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► **To cite this version:**

Gwen-Jiro Clochard, Aby Mbengue, Clément Mettling, Birane Diouf, Charlotte Faurie, et al.. The effect of the 7R allele on the DRD4 risk tolerance locus is independent of background risk in Senegalese fishermen. *Scientific Reports*, 2023, 13 (622), 10.21203/rs.3.rs-1968350/v1 . hal-03930527

HAL Id: hal-03930527

<https://hal.inrae.fr/hal-03930527>

Submitted on 10 Jan 2023

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Article

Keywords:

Posted Date: September 29th, 2022

DOI: <https://doi.org/10.21203/rs.3.rs-1968350/v1>

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The effect of the 7R allele on the DRD4 risk tolerance locus is independent of background risk in Senegalese fishermen

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ABSTRACT

It has been shown that living in risky environments, as well as having a risky occupation, can moderate risk-tolerance. Despite the involvement of dopamine in the expectation of reward described by neurobiologists, a GWAS study was not able to demonstrate a genetic contribution of genes involved in the dopaminergic pathway in risk attitudes and gene candidate studies gave contrasting results. We test the possibility that a genetic effect of the DRD4-7R allele in risk-taking behavior could be modulated by environmental factors. We show that the increase in risk-tolerance due to the 7R allele is independent of the environmental risk in two populations in Northern Senegal, one of which is exposed to a very high risk due to dangerous fishing.

Introduction

Humans need to adapt their behavior as a result of risk. Previous research has shown that risk coping attitudes are partly heritable¹. Genes involved in the regulation of the dopaminergic system are good candidates to explain the heritability of risk behavior. However, many reports on gene and behavior association, based on small-sample candidate gene have found contrasting results, leading to debates in the scientific community^{2,3}.

To overcome this limitation, a genome-wide association study (GWAS), based on over 1 million individuals, identified 99 loci associated with general risk tolerance⁴. Surprisingly, none of identified loci were close to genes involved in the dopamine pathway. Their bioinformatic analysis pointed to the role of genes expressed in brain regions involved in decision-making, although genes involved in dopamine biosynthesis (TH) or receptors (DRD1,2,3 and 4) did not reach statistical significance.

Yet, the evidence that not only these brain regions but the dopamine neurotransmitter itself plays a role in the expectation of reward is compelling: dopaminergic neurons can code the probability of the reward in a primate model⁵. Moreover, a known side-effect of Parkinson (known to impair dopamine production) treatment is to dramatically increase impulsivity⁶. The dopamine receptor gene DRD4 fulfills many criteria as a good candidate gene: it is highly polymorphic^{7,8}, expressed in the prefrontal cortex, it shows an unusually large variable repeat region coding for 16 amino acids in the third cytoplasmic loop, a region interacting with SH3 domain-binding proteins.

While the 4 repeat (4R) variant is the ancestral, and most common allele in all human populations⁹, there exist variations between 2 and 11 repeats (2R to 11R). The different alleles have functional differences¹⁰⁻¹³. The DRD4-7R allele is under strong positive selection in human population¹⁴⁻¹⁶, and has been shown to be linked to more risk-tolerant attitudes¹⁷⁻¹⁹. However, some findings revealed a lack of differences in the domain of financial risk-taking²⁰⁻²³.

The discrepancy between these different studies may come from the fact that GWAS studies underestimate the genetic variance due to gene-gene or gene-environment interactions or an inability to capture rare genetic variants, and candidate-gene studies conducted in specific environments may sometimes benefit from circumstances revealing a genetic variance. For instance, administration of L-DOPA to volunteers did not lead to an increase in gambling propensity unless the subjects carried at least one copy of the 7-repeat allele²⁴. The negative association with DRD4 variation and risk-taking previously reported²³ might have been concealed by the association with MAOA variation, an enzyme catalysing dopamine. It is then possible that a genetic effect of DRD4 on risk-taking behavior can only be shown in some circumstances, and that the GWAS study, by leveling all environmental condition or gene interaction may mask some dopaminergic genetic contribution.

Humans also adapt their risk attitudes as a response to the level of risk in their environment²⁵. In particular, people have been found to be more risk-averse in the presence of unfair background risk²⁶⁻²⁹, in accordance with the “risk-vulnerability” hypothesis^{30,31}. The aim of the present paper is to test the interaction between the influence of the 7R allele on risk-tolerance and the level of risk to which people are exposed.

Results

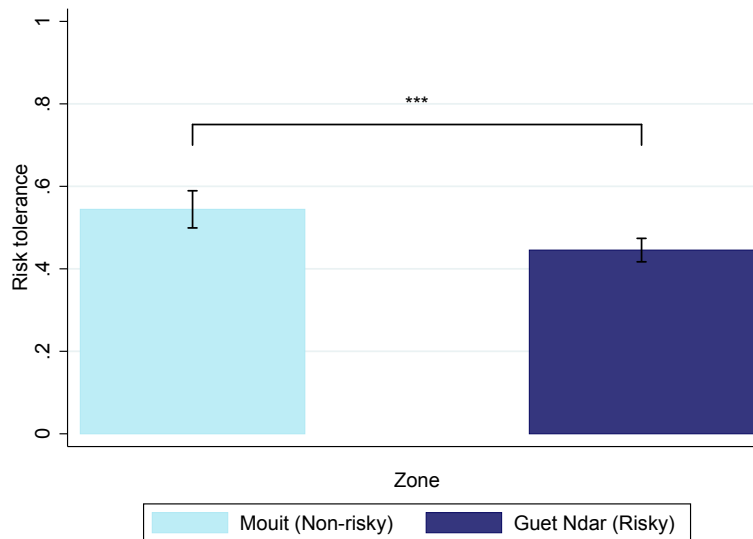
Risk-tolerance by zone

The village of Guet Ndar (Saint-Louis region in Northern Senegal) is famous for its fisheries. Fishing in the area is very dangerous, with authorities reporting 20 deaths due to fishing on average per year over the past 20 years³². Given the demography of the village, with 20 000 inhabitants, among which fishing represents the main occupation of approximately 80% of the adult male workforce, this corresponds to approximately 4% of the male population who died due to fishing in the last 20 years. The prevalence of deaths is strongly linked to the intersection of strong currents coming from the Senegal river and an upwelling current from the ocean³³. However, these currents attract a lot of fish, making fishing more profitable than other activities in the region (fishermen in our sample declare income significantly higher than non-fishermen, $p < 0.01$, Table S.1).

In this paper, we compared populations from the fishing village of Guet Ndar ($N = 601$), which is labelled as the *risky area*, and that of a farming village called Mouit, 23 kilometers away ($N = 263$), labelled the *non-risky area*. Importantly, the two populations are mostly composed of the same ethnic group (the Wolofs, representing approximately 80% of the sample in both areas). Because fishing is an activity predominately performed by men, our sample only consists of men.

Our experimental measure of risk-tolerance was based on a lottery task³⁴. A description of the task is provided in the Supplementary Materials. Results indicate that risk-tolerance varied between the risky and non-risky areas. Participants from the risky area tended to exhibit less risk-tolerance than participants from the non-risky area (Figure 1, Student's t -test $p < 0.01$). The difference remains significant after controlling for age and education (Table S.2). Our data is consistent with field data and laboratory experiments showing that people exposed to high background risk tend to exhibit less risk-tolerance, in accordance with the “risk-vulnerability hypothesis”^{30,31}.

Figure 1. Average level of risk-tolerance by zone



Note: A higher level of risk-tolerance indicates the choice of a riskier lottery by participants in the lottery choice task. Segments represent 95% confidence intervals. Student's *t*-test * $p < 0.1$, ** $p < 0.05$, *** $p < 0.01$.

Genotypes by zone

Genotypes at the DRD4 locus displayed two common alleles (4R and 7R, with 4 and 7 repeats, respectively), which was expected for populations in Sub-Saharan Africa⁹, and 5 minor alleles with negligible frequencies (2R, 3R, 5R, 6R and 8R) leading to 21 different genotypes (Table 1). Within each area, populations were not at Hardy-Weinberg equilibrium ($p < 0.01$ in the non-risky area, $p = 0.02$ in the risky area), and displayed an heterozygote deficiency ($F_{IS} = 0.134$ in the non-risky area, and $F_{IS} = 0.052$ in the risky area).

Because we are primarily interested in the effect of the 7R allele on risk-taking, we combined all other alleles into a single category, identified as allele "X". This combination yields three genotypes: XR/XR, XR/7R and 7R/7R. Hardy-Weinberg equilibrium was rejected ($p = 0.01$) for the non-risky area, but not for the risky area ($p = 0.40$), see Table 1. Deviations from HW equilibrium were $F_{IS} = 0.181$ in the non-risky area, and $F_{IS} = 0.037$ in the risky area.

The genotypic differentiation between the two areas was measured as $F_{ST} = 0.0036$, and was marginally non-significant (exact G test, $p = 0.094$). This level of genotypic differentiation was compared with those displayed by 30 micro-satellite loci. One locus (032) was not polymorphic and was discarded. The other 29 loci displayed between 2 and 15 alleles. Their level of genotypic differentiation ranged between $F_{ST} = -0.0094$ and $F_{ST} = 0.0226$, with an overall average value of $F_{ST} = 0.0035$ (Figure S.2 and Table S.4).

Risk-tolerance by genotype

Risk-tolerance was not independent of genotype at the DRD4 locus (Figure 2 and Table 2, Column 1). The 7R allele demonstrated a significant additive effect ($p = 0.01$), and no dominance effect was found ($p = 0.31$). The 7R allele increases risk-tolerance. Importantly, the result holds after controlling for age, education and the living area (Table 2, Column 2). Our results indicate that the 7R allele is associated with more risk-tolerance than other alleles, in line with previous literature^{17,18}.

Moreover, environmental risk did not appear to significantly moderate the effect of the 7R allele. First, its additive effect holds when analyzing both areas separately (Table 2, Columns 3 and 4, Figure S.1), although the significance levels drop slightly due to sample limitations ($p = 0.05$ and $p = 0.08$ in the non-risky and risky area, respectively). Second, the interaction between the additive effect and the area (Table 2, Column 5) was not significant ($p = 0.25$).

Discussion

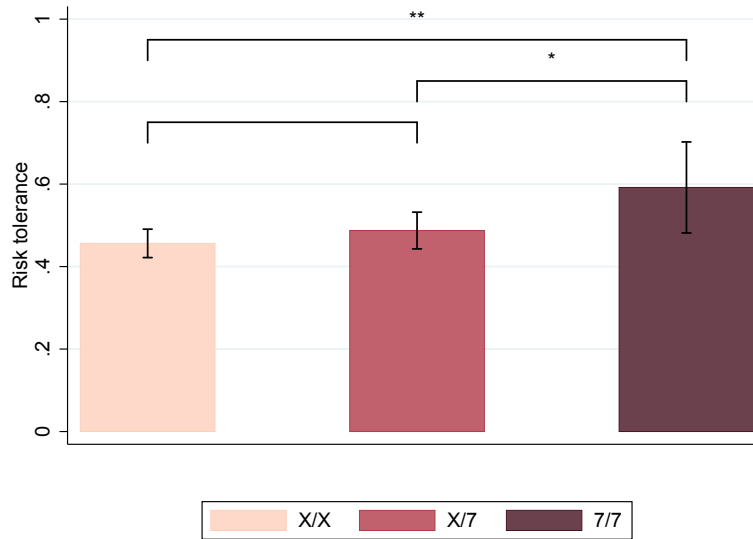
In this paper, we find that the 7R allele of DRD4 affects risk-attitudes by an additivity effect, not a dominance effect. This is in contrast with previous research³⁵ who found that heterozygotes 2R/4R had lower risk tolerance. It is unclear if this difference comes from the type of risky environment considered (background volcanic risk or risky subsidence type), or comes from the

Table 1. Genotypic composition at the *DRD4* locus of populations from the Saint-Louis region in the non-risky and risky areas.

Genotype	Non-risky area		Risky area	
	<i>N</i>	%	<i>N</i>	%
<i>Panel A. Without combination of genotypes</i>				
22	3	1.4	3	0.6
24	7	3.3	14	2.8
25	1	0.5	-	-
27	-	-	2	0.4
34	3	1.4	-	-
36	1	0.5	-	-
37	1	0.5	-	-
44	84	39.1	202	40
45	19	8.8	34	6.7
46	14	6.5	18	3.6
47	48	22.3	149	29.5
48	5	2.3	12	2.4
55	4	1.9	3	0.6
56	-	-	1	0.2
57	4	1.9	13	2.6
58	-	-	2	0.4
66	2	0.9	1	0.2
67	1	0.5	12	2.4
77	15	7	34	6.7
78	3	1.4	4	0.8
88	-	-	1	0.2
<i>HW equilibrium</i>				
<i>p</i>	<0.01		0.023	
<i>Panel B. Allele 7R versus other alleles</i>				
XX	143	66.5	291	57.5
X7	57	26.5	181	35.8
77	15	7.0	34	6.7
<i>HW equilibrium</i>				
<i>p</i>	0.011		0.40	

The p-value (*p*) corresponds to the HW probability exact test. Genotype ij refers to the *DRD4* genotype iR/jR. For *Panel B*, all alleles not 7R are combined in the X allele.

Figure 2. Average level of risk-tolerance by genotype



Note: A higher level of risk-tolerance indicates the choice of a riskier lottery by participants in the lottery choice task. X/X, X/7 and 7/7 represent genotypes, with all alleles not 7R combined into the X allele. Segments represent 95% confidence intervals. Student's *t*-test * $p < 0.1$, ** $p < 0.05$.

Table 2. Differences between genotypes in risk-tolerance

	(1) Without controls	(2) With controls	(3) Non-risky area only	(4) Risky area only	(5) Interaction
7R: additive effect	0.068** (0.027)	0.064** (0.028)	0.097* (0.049)	0.056* (0.032)	0.107** (0.042)
7R: dominance effect	-0.037 (0.036)	-0.026 (0.037)	-0.003 (0.042)	-0.036 (0.042)	-0.027 (0.036)
Age		-0.002* (0.001)			
Education		-0.004 (0.005)			
Risky area		-0.120*** (0.032)			-0.079** (0.036)
Risky area × 7R: additive effect					-0.054 (0.047)
Constant	0.456*** (0.017)	0.613*** (0.053)	0.507*** (0.030)	0.431*** (0.021)	0.509*** (0.029)
R^2	0.009	0.030	0.026	0.006	0.028
No. obs	721	699	215	506	721

The outcome variable is risk-tolerance. A higher level of risk-tolerance indicates the choice of a riskier lottery by participants in the lottery choice task. Standard errors in parentheses. The coefficients are the results of Ordinary Least Square (OLS) estimations. * $p < 0.10$, ** $p < 0.05$, *** $p < 0.01$.

different alleles involved (2R and 4R in Indonesia, or 7R in Senegal).

In addition, we do not find evidence that the 7R allele is associated with novelty seeking, as previously found³⁶. While the sample would satisfy conditions for a genetic adaptation to habitat³⁷ (limited migration with 74% of grandparents of participants of the risky area born in the same village, Table S.3, and a strong economic benefit to living in the area), we find no specific genetic differentiation at DRD4 locus relative to 29 unlinked microsatellites loci (Table S.4 and Figure S.2). Moreover, if there was genetic differentiation, it would move in the opposite direction as the risk-vulnerability hypothesis found in previous work for DRD4³⁵, as the 7R allele, favoring more risk-tolerant attitudes, is more prevalent in the risky area. Altogether, our results indicate that no selection at the DRD4 locus is apparent in our sample. This does not mean that such selection is absent, as many generations of selection are required for gene frequencies to change. This dangerous fishing activity started perhaps around the 16th century³⁸, thus, with 4-5 generations per century, this gives approximately 20-25 generations for which selection could have occurred, which is small. It is thus unclear if selection is acting, but during a too short period of time, or if there is currently no selection at the DRD4 locus.

Another point worth mentioning is that the observed differences between zones could also be the reflect of the effects of occupation on risk attitudes, because of a strong correlation between the living area and the probability of being a fisherman (85% of the sample in the risky area declared their main activity as fishing, vs. 4% in the non-risky area).

Further work should focus on genetic adaptation at other loci, for instance using the loci identified in the GWAS on risk attitudes⁴. Moreover, identifying other solutions for people to cope with risk in risky environments could also be further investigated.

Methods

A field study was conducted in the Saint-Louis region in Northern Senegal between March 2018 and March 2020. All experiments were conducted in accordance with relevant guidelines and regulations. The protocol (including genotyping) was approved by the Senegalese National Ethics Committee (*Comité National d'Ethique en Recherche en Santé*), and informed consent was obtained from all participants. Behavioral measures were made at the same time as samples were collected for genotyping, so genotypes were not established at the time of measure. Investigators were blind to the behavioral measures during the genotyping.

Measure of risk-tolerance We relied on a standard measure of risk-elicitation task from the experimental economics literature³⁴. Instructions were displayed in French (the official written language of the country) and enumerators were present to explain the instructions in Wolof, the vernacular language of Senegal. Participants were invited to choose a card among five. On each card, two amounts were displayed, with an associated color (red or black) and the corresponding amount in coins of XOF 100, in order to have a more visual representation. At the end of the experiment, one ball was randomly drawn by a local child and gains were calculated. The cards ranged from completely risk-free (400 XOF for both balls) to extremely unequal (0 XOF if Red, 1200 XOF if Black). At each new card, the risk is increased, but so is the average amount won. Cards used are displayed in Figure S.3.

Genotyping DRD4 genotyping was done as described in³⁵. In short, DNA was collected on FTA paper and extracted according to the manufacturer's instruction. 505 and 215 samples from the risky and non risky area respectively were of sufficient quality to allow amplification with the appropriate primers. Relevant allele was estimated by the size of the PCR product on a 2% agarose gel.

Microsatellite genotyping was based on high-throughput sequencing technology (SSRseq). 190 samples of each area were picked up randomly with the `sample()` function in R. 30 microsatellite tests were designed according to a streamlined SSRseq development workflow described in³⁹, of which 29 gave differentiation information (one had only one allele for all individuals). The genomic localization of the 29 microsatellites and their corresponding F_{ST} between the 2 populations are presented in Table S.4. Details on the design and analysis are in supplementary materials.

Population genetics DRD4 locus was tested for conformity with Hardy-Weinberg (HW) equilibrium using the exact probability test⁴⁰. Deviations from HW equilibrium were measured using the F_{IS} estimator⁴¹. DRD4 and microsatellite loci genotypic differentiation between populations was tested for by calculating an unbiased estimate of the P-value of a log-likelihood (G) based exact test⁴², a global test over loci was calculated using Fisher's method. Population differentiation was measured using the F_{ST} estimator⁴¹. Calculations were performed using Genepop R package (V. 1.1.7), based on⁴³.

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Acknowledgements

The authors gratefully acknowledge financial support from GENES, the Key initiatives MUSE Sea & Coast and Investissements d’Avenir (ANR-11-IDEX-0003/Labex Ecodec/ANR-11-LABX-0047).

The authors also acknowledge the help in data collection from field research assistants. Microsatellite design and analysis were performed at the PGTB (doi:10.15454/1.5572396583599417E12) with the help of Zoe Delporte.

Author contributions statement

G-J.C., A.M., B.D., O.S. and M.W. collected the data. G-J.C., C.M., C.F., G.H., M.R. and M.W. analyzed the data. E.C., E.G, C.M. and M.R. performed the micro-satellite analysis. G-J.C., C.M., C.F., G.H., M.R. and M.W. wrote the paper. All authors reviewed the manuscript.

Additional information

Supplementary Information accompanies this paper, available [here](#)

Data availability statement: The data used for this paper are available on the repository of the American Economic Association, under the identifier “openicpsr-179321”, and can be accessed using the following [link](#). The sequencing data are registered on the BioProject data base, under the identifier ID PRJNA879442, and are accessible using the following [link](#) (embargo until 2023-10-05).

Competing financial interests: The authors declare no competing financial interests.

Supplementary Files

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