sperm*

As macrocephalic sperm was the only pathological sperm form whose percentage differed between the HR group and the LR group, we performed simple and multiple regression analyses to determine whether the percentage of macrocephalic sperm was associated with EtOH intake and/or sex hormone levels. To avoid multicollinearity between independent variables, we first performed simple regression analyses to determine whether the participants' hormonal parameters were dependent upon their EtOH intake. These analyses revealed that the levels of LH, FSH, T, and FT were not associated with weekly EtOH intake (data not shown). The results of simple regression analyses of the associations between the percentage of macrocephalic sperm, hormonal parameters and EtOH intake are shown in Table 4.

The percentage of macrocephalic sperm was significantly associated with the levels of total testosterone ($R = 0.174$, $P = 0.022$, $\alpha = 0.631$), free testosterone ($R = 0.152$, $P = 0.046$, $\alpha = 0.516$) and weekly EtOH intake ($R = 0.216$, $P = 0.005$, $\alpha = 0.813$).

Only the association between EtOH intake and the percentage of macrocephalic sperm demonstrated a statistical power that exceeded the desired 0.800. The statistical power values identified for the two other significant analyses were lower than 0.800, which raises some doubts regarding whether the relationships observed between macrocephalic sperm percentage

**Table 3** Sperm Pathological Forms of the Participants Classified According to Ethanol Consumption

| Sperm pathological form | LR group<br>*n* = 128 | HR group<br>*n* = 44 | *P* |
|---|---|---|---|
| Amorphous head (%) | 54.5 (44.0 – 65.1) | 54.0 (46.0 – 66.5) | 0.393 |
| Round head (%) | 3 (0 – 10) | 3 (0 – 9) | 0.508 |
| Tapered head (%) | 1.0 (0.0 – 8.6) | 0.5 (0.0 – 5.8) | 0.077 |
| Microcephalic head (%) | 2.0 (0.0 – 6.6) | 1.0 (0.0 – 7.5) | 0.408 |
| Macrocephalic head (%) | 3.0 (0.0 – 7.0) | 4.0 (1.0 – 9.2) | **0.011*** |
| Vacuolated head (%) | 3 (1 – 7) | 4 (1 – 7) | 0.223 |
| Double headed (%) | 0.0 (0.0 – 2.0) | 0.0 (0.0 -1.8) | 0.867 |
| Abnormal middle-piece (%) | 7 (2 – 16) | 7 (3 – 15) | 0.870 |
| Abnormal tail (%) | 7.0 (2 – 16) | 6.5 (3 – 16) | 0.831 |

*Data are medians and 5th – 95th percentiles. EtOH = ethanol; LR = low-risk EtOH intake ≤ 140 g/week; HR = high-risk EtOH intake > 140 g/week. Asterisk and bold typeface indicate statistically significant difference.*

**Table 4** Correlation Coefficients by Simple Regression Analyses between Hormonal Parameters, EtOH Intake and the Percentage of Macrocephalic Sperm ($n$ = 172).

| **Simple Linear Regression** Dependent variable: percentage of macrocephalic sperm | | | |
|---|---|---|---|
| **Independent variable** | **R** | ***P*** | **α** |
| LH (mIU/mL) | 0.074 | 0.330 | 0.162 |
| FSH (mIU/mL) | 0.076 | 0.323 | 0.165 |
| T (ng/mL) | **0.174** | **0.022*** | 0.631 |
| FT (ng/mL) | **0.152** | **0.046*** | 0.516 |
| SHBG (nmol/L) | 0.100 | 0.188 | 0.260 |
| EtOH intake† | **0.216** | **0.005*** | 0.813 |
| **Multiple linear regression** Dependent variable: percentage of macrocephalic sperm | | | |
| **Independent variable** | **β** | ***P*** | **α** |
| T (ng/mL) | 0.086 | 0.365 | 0.902 |
| TC (ng/mL) | 0.111 | 0.175 | |
| EtOH intake† | **0.171** | **0.025*** | |

*LH = luteinizing hormone; FSH = follicle-stimulating hormone; T = total testosterone; FT = free testosterone; SHBG = sex hormone binding globulin; R = correlation coefficient; β = standardized correlation coefficient; α = power of the test; EtOH = ethanol.*

*†/ Weekly EtOH intake (g/kg of body mass). Asterisk and bold typeface indicate statistically significant association.*

and T and FT levels were true associations or occurred only by chance. To clarify this issue, multiple linear regression analysis was performed including the independent variables with $P$ values lower than 0.05 (T, FT levels and weekly EtOH intake) that were identified in the univariate analyses. The results of the multiple linear regression analysis, which are presented in Table 4, revealed that EtOH intake was the only factor influencing the percentage of macrocephalic sperm ($\beta = 0.171$, $P = 0.025$). Associations between the percentage of macrocephalic sperm and hormonal parameters were not supported by the results of this analysis ($P>0.05$). The power of the multiple regression analysis was 0.902, indicating that the results are reliable (Table 4). Therefore, T and FT levels did not seem to be independent factors influencing the percentage of macrocephalic sperm observed and could be removed from the regression model.

## DISCUSSION

In this study, we found that young and healthy adult males recruited from an urban population who reported occasionally consuming more than 140 g EtOH/week, which corresponds to HR EtOH intake,[12] had sperm quality characteristics that were similar to those of their LR counterparts (≤140 g EtOH/week). Our results were in accordance with several earlier reports wherein no decrease in semen-quality parameters was identified among men who were not heavy alcohol drinkers.[9] Thus, it may be concluded that occasional EtOH drinking that exceeds the maximal number of 14 standard alcohol drinks per week, as recommended by the WHO,[12] should not affect male fertility.

The following semen characteristics were evaluated among LR and HR alcohol consumers: ejaculate volume, pH, the presence of leukocytes, the time of liquefaction, sperm concentration, total sperm count,

progressive motility, total motility, vitality and the percentage of normal sperm forms. These are standard semen-quality parameters that are usually evaluated in andrology clinics. Using this set of semen characteristics, information regarding sperm morphology is limited only to the percentage of normal sperm forms.[13,15] However, other authors have proposed that the percentage of some specific morphologically abnormal sperm may have a prognostic value in male fertility.[16] As noted by Mankfeld and collaborators, the cut-off value (4%) for normal sperm morphology is very low and may not to be sufficient for the prediction of male fertility potential.[17,18] These authors suggested that semen evaluations should also include more detailed sperm morphology examinations to provide additional information regarding the distribution of specific abnormal sperm forms. We followed this suggestion, and the semen of the participants was analyzed not only for standard parameters but also for the presence of several pathological sperm forms (Table 3). The only significant difference between the LR and HR groups was the identification of a higher percentage of macrocephalic sperm in HR group members (Table 3). Previous studies have suggested that the decrease in semen quality caused by alcohol consumption may occur as a result of the adverse effects of EtOH on both the metabolism of reproductive hormones and the process of spermatogenesis.[7,8,19] In this study, we did not identify associations between the levels of sex hormones such as LH, FSH, T, FT and the percentage of macrocephalic sperm. Additionally, although simple regression analysis suggested an association between the percentage of macrocephalic sperm and the levels of T and FT, this association was not supported by multiple regression analysis (Table 4). We also did not identify a relationship between reproductive hormone levels and weekly EtOH intake (data not shown). Thus, we concluded that EtOH intake higher than 140 g/week was the only factor associated with an increased percentage of macrocephalic sperm and that the levels of reproductive hormones did not mediate this effect. Previous studies have reported that chronic alcoholism has a significant detrimental effect on sex hormone equilibrium and semen-quality characteristics and causes increased levels of head-defective sperm.[19] The difference between our results and that report probably occurred due to the fact that the participants in this study were men who did not drink alcohol regularly but rather occasionally consumed alcoholic beverages during social events such as family parties, receptions, and in pubs. It is possible that this occasional exposure was too minimal to disturb the equilibrium of sex hormones.

Our results raise a question regarding the mechanism by which EtOH increases the percentage of macrocephalic sperm. As the participants in this study did not report frequent or high EtOH consumption, it seems unlikely that the elevated percentage of macrocephalic sperm was caused by toxic effects of EtOH on spermatogenesis and/or testes, as was previously identified among chronic alcohol consumers.[6] We hypothesize that the observed increase in the percentage of macrocephalic sperm may have been the result of direct toxic actions of EtOH and/or its metabolites on sperm. The results of numerous studies have suggested that the cellular toxicity of EtOH is mediated by the generation of reactive oxygen species (ROS), leading to oxidative stress.[20] As ROS are highly reactive and have no specific targets, these agents can cause many lesions while reacting with cellular components such as nucleic acids, proteins and lipids.[21] Oxidative stress is believed to play an important role in the reduction of sperm fertilization potential, as spermatozoa possess limited capacity to repair DNA damage and are therefore susceptible to oxidative stress.[22] ROS may cause a wide spectrum of chromatin lesions through oxidation, nitration, halogenation and base alkylation, DNA interstrand crosslinking and DNA-protein crosslinking.[23] Due to spontaneous reactions or repair processes, these lesions may be converted into DNA breaks (DNA fragmentation), resulting in chromatin decondensation. Previous studies have reported similar chromatin damage in the spermatozoa of EtOH-consuming rats.[24] Chromatin decondensation and DNA fragmentation were reported to be positively correlated with the percentage of macrocephalic sperm[25] and other types of morphologically defective spermatozoa.[26] Although we have no direct proof, we hypothesize that the association between the percentage of macrocephalic sperm and EtOH consumption observed in this study reflected DNA fragmentation caused by ROS.

Perhaps the most important question raised by our findings is whether having a higher percentage of macrocephalic spermatozoa, which was found to be associated with occasional alcohol intake higher than 140 g/week (HR group of alcohol drinkers), has any clinical significance. The semen-quality parameters that are typically evaluated in andrology clinics were similar in HR and LR alcohol consumers. Therefore, it seems that fertility is not decreased among men who occasionally drink EtOH at amounts higher than that recommended by the WHO[12] (Table 2). The elevated percentage of macrocephalic sperm identified in the HR group also did not seem to affect fertility, as it was not accompanied by a lower percentage of normal sperm forms. However, macrocephalic spermatozoa have been associated with decreased fertility or infertility.[27,29] Therefore, the increase in the percentage of this type of defective sperm in the semen of HR group members may be an early sign of the sperm damage induced in male germ cells by EtOH-derived ROS in addition to several other genotoxic, mutagenic and carcinogenic compounds that are present in alcoholic beverages.[30] This hypothesis is plausible, as human semen has been reported as an early and sensitive biomarker of environmental pollution.[4] Thus, we assume that elevated amounts of macrocephalic sperm should not to be ignored, as they may reflect the accumulation of DNA damage related to EtOH consumption.

This work had some limitations. (1)The sizes of the study groups were relatively small, and therefore, some between-group comparisons had less than the desired 0.8-level of statistical power. Thus, it is possible that the lack of differences observed between semen characteristics (presented in Tables 2 and 3) occurred by chance. (2) We have no data concerning oxidative stress, antioxidant defense in semen, or the fragmentation of spermatozoa chromatin, thus limiting our ability to explain our results in the context of some of the data available in the literature. (3) An additional limitation is that macrocephalic sperm is a surrogate marker of male reproductive health and therefore, more extensive studies are needed to provide proof that occasional alcohol drinking by men is associated with DNA damage in spermatozoa and presents a potential risk for cancer development in offspring or birth defects.

## CONCLUSIONS

The results of this study suggested that fertility was not reduced among young and healthy men who were not alcohol dependent and occasionally drank alcoholic beverages in amounts exceeding the WHO-recommended weekly dose of 140 g (14 standard drinks). However, even occasional EtOH consumption can cause the accumulation of macrocephalic sperm potentially containing damaged DNA. This possibility is of concern, as mutagenic and/or carcinogenic DNA lesions may also be present in spermatozoa that seem to be morphologically normal, which may potentially affect the health of offspring. Therefore, we recommend that men who plan on fathering children reduce or even stop any alcohol consumption at least 3 months before engaging in sexual intercourse that may lead to pregnancy. As the duration of spermatogenesis is approximately 74 days,[31] this time interval seems to be sufficient to replace sperm that may be defective due to EtOH consumption.

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